S. 37

File With _____

SECTION 131 FORM

Appeal NO: ABP 314485

TO: SEO

Defer Re O/H ☐

Having considered the contents of the submission ~~dated~~/ received 23/12/24 from Liam O'Gradaigh recommend that section 131 of the Planning and Development Act, 2000 ~~be~~/not be invoked at this stage for the following reason(s):. no m

E.O.: _____ Date: 2/1/25

To EO: _____

Section 131 not to be invoked at this stage. ☐

Section 131 to be invoked – allow 2/4 weeks for reply. ☐

S.E.O.: _____ Date: _____

S.A.O: _____ Date: _____

M _____

Please prepare BP _____ - Section 131 notice enclosing a copy of the attached submission to: _____

Allow 2/3/4weeks – BP _____

EO: _____ Date: _____

AA: _____ Date: _____

S. 37

File With _____

CORRESPONDENCE FORM

Appeal No: ABP 324485

M _____

Please treat correspondence received on 23/12/24 as follows:

1. Update database with new agent for Applicant/Appellant _____
2. Acknowledge with BP 23
3. Keep copy of Board's Letter ☐

1. RETURN TO SENDER with BP _____
2. Keep Envelope: ☐
3. Keep Copy of Board's letter ☐

Amendments/Comments

4. Attach to file
(a) R/S ☐
(b) GIS Processing ☐
(c) Processing ☑
(d) Screening ☐
(e) Inspectorate ☐

RETURN TO EO ☐

	Plans Date Stamped ☐ Date Stamped Filled in ☐
EO:	AA:
Date: 2/1/25	Date: 2/1/25

Dillon Corcoran

From:	Liam O'Gradaigh <logradaigh@hotmail.com>
Sent:	Monday 23 December 2024 16:36
To:	Appeals2
Subject:	PL06F.314485 (F20A/0668)
Attachments:	Submission_ABP_DraftDecision_LiamOGradaigh_20-12-2024.pdf; 20240624-ppt-Hahad.pdf; advisory-report-the-influence-of-night-time-noise-on-sleep-and-health.pdf; planning-enforcement-complaint-form-65-Flights-summer.pdf; PENF_0133_2023 S153.pdf; PENF_0134_2023 S154 Enforcement Notice.pdf; ENF_24-263_Dublin_Airport_32m_cap.pdf; Transportation_Noise_Pollution_and_Cardiovascular_Health.pdf; Noise_causes_cardiovascular_disease.pdf; Daytime_vs_Nighttime_effects_of_aircraft_noise.pdf; enhealth-guidance-the-health-effects-of-environmental-noise.pdf; Basner_aircraft_noise_exposure.pdf; Basner_Environmental_Noise_and_Effects_on_Sleep.pdf; Basner_effects_on_sleep.pdf; Tech 11 2010 Good practice guide on noise.pdf; Dublin_Airport_Noise_Medical_Report.pdf

Dear An Bord Pleanála,
Please find attached my submission on the draft decision by the Board in relation to PL06F.314485.
I made previous submissions to the Board and do not need to pay a further fee.

Please acknowledge receipt of this submission.

Many thanks
Liam

Liam O'Gradaigh
Ward Cross
The Ward
Co. Dublin

The Secretary
An Bord Pleanála
64 Marlborough Street
Dublin 1
D01 V902
23rd December 2024

RE: DRAFT DECISION BY AN BORD PLEANALA ON PLANNING APPLICATION F20A/0668

Dear Sir/ Madam,

I welcome the opportunity to make a submission on the draft decision by An Bord Pleanála, dated September 17th, 2024. I note the information in the draft decision is very complex and one that should have been facilitated with an Oral Hearing. The Board does not have the relevant expertise to understand the complex details of aircraft noise and an Oral Hearing would have provided an opportunity to clarify many of the issues. This submission highlights a number of serious deficiencies with the proposed application and as a result the permission should be refused. The daa has carried out unlawful development breaching the passenger cap in 2019, 2023 and in 2024. The daa have never respected the 65 nighttime flight limit which ironically is one of the conditions that they are trying to amend in this Relevant Action. The daa have known about PFAS contamination at the time the North Runway was being constructed and decided to withhold this information from the authorities and literally buried the contaminated soil on site. This PFAS contamination has never been screened in any environmental assessment by the daa and has not been assessed in this application. All projects and impacts need to be assessed for cumulative and in-combination effects. In fact, the whole North Runway project has never had a full AA.

There are major concerns with the AA screening in this application and in particular the failure of the Board's ecologist to examine appellant's submissions. Surveys are out of date, lack of cumulative and in-combination project screening, failure to screen for the effects on the Red Kite, failure to understand the real noise levels at the SPAs and SACs along the Dublin Coast.

The daa have failed to show a need for this development. Their own data shows they can achieve 40mppa by 2034 without the Relevant Action. ANCA and the Board have failed to take Health costs into account. The daa provided a sub-standard assessment on awakenings. Awakenings have been assessed at key receptors under both the

North and South Runways and the proposal fails to achieve less than one additional awakening per night on average. The awakenings criteria can only be achieved by a complete ban on nighttime flights. The Board's movement limit doesn't address all awakenings, and the Board has not provided any mitigation measures for those still impacted by awakenings. Insulation is not the panacea that the daa and ANCA claim. A 20k euro grant to insulate bedrooms is an insult to the residents impacted. It doesn't matter what happens in other jurisdictions, especially the UK, which is no longer in the EU, the residents have the right to a good night's sleep and the right that their health is not impacted. EU598/2014 is all about applying the Balanced Approach, but in this application to date there is no balance. Health costs have not been taken into account. The Board needs to revisit the insulation scheme and its adequacy as there are some dwellings not adjacent to the flight paths that would benefit from it. But this is not the case for dwellings immediately adjacent to the airport and under the flight paths.

It is very evident that the Noise Abatement Objective has been breached in 2022 and again in 2023, with no repercussions from ANCA. The noise has increased for those residents exposed to the higher contours of noise. It is only at the lower noise contours where the noise has reduced. But these lower noise contours are where the densely populated areas in Dublin are and so skew the results. ANCA seem happy that the number of people affected from all noise has reduced but fails to address the real issue of increasing noise on those already severely impacted by noise. It is very clear that noise is increasing at Dublin Airport and not reducing. It is pure fiction that quieter aircraft will reduce noise levels when the aircraft movements are increasing. Quiter aircraft have done nothing to reduce noise over the 2 Rounds of the End and there's evidence that it will lead to lower noise levels in the future, with increasing aircraft movements. This is a national scandal, and the lives and health of Fingal and Meath residents are being disregarded in the name of aviation growth. The daa have never provided a business plan to properly address the impacts on residents. They failed to engage with Community groups on the flight path issue stating they cannot discuss them while enforcement proceedings are ongoing. It is crucial that the Board makes a decision on the validity of the flight paths. The Planning Authority has had enforcement proceedings open on the flight paths since 2022 but has been waiting for the Board to adjudicate. From the draft decision, the Board has not come to a conclusion and appears to be passing the issue back to the Planning Authority. Condition 1 of 2007 still applies and the current flight paths are in violation of condition 1. It has been shown that the airport can operate in different runway configurations such as Dependent mode which doesn't require divergence. This alternative has never been submitted for discussion. In addition, no alternative has been proposed to allow for respite from aircraft noise as is in place at Heathrow. At Heathrow the runways alternate at 3pm to offer respite. At Dublin Airport the aim appears to inflict as much damage as possible on the populations under the North Runway flight paths from 6am to 12 midnight without any respite.

The remainder of this submission goes into further detail on the serious issues with this proposed development. I also endorse the submission from the St Margarets The Ward Residents Group. I plead with the Board to refuse permission but in the event that some sort of permission is granted, I ask that the Board put in clear and concise

conditions. The daa have no respect for the Board and have found ways to create legal ambiguity with the previous conditions on the North Runway planning. In 2007 the Board members went against the Inspector and granted permission for the North Runway and imposed two conditions to alleviate the Significance issue. But as soon as permission was granted the daa started to work on ways to get rid of these conditions and effectively ignore them. At this point in time the daa are above the Planning bodies in this country. They have no respect for the Board and will ignore whatever the Board tries to impose on them. The Board needs to assert its authority or else its very existence and future will be called into question.

Yours Sincerely

_____

Liam O'Gradaigh

Flight Paths:

During this process, there have been effectively 3 separate EIARs submitted by the daa. The last Supplementary EIAR included significant changes to the previous EIARs, mainly that whole new flight paths have been submitted. This was the third revision of the EIAR, and one must ask the Board how many chances an applicant gets. In previous submissions to the Planning Authority, ANCA and the Board, it has been highlighted that the flight paths in operation are not the ones used in the original planning permission of 2007. In 2007 they were based on straight out flight routes and all the environmental assessments and baselines were based on these straight-out routes. In 2018, Fingal County Council signed off on compliance for Condition 7 on planning permission in relation to the dwelling insulation scheme. Fingal County Council employed AWN Consulting to review the insulation scheme, and no issues were raised at that time in relation to the noise contours as they were based on straight-out flight paths. In the intervening years, the daa decided they wanted to use divergent flight paths. They presented a 15/75-degree option in a consultation in 2016. At this point in time the daa intended to submit a revised EIS and planning application to the Board. I have received this draft EIS via an AIE request which was initially refused but eventually granted by the OCEI Commissioner. However, the EIS approach was dropped in favour of the Relevant Action approach as part of the Aircraft Noise (Dublin Airport) Regulation Act 2019. But somewhere along the way the daa forgot to include flight paths changes in their planning application. Nowhere in the planning notice does it state that the daa wish to apply for new flight paths.

They began operations on the North Runway in August 2022 and immediately it was noticeable to the public that the flight paths were incorrect. It took the daa 2 weeks before they made contact with the IAA to understand what had happened. Then in February 2023 they revised their flight paths once more. These revised flight paths were again subject to no public consultation or planning permission. These too were never environmentally assessed. But still these flight paths did not adhere to the ones that were environmentally assessed in 2004-2007 and which formed part of Condition 1 of planning. Enforcement investigations have been underway with Fingal County Council for over 2 years now and it's evident that they do not want to rule on this and are leaving it up to the Board to decide. Unfortunately, the Inspector has not made any decision on the flight paths, and we are left in limbo.

Flight paths are a fundamental part of this application, and the Board must adjudicate on them. Failure to do so could set a precedence where flight paths could be changed at any time by the daa without any proper planning consent. The Board must take cognisance of Condition 1. Condition 1 is still valid and the daa never applied to change it. Therefore, the flight paths need to be refused and the daa ordered to apply to change Condition 1. The flight paths are also fundamental to the issue of Significance. Significance was never assessed in the planning of 2007 and the Board's Noise expert and Inspector concluded that planning for the North Runway should be refused due to lack of evidence of Significance. We now have a situation where the Relevant Action has not been compared to 2007 in terms of Significance and therefore the application fails the basic Significance criteria. The Inspector has not grasped the severity of the lack of Significance analysis between the 2007 planning application and the Relevant Action.

In the Infrastructure Application (F23A/0781), the Planning Authority requested a response to the following question which can be viewed on page 359 of the CE's Order of Feb 16th 2024, https://planningapi.agileapplications.ie/api/application/document/FG/907689:

> "*The applicant is invited to provide analysis with narrative explaining the variation over time, of previously modelled aircraft noise contours for Dublin Airport. The analysis should be accompanied by an overlay graphical representation of noise modelling prepared and presented as contours for the currently proposed development shown with each of the following previously presented contours:*
>
> *1) the North Runway application (December 2005 EIS),*
>
> *2) the consented North Runway (EIS Addendum 9th August 2007)*
>
> *3) the modelling agreed for operation of the noise mitigation schemes under that permission (2016)*
>
> *4) the Airport Noise Zones in the Fingal Development Plan 2023. The methodological differences between the various contours and the reasons why they are not directly comparable should be noted.*"

The answer to Question #6 is in the doc *'Part 1 - RFI Response Report B Response to RFIs'*, on page 76 which is page 56 of the Coakley O'Neill report:

https://planningapi.agileapplications.ie/api/application/document/FG/1067909

Coakley O'Neill discuss the evolution in noise contours since 2004:

North Runway Dec 2004:

> "*The flight routes assumed that the North Runway tracks would replicate those on the South Runway. These assumed aircraft turned after a* ***straight segment of around S nm*** *from the end of the runway*"

Noise Mitigation 2016 (insulation scheme compliance):

> "*The flight routes assumed that the north runway tracks would replicate those on the south runway. These assumed that 25% of aircraft turned after a straight segment of around 5 nm from the end of the runway, with the remaining 75% turning earlier, around 2 nm from the end of the runway. This was based on an analysis of a sample of radar flight tracks*"

IA EIAR Dec '23:

> "*The flight routes were based cm an analysis of actual radar tracks. For the South Runway these were similar to previous assumptions.* ***For the North Runway this meant an initial 30 degree right turn shortly after the end of the runway.*** *After this initial turn the routes are similar to previous assumptions.*"

"*This response is written in the context of the Board Inspector's findings in her assessment of the North Runway Relevant Action (NRRA), ABP Ref. No. ABP-314465-22 (F20A/0668), which stated that:*

> *"the Board will note that the flight patterns submitted in the applicant's supplementary information and included far the purpose of the proposed scenario of the EIAR,* ***differ to those submitted in the original EIS*** *for the NR application. The Board will note that the flight patterns submitted to the planning authority for the original Relevant Action also differed from those submitted with the original EIS for the NR application* ***The main difference between the revised EIAR and the amended supplementary EIAR is the divergence north from the NR, earlier than previously indicated in the revised EIAR permitted by the planning authority.***"'

So here for the very first time since the North Runway opened, we have Coakley O'Neill on behalf of the daa holding their hands up in an official submission document, acknowledging and agreeing with the Board's Inspector that the current flight paths are different than originally submitted and planned for. This has serious implications, and this has been pointed out on numerous times to the Planning Authority, ANCA and An Bord Pleanála during this Relevant Action planning application process. Therefore, this is an admittance of Unauthorised Development by the applicant and the Board have no alternative but refuse planning permission or request the applicant apply for retention or substitute consent.

Significance formed a major part of Mr Rupert Thornely-Taylor's evidence to the Board back in 2004-2007. Mr Thornely-Taylor was very clear that Significance was not addressed in the planning for the North Runway, and he recommended refusal on that basis. The Inspector agreed with Mr Thornely-Taylor and recommended refusal. The Board went against the recommendation of the inspector and inserted Conditions 3(d) and 5 to alleviate the Significance problem. However, Significance has not been addressed in this Relevant Action application by comparing the 'Proposed' scenario to what was granted in 2007. Permission was granted for straight out flight paths in 2007 and the Relevant Action has never compared any Proposed scenario with straight out flight paths. Therefore, Significance has not been addressed. The Board is reminded that the Relevant Action just concerns Condition 3(d) and 5 of 2007 and that Condition 1 still remains in force. The Relevant Action does not replace the planning of 2007 but just amends those 2 conditions. Therefore, it is very clear that Significance has not been assessed correctly now, as was the case in 2007, and the Board cannot approve the application with an invalid assessment. The lack of a proper Significance assessment is contrary to the EIAR Guidelines.

If the Board does approve the Relevant Action, the Board must state clearly in their decision that the **flight paths have not been approved by the grant of approval** and that any future flight path changes must go through proper planning and environmental assessment.

Awakenings:

Awakenings have been central to the Vanguardia report and the Inspector's draft report. Submissions have been made to the Board that the awakenings assessment provided by the daa fell very short of that requested by the Board. The daa provided no maps showing the areas impacted by 1, 2 and 3 awakenings.

However, an assessment has been provided by Suono based on the "*WHO Environmental Noise Guidelines for the European Region: A Systematic Review on Environmental Noise and Effects on Sleep*" by Basner and McGuire. The Suono report forms part of the submission by the St Margarets The Ward Residents Group. The Board should be mindful that the residents have had to pay for such an assessment as a proper assessment was not carried out by the daa as requested by the Board.

In the Suono assessment, 5 receptors were chosen, which are daa NMT locations located under the North and South Runway flight paths, and the awakenings calculated for each receptor based on the 2025 Proposed scenario. The assessment calculated awakenings using an external to internal adjustment of 15dB, 21dB and 22dB which allows for insulation. The results are provided in Table 1 of Suono's report:

Table 1 Calculated additional awakenings per night

Annual Average Glazing Reduction	NMT26	NMT28	NMT1	NMT2	NMT20
15 dB	1.8	1.9	0.6	3.0	2.6
21 dB	1.3	1.3	0.5	2.1	1.7
22 dB	1.3	1.3	0.5	2.1	1.7

The results of the assessment show that only NMT1 has less than 1 awakening. NMT1 is located at the Bay Lane and under Westerly departures on the South Runway. With the 2025 Proposed scenario there are very few departures off the South Runway and therefore the awakenings are less than 1. But for all other 4 receptors the awakenings are in excess of 1 awakening on average per night, even with insulation added. This proves that insulation is not the solution for the 2025 Proposed scenario and even with insulation the health of a significant number of residents in Fingal cannot be protected and the scenario fails the awakenings assessment.

NMT2 and 20 are located under the South Runway Easterly arrivals flight paths which traverses the highly populated area of Portmarnock. Even if the dwellings were insulated, the residents of Portmarnock would be subjected to more than 2 awakenings per night on average. This is extremely damaging to Human Health as has been pointed out by Mr Fiumicelli and by the submissions of Dr John Garvey.

The only solution is a complete ban on nighttime flights or a vast reduction in nighttime movements as proposed by the Board in their draft decision. If the Board does decide to grant permission for the Relevant Action with a restricted movement limit such as 13000, the Board must make allowance for those properties where more than 1 awakening would still occur. These dwellings must be offered Voluntary Purchase, relocation or enhanced insulation to protect their health. The Board are very clear in

their draft decision about the health impacts of awakenings and therefore the Board must be cognisant of its duties to protect Human Health.

Population Datasets:

In section 13B.4.1 of Appendix 13 of the Relevant Action Supplementary EAIR from September 2023, it states:

> *"Dwelling data has been acquired from GeoDirectory for **2019 Q2**, which was the dataset utilised in the original EIAR. The same dataset has been used for all assessment scenarios in this EIAR Supplement for consistency."*

However, a later GeoDirectory **2023 Q3** dataset exists and has been used in the Infrastructure Application (F23A/0781).

ANCA have made it clear to the daa on numerous occasions that the most recent population datasets should be used for compliance with the NAO.

The Relevant Action Supplementary EIAR from September 2023 and the Infrastructure Application from December 2023 used different population datasets to calculate the population exposed to >55dB Lnight. The Relevant Action's assessment greatly underestimates the populations exposed to >55dB Lnight in comparison to the Infrastructure Application.

It is incumbent on the Board to request the daa to repeat the analysis of the populations exposed to >55dB Lnight using the **2023 Q3 dataset** as it's obvious that using the 2019 Q2 dataset has led to a misleading lower figure than the true figure. The daa's assessment contravenes the NAO requirements on population datasets and are out of date.

36m Planning Application:

On Friday December 20th 2024, the daa lodged a planning application to increase passenger numbers to 36m without any infrastructure changes. The application is denoted by F24A/1178E).

In Fingal's press release, https://www.fingal.ie/news/planning-application-raise-passenger-capacity-dublin-airport-received, they state that "*There were no pre-planning meetings between the Planning Authority and daa prior to the submission of this application*". This is very worrying that the daa didn't seek advice from the Planning Authority before lodging the submission.

Below is a photo of the site notice for the 36m application:

In the site notice the daa have confirmed that they interpreted the 32m passenger count as meaning one person equals one passenger. But for the 36m application they will now adhere to the IATA Standard. This is a clear admission that they have been skewing the passenger counts in order to breach the 32m limit. This 32m limit was imposed by An Bord Pleanála and the daa have effectively ignored it. The daa are trying to claim that the 32m limit imposed by An Bord Pleanala was related to surface access and road infrastructure. However, that is not the case.

Please refer to section 4.90 of the IAA's final decision on Summer 2025 coordination parameters: https://www.iaa.ie/docs/default-source/car-documents/1c-economic-regulation/s25-final-decision_final.pdf?sfvrsn=a88decf3_1.

> *"The IAA notes the following in respect of the 32mppa Conditions themselves. Certain of the assertions made by airlines (and in particular those of Ryanair and A4A) as to the genesis and primary purpose of the 32mppa Conditions are not correct.* ***It is apparent from the Terminal 2 planning material, in particular the report of the An Bord Pleanála inspector, that the 32mppa Conditions were instead specified as the direct result of a policy objective in a 2006 Dublin Airport Local Area Plan (LAP)****. That LAP contained a high-level objective that terminal passenger capacity beyond 30mppa should be provided by a third terminal on the western campus. The 32m annual limitation on terminals 1 and 2 was set on the basis that, if the capacity of those terminals were to exceed 32m, this might compromise the viability of this putative third terminal on the western campus (2mppa was added to the 30mppa figure for, effectively, contingency/flexibility purposes). It was, expressly,* ***not calculated based on any road traffic concern*** *(which concerns would, of course, not be effectively mitigated by an annual limitation in any case), or otherwise as a mitigation measure to address an environmental concern. We note that daa's submission that the 32mppa Conditions were each attached to the identified grants of planning permission following the carrying out of an environmental impact assessment completed pursuant to Council Directive 2011/92/EU, is also incorrect. The 2006 LAP upon which the 32mppa Conditions were actually based has since lapsed, and been replaced by a new LAP which provides, instead, for 40mppa on the eastern campus."*

So, it is very clear that the daa have deliberately used road infrastructure as a smoke screen to breach the 32m cap. This is again another breach of a condition from An Bord Pleanala and calls into question the integrity and purpose of the Board.

S146A request (ABP ref PL06F.220670)

In 2018, the Dublin Airport Authority made a request to An Bord Pleanála under S.146A to amend the wording of Condition no. 3 (**PL06F.220670)** to remove connecting passengers from the scope of the condition. The amended wording sought to include the words highlighted in bold as follows:

> 3. *The combined capacity of Terminal 2 as permitted together with Terminal 1 shall not exceed 32 million* ***origin-destination*** *passengers per annum unless otherwise authorised by a further grant of planning permission.*

The daa's letter can be viewed at:

https://planningapi.agileapplications.ie/api/application/document/FG/634827

In the letter from the daa, they elaborate on passenger types. This line is extremely relevant:

> ***"In line with international aviation convention such passengers are counted twice, once as an arriving passenger and secondly as a departing passenger*** *eg. 1000 transfer passengers are actually 500 people travelling through the airport."*

Therefore, the daa clearly acknowledged their interpretation that, in line with International Aviation Convention, transfer passengers are counted twice.

Clarification of Passengers Types

For much of its history Dublin Airport operated as primarily an origin-destination airport. This means that Dublin was either the departing or arriving destination for most passengers. At the time of the grant of the T2 planning permission, 99% of passengers were origin-destination passengers.

Connecting passengers are passengers who may travel through Dublin Airport, but Dublin is not their final destination.

The vast majority of connecting passengers are transfer passengers. They may arrive into Dublin on one aircraft and switch aircraft to complete the second leg of their journey towards their final destination. These passengers remain airside, and have no impact on transportation requirements at the airport. In line with international aviation convention such passengers are counted twice, once as an arriving passenger, and secondly as a departing passenger even though it is a single person travelling through the airport. For example, 1,000 transfer passengers is actually 500 people travelling through the airport.

A second type of connecting passenger is a transit passenger. A small number of aircraft stop at Dublin Airport for technical reasons including to refuel. Passengers on these flights are counted as transiting through the airport although they do not generally use the terminal buildings as they remain on the aircraft during the transit stop. It is much clearer that condition no. 3 doesn't apply to such passengers, however we include them for overall context.

Transfer and transit (collectively referred to as connecting passengers) do not impact the transportation network. An airport that facilitates connecting passengers may be referred to as a hub airport.

ABP's Direction of August 2018 stated:

> "*It is considered that the alteration sought would be material in planning terms, and cannot, therefore be considered under S.146A of the Act. The Board considered that the proposed alteration would enable greater throughput of overall passenger numbers through the airport. This greater level of activity would have material planning consequences (in terms of movement and access to the airport, airport capacity, and also in relation to planning policy relation to the airport) and would go beyond what was permitted in the permission granted.*"

The decision on the S.146A application confirms that the limit of 32mmpa applies to **any** passenger type in the terminal buildings.

FS5/036/19

In September 2019, the daa made an application to Fingal County Council seeking a declaration under section 5 on whether development is or is not exempted development. The development consisted of the following:

> *"Three questions in relation to the use by passengers of the airport in excess of 32 million passengers per annum.*
>
> *(a) Is the use of the 'airport' in excess of 32 million passengers per annum (mppa) constitute 'development', if the combined capacity of Terminal 2 as permitted together with Terminal 1 does not exceed 32 mppa and if so, is it exempt development?*
>
> *(b) Is the use of the 'airport' by up to 3 million connecting passengers in excess of 32 million passengers per annum (mppa) constitute 'development' if those connecting passengers are facilitated by the separately permitted transfer facility and the combined capacity of Terminal 2 as permitted together with Terminal 1 does not exceed 32 mppa?*
>
> *(c) Currently a connecting passenger using Dublin Airport is double counted, as both an arriving and department passenger (for the purpose of aviation security measures). If a connecting passenger is counted singly for the purposes of planning, is this development, and if so, is it exempt development?"*

The decision by Fingal County Council was to refer it to An Bord Pleanala.

ABP-305458-19

The question to ABP was whether the 3 questions in FS5/036/19 in relation to the use of in excess of 32mppa is or is not development or is or is not exempted development

ABP's inspector stated in their report:

> *"Use of the "airport" by up to 3 million connecting passengers in excess of 32 million passengers per annum (mppa), if those connecting passengers are facilitated by the Pier 4 passenger transfer facility and the combined capacity of the facility together with Terminal 2 as permitted and Terminal 1 would exceed 32 mppa, would contravene condition no. 3 of PL06F.220670, and is therefore not exempted development."*

Therefore, the Board's inspector's view was that the use of the airport by 3 million connecting passengers was not exempted development. It therefore stands that the daa still needs to apply for planning permission to increase passenger numbers beyond 32mppa.

PPC 106276 & PPC 106336:

In a pre-planning document dated February 25th 2020 (reference Number: PPC 106276 & PPC 106336) between the daa, ANCA and Fingal County Council a discussion arose in relation to the interpretation of the 32mppa cap with regard to types of passengers:

- Discussion on the interpretation of the 32mppa passenger capacity cap with regard to types of passengers, in particular the transfer/ transit passengers.
- The P&SI Dept advises the applicant that, with reference to ABP decisions and known international, European and national methods of counting passengers at airports, the 32mppa passenger cap included in Condition 3 of F06A/1248 (PL 06F 220670) and Condition 2 of F06A/1843 (PL 06F 223469) is considered to be a cumulative, annual figure comprising all passengers using (traveling to, through and from) Dublin Airport.
- The P&SI Dept advises the applicant that as the 32mppa cap is considered to be all inclusive figure, it is not considered possible/ practical for planning assessment and subsequent enforcement purposes, to make any differentiation between different types of passengers.

End

Phillippa Joyce

3/3/2020

This is very clear advice from the Planning and Strategic Infrastructure Dept that the 32mppa is considered to be a cumulative, annual figure comprising **all** passengers using (traveling to, through and from) Dublin Airport. There is to be no differentiation between different types of passengers.

This new 36m planning application confirms that the daa deliberately misled the Planning Authorities and Judiciary on passenger numbers. They breached the cap in 2019, 2023 and again at the end of November 2024. They knew exactly what the passenger counting convention is and were told by the local Planning Authority. Therefore, they are knowingly carrying out Unlawful Development. The Relevant Action cannot be granted while the daa are knowingly carrying out Unlawful Development and the Board must refuse the Relevant Action on that basis or make the daa apply for retention.

F23A/0781:

Another worrying feature of the 36m planning notice is that:

> "*The proposed development would come into effect only in the event of, and subject to, a grant of planning permission for the change to permitted runway operations as proposed under ABP Ref. No. PL06F.314485 (F20A/0668)*".

This is the daa's attempt to blackmail and pressurise the Board into granting the Relevant Action. This is serious interference in the Planning process and the Board should not be intimidated by such actions.

The daa are giving the impression that the Relevant Action is needed to increase passenger numbers to 36m. I'm sure this will form part of the daa's submission on the draft decision. This is not the case and it's critical that the Board doesn't fall for this approach. In the 40m Infrastructure Application (F23A/0781), the daa submitted responses to a further information response from the Planning Authority.

In the EIAR submitted, Table 9-1 provides a breakdown of various assessments with and without the Relevant Action (NRRA) for different years:

Table 9-1: Assessment Years, Scenarios, Passengers and Flights

Assessment Year and Scenario	Predicted Annual Passengers (PAX) (millions per annum)	Aircraft Movements ('000s per annum)
Current State	32.8	234
2027 without Proposed Development without NRRA	32.0	228
2027 with Proposed Development without NRRA	33.2	233
2027 without Proposed Development with NRRA	32.0	240
2027 with Proposed Development with NRRA	35.6	256
2031 without Proposed Development with NRRA	32.0	240
2031 with Proposed Development with NRRA	40.0	279
2034 without Proposed Development without NRRA	32.0	228
2034 with Proposed Development without NRRA	40.0	269
2046 without Proposed Development without NRRA	32.0	228
2046 with Proposed Development without NRRA	40.0	269
2046 without Proposed Development with NRRA	32.0	240
2046 with Proposed Development with NRRA	40.0	279

By 2034 the daa can achieve 40mppa without the NRRA. Therefore, achieving 40mppa is not reliant on the Relevant Action being granted.

Even in 2027 the passenger numbers can increase to 33.2mppa without the Relevant Action. These figures are from the daa themselves and therefore they are

not being truthful when they say that the Relevant Action is needed to achieve either 36m or 40m passengers.

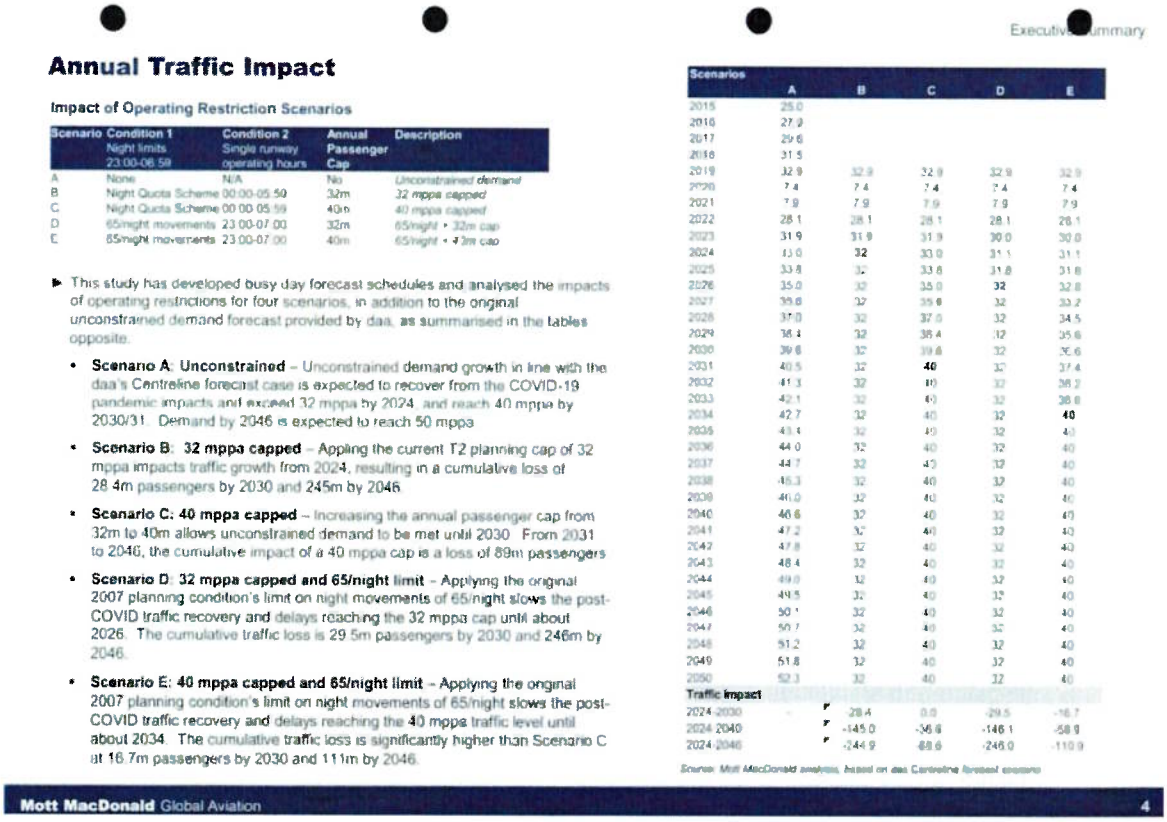
Section 9.1.23 under Table 9-1 is also very relevant:

> 9.1.23 The overall effect of the Proposed Development on the annual aircraft movements once 40mppa is reached (i.e. in the scenarios for 2031 and later) is an increase of 18% without the NRRA, or 16% with the NRRA. Considering the activity at night, there is no change due to the Proposed Development without the NRRA, and an increase of 14% with the NRRA.

There will be a 14% increase in activity at night with the NRRA. Section 9.1.23 states that there will be an increase of 18% without the NRRA as opposed to 16% with the NRRA.

Also included in the RFI material is a Mott MacDonald report titled "*Dublin Airport Operating Restrictions – Quantifications of Impacts on Future Traffic, Growth from 32m to 40million annual passenger – Fleet modernisation to 2046*", which can be found at page 1129 of https://planningapi.agileapplications.ie/api/application/document/FG/1067919.

On slide 4, Mott MacDonald compare various scenarios. Scenario E is noteworthy as it is the scenario without the Relevant Action being granted up to a cap of 40m passengers:



Executive Summary

Annual Traffic Impact

Impact of Operating Restriction Scenarios

Scenario	Condition 1 Night limits 23:00-06:59	Condition 2 Single runway operating hours	Annual Passenger Cap	Description
A	None	N/A	No	*Unconstrained demand*
B	Night Quota Scheme	00:00-05:59	32m	*32 mppa capped*
C	Night Quota Scheme	00:00-05:59	40m	*40 mppa capped*
D	65/night movements	23:00-07:00	32m	*65/night + 32m cap*
E	65/night movements	23:00-07:00	40m	*65/night + 40m cap*

- This study has developed busy day forecast schedules and analysed the impacts of operating restrictions for four scenarios, in addition to the original unconstrained demand forecast provided by daa, as summarised in the tables opposite.
  - **Scenario A: Unconstrained** – Unconstrained demand growth in line with the daa's Centreline forecast case is expected to recover from the COVID-19 pandemic impacts and exceed 32 mppa by 2024, and reach 40 mppa by 2030/31. Demand by 2046 is expected to reach 50 mppa.
  - **Scenario B: 32 mppa capped** – Appling the current T2 planning cap of 32 mppa impacts traffic growth from 2024, resulting in a cumulative loss of 28.4m passengers by 2030 and 245m by 2046.
  - **Scenario C: 40 mppa capped** – Increasing the annual passenger cap from 32m to 40m allows unconstrained demand to be met until 2030. From 2031 to 2046, the cumulative impact of a 40 mppa cap is a loss of 89m passengers.
  - **Scenario D: 32 mppa capped and 65/night limit** – Applying the original 2007 planning condition's limit on night movements of 65/night slows the post-COVID traffic recovery and delays reaching the 32 mppa cap until about 2026. The cumulative traffic loss is 29.5m passengers by 2030 and 246m by 2046.
  - **Scenario E: 40 mppa capped and 65/night limit** – Applying the original 2007 planning condition's limit on night movements of 65/night slows the post-COVID traffic recovery and delays reaching the 40 mppa traffic level until about 2034. The cumulative traffic loss is significantly higher than Scenario C at 16.7m passengers by 2030 and 111m by 2046.

Scenarios	A	B	C	D	E
2015	25.0				
2016	27.2				
2017	29.6				
2018	31.5				
2019	32.9	32.9	32.9	32.9	32.9
2020	7.4	7.4	7.4	7.4	7.4
2021	7.9	7.9	7.9	7.9	7.9
2022	28.1	28.1	28.1	28.1	28.1
2023	31.9	31.9	31.9	30.0	30.0
2024	33.0	32	33.0	31.1	31.1
2025	33.8	32	33.8	31.8	31.8
2026	35.0	32	35.0	32	32.8
2027	35.6	32	35.6	32	33.2
2028	37.0	32	37.0	32	34.5
2029	38.4	32	38.4	32	35.6
2030	39.6	32	39.6	32	36.6
2031	40.5	32	40	32	37.4
2032	41.3	32	40	32	38.2
2033	42.1	32	40	32	38.8
2034	42.7	32	40	32	40
2035	43.4	32	40	32	40
2036	44.0	32	40	32	40
2037	44.7	32	40	32	40
2038	45.3	32	40	32	40
2039	46.0	32	40	32	40
2040	46.6	32	40	32	40
2041	47.2	32	40	32	40
2042	47.8	32	40	32	40
2043	48.4	32	40	32	40
2044	49.0	32	40	32	40
2045	49.5	32	40	32	40
2046	50.1	32	40	32	40
2047	50.7	32	40	32	40
2048	51.2	32	40	32	40
2049	51.8	32	40	32	40
2050	52.3	32	40	32	40
Traffic impact					
2024-2030	-	-28.4	0.0	-29.5	-16.7
2024-2040	-	-145.0	-36.6	-146.1	-58.9
2024-2046	-	-244.9	-88.6	-246.0	-110.9

Source: Mott MacDonald analysis, based on daa Centreline forecast scenario

Mott MacDonald Global Aviation 4

It is very clear that 40m passengers can be achieved using Scenario E by 2034. Also, it shows that 36.6m passengers can be achieved by 2030. The only impact the with or without Relevant Action has is the rate of growth of passenger numbers. Without the Relevant Action still achieves the goals of the National Aviation Policy.

This is very critical to highlight – Not granting the Relevant Action does not impinge on the goals of the National Aviation Policy.

40mppa will be achieved by 2034 with or without the Relevant Action. Therefore, if the Board does grant permission for the Relevant Action, it cannot be based on the aims of the National Aviation Policy. Also, the Board will need to justify why it is inflicting so much adverse health effects at night on residents for no gain in passenger numbers in 2034. The Board will have to justify the costs involved with the grant of the Relevant Action and how the health costs (750m euro annually) can be borne by the Irish taxpayer to subsidise the aviation industry. This is clearly not a Balanced Approach.

PFAS Contamination:

The known PFAS contamination at Dublin Airport has not been addressed by the Board. It is public knowledge that there's a sizeable PFAS contamination issue at Dublin Airport:

https://www.irishtimes.com/transport/2023/03/17/dublin-airport-operator-examining-potential-impact-of-forever-chemicals/

At a DAEWG meeting on the 15th of March 2023, the daa's Head of Environmental Sustainability advised members that:

> *"daa is examining the potential impact of PFAS at Dublin Airport and is engaging with the relevant environmental regulators to ensure best practice in managing this issue*".

https://www.dublinairport.com/docs/default-source/community-engagement/15-march-2023---daewg-meeting-minutes-approved.pdf

It has also been reported that Geminor shipped 150,000 tonnes of PFAS contaminated soil from Dublin Airport to Norway for processing:

https://www.wastetodaymagazine.com/news/geminor-pfas-dublin-soil-treatment/

This work by Geminor also has not formed part of any planning application or environmental assessment and has involved no public consultation. Therefore, this work is unauthorized development and needs immediate assessment and planning permission.

Because the PFAS contamination did not form part of a planning application, the cumulative effects of the PFAS works has not been taken into account in any planning applications. This is a serious omission and this unlawful development has had serious knock-on consequences to other developments at Dublin Airport. The impacts of the PFAS contamination has not been environmentally assessed for its impact on the environment and especially the SACs and SPAs that are hydrologically linked to Dublin Airport. The impact on human health have also not been addressed in any planning context.

The daa first became aware of the impacts of PFAS during the North Runway construction. The daa decided not to alert any relevant authority and continued construction with the Norh Runway. They knowingly continued to construct the runway and therefore these works should be categorised as Unauthorised. A full AA has never been carried out on the whole North Runway projects.

In April 2024 the daa uploaded 4 documents to their website at https://www.dublinairport.com/corporate/environmental-social-governance/sustainability

1) Daa Statement April 2024
2) PFAS FAQ April 2024
3) 2021 – 2023 Environmental Monitoring Non-Technical Summary
4) 2021-2023 Environmental Monitoring Report

In section 5.1 of the document '*2021 – 2023 Environmental Monitoring Non-Technical Summary*', it states:

- **Groundwater**:
  - The highest Sum of 20 PFAS concentrations in groundwater were detected at the site of a former firefighting training ground, where maximum concentrations of **4,111ng/l** were reported.
- **Surface Water**:
  - The highest PFOS concentration in surface water was detected in the Cuckoo Stream at 50.6ng/l (May 2023).
  - The highest PFOS concentration in airside surface water (**1,430ng/l** in March 2022) was recorded in a manhole to the north of the North Apron. The source of PFOS is indicated to be from the Former Fire Station at the North Apron.
- **Soil/Concrete**:
  - The highest concentrations of individual PFAS constituents in soils/concrete were **568µg/kg** in Apron 5H.

These are alarming levels of PFOS / PFAS.

Further documents were released by way of an appeal to the OCEI Commissioner: https://ocei.ie/en/ombudsman-decision/7db6a-daa-public-limited-company-and-fingal-county-council/

Upon release, the daa made the documents available on their website:

https://www.dublinairport.com/corporate/airport-development/north-runway/environment/soil-and-water-management

The two documents are different to the documents previously made available by the daa. These two new documents were undertaken by Fehily Timoney who were retained by RoadBridge to undertake a Risk Assessment of PFAS contamination of groundwater and surface water at the **former Fire Training facility at the Dublin Airport, North Runway development (APEC 5).** RoadBridge were the contractors responsible for the construction of the North Runway.

The report titled '*Groundwater and Surface Water Risk Assessment and Remediation Options Appraisal*', states in section 1.1 that:

"The detected concentrations of Total PFOS at the off-site surface water monitoring points sampled between January 2018 and July 2021 exceeded the:

- *0.65 ng/l (the annual Average Environmental Quality Standards (EQS) for Inland Surface Waters for Total PFOS set by S.I. No. 386 of 2015)."*

"A number of the groundwater monitoring locations during the period January 2018 and October 2018 exceeded the Total PFOS 0.07 µg/l threshold value (defined by the United States Environmental Protection Agency (USEPA) Drinking Water Advisories for PFOS and PFOA)."

The Board cannot grant permission to the Relevant Action when Unauthorised Development has taken place and where the PFAS contamination has not been screened in any of the three environmental assessments. The Board cannot claim that it does not know of PFAS as it has been raised in submissions. It is worth mentioning the **MetroLink** project which will involve works at the airport. TII are taking PFAS very seriously and is including it in their Cumulative Impact Assessment and In-Combination Assessment for NIS. While the daa are taking the opposite approach and failing to adequately address the issue.

The daa have known about PFAS contamination since as early as 2016 during construction of the North Runway and yet none of their Environmental Assessments since then even mention PFAS yet alone provide mitigation and remedial measures. The dangerous levels of PFAS / PFOS have been known for a long number of years now and the daa have only recently contacted the relevant authorities. The response from the daa was to initially remove and bury known contaminated soil from the North Runway site around attenuation tanks and continue with the North Runway development. This was a major mistake as the PFAS levels under the North Runway are at dangerous levels. PFAS contaminated soil has also been found at other sites at the airport and large amounts of contaminated soil from the Apron 5H development has been shipped to Norway for remediation.

The cumulative impacts of the contamination at the Apron 5H development site should be assessed in conjunction with this Relevant Action application. The whole airport site needs to be addressed for PFAS / PFOS contamination as a whole and not the piecemeal approach thus far. The need for Cumulative Assessment and In Combination Assessment are highlighted in the advice given to TII for MetroLink. TII are taking the PFAS situation very seriously and understand their obligations which are clearly lacking with the daa. TII acknowledge that their development will lead to PFAS release into the environment.

The daa have been aware since 2016 of the PFAS issue and decided to literally bury the evidence in order that the North Runway project would not be delayed. No consultation with State Authorities was carried out at the time. We note that no full AA was ever carried out on the North Runway. The daa knew of the PFAS contamination and yet still went ahead without addressing it and even got a time extension and defended High Court proceedings while still burying knowledge of this contamination. The North Runway should be classed as **Unauthorised Development**, and we ask that the Board make a ruling on this.

An Bord Pleanála are mandated to refuse planning permission based on the total lack of screening and assessment of PFAS / PFOS contamination and its impact on European sites.

Nighttime insulation grant

It is proposed in the draft decision to offer a grant of 20k euro for nighttime insulation of bedrooms. The Inspector has accepted the process that ANCA has conducted. The award of any grant should be costed by an organisation such as the Chartered Surveyors of Ireland or Engineers Ireland. 20k euro will not achieve much in 2025 and beyond. It is a derisory sum. Nighttime noise impacts more on health than daytime noise. Yet the daytime insulation scheme for those contained in the 63 LAeq16 contour offers full house insulation. The Board have not explained how 20k euro can achieve any satisfactory level of insulation to protect human health. In fact, the Board have not shown the competence with which they can arrive at that decision. The Board needs to engage proper Engineering and Surveying competence to make any determination on insulation.

Schedules:

In the daa's 2023 Annual Compliance report, https://www.fingal.ie/sites/default/files/2024-09/d00001-daa-xxx-xx-xxx-rp-v-xxx-0003-annual-compliance-report-section-19-2023-v1.0_0.pdf, Appendix 2 on page 54 lists the percentage of arrivals and departures per hour:

App 2 Arrivals and Departures by Hour

Hour	Arrivals	Departures
0	3.4%	0.4%
1	2.0%	0.2%
2	0.5%	0.4%
3	0.4%	0.1%
4	2.3%	0.3%
5	1.4%	1.2%
6	1.3%	7.9%
7	3.0%	9.2%
8	5.1%	5.9%
9	5.9%	5.2%
10	5.7%	5.2%
11	6.1%	6.0%
12	6.3%	6.0%
13	5.8%	6.7%
14	5.6%	5.7%
15	4.6%	6.1%
16	5.3%	5.7%
17	5.6%	6.1%
18	5.3%	6.0%
19	4.5%	5.2%
20	4.5%	4.3%
21	4.8%	3.4%
22	5.7%	2.0%
23	4.9%	0.8%
Total	100%	100%

The daa have always claimed that the 6-7am slot and 23-24pm slot are their busiest hours of operation. Assuming there's an even split of arrivals and departures for the entire day, the 6-7am slot has 9.2% (1.3 + 7.9) of total movements. However, when summing up the totals of arrivals and departures for every hour, it can be seen that the 6-7am slot is only the 14th busiest hour, and the 23-24pm slot is the 18th busiest hour. This makes a mockery of the daa's claims. Submissions on the schedules have been made repeatedly during this planning process by the St Margarets The Ward Residents Group and the Inspector has failed to date to understand how important the evidence in these schedules is and what can be learned from them that runs contrary to what the daa are saying. There has been no evidence provided in the draft decision that shows the Board understands the schedules or has taken the numerous submissions into account. The Board must interrogate the schedules, and will no doubt come to the same conclusion that the 6-7am timeslot is not the busiest.

Hour	Arrivals	Departures	Total
13	5.80%	6.70%	12.50%
12	6.30%	6.00%	12.30%
7	3.00%	9.20%	12.20%
11	6.10%	6.00%	12.10%
17	5.60%	6.10%	11.70%
14	5.60%	5.70%	11.30%
18	5.30%	6.00%	11.30%
9	5.90%	5.20%	11.10%
16	5.30%	5.70%	11.00%
8	5.10%	5.90%	11.00%
10	5.70%	5.20%	10.90%
15	4.60%	6.10%	10.70%
19	4.50%	5.20%	9.70%
6	1.30%	7.90%	9.20%
20	4.50%	4.30%	8.80%
21	4.80%	3.40%	8.20%
22	5.70%	2.00%	7.70%
23	4.90%	0.80%	5.70%
0	3.40%	0.40%	3.80%
5	1.40%	1.20%	2.60%
4	2.30%	0.30%	2.60%
1	2.00%	0.20%	2.20%
2	0.50%	0.40%	0.90%
3	0.40%	0.10%	0.50%
Total	100%	100%	

I reiterate that this data comes from a daa Compliance Report for 2023.

Independence:

The Director of ANCA, Ms Ethna Felten, is also Deputy CEO of Fingal County Council. This is a clear breach of EU598/2014 and the Aircraft Noise (Dublin Airport) Regulation Act 2019.

> *(13) The competent authority responsible for adopting noise-related operating restrictions should be* ***independent*** *of any organisation involved in the airport's operation, air transport or air navigation service provision, or representing the interests thereof and of the residents living in the vicinity of the airport. This should not be understood as requiring Member States to modify their administrative structures or decision-making procedures.*

Article 3:

> *2. The competent authorities shall be* ***independent*** *of any organisation which could be affected by noise-related action. That independence may be achieved through a functional separation.*

On the recent Dublin Airport Noise Action Plan, ANCA and their consultants worked in tandem with Fingal County Council. This does not seem fitting for an independent body.

The Board should clarify if ANCA's position is in accordance with EU598/2014 legislation.

Enforcement:

The flight paths issue is just one condition of planning that Fingal County Council's enforcement department are dealing with. Fingal has taken enforcement proceedings against the daa over breaching Condition 5 and not adhering to 65 nighttime flights. This matter is subject to High Court proceedings. The daa are not adhering to a condition of planning imposed by the Board and have sought a stay via the courts.

In section 12.4.8 of the Inspector's report, it states:

> *"I have no evidence before me to suggest the proposal for the RA is to address any unauthorised action. A response to the supplementary information was received by both ANCA and the PA and no issues relating to unauthorised development have been raised. Any non-compliance with the original NR permission and enforcement issues are a matter for the PA".*

The Board has an obligation to seek information from relevant authorities if required. It appears that the Inspector relied on a lack of material from the PA and ANCA. However, information should have been sought under the Board's powers.

I attach the Enforcement Notice, *PENF_0134_2023 S154 Enforcement Notice.pdf*, from Fingal County Council dated July 28th 2023. I also attach the record of Fingal's CEO, *PENF_0133_2023 S153.pdf*, which clearly states that the development is unauthorised:

- Taking account of the foregoing, it is therefore concluded that by virtue of the scheduled and actual operations reported, the frequency of night flights in Dublin Airport is not in conformity with Condition 5 of the North Runway permission and is for that reason unauthorised development. The 2000 Act, including s.154(5)(a)(ii) provides that the planning authority can issue an Enforcement notice to require the daa, to proceed with a development in conformity with Condition 5;

- Unauthorised development is occurring and will continue to occur in non-conformity with Condition 5 and that unauthorised development is occurring at the Lands and development is not being carried out in conformity with Condition 5 of the North Runway Permission (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429);

- The daa has not sought to remedy the said unauthorised development, there are no compelling reasons for not taking enforcement action, having regard to the nature of the unauthorised development at issue and the nature of Condition 5, including the reason/purpose of same;

- In circumstances where unauthorised development is occurring and will continue to occur at Dublin Airport, contrary to Condition 5 of the North Runway Permission (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429) comprising the continued and ongoing exceedance of the permitted average number of night-time (between 2300 hours and 0700 hours) aircraft movements at the airport – being a permitted average of 65 aircraft movements per night when measured over the 92-day modelling period;

I also attach an enforcement complaint form, *planning-enforcement-complaint-form-65-Flights-summer.pdf*, which lists out flights on June 25th/26th inside the 92-day Sumer period where 106 movements were recorded between 23:00-07:00.

These records provide proof to the Board that Unauthorised Development has been occurring in relation to Condition 5 (65 nighttime limit). The Board has a duty to recognise this Unauthorised Development and refuse the Relevant Action as it's now a case of retention permission.

The daa have also breached the 32m passenger cap conditioned by the Board as part of Terminal 2's planning. They breached it in 2019 and 2023 and have breached it again at the end of November 2024. The passenger numbers can be viewed on daa's own corporate website at https://www.daa.ie/wp-content/uploads/2024/12/daa-Monthly-Statistics-November-2024.pdf. It shows that Dublin Airport has handled 32,250,020 passengers at the end of November. This webpage is accessed via the daa's 'investor relations' webpage. These are the passenger numbers they show off to their investors. This 32m passenger cap was another planning condition imposed by the Board when granting planning permission for Terminal 2. This once again shows the lack of respect for the Board by the daa and they believe they are above the planning laws of this country.

Dublin Airport - November 2024 Statistics

Region	Nov 2024	Nov 2023	% Change	YTD 2024	YTD 2023	% Change
Domestic	12,195	12,678	-4%	162,943	142,996	14%
Great Britain	786,037	769,810	2%	8,981,214	8,712,705	3%
Rest of Europe	1,229,461	1,191,001	3%	18,129,892	17,427,641	4%
Transatlantic	240,924	244,484	-1%	3,904,885	3,651,362	7%
Other International	91,358	80,508	13%	1,062,488	932,492	14%
Transit	915	1,235	-26%	8,598	248,957	-97%
Total Passengers	**2,360,890**	**2,299,716**	3%	**32,250,020**	**31,116,153**	4%
Commercial ATM's	16,597	16,709	-1%	219,717	215,841	2%

I attach correspondence, *ENF_24-263_Dublin_Airport_32m_cap.pdf*, from the enforcement section of Fingal County Council where they state that a Warning Letter pursuant to Section 152 of the Planning and Development Act 2000, as amended, was issued to the daa on December 17th 2024.

The daa breached the cap in 2019 (32.9m), 2023 (33.522m) and now again at the end of November 2024 (32.25m). The final figure will be above 34.6m passenger. This is the repeated ignoring of a planning condition imposed by An Bord Pleanála and is therefore Unauthorised Development. The Board must acknowledge this breach and have the daa apply for retention.

Climate:

Another major issue central to the nighttime flights application and the expansion of aviation is the significant increase in Green House Gases (GHG) emissions. GHG emissions were never assessed for significance in the original planning for the North Runway and therefore no Baseline for emissions was established. Therefore, all emissions from the proposed Relevant Action need to be accounted for and these are '*major adverse*' when accounted for based on the IEMA Guidelines. It is highly significant that the SEAI recently published a report, Energy in Ireland 2024 (https://www.seai.ie/sites/default/files/publications/energy-in-ireland-2024.pdf), estimating that "*Ireland's emissions from International aviation amounted to 3.4 MtCO2eq, equivalent to approximately 11% of national energy-related emissions*."

Table 7.1: Energy-related CO_2eq by sector (share)

GHG [$MtCO_2eq$]	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023
Electricity generation	11.70	11.53	12.13	12.86	12.05	10.70	9.45	8.86	10.36	10.14	8.03
Transport (excl. int. aviation)	10.92	11.20	11.69	12.21	12.05	12.22	12.22	10.29	10.97	11.64	11.68
Industry	3.39	3.61	3.59	3.71	3.83	4.05	3.97	4.02	4.04	3.81	3.62
Residential	7.07	6.27	6.71	7.00	6.51	7.00	6.73	7.34	6.87	5.75	5.35
Services	1.50	1.41	1.54	1.45	1.39	1.51	1.50	1.31	1.41	1.39	1.35
Agriculture	0.59	0.53	0.51	0.54	0.55	0.59	0.61	0.62	0.62	0.85	0.76
Fisheries	0.08	0.07	0.07	0.06	0.07	0.08	0.07	0.06	0.06	0.05	0.06
Other	0.48	0.44	0.53	0.42	0.47	0.52	0.46	0.48	0.47	0.47	0.42
Total (excl. int. aviation)	**35.72**	**35.06**	**36.77**	**38.24**	**36.92**	**36.67**	**35.02**	**32.99**	**34.79**	**34.11**	**31.27**
International aviation	2.02	2.24	2.54	2.60	3.06	3.31	3.34	1.19	1.32	3.04	3.44
Total (incl. int. aviation)	**37.74**	**37.30**	**39.30**	**40.84**	**39.98**	**39.98**	**38.36**	**34.17**	**36.12**	**37.15**	**34.71**

It also showed that Jet kerosene contributed 22.8% of energy related CO2 emission in transport:

Table 7.3: Quantities and shares of energy-related CO_2eq emissions in transport (share)

GHG [$MtCO_2eq$]	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023
Diesel / gasoil	7.34	7.80	8.46	9.16	9.29	9.69	9.82	8.50	9.07	9.48	9.38
Jet kerosene	2.03	2.25	2.55	2.61	3.07	3.32	3.36	1.20	1.34	3.06	3.46
Gasoline	3.54	3.35	3.17	2.96	2.67	2.43	2.30	1.70	1.81	2.06	2.19
Electricity	0.02	0.02	0.02	0.02	0.02	0.03	0.03	0.03	0.05	0.07	0.08
Biodiesel	0.01	0.02	0.02	0.02	0.03	0.03	0.03	0.03	0.03	0.04	0.05
Natural gas	0.01	0.01	0.01	0.05	0.05	0.05	0.04	0.04	0.04	0.04	0.04
LPG	0.00	0.01	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00
Bioethanol	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Fuel oil	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Total	**12.96**	**13.46**	**14.24**	**14.83**	**15.14**	**15.55**	**15.59**	**11.51**	**12.35**	**14.76**	**15.20**

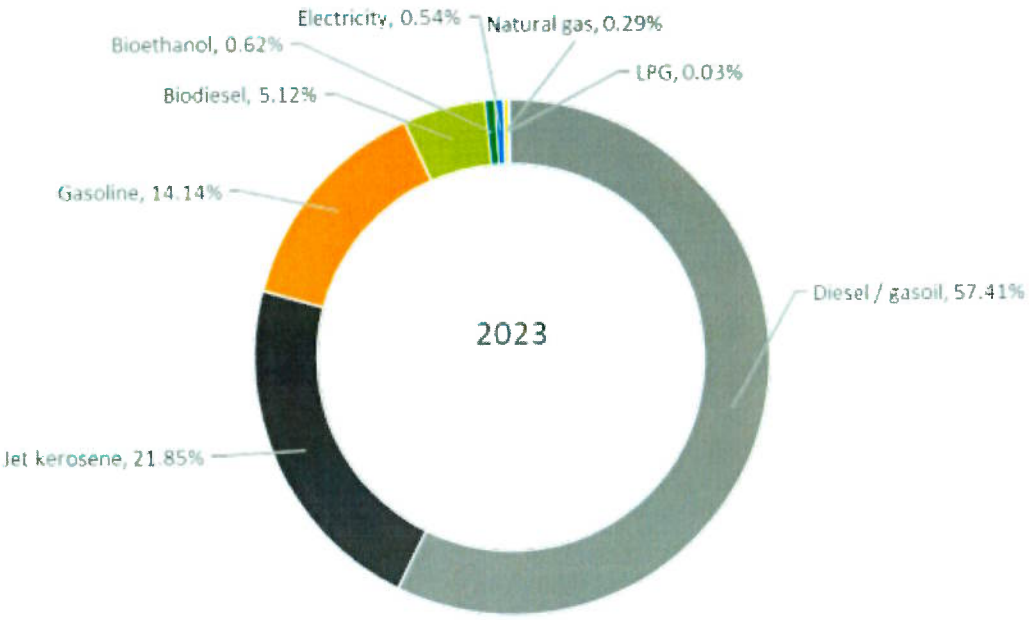
Jet Kerosene use in 2023 surpassed the previous yearly high in 2019:

Table 5.4: Final energy in transport sector by energy types (share)

Energy [TWh]	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023
Diesel / gasoil	27.50	29.25	31.72	34.32	34.80	36.31	36.80	31.83	33.98	35.50	35.10
Jet kerosene	7.85	8.70	9.84	10.10	11.88	12.83	12.98	4.63	5.18	11.84	13.36
Gasoline	13.93	13.18	12.50	11.66	10.52	9.59	9.08	6.73	7.13	8.12	8.65
Biodiesel	0.86	1.04	1.14	1.00	1.52	1.48	1.90	1.82	1.87	2.37	3.13
Bioethanol	0.33	0.31	0.35	0.38	0.34	0.32	0.30	0.23	0.24	0.27	0.38
Electricity	0.04	0.04	0.04	0.05	0.05	0.07	0.09	0.10	0.15	0.22	0.33
Natural gas	0.04	0.03	0.05	0.25	0.24	0.26	0.20	0.18	0.19	0.19	0.18
LPG	0.02	0.02	0.03	0.03	0.03	0.02	0.02	0.01	0.01	0.02	0.02
Fuel oil	0	0	0	0	0	0	0	0	0	0	0
Total	**50.57**	**52.58**	**55.67**	**57.79**	**59.38**	**60.87**	**61.36**	**45.53**	**48.74**	**58.53**	**61.14**

Jet Kerosene accounted for 21.85% of all transport energy use:

Figure 5.7: Shares of energy types in transport final energy



It's imperative that these highly significant GHG emissions from aviation are kept in line with Ireland's obligation under the Paris Agreement.

Significance:

Significance of effects was never established in the original planning application. As a result, the daa has no baseline on which to determine significance with their proposal. It is not just the difference between the Permitted and Proposed scenarios that determines those significantly affected as the Permitted scenario was never assessed for significance. It also needs to be pointed out that the Permitted scenario is not equal to the baseline situation in 2007 or the consented scenario with straight out flight paths. It is also of note that the change to the new flight paths, acknowledged by the daa, was not stated on the Public Notice for the Relevant Action or on the Public Notice from An Bord Pleanála. This is a grave error and totally misleads the public who were not anticipating divergent flight paths.

The Board should be made aware of the RFI responses from the daa's planning consultants, Coakley O'Neill, for the Infrastructure Application, F23A/0781. The planning authority made the following request in question number 6:

> "*The applicant is invited to provide analysis with narrative explaining the variation over time, of previously modelled aircraft noise contours far Dublin Airport. The analysts should be accompanied by an overlay graphical representation of noise modelling prepared and presented as contours for the currently proposed development shown with each of the following previously presented contours: 1) the North Runway application (December 2005 515),*

2) the consented Worth Runway (EIS Addendum 9th August 2007) 3) the modelling agreed for operation of the noise mitigation schemes under that permission (2016) 4) the Airport Noise Zones in the Fingal Development Plan 2023. The methodological differences between the various contours and the reasons why they are not directly comparable should be noted."

The response can be accessed in this document: https://planningapi.agileapplications.ie/api/application/document/FG/1067909. Coakley O'Neill provide a timeline of events, some of which are:

- *"North Runway Application (December 2004 EIS):*
  - *The flight routes assumed that the North Runway tracks would replicate those on the South Runway. These assumed aircraft turned after a straight segment of around 5 nm from the end of the runway*
- *Modelling agreed for operation of the noise mitigation schemes (2016):*
  - *The flight routes assumed that the north runway tracks would replicate those on the south runway. These assumed that 25% of aircraft turned after a straight segment of around 5 nm from the end of the runway, with the remaining 75% turning earlier, around 2 nm from the end of the runway. This was based on an analysis of a sample of radar flight tracks.*
- *IA EIAR (December 2023):*
  - *The flight routes were based cm an analysis of actual radar tracks. For the South Runway these were similar to previous assumptions. For the North Runway this meant an initial 30 degree right turn shortly after the end of the runway After this initial turn the routes are similar to previous assumptions.*

This response is written in the context of the Board Inspector's findings in her assessment of the North Runway Relevant Action (NRRA), ABP Ref. No. ABP-314465-22 (F20A/0668), which stated that:

> *"the Board will note that the flight patterns submitted in the applicant's supplementary information and included far the purpose of the proposed scenario of the EIAR, differ to those submitted in the original EIS for the NR application. The Board will note that the flight patterns submitted to the planning authority for the original Relevant Action also differed from those submitted with the original EIS for the NR application The main difference between the revised EIAR and the amended supplementary EIAR is the divergence north from the NR, earlier than previously indicated in the revised EIAR permitted by the planning authority""*

This is irrevocable proof that the daa have come clean and are agreeing with the Inspector that the flight paths have changed. This is in complete contrast to what the daa has been saying to the Planning Authority, ANCA, Irish people and the Oireachtas. Here are examples from the Oireachtas Transport Committee:

January 18th 2023:

https://www.oireachtas.ie/en/debates/debate/joint_committee_on_transport_and_communications/2023-01-18/3/

Deputy Darren O'Rourke

There was an application in 2007. Ms Gubbins is referencing 2016. I am aware Fingal County Council has issued an enforcement notice. I wonder about the basis on which the DAA is operating now and the basis on which it will operate on 23 February in the context of planning and the regulator. Will the applications have been adequately through those processes or does the DAA need to seek retention? Are other enforcement notices expected? Is the DAA of the opinion that from 23 February it will be entirely sound to operate those flight paths with regard to the regulators and the Aircraft Noise Competent Authority, ANCA, and all the various parameters that one must live within?

Ms Catherine Gubbins

There are two separate issues at play here. The Deputy referred to the deviation in the flight path. That specific issue will, hopefully, be resolved on 23 February, as we have discussed. The actual flight paths, whether they are the deviated or the original, are not a factor of our planning permission. The Deputy referred to the 2007 application. The north runway was constructed under a planning permission that was granted in 2007. The Deputy is absolutely right that we are currently engaged in a process, which the noise regulator ANCA, has made a decision on. This has been appealed to An Bord Pleanála. We are actually in the process of engagement around separate conditions to do with that 2007 planning permission, which to our mind is a completely separate process to the flight path deviation issue that we had previously discussed. As of 23 February, we are very hopeful that the new flight path will be in place, and hopefully that issue will be resolved. We are in the middle of a statutory process, which absolutely needs to run its course over the next few months.

Deputy Darren O'Rourke

Picking up from that point, I presume that in its assessment the DAA has been advised on this. The community might reasonably ask if the 2007 planning permission is being contested but the runway is being operated at the same time, whether the DAA is of the opinion that it is operating the north runway on a sound basis, despite the fact there are ongoing legals in relation to that planning?

The video for this meeting can be accessed at Joint Committee on Transport and Communications debate - Wednesday, 18 Jan 2023. At 1:23:10 into the video the Acting Chairman Senator Gerry Horkan asks Ms Catherine Gubbins the following question:

Acting Chairman (Senator Gerry Horkan)

For my own benefit, can I just confirm that the DAA is operating the north runway on the basis of the existing planning permission as opposed to what the DAA would like the planning permission to be into the future? Yes

Ms Gubbins replied 'Yes' that the daa were operating the North Runway on the basis of the existing planning permission.

November 22nd 2023:

https://www.oireachtas.ie/en/debates/debate/joint_committee_on_transport_and_communications/2023-11-22/2/

Mr. Kenny Jacobs

Flight paths are complicated and they take a long time to work through. When flights commenced on the north runway from August to February, there was a slight deviation for some flights. A small number of aircraft were marginally overflying parts of a community that were not consulted with. That has been corrected from February, which is the most important thing. That was a mistake that we had made. We apologised for it. The flight paths that operate now are fully compliant. They are the flight paths that were intended and are over the communities that were consulted with.

Deputy Steven Matthews

I am glad to hear that the DAA met with representatives of the Kilcoskan school recently. I have been contacted by residents and parents of children who attend the school as they really have difficulty with some of the noise issues. From February 2023, the flight paths relating to the north runway are as proposed in the original planning application from 2007 and the amended one.

Mr. Kenny Jacobs

Exactly, yes.

Example from the daa's own website:

https://www.dublinairport.com/latest-news/2023/03/15/daa-rejects-any-claims-that-it-is-in-breach-of-planning-permission-granted-in-respect-of-the-north-runway

Example from PrimeTime:

https://www.youtube.com/watch?v=jV78GFDwA6Y (3:30 into the audio)

This is proof that the daa misled the Oireachtas and Irish people.

Health costs:

Another serious concern with the daa's submission is that the health costs of nighttime noise have not been assessed. The public have gone to great lengths to point out the strong recommendations of the WHO and the submissions from the HSE, yet the daa and ANCA fail to address health. Neither the daa nor ANCA assess the health costs and other negative impacts of increased aviation activity. Aviation cannot be subsidised, and the impact and cost picked up by the public and Health system. It has been estimated that the health costs associated to just the number of people Highly Annoyed and Highly Sleep Disturbed amounted to **€750million in 2023** alone based on the methodology used in a report commissioned by the Belgian Superior Health Council:

https://www.health.belgium.be/sites/default/files/uploads/fields/fpshealth_theme_file/20240506_hgr-9741_vliegtuiglawaai_en_andere_emissies_vweb.pdf.

https://wakeupkraainem.be/wp-content/uploads/2023/06/ENVISA_Health-Economic-Impact-Brussels-Airport_March-2023.pdf,

The research used €132,000 as the cost of a Disability Adjusted Life Year (DALY). For Dublin Airport the number of DALYs attributed to High Annoyance was 1,428 and for Sleep Disturbance 2,279. The combined estimate of High Annoyance and High Sleep Disturbance amounts to €489m. An estimate for CVD adds another €300 amounting to €789m in total for just 2023 alone. These figures have never been addressed by ANCA, the Planning Authoriity or the Inspector.

Project Ireland 2024:

Another major flaw in the daa's proposal is that it is contrary to the objectives of Project Ireland 2024 and Balanced Regional Development. 90% of international aviation into Ireland is via Dublin Airport. The other airports must fight for the remaining 10%. As a result, the economic benefits of Dublin Airport are totally lob sided to Fingal, Dublin, and the Leinster region. How can Fingal County council be independent in its decision making when the economic benefits of Dublin Airport are felt strongest in Fingal?

Appropriate Assessment:

The AA assessment by the applicant and the AA assessment from ANCA fail to assess the impacts on the Red Kite, which is an Annex I species.

A full NIS was never carried out on the whole North Runway project. This is classic project-splitting and piecemeal development.

The most recent Bird Survey was carried out in 2018 which is out of date and the Board cannot make a determination on AA based on such out-of-date surveys. This goes against the advice of the CIEEM.

The Board's ecologist never read any of the appeals or other submissions made on AA.

The AECOM report misled the authorities on the noise levels at the various SPAs and SACs. The noise levels are far higher than reported and can be seen in the daa's Noise snf Flight Track Monitoring Reports: https://www.dublinairport.com/corporate/environmental-social-governance/noise/noise-management/airport-noise-plans-and-reports. Regular exceedance of 70dB LAmax is achieved at the SPAs and SACs which does impact on birds according to the scientific literature.

No bird surveys taken under the Norh Runway flight path or at night.

No Cumulative or In-Combination assessments were carried out with other projects which is in breach of Article 6(3) of the Habitats Directive.

ANCA's AA only dealt with noise in isolation, and it too is defective with regard to noise levels.

The AA screening report by the Planning Authority is dated August 2022 before the North Runway opened and therefore insufficient, and no other projects were assessed for in-combination effects.

Noise Abatement Objective (NAO):

ANCA reviewed the mitigation effectiveness at Dublin Airport for 2022 and 2023 and reported that Dublin Airport failed the Noise Abatement Objective (NAO) in both years. This was mainly due to more of the population exposed to >55dB Lnight in close proximity to Dublin Airport.

https://www.fingal.ie/sites/default/files/2023-08/Noise%20mitigation%20effectiveness%20review%20report%20for%202022.pdf

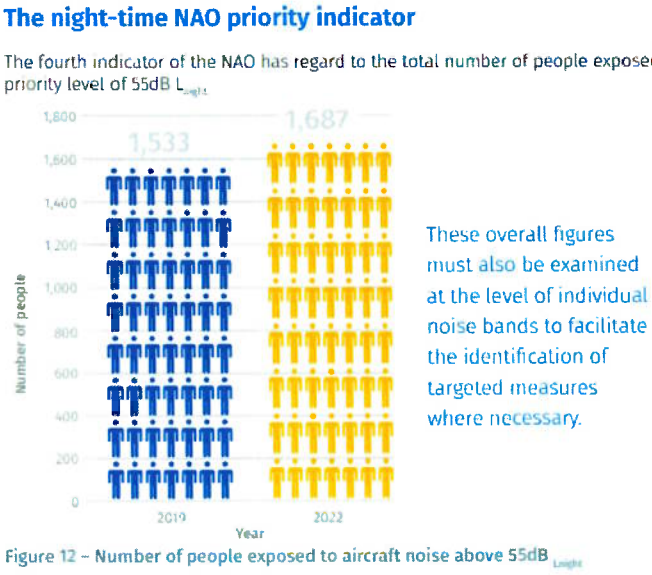


Figure 12 – Number of people exposed to aircraft noise above 55dB L_{night}

https://www.fingal.ie/sites/default/files/2024-08/noise-mitigation-effectiveness-review-report-for-2023.pdf

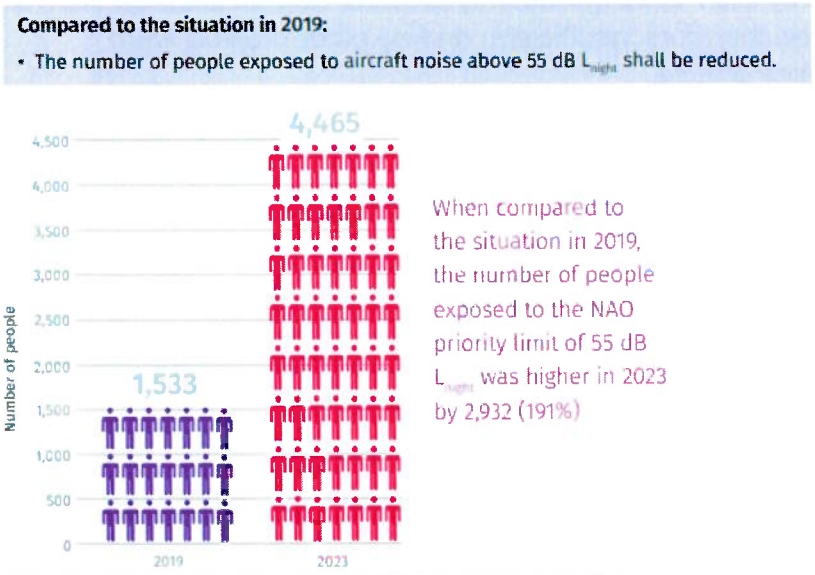


Figure 14 – Number of people exposed to aircraft noise above 55db L_{night}

The number of people exposed to >55dB Lnight was 1687 in 2022 and escalated to 4465 in 2023. Note ANCA took no measures as a result of these breaches. ANCA cannot be relied upon to protect the health of residents of Fingal. This is very

disturbing and calls into question ANCA's role as an Independent Regulator. Its own reports clearly shows that people's lives are being put at risk. What is very worrying about ANCA is that they have never engaged medical experts or professionals to understand the effects of aircraft noise on Human Health. EU598/2014 clearly states that health should be taken into account, but ANCA have refused to engage.

Notwithstanding these breaches of the NAO in 2022 and 2023, 2019 is a flawed year to use as the NAO baseline year. In 2019, Dublin Airport facilitated 32.9m passengers, breaching the planning condition of 32m imposed by An Bord Pleanála. The Baseline year for the NAO should be in line with the EU Action Plan '*Towards a Zero Pollution for Air, Land and Soil*', which refers to 2017.

It is also worth highlighting from the 2023 report the population exposed to noise compared to the EIAR Supplement.

EIAR Supplement:

- 53,854 people Highly Annoyed
- 23,844 people Highly Sleep Disturbed

2023 Noise Mitigation Effectiveness Report:

- 71,388 people were Highly Annoyed
- 32,562 people were Highly Sleep Disturbed

This clearly shows that the Supplementary EIAR is vastly underreporting and underpredicting the real noise levels. The 2023 noise figures are real noise data and not modelled. The Relevant Action has fictional scenarios with a 32m passenger limit. This is pure fabrication, and the Board need to take on board the real 2023 noise levels when determining the impacts of noise.

The HA and HSD numbers on their own are not that meaningful. The formulae to derive the HA and HSD figures are based on Exposure Response Functions that are described in the WHO 2018 Guidelines.

Table 30. The association between exposure to aircraft noise (L_{den}) and annoyance (%HA)

L_{den} (dB)	%HA
40	1.2
45	9.4
50	17.9
55	26.7
60	36.0
65	45.5
70	55.5

At 40dB Lden, 1.2% of the exposed population are highly annoyed, rising to 55.5% of the population exposed at 70dB Lden. The % increases as the noise increases.

Table 32. The association between exposure to aircraft noise (L_{night}) and sleep disturbance (%HSD)

L_{night}	%HSD	95% CI
40	11.3	4.72–17.81
45	15.0	6.95–23.08
50	19.7	9.87–29.60
55	25.5	13.57–37.41
60	32.3	18.15–46.36
65	40.0	23.65–56.05

At 40dB Lnight, 11.3% of the exposed population are highly sleep disturbed, rising to 40% of the population exposed at 65dB Lnight. Again the % increases as the noise increases.

The calculation of HA and HSD can be simplified as the sum of the population in each noise band multiplied by the %HA or %HSD for each band.

The ANCA 2023 Noise Mitigation Effectiveness Report breaks down the number of people in each band for both HA and HSD:

HA:

	45-49 dB	50-54 dB	55-59 dB	60-64 dB	65-69 dB	70-74 dB	>75 dB
2019	74,905	29,814	8,546	2,328	126	15	4
2023	37,959	20,983	8,753	3,532	148	13	0

HSD:

	40-44 dB	45-49 dB	50-54 dB	55-59 dB	60-64 dB	65-69 dB	>70 dB
2019	36,339	7,622	2,665	380	34	5	0
2023	20,101	7,252	4,003	1,147	55	4	0

It's very evident that from 2019 to 2023 the number of people HA reduced in the bands 45-49dB and 50-54dB but increased in all other bands.

It's also very evident that the number of people HSD reduced in the bands 40-44dB and 45-49dB but increased in other bands.

What this shows is that the numbers in the bands with the lowest noise levels have reduced but the numbers in the bands with the highest noise levels have increased.

ANCA is fixated on reducing the numbers of HA and HSD and is not concerned about the makeup of these numbers. ANCA is quite content that the overall numbers are reducing but has no interest that the number of people exposed to the highest levels of noise are increasing.

This is clear evidence that the overall HA and HSD numbers mask the effect that higher noise levels are impacting a larger cohort of people.

A worthwhile exercise is to compute the HA and HSD number based on the Environmental Noise Directive (END) reporting limits of 50dB Lnight and 55dB Lden.

Using the tables in the ANCA 2023 report which were shown above and summing the numbers in the bands from 50-54dB Lnight upwards and from 55-59dB Lden upwards:

Year	HA	HSD
2019	11,019	3,084
2023	12,446	5,209

These values paint a very different picture and show that the number of HA and HSD rose between 2019 and 2023 when you start counting at the END limit thresholds. The numbers being relied upon by ANCA in their NAO are skewed by the numbers in the lowest noise bands.

It's also worth highlighting that these lowest noise bands are where the largest populations in Dublin reside. A marginal effect at the lowest noise bands has a significant effect on the HA and HSD numbers.

From data extracted from the ANCA Reporting Templates for the Relevant Action and Reporting Template for 2023, a comparison can be made of the population in the Lden and Lnight contours for 2019 and 2023:

dB Lden	2019	2023
>=45	754135	419796
>=50	174146	132890
>=55	34097	37037
>=60	6279	9102
>=65	285	320
>=70	31	22
>=75	6	0

dB Lnight	2019	2023
>=40	344912	220460
>=45	59307	65227
>=50	13838	22417
>=55	1533	4339
>=60	110	159
>=65	13	8
>=70	0	0

From the Lden figures, 579,989 people resided in the 45-49dB Lden band in 2019 which is 77% of the total population exposed to greater than 45dB Lden.

From the Lnight figures, 285,605 people resided in the 40-44dB Lden band in 2019 which is 83% of the total population exposed to greater than 40dB Lnight.

Therefore, it's evidently clear that the quietest bands have a disproportionate number of people residing in the bands and therefore have a huge effect on the HA and HSD numbers if the noise contours change ever so slightly at the lowest bands.

From the tables above, based on the END reporting limits, 37,037 were exposed to >55dB Lden in 2023 compared to 34,097 in 2019 and 22,417 were exposed to >50dB Lnight in 2023 compared to 13,838 in 2019.

This is the reason that the HA and HSD figures above based on the END reporting limits are higher in 2023 than in 2019. The number of people exposed to the higher noise levels have been increasing.

Health

Attached to this submission are a list of scientific papers that provide expert advice on the impacts of Aviation Noise on health. Some of these papers are new and are after the WHO 2018 Guidelines were published. I encourage the Board to get the relevant expertise who can read and understand this literature and help make informed decisions. The Board must consult with medical experts if it doesn't have the necessary expertise to make an informed determination by taking this advice.

- *Transportation_Noise_Pollution_and_Cardiovascular_Health.pdf*
- *advisory-report-the-influence-of-night-time-noise-on-sleep-and-health.pdf*
- *Basner_aircraft_noise_exposure.pdf*
- *Basner_effects_on_sleep.pdf*
- *Basner_Environmental_Noise_and_Effects_on_Sleep.pdf*
- *Daytime_vs_Nighttime_effects_of_aircraft_noise.pdf*
- *20240624-ppt-Hahad.pdf*
- *enhealth-guidance-the-health-effects-of-environmental-noise.pdf*
- *Noise_causes_cardiovascular_disease.pdf*
- *Tech 11 2010 Good practice guide on noise.pdf*

I also attach the *Dublin_Airport_Noise_Medical_Report.pdf* from Professor Thomas Münzel, one of the world's leading experts on aviation noise and its effects on Human Health. Some of the key conclusions on his assessment of the Relevant Action are:

- *In addition to the fact that noise is now recognized as a cardiovascular risk factor, all possible measures must be taken to protect people who live near airports from the health consequences of noise.*
- *Based on the current study situation, it should be assumed that average outside noise levels caused by aircraft noise over a period of 24 hours, beginning around 40 dB (A), are associated with harmful effects. From this area on, increased noise pollution is to be expected, which is considered an effect modifier when communicating negative health consequences. Since night-time aircraft noise in particular has negative effects on health, stricter*

measures must be used in order to comply with the WHO recommendation (indoor noise level of less than 25 dB Lnight)

- *The noise study conducted on dwellings in close proximity to Dublin Airport shows that mitigation through insulation cannot reduce the noise to safe levels.*
- *Due to the new data on the negative health effects related to night-time aircraft noise, the number of night flights must remain limited and, in our opinion, cannot be increased any further.*
- *Due to the fact that night aircraft noise in particular is harmful to health, air traffic should, if unavoidable, be shifted more to the daytime.*
- *The legally defined night's sleep from 11:00 p.m. to 7:00 a.m. should be aimed for.*

It is unclear from the Inspector's report whether the Inspector has read this report and understands it. There is no evidence available in the Inspector's report to show that the Board received expert advice and obtained the expertise to thoroughly understand the evidence provided. Had an Oral Hearing been granted, the Inspector would have had the opportunity to hear first hands from one of the leading medical experts in transportation noise. It is clear from the Inspector's report that the Board have not provided proof of their expertise to understand the content of such a report. **The Board should now take this opportunity to obtain expertise from the appropriate experts to ensure they can come to a qualified determination.**

Noise Monitoring and Modelling:

It is very apparent that the Relevant Action leads to a serious deterioration in the noise situation for Fingal residents. Serious questions need to be asked about the daa's noise modelling. Only one portable noise monitor was used to calibrate the North Runway. They have used fixed monitors from the South Runway to attempt to calibrate divergent flight paths on the North Runway. The North Runway has been in operation for over 2 years now. The daa and ANCA have had plenty of time to collect reliable real measurements from under the North Runway.

Due to the lack of monitoring the local community have had to go to great lengths and cost to carry out their own independent monitoring. Monitoring was performed at 3 locations under the North Runway flight paths for the entire 92-day summer period in 2023 and 2024. The results of this monitoring show that the modelling presented in the EIAR Supplement is unreliable and very inaccurate for the North Runway, leading to variations of 2dB. This modelling cannot be trusted. The community engaged independent Acoustic experts to provide an expert opinion on the modelling and that evidence is attached to this submission.

Further comments:

The proposed insulation scheme and mitigation measures proposed by the daa are insufficient to ensure that all significant effects are avoided, prevented, or reduced. The effects of aircraft noise on the cardiovascular system are indisputable and it can exacerbate preexisting cardiovascular disease. The WHO 2018 Guidelines evaluated the scientific literature up to 2015. Since then, there is increasing evidence supporting the adverse effects of aircraft noise, nighttime noise in particular, on health. The vulnerable in society are more susceptible. Aircraft noise can have long term and permanent effects on children's cognitive ability, mental and physical well-being. Sleep is disrupted by aircraft noise. The pattern and frequency of aircraft noise renders it more likely to cause sleep disturbance. With the proposal, communities impacted by the North Runway are somehow expected to get their full night's sleep in a restricted 6-hour timeframe (24:00-06:00). This is extremely unhealthy when sleep is disturbed and limited. This additional use of the North Runway at night also increases the significant adverse effects of the North Runway, contrary to the planning permission conditioned by ABP in 2007. Why increase the number of people significantly adversely affected and inflict serious noise and health problems on a whole new cohort of the population when there are alternatives available?

The Board's draft decision has recommended the approval of the NQS proposal from the daa along with a movement limit of 13000 movements. The Quota Count itself should also be adjusted far below 16260 in line with counterparts in the UK.

The biggest impact on my family is the result of Westerly arrivals into the North Runway. The aircraft are so low and noisy, and it impacts severely on the use of our house. The external amenity of our house is obliterated during these Westerly arrivals. Thankfully we are only exposed to these westerly arrivals on the North Runway 30% of the time due to the Wind direction. However, the daa and ANCA average out these extremes of noise into annual averages. This takes no account of the extreme torment suffered during the 30% of the year. It should be noted that the Planning Authority took account of 100% directional use when developing the Noise Zones to ensure that on any given day that no new dwelling would be exposed to high levels of noise. Unfortunately, neither ANCA nor the Planning Authority applied this same logic to existing dwellings. The Planning Authority has deemed it a serious health risk for any new dwellings in Zone A, yet they see it as ok to inflict this same level of noise on existing dwellings in Zone A. This is a serious issue, and the noise zones show that existing dwellings in Zone A should be afforded immediately relief from the severe noise levels. Failure to do so contravenes the Fingal Development Plan.

Notwithstanding the complexity of this application, we urge the Board to make a swift decision on this case. The local communities are suffering severe physical health, mental health and emotional health issues in relation to ongoing breaches by the daa of planning conditions imposed by the Board in 2007 when granting permission for the North Runway. How many breaches of planning are acceptable by the Board? Why are the daa allowed to carry on with impunity? An Bord Pleanála has a duty to ensure all planning and environmental laws are respected in their decision making. They also should take into account the proven track record of the daa breaching the very conditions laid down by the Board.

Previous submissions to the Planning Authority and ANCA from the '*St Margaret's The Ward Residents Group*' included relocation options for the dwellings most impacted by noise and where ANCA's decisions would leave these people vulnerable to the adverse effects of Aircraft Noise. An Bord Pleanala have the power to remove/amend the night-time restrictions and therefore the onus is on the Board to find a safe environment for these people and their families to live. In their current draft decision, the Board have not explored relocation options or taken on board the residual health effects and costs associated with their decision. The community has proposed Thornton Hall as such a site that would be acceptable to the community and the Board could make such a recommendation to explore this option in depth. To finance this relocation scheme, the community has advocated an increase to the passenger charge imposed on travellers along the lines of the '***Polluter Pays'*** principal. The monies raised from such a charge could be ring fenced to purchase Thornton Hall or equivalent site and provide housing for the displaced residents. The cost is borne by the 'Polluter' and not by Government or the daa. The community most impacted knows that it cannot stand in the way of Dublin Airport but it wants proper recognition for the harms inflicted on them and for the community to be provided with proper relocation so they can continue to live amongst their community and families. The option of

voluntary purchase is meaningless if you are displaced from your family and community.

In conclusion, we call on An Bord Pleanála to reject this Planning application and regulatory decision as there's no justification for it except inflicting health costs and carbon costs on the public. Planning is an afterthought for the daa. Their actions show they do not respect the decisions of the Board. It is 2 years now since the North Runway opened. Fingal County Council has taken enforcement proceedings against the daa in relation to the breach of Condition 5 (65 nighttime flights). The Council is also investigating the alleged illegal divergent flight paths off the North Runway. Unfortunately, for residents, the Council seems incapable of coming to a swift decision and appears to be waiting on the Board's decision in this Relevant Action application. It is therefore of upmost importance that the Board makes a decision in a timely manner to refuse permission for the Relevant Action application.

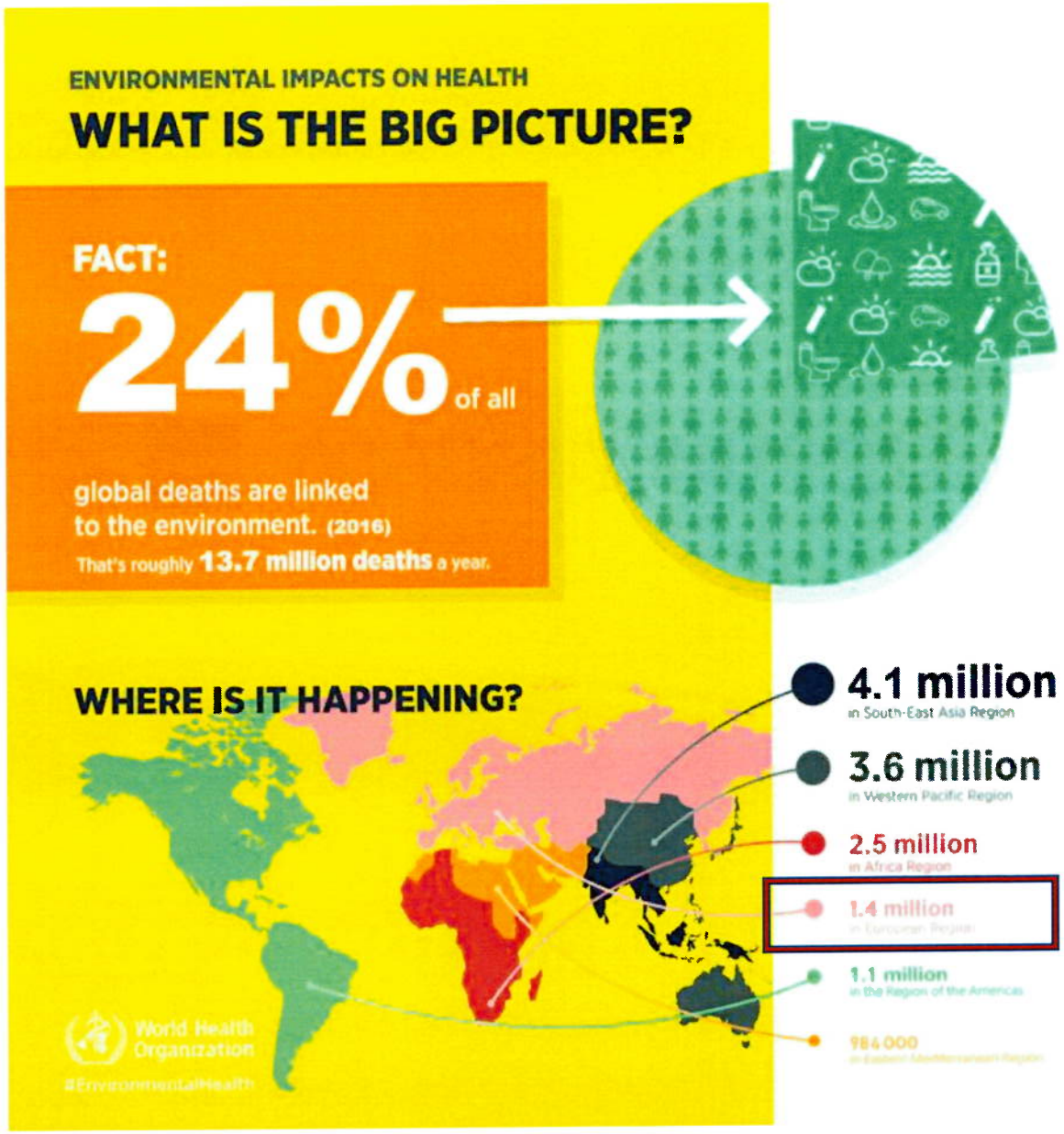
Adverse health consequences of noise

uecna
European Union Against Aircraft Nuisances

JG|U UNIVERSITÄTSmedizin. MAINZ

Omar Hahad
University Medical Center Mainz, Germany
Department of Cardiology, Cardiology I
24th of June 2024

The environment is important

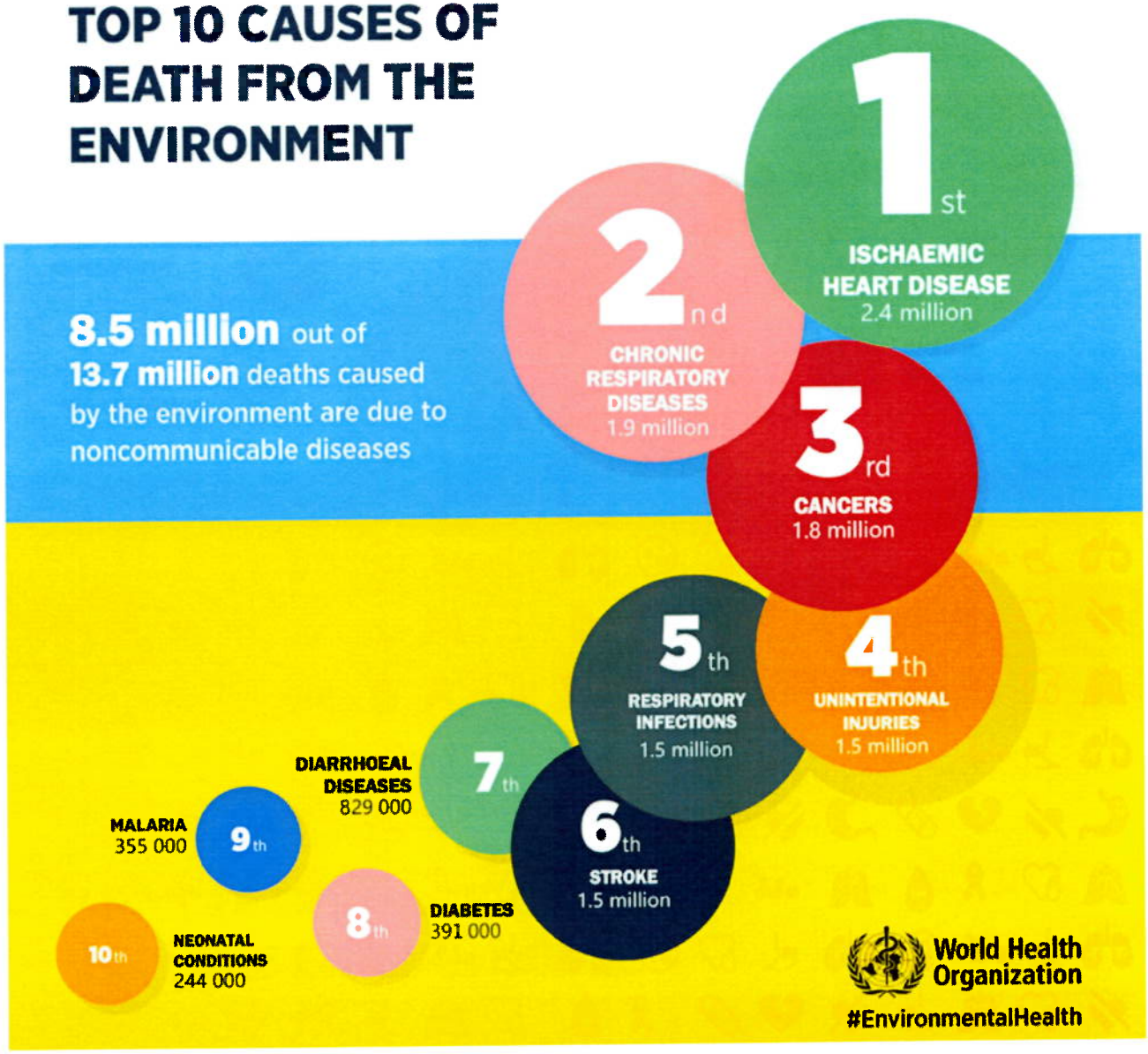


https://www.who.int/activities/environmental-health-impacts

Environmental risk factors

https://www.who.int/activities/environmental-health-impacts

Environmental causes of death



https://www.who.int/activities/environmental-health-impacts

Who is at risk?

https:// www.who.int / activities /environmental- health -impacts

WHO IS MOST IMPACTED BY THE ENVIRONMENT

Children under five and adults between 50 and 75 years old are most affected by the environment.

Environmental impacts on health are uneven across age and mostly affect the poor.

Low- and middle-income countries bear the greatest share of environmental disease.

Men
are slightly more affected due to occupational risks and injuries.

Women
bear higher exposures to traditional environmental risks such as smoke from cooking with solid fuels or carrying water.

YEARLY

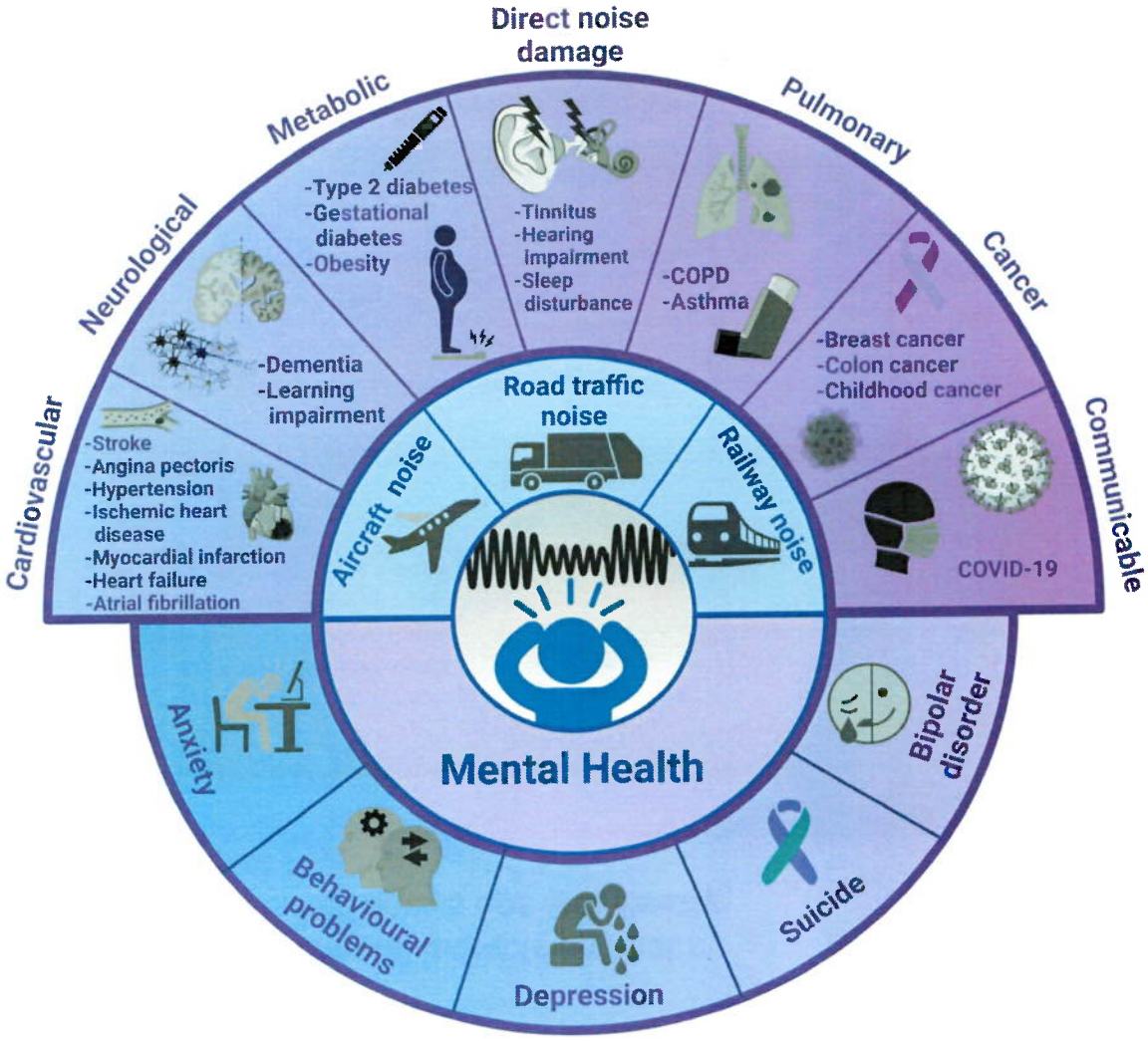
5.2 MILLION
Deaths in adults
between 50 and 75 years. The most common causes are noncommunicable diseases and injuries.

1.6 MILLION
Deaths in children
under five. The most prominent causes are lower respiratory infections and diarrhoeal diseases.

World Health Organization

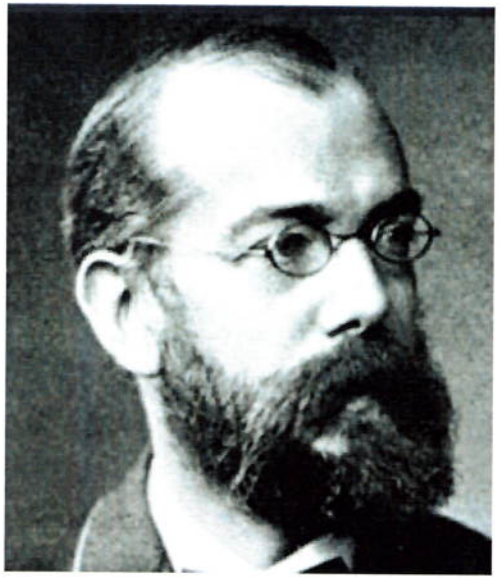
#EnvironmentalHealth

Is noise harmful to health?



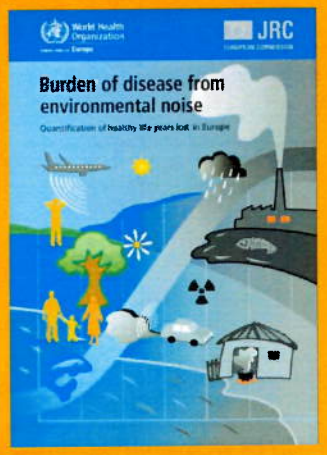
Hahad O et al. Noise and mental health: evidence, mechanisms, and consequences. J Expo Sci Environ, 2024.

Noise and health



„One day man will have to fight noise as fiercely as cholera and pest."

Nobel Prize winner Robert Koch (1910).

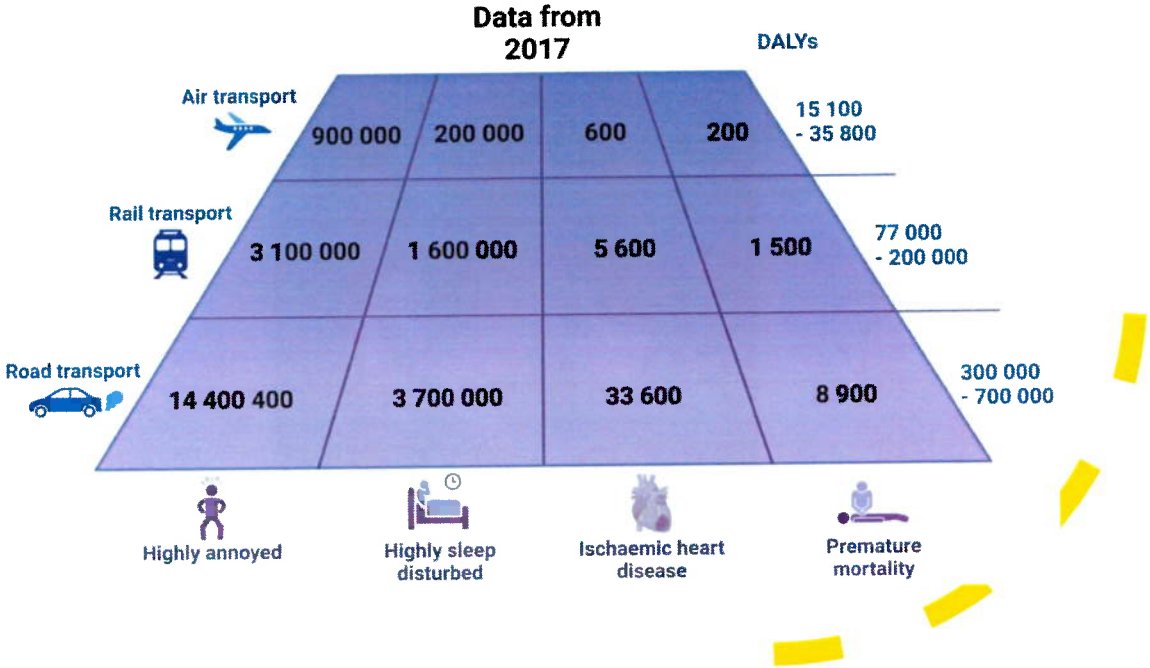


European Environment Agency

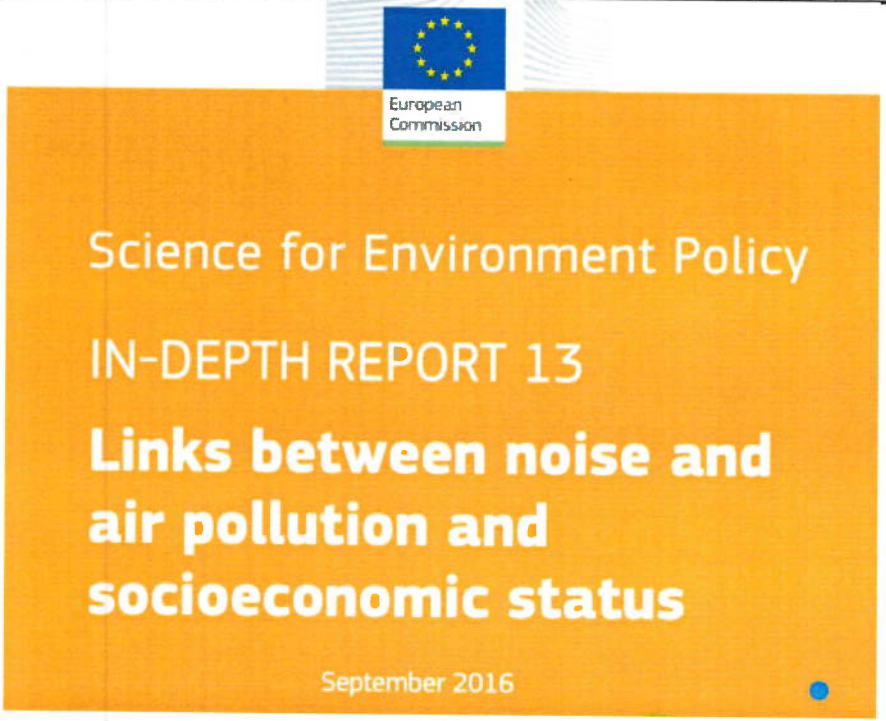
https://www.who.int/publications/i/item/9789289002295

https://www.eea.europa.eu/data-and-maps/figures/additional-information-on-health-impacts

Environmental noise causes the loss of up to **1.6 million healthy life years** (healthy life years lost due to illness, disability and premature death) in Western European countries every year



Noise rarely comes alone



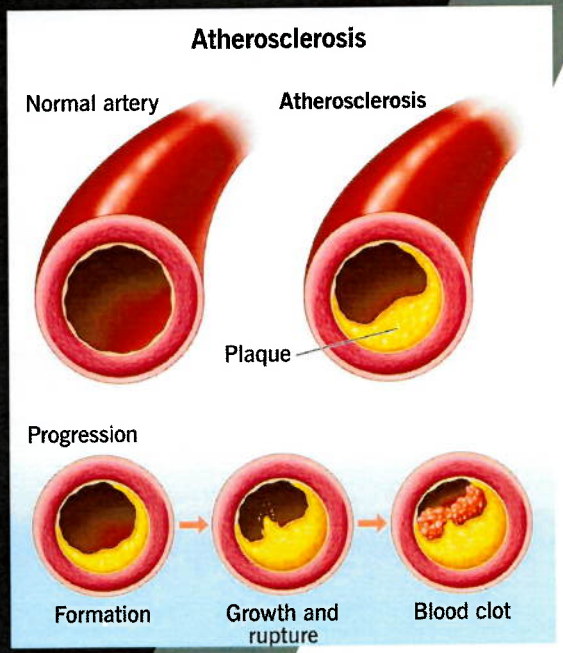
https://op.europa.eu/en/publication-detail/-/publication/1a3f0657-9a83-11e6-9bca-01aa75ed71a1/language-en

- Research suggests that the **social cost of noise and air pollution** in the EU - including death and disease — could be nearly **€1 trillion**.

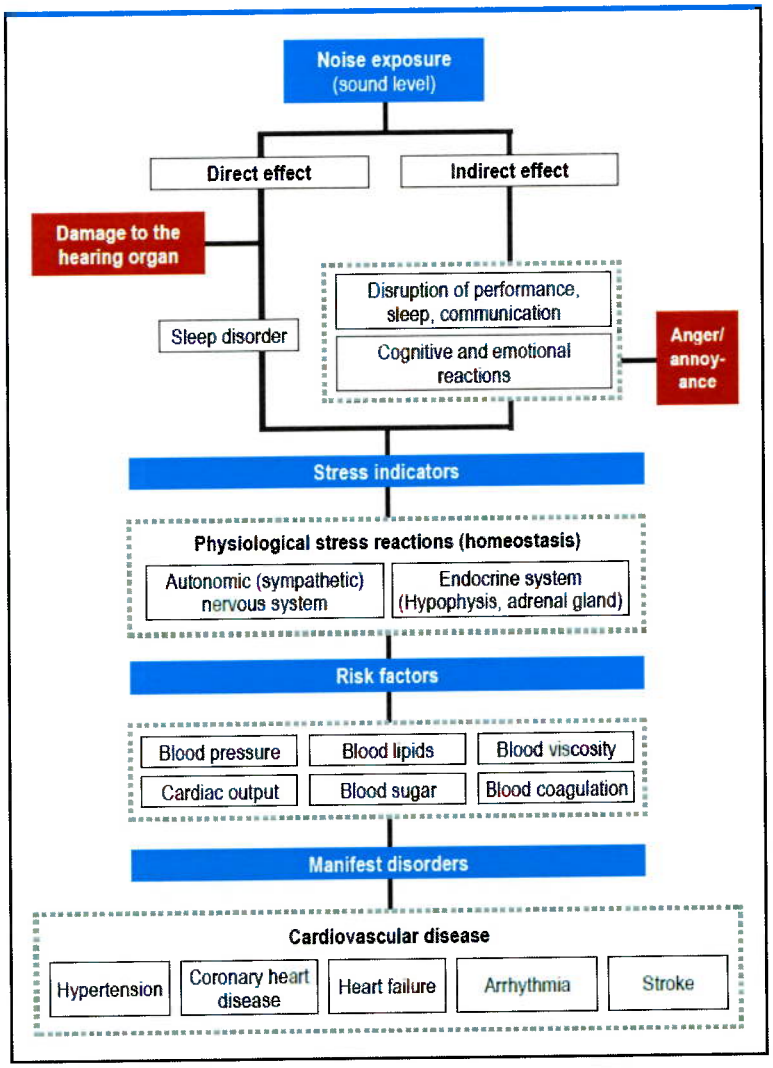
- For comparison, the social cost of **alcohol** in the EU has been estimated to be **€50-120 billion** and **smoking at €544 billion**.

How does noise make us sick?

Noise reaction scheme according to W. Babisch

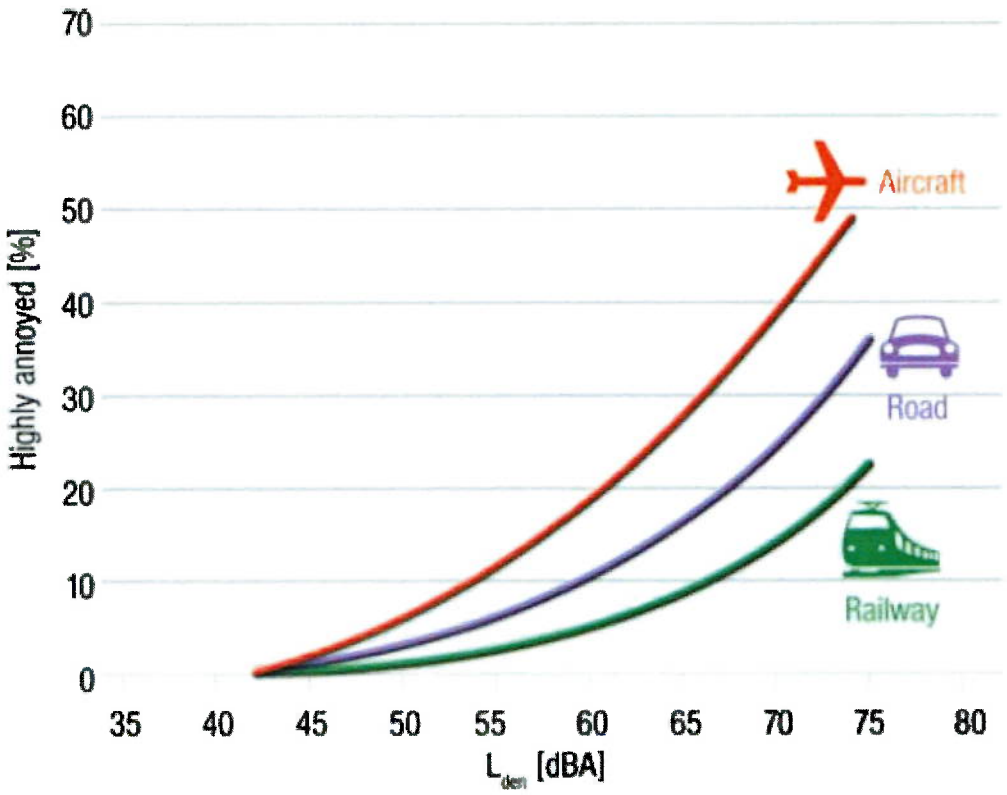


?



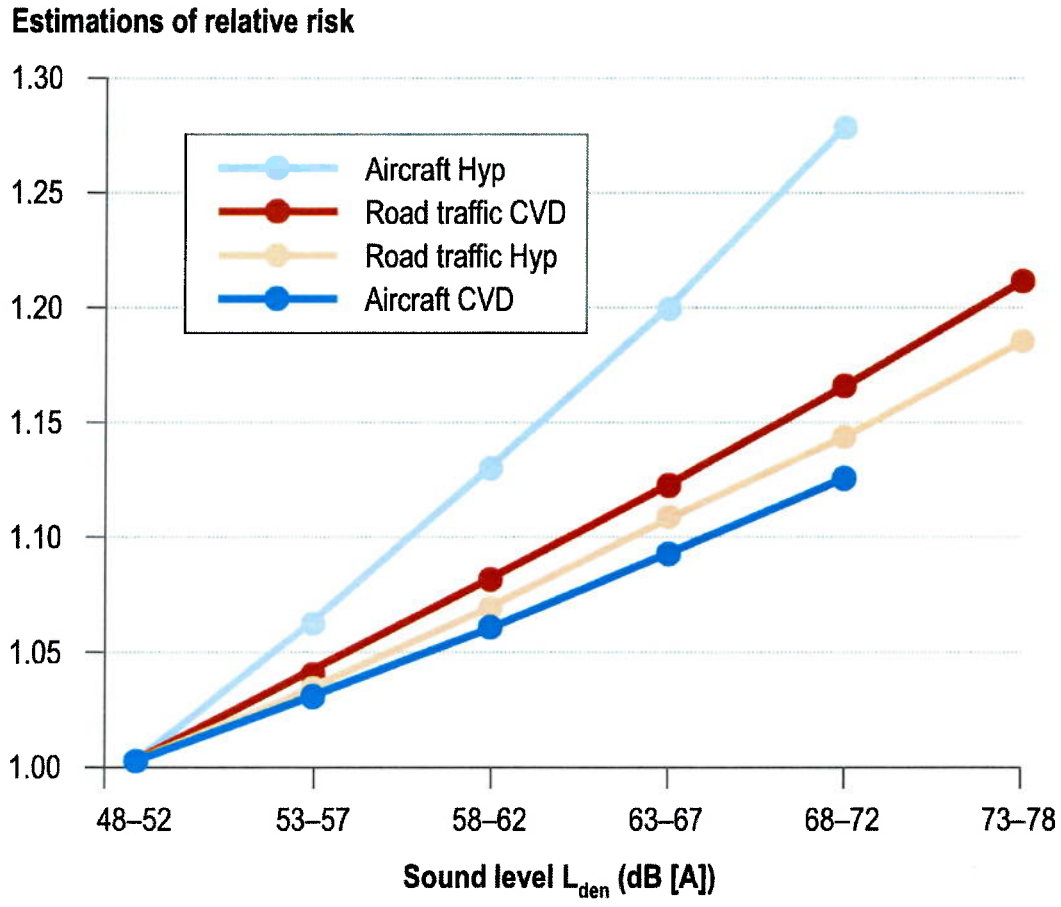
Hahad O et al. The Cardiovascular Effects of Noise. Dtsch Arztebl Int, 2019.

Aircraft noise bothers us the most



Münzel et al. Cardiovascular effects of environmental noise exposure. European Heart Journal, 2014.

Risk of high blood pressure and coronary heart disease due to aircraft noise

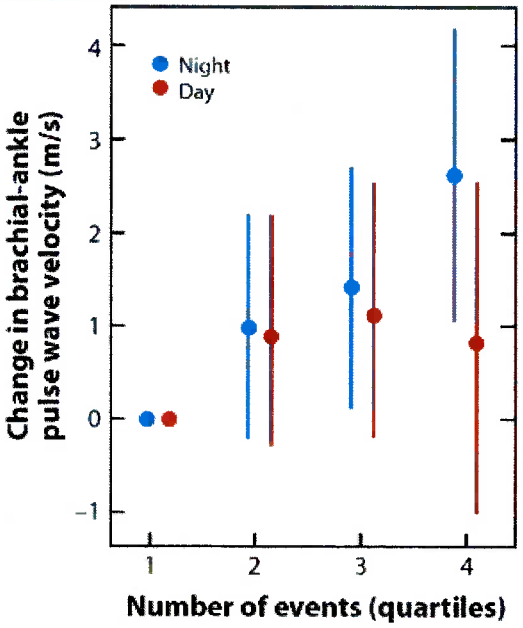


Hahad O et al. The Cardiovascular Effects of Noise. Dtsch Arztebl Int, 2019.

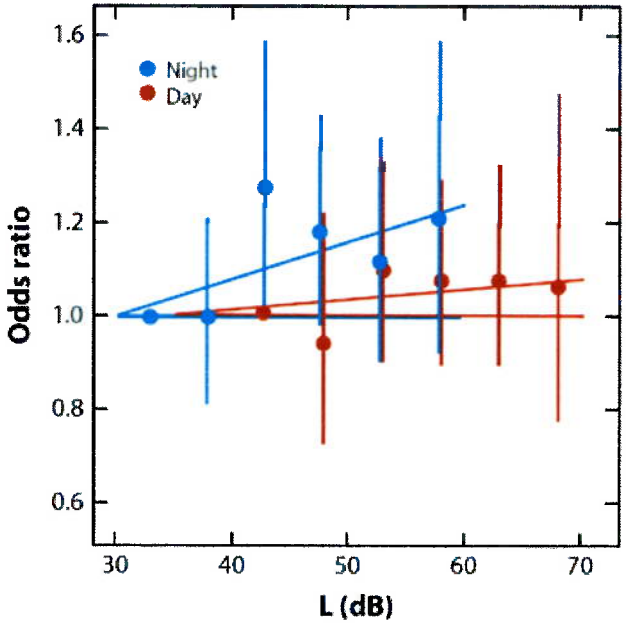
Important: Nocturnal aircraft noise

In particular, nighttime noise events (in blue) are responsible for increased vascular stiffness and high blood pressure compared to daytime noise events

Stiffness of the vessels



high blood pressure



Münzel T. et al. Adverse Cardiovascular Effects of Traffic Noise with a Focus on Nighttime Noise and the New WHO Noise Guidelines. 2020. Annual Reviews of Public Health .

Acute overflight and hypertension

European Heart Journal Advance Access published February 12, 2008

European Heart Journal
doi:10.1093/eurheartj/ehn013

CLINICAL RESEARCH

Acute effects of night-time noise exposure on blood pressure in populations living near airports

Alexandros S. Haralabidis[1], Konstantina Dimakopoulou[1], Federica Vigna-Taglianti[2], Matteo Giampaolo[3], Alessandro Borgini[4], Marie-Louise Dudley[5], Göran Pershagen[6], Gösta Bluhm[6], Danny Houthuijs[7], Wolfgang Babisch[8], Manolis Velonakis[9], Klea Katsouyanni[1]*, and Lars Jarup[5] for the HYENA Consortium

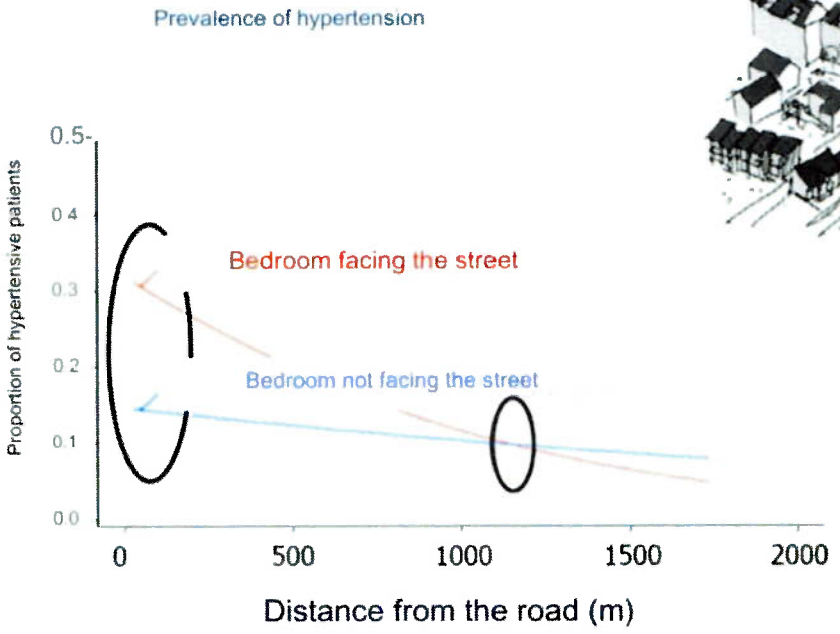
Aims Within the framework of the HYENA (hypertension and exposure to noise near airports) project we investigated the effect of short-term changes of transportation or indoor noise levels on blood pressure (BP) and heart rate (HR) during night-time sleep in 140 subjects living near four major European airports.

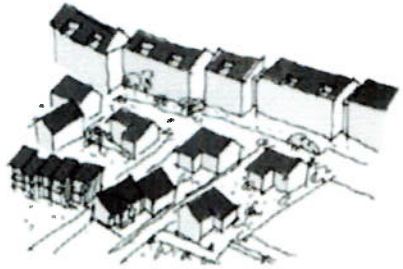
Methods and results Non-invasive ambulatory BP measurements at 15 min intervals were performed. Noise was measured during the night sleeping period and recorded digitally for the identification of the source of a noise event. Exposure variables included equivalent noise level over 1 and 15 min and presence/absence of event (with LAmax > 35 dB) before each BP measurement. Random effects models for repeated measurements were applied. An increase in BP (6.2 mmHg (0.63–12) for systolic and 7.4 mmHg (3.1, 12) for diastolic) was observed over 15 min intervals in which an aircraft event occurred. A non-significant increase in HR was also observed (by 5.4 b.p.m.). Less consistent effects were observed on HR. When the actual maximum noise level of an event was assessed there were no systematic differences in the effects according to the noise source.

Conclusion Effects of noise exposure on elevated subsequent BP measurements were clearly shown. The effect size of the noise level appears to be independent of the noise source.

Keywords Environmental noise • Blood pressure • Night-time sleep • Acute effects • Epidemiological study

Room orientation and high blood pressure





Lercher et al. (2000)

Insufficient sleep adversely affects both physical and mental well-being

European Heart Journal (2011) 32, 1484–1492
doi:10.1093/eurheartj/ehr007

CLINICAL RESEARCH
Prevention/epidemiology

Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies

Francesco P. Cappuccio[1*†], Daniel Cooper[1], Lanfranco D'Elia[2], Pasquale Strazzullo[2], and Michelle A. Miller[1†]

[1]Warwick Medical School, University of Warwick, CSB Building, UHCW Campus, Clifford Bridge Road, Coventry CV2 2DX, UK; and [2]Department of Clinical and Experimental Medicine, Federico II Medical School, University of Naples, Naples, Italy

Received 7 August 2010; revised 13 December 2010; accepted 13 January 2011; online publish-ahead-of-print 7 February 2011

Journal of Adolescent Health
Volume 66, Issue 5, May 2020, Pages 567-574

Original article

Sleep Disturbance Predicts Depression Symptoms in Early Adolescence: Initial Findings From the Adolescent Brain Cognitive Development Study

Aimée Goldstone Ph.D. [a, ✉], Harold S. Javitz Ph.D. [a], Stephanie A. Claudatos [a], Daniel J. Buysse M.D. [b], Brant P. Hasler Ph.D. [b], Massimiliano de Zambotti Ph.D. [a], Duncan B. Clark M.D., Ph.D. [b], Peter L. Franzen Ph.D. [b], Devin E. Prouty Ph.D. [a], Ian M. Colrain Ph.D. [a, c], Fiona C. Baker Ph.D. [a, d]

Nighttime aircraft noise around Zurich Airport

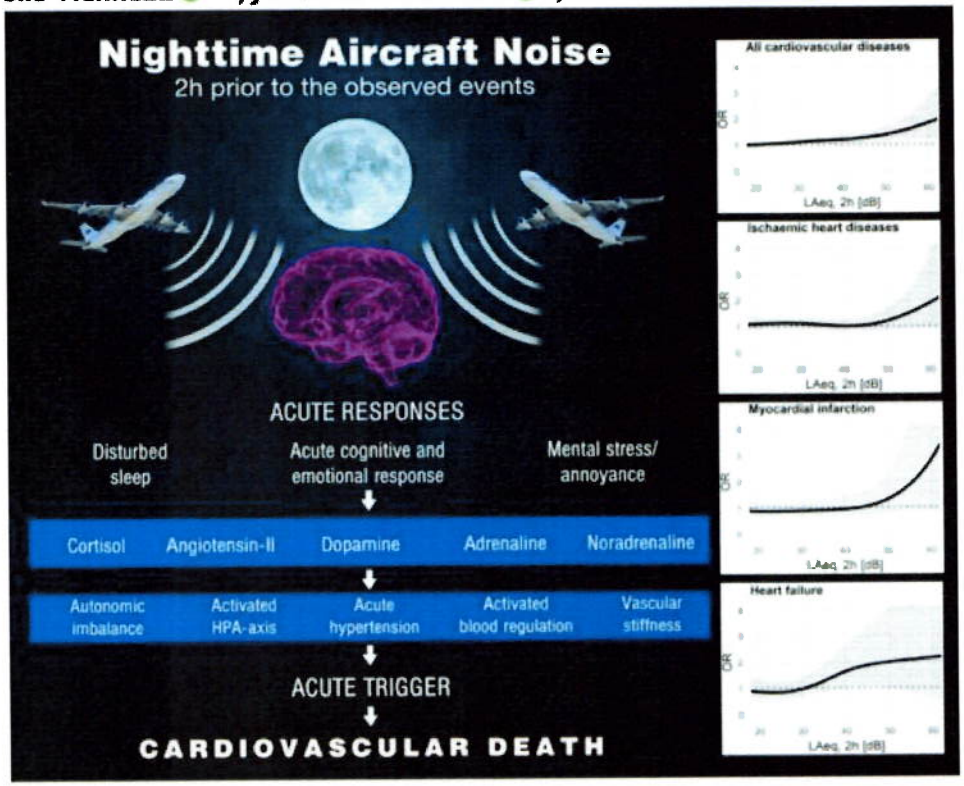
→The study found that the risk of cardiovascular death increases by 33 percent with nighttime noise levels betwee 40-50 decibels and by 44 percent with noise levels of 55 decibels.

ESC European Society of Cardiology
European Heart Journal (2020) 00, 1–9
doi:10.1093/eurheartj/ehaa957

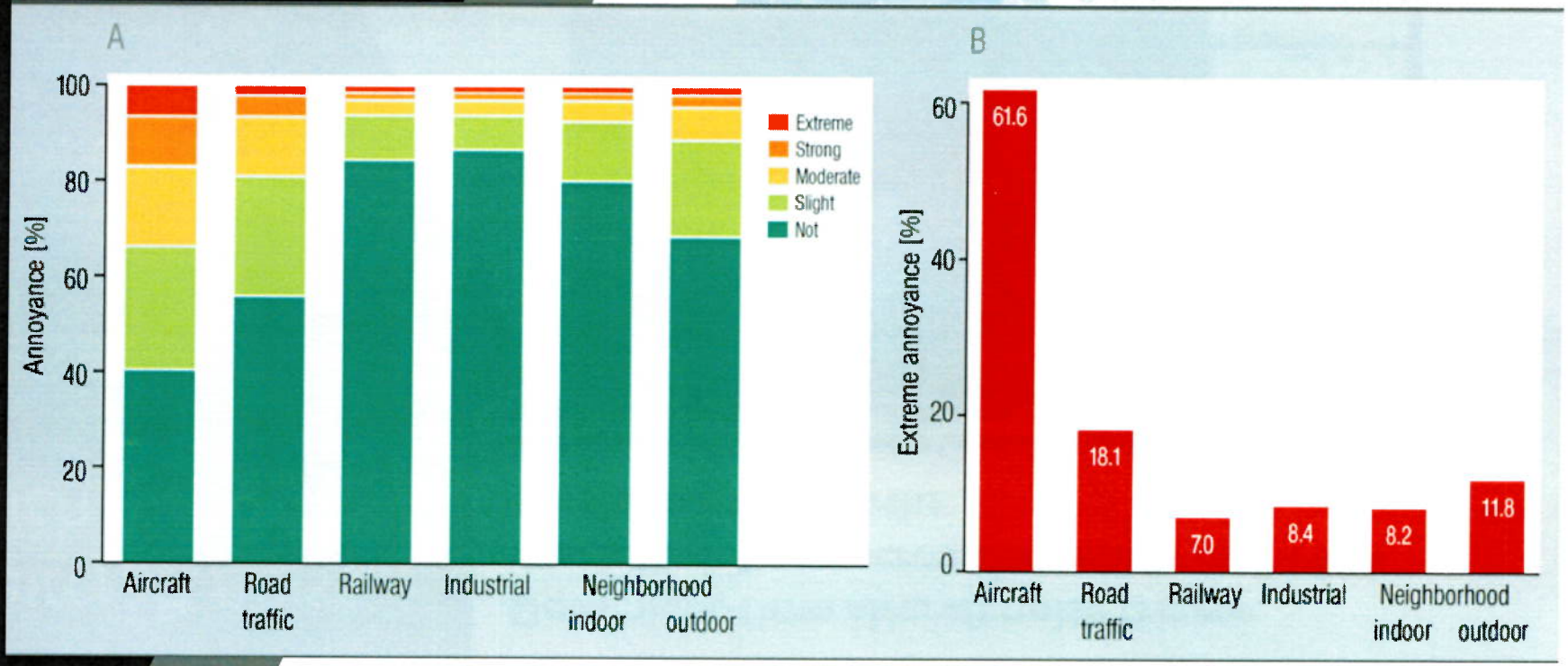
CLINICAL RESEARCH
Epidemiology

Does night-time aircraft noise trigger mortality? A case-crossover study on 24 886 cardiovascular deaths

Apolline Saucy [1,2], Beat Schäffer [3], Louise Tangermann [1,2], Danielle Vienneau [1,2], Jean-Marc Wunderli [3], and Martin Röösli [1,2]*



Our noise studies: Aircraft noise is dominant



Aircraft noise accounts for the largest share of total extreme noise pollution (right figure).

Aircraft noise annoyance in the general population

→more depression
→more atrial fibrillation

PLOS ONE

RESEARCH ARTICLE

Noise Annoyance Is Associated with Depression and Anxiety in the General Population- The Contribution of Aircraft Noise

Manfred E. Beutel[1]*, Claus Jünger[2], Eva M. Klein[1], Philipp Wild[3,4,5], Karl Lackner[6], Maria Blettner[7], Harald Binder[7], Matthias Michal[1], Jörg Wiltink[1], Elmar Brähler[1], Thomas Münzel[2]

1 Department of Psychosomatic Medicine and Psychotherapy, University Medical Center of the Johannes Gutenberg University Mainz, Mainz, Germany, 2 Medical Clinic for Cardiology, Angiology and Intensive Care Medicine, University Medical Center of the Johannes Gutenberg University Mainz, Mainz, Germany, 3 Preventive Cardiology and Preventive Medicine, Department of Medicine 2, University Medical Center of

International Journal of Cardiology 264 (2018) 79–84

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Annoyance to different noise sources is associated with atrial fibrillation in the Gutenberg Health Study

Omar Hahad [a], Manfred Beutel [b], Tommaso Gori [a], Andreas Schulz [c], Maria Blettner [d], Norbert Pfeiffer [e], Thomas Rostock [h], Karl Lackner [f], Mette Sørensen [g], Jürgen H. Prochaska [a], Philipp S. Wild [a], Thomas Münzel [a,*]

[a] Center of Cardiology, Cardiology I, University Medical Center of the Johannes Gutenberg-University Mainz and DZHK Standort Rhein-Main, Mainz, Germany
[b] Department of Psychosomatic Medicine and Psychotherapy, University Medical Center of the Johannes Gutenberg-University Mainz, Germany
[c] Preventive Cardiology and Preventive Medicine, Department of Medicine II, University Medical Center of the Johannes Gutenberg-University Mainz, Germany
[d] Institute of Medical Biostatistics, Epidemiology & Informatics, University Medical Center of the Johannes Gutenberg-University Mainz, Germany
[e] Department of Ophthalmology, University Medical Center Mainz, Germany
[f] Institute of Clinical Chemistry and Laboratory Medicine, University Medical Center of the Johannes Gutenberg-University Mainz, Germany
[g] Danish Cancer Society Research Center, Copenhagen, Denmark
[h] Center of Cardiology, Cardiology I, University Medical Center of the Johannes Gutenberg University Mainz, Germany

Field Study 1

European Heart Journal (2013) **34**, 3508–3514
doi:10.1093/eurheartj/eht269

CLINICAL RESEARCH

Effect of nighttime aircraft noise exposure on endothelial function and stress hormone release in healthy adults

Frank P. Schmidt[1], Mathias Basner[2], Gunnar Kröger[1], Stefanie Weck[1], Boris Schnorbus[1], Axel Muttray[3], Murat Sariyar[4], Harald Binder[4], Tommaso Gori[1], Ascan Warnholtz[1], and Thomas Münzel[1]*

[1]Department of Medicine II, University Medical Center, Johannes Gutenberg University Mainz, Langenbeckstrasse 1, 55131 Mainz, Germany; [2]Unit of Experimental Psychiatry, Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, USA, [3]Institut für Arbeits-, Sozial- und Umweltmedizin, University of Mainz, Mainz, Germany; and [4]Institute for Medical Biometry, Epidemiology and Informatics, University of Mainz, Mainz, Germany

Received 31 January 2013; revised 6 June 2013; accepted 20 June 2013; online publish-ahead-of-print 2 July 2013

Noise scenarios

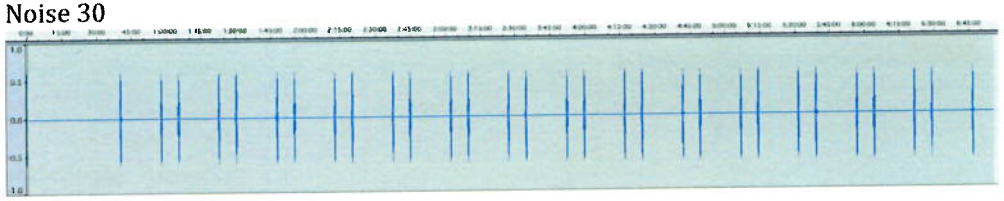
- **75 healthy subjects were** exposed to **simulated nighttime aircraft noise** at home while they slept

- **3 scenarios** : control scenario (no noise exposure), Noise30 (30 aircraft noise events) and Noise60 (60 aircraft noise events)

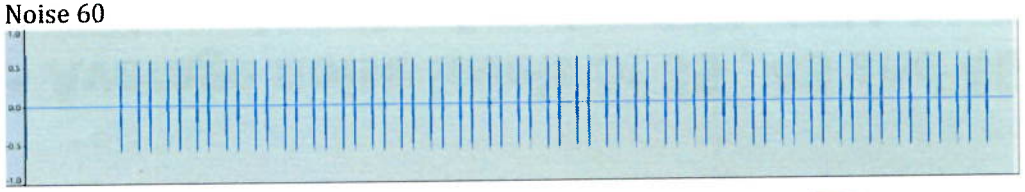
- **Average noise levels** of **35** , **43** and **46 dB(A)** and **peak noise levels of 60 dB(A**)

Setting

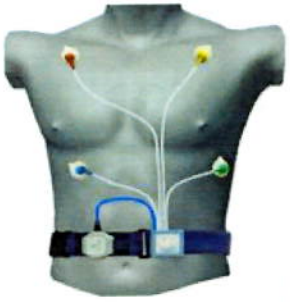
Noise 30

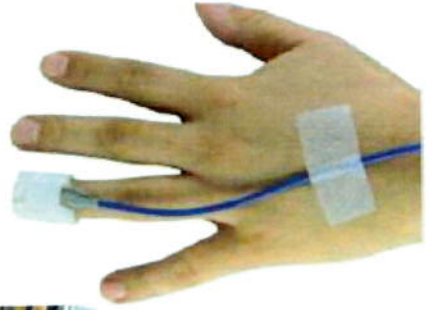


Noise 60

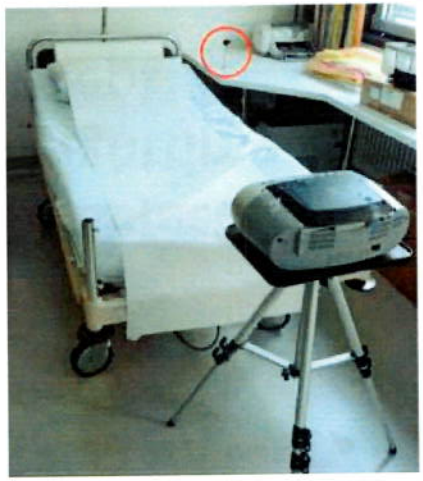


Noise scenaric



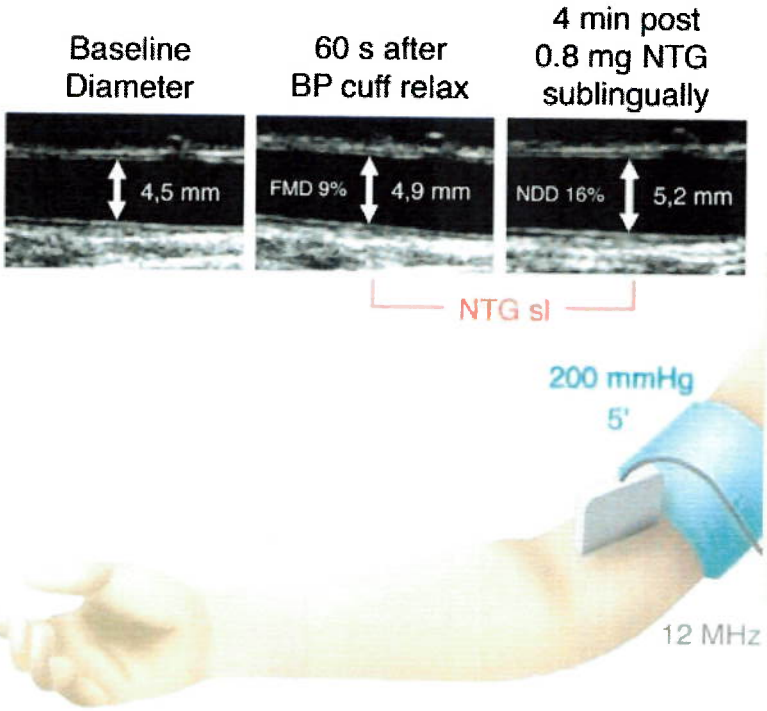


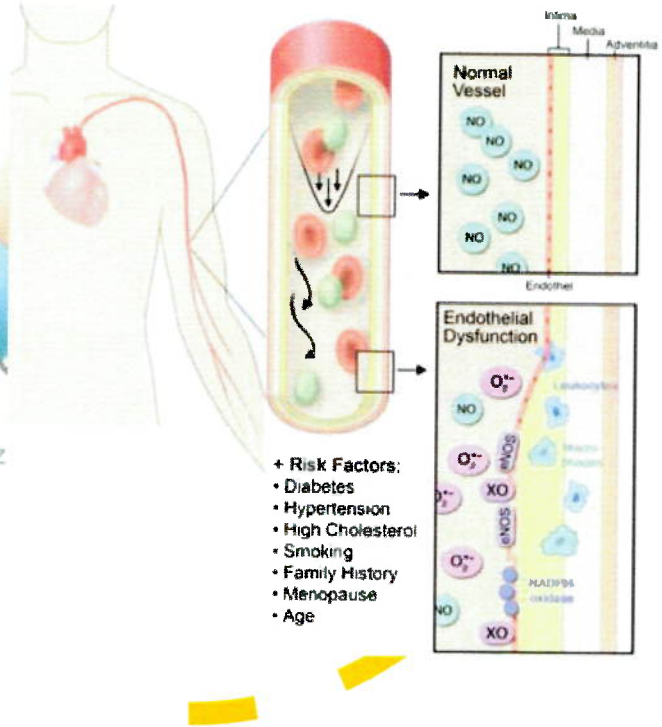
Polygraphy



Player

Measurement of vascular endothelial function





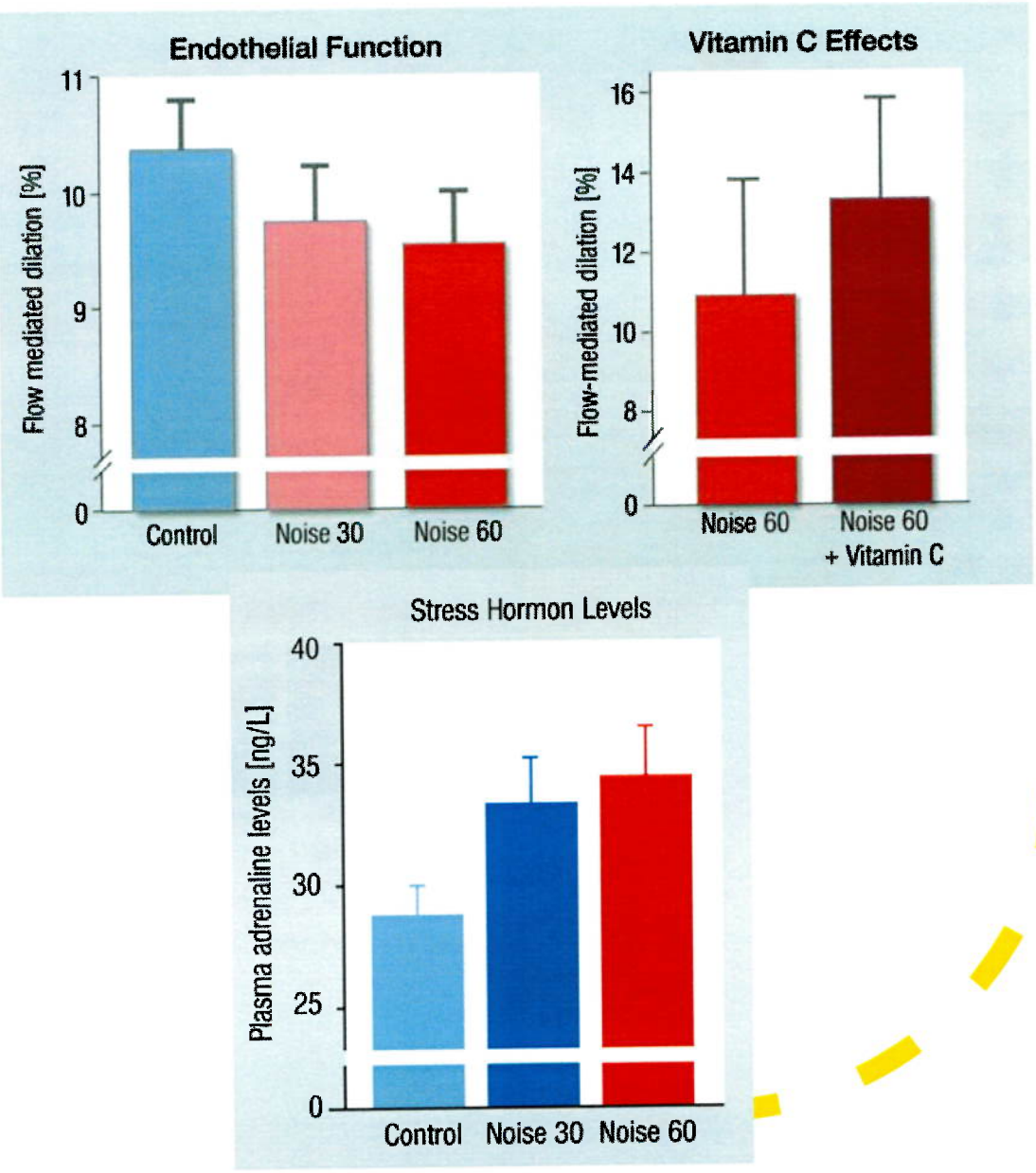
Results :

Significant deterioration in **sleep quality**

Increased release of **adrenaline**

Deterioration in **endothelial function**

Interesting: **Vitamin C** improves vascular function after noise exposure



Field Study 2: Patients with coronary heart disease

In patients with existing coronary heart disease, the aircraft noise effects were significantly more pronounced

Clin Res Cardiol (2015) 104:23–30
DOI 10.1007/s00392-014-0751-x

ORIGINAL PAPER

Nighttime aircraft noise impairs endothelial function and increases blood pressure in patients with or at high risk for coronary artery disease

Frank Schmidt · Kristoffer Kolle · Katharina Kreuder · Boris Schnorbus · Philip Wild · Marlene Hechtner · Harald Binder · Tommaso Gori · Thomas Münzel

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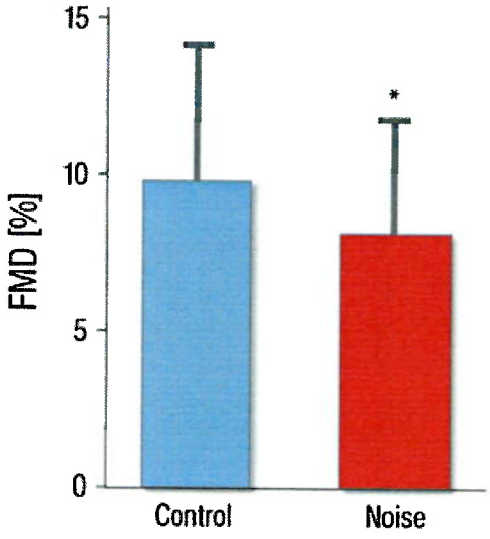
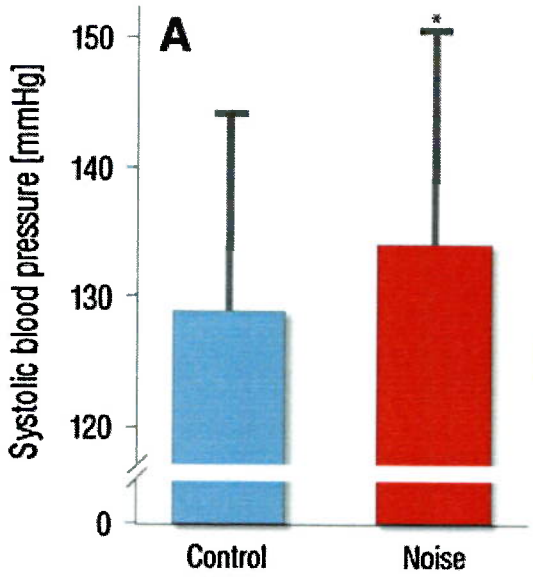




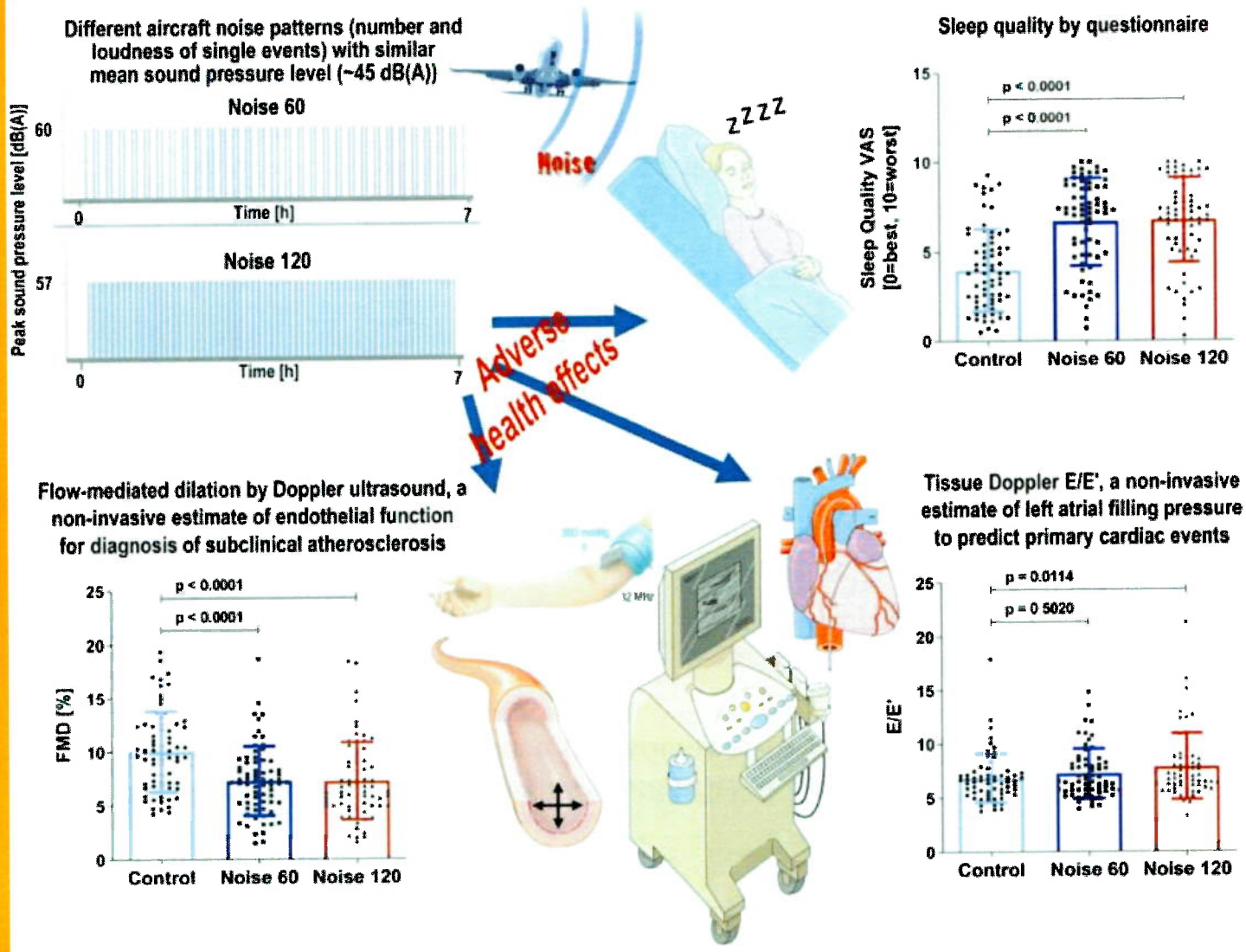
ESC

The impact of aircraft noise on vascular and cardiac function in relation to noise event number: a randomized trial

Frank P. Schmidt[†], Johannes Herzog[†], Boris Schnorbus[†], Mir Abolfazl Ostad[†], Larissa Lasetzki[†], Omar Hahad[†,‡], Gianna Schäfers[†], Tommaso Gori[†,‡], Mette Sørensen[§], Andreas Daiber[†,‡], and Thomas Münzel[†,‡*]

Field Study 3: Little loud vs. many quiet Aircraft noise events

result in comparable damage



What we need:

- Noise should be officially recognized as a manifest (cardiovascular) risk factor
- Integration into medical guidelines for prevention
- Legal regulation in line with WHO limits
- Restriction of nighttime noise (no-fly times, etc.)

Thank you for your attention

The Influence of Night-time Noise on Sleep and Health

Gezondheidsraad **President**
Health Council of the Netherlands

To the State Secretary for Housing, Spatial Planning and the Environment
PO Box 20951
2500 EZ The Hague

Subject	: Report on night-time exposure to noise
Your reference	: LMV 2003003076
Our reference	: U 1007/WP/718-K
Enclosures	: 1
Date	: 22 July 2004

Mr State Secretary,

Further to your letter, reference LMV 2003003076, I am pleased to enclose an advisory report on night-time exposure to noise. At my request, the report has been drawn up by a specially formed Health Council Committee. The report has been reviewed by the Standing Committee on Medicine and the Standing Committee on Health and Environment.

The report is based upon the compiling Committee's assessment of the findings of available scientific research into the influence that night-time exposure to noise has on sleep and health. In order to obtain a good overview of the relevant themes, the Committee began its deliberations with a workshop for experts from the Netherlands and other countries. The workshop took place on 2 July 2003 as part of the *8th International Congress on Noise as a Public Health Problem* (ICBEN2003) in Rotterdam. In addition, interested parties were invited—both in direct correspondence from myself and in an advertisement placed in the *Government Gazette* of 22 July 2003—to submit any information that they felt might be helpful to the Committee. The Committee took account of the eleven responses to this invitation that were received when preparing its report, and each respondent received an individual reply from the Committee.

The Committee paid particular attention to the strength of the evidence for a link between exposure to night-time traffic noise and increased risk of hypertension. Almost all the studies that have looked at hypertension and ischemic cardiovascular disease have focused exclusively on associations with noise exposure *during the daytime and evening*. A recent study, to which you also made reference in your letter, has suggested that night-time noise and its effects on and during sleep are much more significant than daytime noise for the development of hypertension. Although the Committee considers it plausible that a causal relationship exists between exposure to *night-*

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time noise and increased risk of hypertension, the Committee has concluded that the evidence for such a relationship is limited.

The Committee has noted that very little is known about the biological effects on children of exposure to noise when sleeping, or about the impact on children's health and well-being. The findings of the European research project *Road traffic and Aircraft Noise exposure and children's Cognition and Health* (RANCH) are due to be published shortly (probably in the summer of 2004). However, the Dutch participants in this project point out that these results will not entirely eliminate our lack of knowledge regarding the issue of childhood exposure to noise when sleeping.

I am also sending a copy of the enclosed advisory report to the Minister of Health, Welfare and Sport and another to the State Secretary for Transport, Public Works and Water Management.

Yours sincerely,
(signed)
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The Influence of Night-time Noise on Sleep and Health

to:

the State Secretary of Housing, Spatial Planning & the Environment

No. 2004/14E, The Hague, 22 July 2004 (corrected version, 27 January 2005)

The Health Council of the Netherlands, established in 1902, is an independent scientific advisory body. Its remit is "to advise the government and Parliament on the current level of knowledge with respect to public health issues..." (Section 21, Health Act).

The Health Council receives most requests for advice from the Ministers of Health, Welfare & Sport, Housing, Spatial Planning & the Environment, Social Affairs & Employment, and Agriculture, Nature & Food Quality. The Council can publish advisory reports on its own initiative. It usually does this in order to ask attention for developments or trends that are thought to be relevant to government policy.

Most Health Council reports are prepared by multidisciplinary committees of Dutch or, sometimes, foreign experts, appointed in a personal capacity. The reports are available to the public.

This report can be downloaded from www.healthcouncil.nl.

Preferred citation:
Health Council of the Netherlands. The Influence of Night-time Noise on Sleep and Health. The Hague: Health Council of the Netherlands, 2004; publication no. 2004/14E.

ISBN: 90-5549-550-6

Contents

Executive summary

Why this report?

Sleep is very important. It is therefore understandable that unintentional noise-related disturbance of sleep is a serious problem. Since it is not always easy to reduce disturbing noise, which is frequently associated with activities that are of value to the community at large, such as travel and transport, a debate has arisen regarding the health and well-being implications of sleep disturbance by environmental noise.

Like other countries, the Netherlands has regulations designed to limit public exposure to environmental noise, primarily with a view to managing the associated nuisance. Most of the limits relate to exposure over a complete twenty-four-hour period and do not therefore focus specifically on the period during which most people sleep. However, regulations are presently being prepared at EU level that do concentrate on night-time noise exposure. In due course, Dutch law will be brought into line with the new EU legislation.

Against this background, the State Secretary for Housing, Spatial Planning and the Environment wrote to the Health Council on 3 February 2003, asking for its advice regarding the influence of night-time noise on sleep, health and well-being. This report has been compiled by the Council's Noise, Sleep and Health Committee and addresses the questions posed by the State Secretary.

Exposure to night-time noise when sleeping

Environmental noise may originate from a wide variety of sources: air, road or rail traffic; industry and other localised activities; neighbours or one's general neighbourhood.

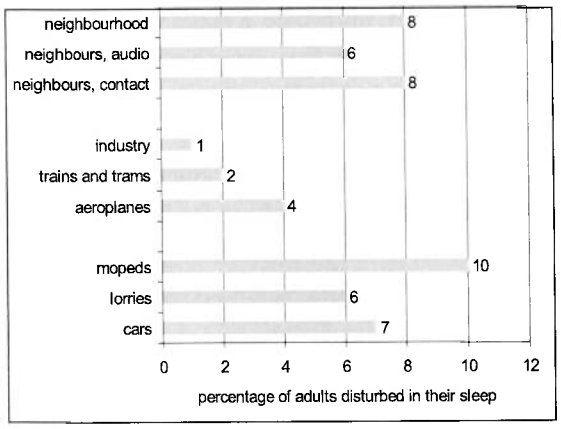
The consequences of exposure to night-time noise when sleeping have mainly been studied in relation to traffic noise. In the vast majority of cases, night-time traffic noise involves individually distinguishable noise events, such as the passage of a train, car or aeroplane.

Little research has been conducted into sleep disturbance from localised noise sources such as factories, firing ranges, shunting yards, wind turbines, climate control systems, building or demolition work. However, the Committee believes that the effects of noise from such sources are unlikely to differ essentially from the effects of traffic noise.

To date there has been no published research into a possible relationship between exposure to the other types of noise in the neighbourhood (recreational activities, children playing) and sleep disturbance. The Committee was therefore unable to assess the influence that such noise has on sleep.

Published research findings indicate that a variety of non-acoustical factors determine whether people are disturbed in their sleep by noises from neighbouring homes (voices, toilet flushing, footsteps, radio, television). The existence and complexity of these factors imply that it is not possible to establish meaningful relationships between night-time noise from neighbouring dwellings and the degree of sleep disturbance one suffers.

Research into the extent to which Dutch people claim to be disturbed by night-time noise during sleep is summarised below:



Effects of night-time noise during sleep

The Committee divided the effects of environmental noise during sleep into two general categories: biological responses and effects on health and well-being.

Biological responses to environmental noise occur because, even when asleep, an organism has to appraise and process stimuli from its environment. Such responses include waking up, having difficulty falling asleep again and increased motility.

It is plausible that, in the event of repeated exposure to night-time noise and under certain circumstances, some biological responses will have long-term implications for health and well-being. The Committee distinguishes five categories of effects:

- reduced sleep quality
- reduced general well-being
- impaired social interaction and reduced concentration during day-time
- specific disease symptoms
- loss of years of life (premature mortality).

Individuals differ from one another both in terms of their biological responses to night-time noise and in terms of the effects on their health and well-being. Thus, one person may take potentially harmful noise exposure levels in his or her stride without any significant adverse effects, while the health and well-being of someone else in a similar situation will deteriorate. In this context, much depends on the extent to which a variety of inherent and acquired personal factors interact with environmental factors.

Evidence

In order to assess the degree of certainty concerning the relationship between exposure to night-time noise and a particular effect, the Committee rates the available evidence as *sufficient*, *limited* or *insufficient*. Evidence is deemed sufficient if an indisputable relationship exists between exposure to night-time noise during the sleeping period and the effect in question, and if it is plausible from a biological model that the effect is attributable, at least in part, to the exposure. Evidence is rated as limited if a relationship between exposure and effect has been observed and a causal relationship is credible and plausible, but where the possibility of bias attributable to other factors cannot be excluded. The Committee also rates the evidence as limited when a relationship is plausible, and it has been observed that night-time noise exposure has an intermediary effect, which is known from other research to lead to the ultimate effect under consideration. Evidence is regarded as insufficient if the underlying research lacks the quality, consis-

tency or weight necessary to support a conclusion regarding the existence of a causal relationship.

Biological responses

There is sufficient evidence that night-time noise events cause direct biological responses, such as increased heart rate, reduced depth of sleep, increased motility and awakening.

Most biological responses begin to manifest themselves at an *SEL* in the bedroom of approximately 40 dB(A) (*LAmax* in the bedroom of at least 32 dB(A))*. Behavioural awakening (established by pressing a button) occurs when the bedroom *SEL* exceeds 55 dB(A).

The Committee also concludes that there is sufficient evidence of a relationship between exposure to night-time noise and a variety of biological responses exhibited before, during and after sleeping. Some of these are consequences of the direct responses already referred to: increased average heart rate, increased average level of motility, more frequent behavioural awakenings, and longer intervals of wakefulness. It additionally appears that average motility in people exposed to night-time traffic noise is greater at higher noise levels than might be expected on the basis of the direct responses. Higher levels of average motility are closely related to more frequent awakening, lower perceived sleep quality and increased daytime drowsiness.

Furthermore, there is sufficient evidence that people who, while attempting to sleep, are exposed to environmental noise or are concerned about being disturbed by noise in the night, have more difficulty falling asleep. After the sleep period, those who were exposed to night-time noise perceive the quality of their sleep to be impaired, find that their daytime mood is adversely affected and experience greater drowsiness, fatigue and irritability, especially in the morning.

There is limited evidence that under certain circumstances night-time noise can influence stress hormone levels. This effect was observed in women who were annoyed by noise at night and unable to protect themselves adequately to prevent the annoyance.

Implications for health and well-being

The Committee believes there is sufficient evidence that night-time noise has an adverse effect on quality of sleep and general well-being. Limited evidence exists that exposure to night-time noise has a negative impact on social interaction, on the performance of

* In acoustics, the following two values are employed to specify a noise event: *LAmax*, the maximum sound level during a noise event, and *SEL* (sound exposure level), a particular summation of all sound levels during a noise event.

concentration-sensitive tasks during the day, on specific complaints or disease symptoms and on loss of life years due to fatal accidents at work.

Reduced sleep quality is evident from studies on reduced self-reported sleep quality, difficulty falling asleep and remaining asleep, more frequent awakening during the night, shorter sleep periods and increased motility during sleep. A reduction in general well-being due to night-time noise is evident from self-reported sleep disturbance, self-reported health problems, use of sleeping pills and sedatives, and adversely affected daytime mood. Among older people in particular, the use of sleeping pills and sedatives is increased by night-time noise.

The medical conditions that may be linked to exposure to night-time noise are insomnia, high blood pressure and cardiac disease, as well as depression in females. Where insomnia is concerned, the Committee considers the evidence of a causal relationship as sufficient, while there is limited *indirect* evidence for the three latter conditions. There is also limited *indirect* evidence of an increased risk of involvement in a fatal accident at work as a result of sleeping problems and insomnia associated with exposure to night-time noise.

The Committee has estimated the extent of the impact of night-time noise on the health and well-being of the Dutch people in the year 2003 in terms of people who report to be highly sleep disturbed and people suffering from insomnia. The results have been based on data regarding cumulative night-time exposure to road, rail and aircraft noise, provided by the Netherlands National Institute for Public Health and the Environment (RIVM).

Effect	Prevalence in 2003 Number of people affected (thousands)
Self-reported high sleep disturbance	100-1000
Insomnia	10-100

The number of adults in the Netherlands in 2003 who reported to be highly sleep disturbed due to night-time traffic noise is between one hundred thousand and one million. The increase in the number of people with insomnia attributable to exposure to night-time traffic noise is estimated at 2 per cent of the number of people who reported to be highly sleep disturbed.

Using data on the specific exposures to road, rail and air traffic, the Committee estimates the number of adults who reported to be highly sleep disturbed to be more than 100,000 for each noise source (data for the year 2000; data for 2003 are not available as yet). This number for road traffic noise is about two to four times as large as the numbers for rail and aircraft traffic noise. The increased number of individuals with insom-

nia attributable to road and rail traffic noise amounts to between 1000 and 10,000 in each case. For air traffic noise in the region of Amsterdam Schiphol Airport the corresponding figure is between 100 and 1000 individuals.

Recently the collective burden of disease has been quantified in terms of *disability adjusted life years* or DALYs. Using data from an initial study by RIVM into the severity of various health effects, the Committee has calculated that high sleep disturbance resulting from traffic noise results in a burden of disease amounting to several tens of thousands of DALYs. The equivalent figure for insomnia is certainly an order of magnitude less than this. In spite of the uncertainties associated with such estimates, it does appear that, by affecting sleep, night-time traffic noise is one of the most important effects exerted by the physical environment on health.

Groups at higher risk

Direct cardiovascular responses to night-time noise may be more common in people with cardiovascular problems, people who consider themselves sensitive to noise, and in children. Due to lack of research, it is at present impossible to indicate whether children are possibly more sensitive than adults to other direct biological effects of night-time noise.

People with insomnia are at greater risk of biological effects due to night-time noise than good sleepers. Environmental noise exposure increases the time it takes to fall asleep, especially in people who are worried when they go to sleep. In addition, they also perceive their sleep quality as lower.

The Committee also considers it plausible that exposure to night-time noise is more likely to have an adverse effect on the health and well-being of the following groups: older people, pregnant women, women who have given birth within the preceding 12 months or so, people who regularly work at night, people with sleep disorders, physical pain, dementia, depression, hypertension, heart disease and pulmonary disease.

A special metric for night-time noise

In the Netherlands, special rules covering night-time noise are applied only in relation to scheduled overnight aircraft movements. However, from a scientific point of view, there is no reason why night-time noise from road traffic, rail traffic and industrial activities should be regarded as different from aircraft noise with respect to possible effects on health and well-being. In 1997, the Health Council recommended a system with two noise indicators to protect the public from traffic and industrial noise in the living environment. The Committee has taken up this proposal. According to the system put forward in 1997 the metric of exposure to noise over a twenty-four-hour period should be

representative of general annoyance, while the night-time noise metric should be related to sleep disturbance. Such an approach is rational since there is only a limited degree of comparison between the working mechanisms and effects of night-time noise on the one hand and general annoyance on the other hand.

In addition to *Lden*, the indicator of noise over a twenty-four-hour period, the European Union has adopted *Lnight*, an indicator to be used in the regulation of night-time noise. *Lnight* represents the noise exposure at the most exposed façade, calculated for an eight-hour night-time period (11pm to 7am), and averaged over a full year. In the calculations, more weight is given to the louder noise events than to the quieter ones. Since *Lnight* relates to the outdoor situation, the noise exposure in a person's bedroom may in practice be considerably higher than *Lnight* minus the average noise attenuation of a Dutch home. This is partly because homes differ considerably in the attenuation they provide (in the Netherlands, only newly built homes have to meet noise attenuation standards), and partly because most Dutch people choose to sleep with their bedroom windows at least slightly open. Furthermore, requirements on the basis of *Lnight* can never provide complete protection against sleep disturbance, since many Dutch people go to bed before 11pm and still more (roughly half of all adults) sleep beyond 7am.

Nevertheless, the Committee sees no benefit in adopting an alternative to *Lnight*, since it realises that it is impossible to address every conceivable factor by means of a regulatory noise metric. Furthermore, the Committee is of the opinion that regulations based on the use of *Lnight* (as well as *Lden*) could provide a considerable degree of protection against noise during sleep.

Additional metrics

In addition to setting standards based on *Lnight*, exposure limits could also be imposed on noise events, possibly by limiting the maximum permissible sound level or the number of events per night.

At a given *Lnight* value, the most unfavourable situation in terms of a particular direct biological effect of night-time noise is not, as might be supposed, one characterised by a few loud noise events per night. Rather, the worst scenario involves a number of noise events all of which are roughly 5 dB(A) above the threshold for the effect in question. Where motility is concerned, for example, the worst situation is one where all noise events have an *SEL* of roughly 45 dB(A) inside the bedroom. However, limiting the *SEL* inside the bedroom to less than the biological effect threshold levels is not a technically realistic option at the present time. Depending on how *Lnight* is regulated, one option might also be to limit the number of noise events.

An average adult experiences one or two 'spontaneous' behavioural awakenings during a typical night. The more noise events occur each night, the more likely it is that

a sleeper who awakens 'spontaneously' during an event will hear the noise, be annoyed by it, and then have trouble getting back to sleep. In extreme cases, a person can hear a noise up to ten times a night without being awoken by it. This would tend to argue in favour of limiting the number of events. Depending on the level to which *Lnight* is limited and the level of protection opted for, it could therefore be possible to limit the number of noise events (e.g. the number of trains, cars or aeroplanes per night). The effectiveness of applying such limits can only be estimated very roughly.

Adjustment of *Lnight* to take account of special noises

The Committee has considered the following 'special' environmental noises: low frequency noise (humming), noise containing low frequency components, tonal noise, impulse noise (noise that rapidly rises), industrial noise and sporadic but very loud noise events. Although little information is available concerning the influence on sleep of exposure to noise with these special characteristics, the Committee believes that there are reasons to assume that in some cases the effects are more pronounced than the effects of exposure to 'ordinary' traffic noise. In cases involving noise that contains low frequency components, tonal noise and impulse noise, the Committee suggests using the same adjustment factors for *Lnight* as proposed in the Health Council's 1997 report *Assessing Noise Exposure For Public Health Purposes*. Like its predecessor, the Committee is unable to propose an adjustment factor for low-frequency noise that consists entirely of humming, such as that associated with transformers and wind turbines. In cases involving noise from industrial activities, the Committee takes the view that research conducted since 1997 has shown that adjustments to match the effect of such noise to road traffic noise are not necessary.

It is not known whether sporadic but very loud noise events have any special consequences for sleep. The Committee is therefore unable to produce any scientifically based conclusion regarding these events.

Protective measures

In response to the State Secretary's question regarding ways in which the public may be protected against night-time noise, the Committee adopts the generally accepted environmental management and occupational health and safety strategies. Hence, the first step should be to reduce the noise at the source (and to reduce the number of sources), followed by measures designed to address the transfer of noise from the source to the 'receiver', and finally 'receiver-oriented' measures might be considered.

Many of the noise-reducing measures already in place are concerned primarily with limiting the impact of exposure to noise over a twenty-four-hour period. Additional

noise attenuation of the façade of bedrooms is one of the few measures that are taken to deal with night-time noise.

Little scientific research has been conducted into the effectiveness or efficiency of measures intended to protect against the consequences that either general noise exposure or night-time noise exposure has for health and well-being. Consequently, there is no sound scientific basis for making any statement regarding the effectiveness of any protective regime. Furthermore, increasing mobility is liable to offset the benefit that might be gained from many traffic noise reduction measures.

Furthermore, the Committee would like to emphasise the importance of instruction and communication as the final elements among the measures needed to keep the adverse effects of night-time noise within acceptable limits.

Often, there is no choice but to take both source-oriented and transfer-oriented measures, sometimes complemented by recipient-oriented measures. This is because – even disregarding the issues of effectiveness and efficiency – none of the possible forms of intervention is easy to implement. The Committee does not consider the introduction of personal hearing protectors an appropriate collective response to environmental noise, although such protectors may offer relief in specific cases.

Recommendations for future research

The Committee recommends that studies be carried out into various topics, in order to fill what it considers to be the most important gaps in our knowledge regarding exposure to night-time noise. These topics are the long-term effects of night-time noise on health and well-being, the effects of night-time noise on children, the effectiveness and efficiency of noise attenuation measures for façades and between dwellings, and the effects of noise produced by neighbours or by one's general neighbourhood. The Committee advocates that such studies be linked to international programmes, as the Health Council has indeed already proposed in its advisory report entitled *Gezondheid en milieu: Kennis voor beleid* (*Environmental Health: Research for Policy*).

Glossary of terms used in this report

Table 1 provides brief definitions of the terms used in this report. Several groups of terms are distinguished: terms relating to sleep and the measurement of sleep parameters, terms relating to the acute effects of exposure to night-time noise when sleeping, terms relating to health and well-being, and terms relating to the indexes of noise exposure used in this report.

Table 1 Glossary of general (sleep-related) terms, biological phenomena, terms relating to health and well-being, and indexes of noise exposure.

Term	Definition
General sleep-related terms	
Sleep inception time	The point in time when a person falls asleep.
Awakening time	The point in time when a person wakes up, as a precursor to arising and becoming active.
Sleep latency/inception period	The length of time taken to fall asleep, i.e. the interval between the point at which a person begins trying to go to sleep or allowing him/herself to go to sleep and sleep inception time.
Sleep period/sleeping time, sometimes referred to as 'sleep'	Period between sleep inception time and awakening time, including any interim intervals of wakefulness.
Time in bed	The sum of a sleep period and the associated sleep latency period.
Polysomnography	The measurement during a subject's time in bed of his or her brain activity by means of EEG, EOG and EMG. In this report, the term EEG measurement or scanning is used. The technique involves the use of electrodes to record electrical potentials in the brain. On the basis of international standards, the data collected can be used to identify phenomena such as the stages of sleep.

Sleep EEG	Graph created using data from EEG scanning during a subject's time in bed, showing the various stages of sleep as a function of time. From such a graph, it is possible to draw conclusions regarding the structure of the subject's sleep.
Actimetry	The measurement of accelerations associated with the movement of an actimeter. In scientific research, an actimeter is a device resembling a wristwatch, which measures how much the wearer moves (by recording accelerations above a given threshold) over a predetermined time interval, typically between one second and one minute. The curve representing the amount of movement as a function of time is known as an actigram.
Measurement of stress hormones	Measurement of the quantity of (stress) hormones – typically cortisol, adrenaline (epinephrine) and noradrenaline (norepinephrine) – in the blood, urine or saliva.
Registration of wakefulness	The indication by a subject (for the benefit of an investigator) that he or she is awake, typically after waking up in the course of or at the end of his or her sleep period, by pressing a button or performing some other conscious act.
Acute phenomena	
Heart rate acceleration	A temporary rise in heart rate relative to the average heart rate assessed shortly before a noise event.
Change in the quantity of a stress hormone	The difference in the quantity of a stress hormone in blood, urine or saliva samples collected at two successive points in time.
Sleep stage change (from deeper to less deep sleep)	Change from a deeper stage of sleep to a less deep stage, as determined by a sleep EEG.
EEG awakening	Transition from a state of sleep to a state of consciousness, as determined by a sleep EEG.
Motility	The presence of movement in a short time interval, as recorded on an actigram.
Motility onset	The presence of movement in a short time interval, following an interval without movement.
Subject-registered awakening (behavioural awakening)	Awakening that is registered by the subject by means of a conscious action.
Phenomena relating to one or more sleep periods or sleep latency periods	
Average sleep latency period	The average length of the sleep latency period on a number of occasions.
Average heart rate	The average speed at which the heart beats when asleep.
(Stress) hormone concentration	The concentration of (stress) hormone in blood, urine or saliva collected during and/or after a sleep period.
Duration of a sleep stage	The number of minutes that a sleeping person is in a particular stage of sleep.
Sleep fragmentation	Within a sleep period, the frequency and duration of intervals of wakefulness recorded on a sleep EEG or intervals of motility recorded on an actigram.
Average motility/motor unrest	Within a sleep period, the recorded number of intervals involving motility divided by the total number of intervals making up the sleep period.
Average motility onset frequency	Within a sleep period, the recorded number of intervals in which motility begins, divided by the total number of intervals making up the sleep period.
Perceived quality of sleep	The quality of sleep, as perceived by a subject and described in a questionnaire response or journal entry.
Sleeping problems: difficulty falling or staying asleep	Difficulty falling or staying asleep, as perceived by a subject and described in a questionnaire response or journal entry.

Sleep disturbance	Disturbance of sleep by night-time noise, as perceived by a subject and described in a questionnaire response or journal entry.
Health problems	Problems with health, as perceived by a subject and described in a questionnaire response or journal entry
Insomnia	Sleeping disorder consistent with an internationally accepted definition, which takes account of difficulty falling or staying asleep, the daytime implications and the duration of the problems.
Raised blood pressure/hypertension	A condition characterised by systolic blood pressure higher than 160 mmHg and/or diastolic blood pressure higher than 100 mmHg (internationally recognised definition).
Noise exposure indexes	
Sound pressure level at a given point in time	The intensity of a noise at a given point in time, expressed in dB(A) (A-curve decibels).
LAmax	Maximum outdoor sound pressure level associated with an individual noise event.
LAmax_i	Maximum indoor sound pressure level associated with an individual noise event.
Equivalent sound pressure level over a given time interval *T*: *LAeq,T*	Exposure to noise for the duration of a given time interval *T* (a twenty-four hour period, a night, a day, an evening) is expressed as an equivalent sound pressure level (measured in dB(A)) over the interval in question. The equivalent sound pressure level is an 'exponential average' of the sound pressure levels occurring during the interval in question, i.e. an 'average' calculated by a method that attributes greater weight to higher sound pressure levels.
SEL (sound exposure level)	Equivalent outdoor sound pressure level associated with an individual noise event, with the equivalent level standardised at one second.
SEL_i[a]	Equivalent indoor sound pressure level associated with an individual noise event, with the equivalent level standardised at one second.
Lnight	Equivalent outdoor sound pressure level associated with a particular type of noise source between 11pm and 7am, calculated over a period of a year.
Lnight_i	Equivalent indoor sound pressure level associated with a particular type of noise source between 11pm and 7am, calculated over a period of a year. *Lnight_i* equals *Lnight* minus a sound attenuation value specific to the fabric of the individual building and the particular type of noise source.
Lden (d: day, e: evening, n: night)	Equivalent outdoor sound pressure level attributable to a particular type of noise source, over a twenty-four-hour period, adjusted using evening and night factors, calculated on an annual basis.
Li	Equivalent sound pressure level representative of exposure to a particular type of noise source, occurring in an individual's bedroom while he or she is asleep.
I_{lu} and $I_{lu;k}$	Indexes of the attenuation of airborne noise by a screening surface (wall, floor, ceiling) between dwellings; $I_{lu;k}$ is based upon a reception room of standardised dimensions.
I_{co}	Index of the attenuation of contact noise by a screening surface (wall, floor, ceiling) between dwellings.

[a] If a noise event lasts for one second, the *SEL_i* for the event is the equivalent noise level during that second (*LAeq,1s*). If a noise event lasts for a hundred seconds, the *SEL_i* for the event is the equivalent noise level during those hundred seconds: (*LAeq,100s*) plus 10*lg100 = *LAeq,100s* + 20. A constant-level noise event that lasts for a hundred seconds therefore has an *SEL_i* that is 20 dB(A) higher than the *SEL_i* of a noise event of the same constant level that lasts for one second.

Chapter 1

Noise, sleep and health

1.1 Background

People cannot function without sleep. It is therefore understandable that any disturbance of sleep by environmental factors, in particular noise, should be a cause for concern. Since it is not always easy to reduce sleep-disturbing noise, which is frequently associated with activities that are of value to the community at large, such as travel and transport, a debate has arisen regarding the health implications of sleep disturbance by environmental noise. It is undeniably the case that noise tends to disturb sleep[1,2]. However, the precise significance of such disturbance for perceived health and the development of illness is less clear[1].

Like other countries, the Netherlands has legal controls designed to limit public exposure to environmental noise, primarily with a view to managing the associated annoyance. Most of the limits that exist are concerned with exposure over a complete twenty-four-hour period and do not therefore focus specifically on the period during which most people sleep. In the Netherlands, special rules covering night-time noise are applied only in relation to scheduled overnight aircraft movements. However, legislation is presently being prepared at the EU level that does seek ultimately to reduce night-time exposure. In due course, Dutch law will be brought into line with the new EU legislation.

1.2 Ministerial commission and establishment of the Committee

Against this background, the State Secretary for Housing, Spatial Planning and the Environment wrote to the Health Council on 3 February 2003, asking that an advisory report should be prepared on the effects of night-time noise on sleep and health (see Annex A). Specifically, the State Secretary asked the Council to address the following questions:

a What are the effects (expressed in quantitative terms as far as possible) of exposure to noise when sleeping?
b How do such effects compare with other effects on health, in terms of seriousness and magnitude?
c Is it necessary to take special account of any population groups that are at particular risk?
d In view of the effects referred to, would it be advisable to introduce special rules, similar to those contained in Directive 2002/49 and the Aviation Act, for night-time noise from sources other than air traffic?
e If so, is it sufficient for such rules to be based on *Lnight*, or are additional indexes of exposure required, with a view to regulating impulse-like noises and situations involving relatively infrequent but high-intensity noise events?
f Could the public be protected by the use of a. performance-related or design requirements for residential buildings, b. personal protective gear, c. rules regarding sound pressure levels outside buildings, d. rules relating to vehicles and machinery, or e. a combination of these measures?

In response, the President of Health Council established the Committee on Sleep, Health and Noise, referred to below simply as the Committee. The members of the Committee are listed in Annex B.

1.3 Methodology

Over the last few decades, the Health Council has produced several advisory reports relating, at least in part, to the influence on sleep of exposure to noise[1,2,6-8]. The present report builds upon these earlier publications and updates their findings where justified by the subsequent emergence of further scientific information.

To support the Committee's deliberations, the secretary produced a summary of available information concerning the interrelationships between noise, sleep and health. This involved carrying out a number of literature searches. The file of relevant literature was complemented by pertinent data supplied by members of the Committee.

In addition, interested parties were invited – both in direct correspondence and in an advertisement placed in the *Government Gazette* of 22 July 2003 – to submit any information that they felt might be helpful to the Committee. The bodies and individuals that responded to this invitation are listed in Annex C.

On 2 July 2003, the Committee organised an international workshop, which was attended by experts from the Netherlands and other countries. The workshop formed part of the *8th International Congress on Noise as a Public Health Problem* (ICBEN2003), which took place between 30 June and 3 July 2003 at *De Doelen* in Rotterdam. The Committee drew upon the information obtained at the workshop when preparing this report.

The Committee finalised the text of this report in the course of six meetings.

1.4 The collation of available scientific data

Relevant publications and reports were collected by several means:

- A search of the document library at TNO Inro's Department of Health and Environment was carried out for material relating to sleep and the influence of noise on sleep. A collection of relevant documentation was compiled in connection with preparation of the 1994 advisory report *Noise and Health*[2], and efforts have been made to keep the collection up to date over the intervening decade. In addition, reports on international (acoustic) conferences were screened for publications on the effect of noise on sleep.
- The library staff at the Health Council carried out searches of *Medline*, *Biosis*, *Embase* and *PsychInfo* for relevant documents published since 1994. These searches were performed using combinations of the keywords 'noise', 'sleep' and 'effect', with the latter linked to numerous parameters. Some of the effect parameters used were as listed in the first columns of Tables 12, 13 and 14*. Searches were also carried out using the effect variable specifications** referred to. Information about sleep disorders was sought by the Committee secretary using the keywords 'insomnia', 'prevalence' and 'sleep apnoea', 'prevalence' and 'narcolepsy', and 'prevalence' and 'restless legs syndrome'. Searches were also carried out using the names of a number of researchers known to be active in the field of noise-related sleep disturbance.
- Individual members of the Committee supplied literature concerning their specialist fields.

* The direction of the change in a given effect parameter was not specified. So, for example, searches were made on 'sleep stage', not on transition from a deeper stage of sleep to a less deep stage.

** So, for example, in addition to searching on 'stress hormone', searches were carried out using the terms 'adrenaline', 'noradrenaline' and 'cortisol'.

1.5 Structure of the report

The structure of this advisory report is as follows. Chapter 2 outlines the terminology used. Chapter 3 summarises the results of research into the effects of exposure to night-time noise when sleeping. Next, a number of acoustic issues are considered in chapter 4. In chapter 5, the Committee directly addresses the six questions posed by the State Secretary. The main body of the report concludes with a list of references.

Appendices A, B, and C set out, respectively, the content of the State Secretary's letter, the composition of the Committee, and the names of bodies and individuals who responded to the invitation to submit information for consideration by the Committee. Annex D contains a discussion of research into consequences of exposure to night-time noise when sleeping. Annex E describes the situation with regard to sleep disorders and sleeping problems in the population at large, and Annex F summarises the most recent Health Council advisory report on environmental noise (*Assessing Noise Exposure for Public Health Purposes*)[8]. Annex G reproduces the text of an attachment to a letter from the RIVM containing recent information on the noise exposure in the Netherlands.

Chapter 2

Central concepts

In this chapter, the Committee begins by presenting a summary of the different types of environmental noise (2.1). Section 2.2 explains the indexes used in this report to characterise exposure to noise, while section 2.3 is devoted to various aspects of sleep. In section 2.4, a model is presented that describes the influence of environmental factors on health and well-being. Finally, an assessment of the evidence for the effects of nighttime noise is made in section 2.5.

2.1 Research into the relationship between environmental noise and sleep and health

Environmental noise can be divided into a number of types on the basis of source:

- Traffic sources: aviation, road traffic, rail traffic and shipping
- Stationary environmental sources, such as factories, shooting ranges, shunting yards, wind turbines, climate control systems, (temporary) building and demolition sites
- People and human activities in the neighbourhood not covered by the first two categories (neighbourhood noise)
- People and human activities in adjacent dwellings (noise from neighbours)

Research into exposure to environmental noise may be divided into two broad types:

- Research into the prevalence of the effects of exposure to environmental noise (inventory research)

- Research into the relationship between exposure and the extent to which an effect occurs: epidemiological research with population groups and laboratory research with human subjects.

A nationwide Dutch inventory study was undertaken in 1998, in the context of which four thousand people aged sixteen and above completed questionnaires[9]. This study indicated that passenger cars, lorries and mopeds were the types of vehicle most often associated with sleep disturbance in the Netherlands (being mentioned as causes of disturbance by 7, 6, and 10 per cent of respondents, respectively). Sleep disturbance is (much) less frequently associated with noise from aviation or rail traffic, or from factories and other economic activities. Where noise from neighbours is concerned, the most frequently mentioned problems were contact noise (people going up stairs, slamming doors, etc) and noise from audio equipment, being referred to by 8 and 6 per cent of respondents, respectively. Neighbourhood noises also proved to be a significant cause of sleep disturbance, mentioned by 8 per cent of respondents. See Figure 1; further details are presented in Table 21 in Annex D.

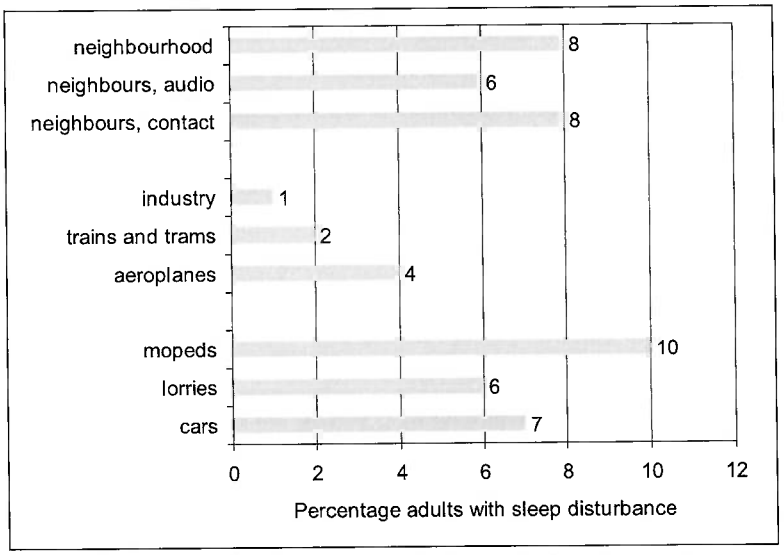


Figure 1 Percentage of adults in the Netherlands experiencing sleep disturbance due to particular noise sources in the residential environment[9]. The national inventory study carried out in 1998 asked respondents to indicate the extent to which their sleep was disturbed by noise from various sources, by giving a number between 0 and 10, where 0 = not disturbed at all and 10 = very highly disturbed. A standardised method was then used to calculate the percentage of respondents reporting sleep disturbance and high sleep disturbance. This involved transforming the 11-point scale into a continuous scale from 0 to 100. Respondents who scored 50 or more on this scale were deemed to suffer from sleep disturbance.

Most of the epidemiological and laboratory research that has been conducted into the relationship between, on the one hand, sleep and health characteristics and, on the other, exposure to night-time noise has focused on *noise from traffic sources* (other than shipping). Epidemiological research into the influence of *stationary environmental sources*, such as industrial premises, has been confined to self-reported noise-related annoyance over a twenty-four-hour period. However, there have also been some isolated laboratory studies that have looked at the effect of specific noise characteristics that can be associated with stationary environmental sources, such as a very rapid rise in intensity at the start of a noise event. The Health Council published a report on this topic in 1997[8]. The way in which the specific characteristics of environmental noise help to determine its effect is briefly explained in Annex F. The Committee returns to this matter in chapter 4, and in its answers to the State Secretary's questions.

Noise from neighbours comes in many different forms. Furthermore, research has shown that the factors which determine whether people are disturbed in their sleep by such noise are both numerous and very varied. As a result, it is not possible to determine the relationship between exposure to noise from such sources and the degree of sleep disturbance. However, in this report, the Committee does comment on the influence of features designed to attenuate noise transmitted between dwellings and on certain matters relating to the disturbance of sleep by noise from neighbours.

So far as the Committee has been able to ascertain, no research has been done into a possible link between exposure to *neighbourhood noise* and sleep disturbance. The Committee has therefore been obliged to disregard this topic.

To sum up, therefore, the nature of the scientific data research available is such that this advisory report necessarily concentrates on the consequences of night-time traffic noise (from sources other than shipping) on sleep and health characteristics.

2.2 Characterisation of exposure to night-time noise

The characteristics of a noise include its intensity and its pitch. The louder a noise is, the greater its intensity. The *intensity* of a noise is expressed in decibels (dB). *Pitch* is an expression of acoustic frequency: a buzzing noise is a low-pitch sound, while a hissing noise is a high-pitch sound. Most environmental noises have both high-pitch and low-pitch components. However, the ear is not equally sensitive to all such components. Consequently, when measuring the intensity of an environmental noise, a filter is normally used to reflect the range of human perception. The most widely used filter is known as the 'A filter', for the determination of a *sound pressure level* in dB(A). The 'A' in 'dB(A)' indicates that the figure is adjusted by an internationally standardised method to reflect the relative sensitivity of the ear to low-frequency and high-frequency

components ('A-weighting'). Another commonly used filter is the C filter; the main difference between the A filter and the C filter is that the latter allows through more low-frequency sound than the former.

The sound produced by most sources of environmental noise does not remain at a constant level over time. The noise from an aeroplane or train, for example, consists of a number of temporally distinct passages (noise events). By contrast, the noise from a motorway, when heard from a distance, is more of a constant drone. Exposure to constant or fluctuating noise for a given time interval (e.g. a twenty-four-hour period, night, day or evening) is expressed as an *equivalent sound pressure level* (in dB(A)) for the interval in question. An equivalent sound pressure level is a sort of average of the sound pressure levels occurring during the relevant time interval. However, it is not a true arithmetical average, since more weight is given to higher sound pressure levels than to lower sound pressure levels. Equivalent sound pressure levels for particular parts of the twenty-four-hour period are used as indexes of exposure both in research and for regulatory purposes.

The indexes used to characterise noise in this advisory report (as previously listed in Table 1) are briefly discussed below.

The *intensity of a noise event, as perceived in the bedroom* is characterised by *LAmax_i* and *SEL_i* (*i* stands for *indoor*). *LAmax_i* is the maximum sound pressure level during a noise event, while *SEL_i* is the equivalent sound pressure level of a noise event for a standardised one-second period. The *LAmax_i* and *SEL_i* for a given type of noise source are often closely related, as are the *LAmax* and *SEL*; so, for example, the correlation for indoor values of aviation noise was found to be 0.94 and that for outdoor values of lorry noise to be 0.99[10-13].

The long-term outdoor night-time noise exposure at a particular location associated with a particular noise source is characterised using *Lnight*, the annual equivalent sound pressure level between 11pm and 7am attributable to that source. Within the EU, *Lnight* is designated as the index of the night-time noise exposure attributable to a given noise source that should be used for certain purposes[3,4].

The *long-term night-time noise exposure in dwellings* can be characterised using *Lnight_i*. This index of equivalent sound pressure level is calculated by deducting from *Lnight* the average attenuation provided by the fabric of the walls. The Building Decree lays down requirements regarding the noise-attenuating properties of the walls of dwellings and other noise-sensitive buildings. The attenuation provided by the wall of a new building has to be at least 20 dB(A)[14].

The *long-term outdoor noise exposure at a particular site, as associated with a given noise source* is characterised with *Lden*, the annual equivalent sound pressure

level over a twenty-four-hour period. In the calculation of this figure, the equivalent sound pressure levels during the evening (7pm to 11pm) and the night (11pm to 7am) are increased by 5 and 10 dB(A), respectively. *Lden* is used in EU directives as an index of noise exposure over a twenty-four-hour period[3,4].

Li is an expression of the *personal noise exposure when sleeping associated with a given noise source*. It is an index of the equivalent sound pressure level in an individual's bedroom during the sleep period, as attributable to a given noise source over an extended period of time. It therefore expresses the individual's noise exposure when sleeping, taking account of the length of his or her sleep period, the time he or she goes to sleep and gets up, the outdoor noise exposure and the difference between the individual outdoor and indoor noise exposure. Calculation of the latter difference also takes account of whether the person in question has his or her bedroom window open or closed. Hence, while the *Lnight_i* for a given noise source may be constant throughout a particular part of a residential site, the *Li* values for the individual residents may differ significantly, due to behavioural differences or variations in the properties of the dwellings.

Sound attenuation between dwellings can be quantified using an index for the attenuation of airborne noise, I_{lu}, while $I_{lu;k}$ is a similar index which also takes account of the volume of the reception room and the area of the common screening structure, given its characteristic sound attenuation properties. A screen's ability to attenuate contact noise transmitted between two dwellings is quantified using the index for contact noise, I_{co}[14].

To give an impression of the environmental noise situation in the Netherlands, Figure 2 shows the distribution of the traffic-related outdoor noise exposure (*Lden, Lnight)* on dwellings in the year 2000, broken down by source category (motorway traffic, provincial road traffic, municipal road traffic, rail traffic and air traffic)[15]. From the graphs, it will be very clear that municipal road traffic generates the most noise, both at night and over a twenty-four-hour period.

To give another example, 40 dB(A) is a widely used limit for twenty-four-hour noise exposure (equivalent sound pressure level) in Dutch nature reserves and recreational areas. In the Central Veluwe Nature Reserve, the noise exposure (twenty-four-hour equivalent sound pressure level) associated with motorway traffic, provincial road traffic, rail traffic and air traffic accounts for, respectively, 19, 12, 6, and 0 per cent of all environmental noise in areas where this limit is exceeded[16]. Across the reserve as a whole, the average equivalent sound pressure level associated with all noise sources together is 53 dB(A); across areas where cycling is possible, the corresponding figure is 57 dB(A) and across areas where walking is possible, it is 52 dB(A).

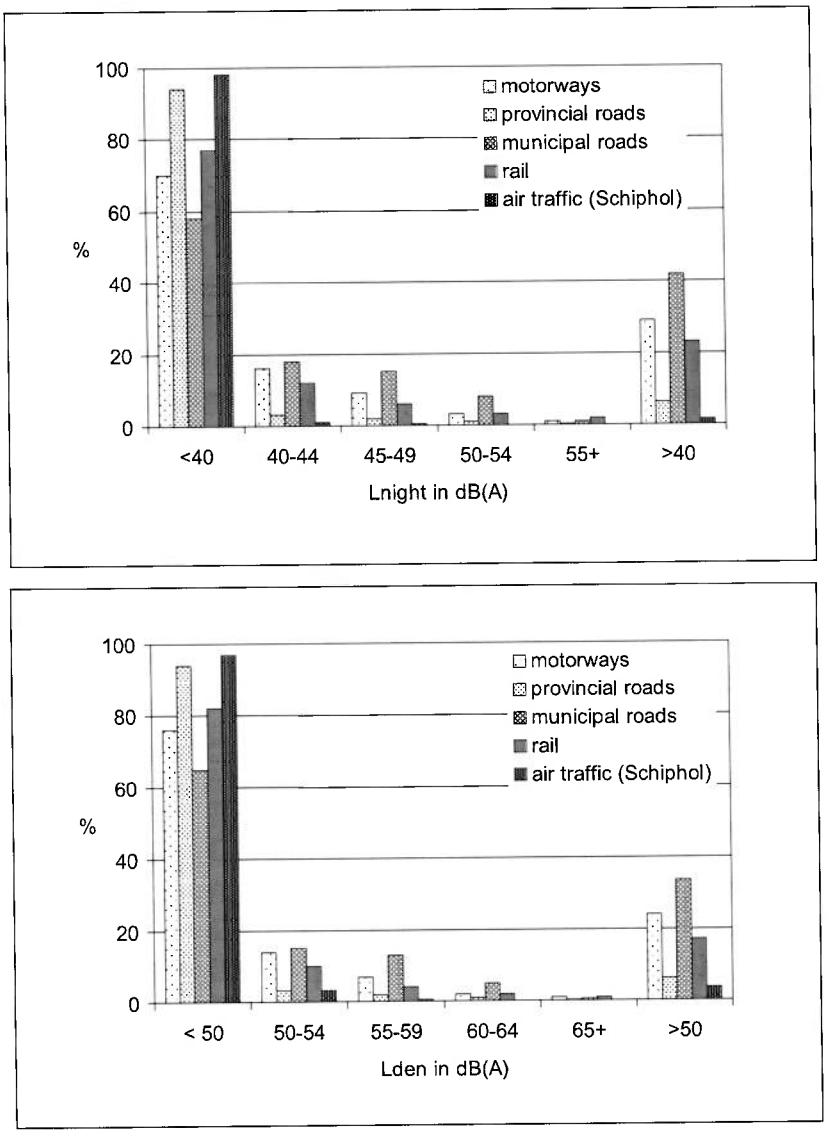


Figure 2 The distribution of traffic-related outdoor noise exposure (*Lnight* in the top graph, *Lden* in the bottom graph) on dwellings in the Netherlands in the year 2000, broken down by source category (motorway traffic, provincial road traffic, municipal road traffic, rail traffic and air traffic)[15].

2.3 Sleep

2.3.1 *What is sleep*?*

In the background study document[17] for the Health Council's 1991 report *Aviation Noise and Sleep*, Hofman – following the textbook *Principles and Practice of Sleep Medicine*[18] – described sleep as a periodically occurring state of apparent inactivity, in which the organism's responses to environmental stimuli are modified to an extent which is not uniform for all stimuli and which differs from one individual to another. Sleep should not be regarded as the mere absence of consciousness, but as a cyclical, active neurophysiological process[6].

By sleeping, people recover physically and mentally from their efforts. In addition, they process the information that they have absorbed during the day. Finally, sleeping is also enjoyable[19].

Human beings have an internal biological clock with a cycle of roughly twenty-four hours (the circadian rhythm: *circa* = approximately, *dies* = day). Sleep is also a cyclical phenomenon: in adults, it generally consists of roughly five periods of approximately 90 minutes, in each of which there is a spell of so-called 'REM sleep' (or 'dreaming sleep'; REM stands for *rapid eye movement*) and a spell of non-REM sleep. Non-REM sleep is itself divided into four stages, discernible from distinctive electroencephalogram (EEG) patterns. Stages 1 and 2 are referred to more generally as light sleep and stages 3 and 4 as deep or SWS sleep (SWS stands for *slow wave sleep*, a phrase that refers to the extended delta waves that characterise deep sleep on an EEG). When a person is awake, his or her EEG is characterised by so-called alpha and beta waves. Deep sleep tends to occur more towards the start of a period of sleep and REM more towards the end. As one gets older, the amount of deep sleep one needs declines. Waking up from time to time in the course of a period of sleep is part of a normal sleeping pattern[18]. Such waking periods may be brief or may last some while. 'EEG awakenings' of short duration, lasting between three and fifteen seconds, are referred to as *(cortical) arousals*.

It is generally believed that deep sleep and REM sleep are the most important sleep components, and that stages 1 and 2 are transitional stages. Both deep sleep and REM sleep are necessary for the processing of information taken in during the period prior to sleeping[20-31].

When one is asleep, changes also occur in one's hormone balance.

* See table 1 for explanations of the terms used.

2.3.2 *What is normal sleep?*

The term 'normal sleep' is defined in various ways in the published literature, by reference to both objective and subjective criteria. The objective criteria used include sleep duration, the length of time taken to fall asleep (sleep latency period), sleep efficiency (the time that one spends asleep as a percentage of the time one spends in bed), and the number of EEG awakenings, including cortical arousals. As well as being generally age-related and sometimes gender-related, these sleep characteristics vary substantially from one individual to the next. The subjective criteria used to define normal sleep are based on self-reported sleep characteristics, such as satisfaction with one's sleep, the feeling of being well-rested when one wakes up, and alertness during the day. As long as the values for all three characteristics are within a given range, the subject's sleep may be regarded as 'normal'.

People without sleep disorders who are not while sleeping exposed to loud noises (whether environmental noise, noise from inside their own dwellings or neighbouring dwellings) typically report waking up (subject-registered, behavioural awakening) one and a half to two times during an average sleep period, not counting the occasion that they wake up prior getting up[52]. The number of EEG awakenings, including cortical arousals, averages ten to twelve per night (although there is considerable individual variation)[52] Such events are therefore approximately six to seven times more frequent than spontaneous subject-registered awakenings. The general figures of one and a half to two subject-registered awakenings per night and ten to twelve EEG awakenings per night can be seen as defining the range of spontaneous awakening frequencies in a population unaffected by sleep disorders or sleep disturbance.

2.3.3 *Sleep and quality of life*

The phrase 'quality of life' is used to mean various things, three of which are taken into account here. First, 'quality of life' can refer to satisfaction with one's health: health-related quality of life. The phrase can sometimes also be an expression of satisfaction with life in general. In the latter sense, 'quality of life' is synonymous with 'happiness'. The third relevant meaning of the phrase is satisfaction with the environment in which one lives. It is in this third sense that 'quality of life' is most often used by researchers concerned with the annoyance caused by night-time noise. Nevertheless, in the Netherlands in particular, more attention has in recent years been given to quality of life in the first sense[32-35].

In order to measure any diminution of *health-related quality of life* associated with a given cause, such as night-time noise, one first has to specify the nature of the health

diminution. Where health diminution is detected (or assumed), an assessment of the subject's quality of life ('satisfaction'), in the form of a weighting factor, can be linked to the diminished state of health. In this way, diminution in health-related quality of life can be determined in quantitative terms. One expression of such diminution is Murray's DALY (*Disability Adjusted Life Year*)[36].

Diminished satisfaction with the environment in which one lives can be determined relatively easily by obtaining information from the subject using a questionnaire.

As a 'condition', sleep is also seen as a component of health. Thus, if a person is not sleeping well, the direct consequences – fretful waking periods in the night, tiredness the next day and the real or supposed impact of tiredness on daytime activities – lead to a diminution of his or her health-related quality of life. Such diminution can be substantial, as illustrated by the quality-of-life weighting system developed by Stolk *et al*[37]. Insomnia, as diagnosed by a GP, has a quality-of-life weighting of 0.83, compared with 0.93 for a spastic colon and 0.68 for localised lumbar pain.

The sleep disorders – particularly insomnia – and sleeping problems prevalent in the population at large are reviewed in Annex E. Following the examination of the influence that night-time noise has on sleep, the Committee considers whether there may be correspondences between, on the one hand, certain sleep disorders and sleeping problems in the population at large and, on the other, noise-related sleep disturbance and, if so, whether certain conclusions may reasonably be drawn concerning the influence of noise on sleep.

2.4 Environment and health

In several recent reports, the Health Council has presented its view of the relationship between environmental factors and health[1,38]. Figure 3 is a schematic illustration of the way that factors in the environment exert an influence and thus can have implications for human health and well-being. The use of the phrase 'health and well-being' in this context is indicative of the fact that, in considering the relationship between environmental factors and health, account is taken of subjective perceptions of health[39].

People are not passive under the influence of environmental factors. External influences trigger responses designed to modify their effects and, insofar as the influences are harmful, to counter them or compensate for them. Environmental factors will therefore always have an effect on a person, which is demonstrable in many cases. However, such effects do not necessarily have negative long-term implications for health and well-being.

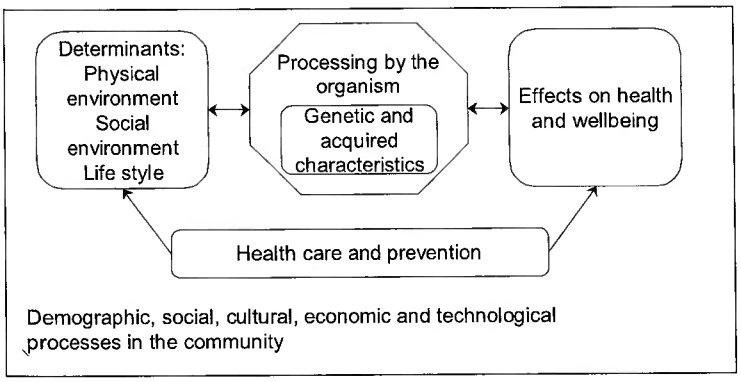


Figure 3 Model of the interrelationships between an individual's environment and his or her health and well-being (from earlier Health Council reports[1,38]).

The way an individual responds to external factors depends on a combination of inherent and acquired characteristics. Consequently, the effects of such factors and their implications for the health and well-being of the individual differ from person to person. A given potentially harmful influence may be tolerated by one person, but may adversely affect the health and well-being of another. Furthermore, the effect that an environmental factor has can be influenced by the extent to which other factors are simultaneously at work.

2.4.1 *Cause-effect chain*

The study of links between environmental factors and health generally involves following cause-effect chains[40] (see Figure 4).

At the point of progression from each block to the next, two questions have to be addressed:

- Is there a causal relationship: is the next event a consequence of the last?
- What influence do other factors have?

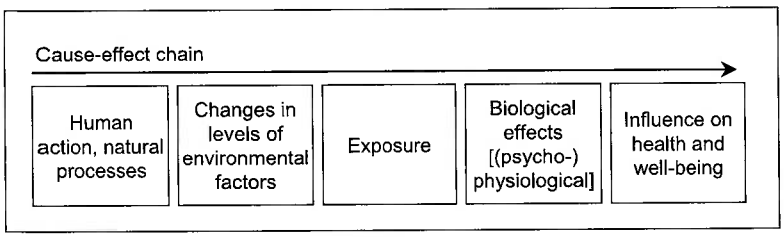


Figure 4 Cause-effect chain.

The 'exposure' block plays a special role. It may be regarded as a 'filter', which under certain circumstances connects the first two blocks to the last two. In line with the model shown in Figure 4, exposure leads to what are described in the diagram as 'biological' effects: physiological and psycho-physiological responses by the individual. These responses are sometimes predictors of brief or prolonged declines in the individual's health and well-being.

2.5 Assessment of the effects of night-time noise

For an assessment of the relationship between exposure to night-time noise when sleeping and effects on an individual's health and well-being, the Committee considers the following to be of particular importance:

- The distinction between biological effects and health effect (see Figure 4)
- The 'significance' of a health effect
- The exposure-effect process
- The strength of the evidence for each relationship.

These four topics are considered in more detail below.

2.5.1 *The distinction between biological effects and health effects*

In the model illustrated in Figure 3, environmental noise triggers biological responses from the individual because, even when sleeping, he or she still needs to assess and process 'stimuli' from the environment. The biological responses that are liable to occur include waking up, difficulties getting off to sleep and increased motor unrest while sleeping. To some extent, these responses involve acute changes during exposure to a noise, and to some extent they involve changes that manifest themselves over a night (before, while and after sleeping). In research, such effects are often used as markers of change in an individual's state of health and well-being. However, this makes it necessary to consider whether a given biological effect is in fact a predictor of long-term decline in health and well-being, which may or may not depend upon the nature and duration of the exposure. To this end, the Committee distinguishes between biological effects and effects on an individual's health and well-being. The former manifest themselves at the time of exposure and in the course of a sleep period, while the latter become apparent only in the longer term.

2.5.2 *The significance of a health effect*

The 'significance' of a health effect is a concept that includes the seriousness of the effect. The Committee has grouped the relevant effect parameters under five headings: quality of sleep; general well-being; social contacts and concentration; medical conditions; reduction in life expectancy.

2.5.3 *The exposure-effect process*

Not all levels of night-time noise have an effect. It is therefore desirable to be familiar with the exposure process that is liable to induce an effect. The Health Council's 1994[2] report on the consequences of exposure to noise introduced the term 'observation threshold' for use in this context. This term is defined as follows:

> The lowest level of exposure at which epidemiological research has shown noise typically has an effect on health. Where an exposure-effect function has been calculated for a given effect (...) the observation threshold will be obtained from that function.

In the current report, the term 'observation threshold' is also applied to effects that are not *necessarily* health effects. In most cases, epidemiological research with human subjects has found that effects occur only when exposure exceeds a certain level. It is possible that effects also occur – in some people, at least – at lower levels, but this possibility is usually not easy to investigate in practice.

2.5.4 *Strength of the evidence*

In order to define the degree of certainty concerning the relationship between exposure to night-time noise and a particular effect, it is normally necessary for a researcher to describe his/her findings in detail, since this is the only way to give a proper account of what is and is not known. However, when preparing a report for policy support purposes, it is desirable to indicate the degree of (un)certainty using a simple scale. The Committee has accordingly introduced a simple uncertainty scale, based on those developed by IARC[41] and a research team in Jülich, Germany[42].

Since this advisory report draws mainly on *epidemiological* research into the influence that night-time noise has on people's sleep, supplemented by a small number of laboratory studies, assessment of the strength of the evidence concerning a given relationship here involves determining the extent to which there is a statistically significant correlation between exposure and effect, so that a conclusion may be drawn concerning

causation by applying the so-called 'Hill criteria'[43] (about which more will be said later) and taking account of any other relevant considerations.

In line with the position taken by the IARC and the findings of the 1994 advisory report *Noise and health*, the following definitions have been adopted for the three categories of evidence.

Table 2 The strength of evidence concerning a relationship: definitions of the three levels.

Sufficient	A causal relationship has been demonstrated between exposure to night-time noise during the sleep period and a given effect. A relationship has been observed between exposure and effect in research which may reasonably be deemed to exclude the possibility of coincidence, bias and distortion, and it is plausible that the effect is attributable, at least in part, to the exposure.
Limited	A relationship between exposure and effect has been observed, and a causal relationship is credible, but the possibility of coincidence, bias or distortion cannot confidently be excluded. The presence of a relationship is generally plausible.
	No direct link has been established between exposure and effect, but there is good quality indirect empirical evidence for such a link, and the presence of a link is plausible. Indirect evidence may be said to exist if it has been observed that exposure has an intermediary effect, which is known from other research to lead to the ultimate effect under consideration.
Insufficient	The underlying research lacks the quality, consistency or weight necessary to support a conclusion regarding the existence of a causal relationship between exposure and effect. A link is not particularly plausible or is implausible.

The definitions given in Table 2 incorporate those developed by the IARC, but additionally make reference to the plausibility of a relationship. Hence, for the evidence of a relationship to be classed as 'sufficient', it is necessary for the causal link to be plausible. Otherwise, the evidence is classed as 'insufficient'. Furthermore, a subcategory of limited evidence not recognised by the IARC has been added: there is deemed to be limited evidence of a relationship where there is indirect empirical evidence that exposure has an intermediate effect, which is known from other research to lead to the ultimate effect under consideration. Inclusion of a relationship within this category depends on examination of its plausibility, with particular emphasis on the differences and similarities in nature and seriousness of the intermediate effect in each case (see Figure 5).

Hill criteria for assessing degree of certainty

When assessing epidemiological research findings to determine whether there is evidence of a causal relationship between exposure and effect, use is often made of the so-called Hill criteria[43]. In a speech to the *Section on Occupational Medicine* of the *Royal*

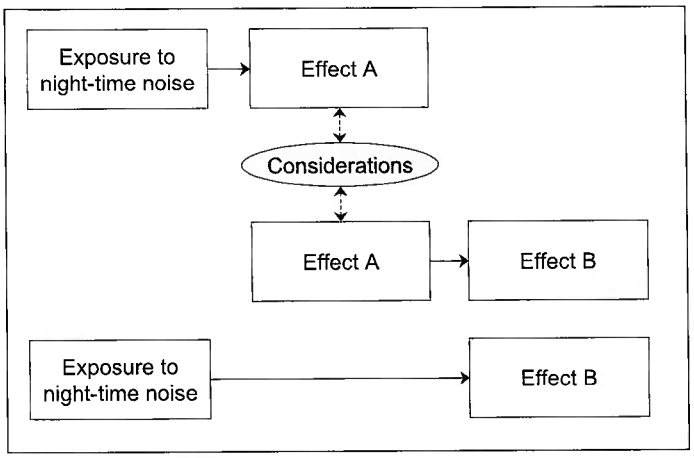


Figure 5 Indirect evidence. Indirect evidence that exposure to night-time noise has an effect (effect B). There is sufficient evidence for a link between exposure to night-time noise and effect A, and there is sufficient evidence that effect A leads to effect B in the general population. Furthermore, it is plausible that noise-induced effect A is consistent with effect A in the general population. Hence, there is limited evidence of a link between exposure to night-time noise and effect B.

Society for Medicine, the section chairman, Professor Austin Bradford Hill, put forward the following criteria for establishing an argument of causation:

- Strength of the relationship
- Consistency
- Specificity
- Temporal sequence
- Biological gradient
- Biological rationale
- Coherence
- Experimental evidence
- Analogous evidence.

Hill pointed out that it was not possible to provide absolute rules for the application of his criteria. What was required was careful assessment of the data, using the criteria for guidance. In practice, decisive criteria tend to be 'consistency', 'biological rationale', 'strength of the relationship' and 'biological gradient'[44].

Hill also said that statistical significance was of secondary importance, except insofar as a significance test served to remind the assessor that a study's observations might have been the product of mere chance. In recent years, meta-analytical methods have been developed to enable conclusions to be drawn by examining a number of studies

collectively. However, whether the application of such methods can ever substitute for careful, well-informed analysis is open to question[45].

Irrespective of the merits of meta-analysis for the extraction of evidence, the Committee does not believe that the available research data lends itself to quantitative meta-analysis with a view to reaching conclusions regarding the relationship between exposure to noise during the sleep period and (ultimate) effects on health and well-being. What is necessary is to reach consensus regarding the significance of the research findings, in which context the Hill criteria can, as indicated above, play a useful role.

Chapter 3

Effects of exposure to noise when sleeping

In sections 3.1 to 3.4, the Committee presents a survey of the effects of exposure to noise when sleeping, and draws a number of conclusions regarding the correlations between, on the one hand, certain sleep disorders and sleeping problems in the population at large and, on the other, the consequences of noise-related sleep disturbance. In Section 3.5, an estimate is made of the prevalence of some of the consequences of exposure to night-time noise for health and well-being and the associated disease burden in the Dutch population.

3.1 Laboratory and field research

In the following discussion of the available research data on the effects of exposure to noise when sleeping, the Committee concentrates on the findings of field research. The reason being that laboratory research does not always take proper account of the habituation to noise that tends to take place in practice. (Although it was, in fact, laboratory research that first demonstrated this effect twenty-five years ago (see Figure 6)[46].)

Figure 6 shows that, in the course of an experiment, the probability of EEG awakening decreases substantially, although there is barely any alteration in the probability of change from a deeper stage of sleep to a lighter stage. It may therefore be concluded that habituation does not influence all the effects of exposure to noise when sleeping to a similar extent. It should be pointed out that laboratory research has also shown that the probability of exposure to noise having a given effect can *increase* in the later nights of a study[47].

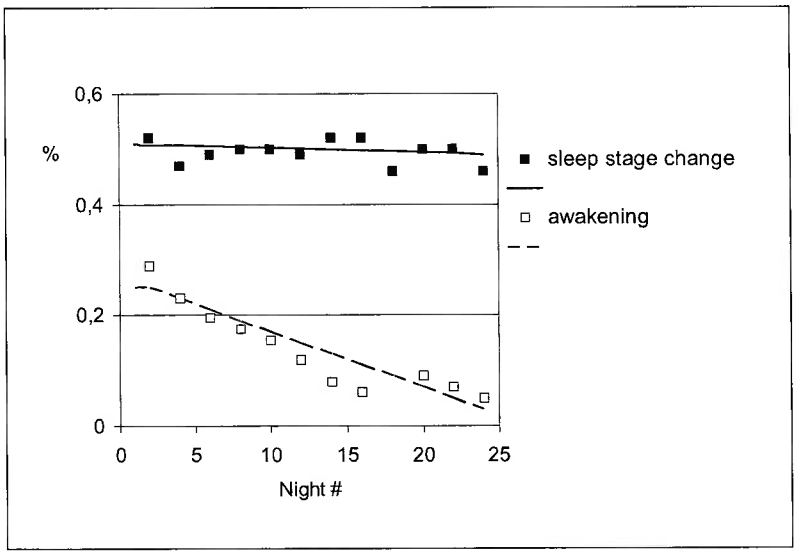


Figure 6 Average probability of the noise of a lorry with an *LAmax_i* of 65 dB(A) resulting in a change from a deeper stage of sleep to a less deep stage, or in EEG awakening, as a function of the night of exposure[46].

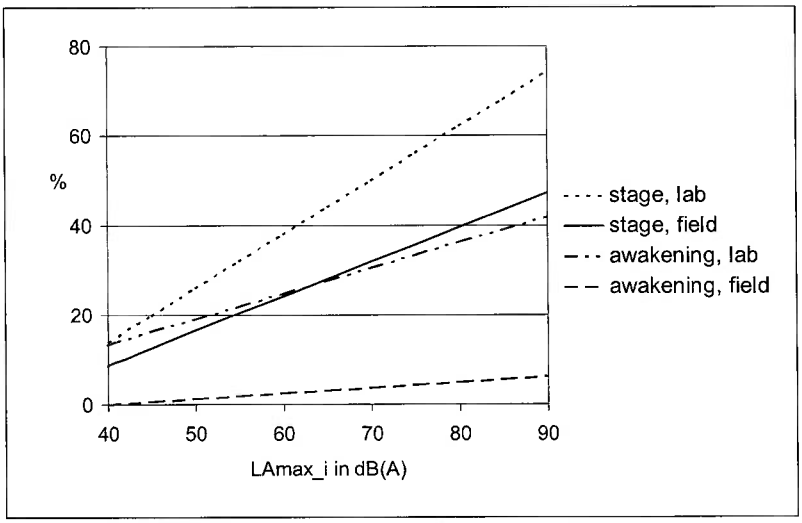


Figure 7 Average probability of sleep stage change and of EEG awakening as functions of *LAmax_i* for laboratory research subjects and for field research subjects exposed at home[48].

The fact that laboratory research sometimes sheds little light on the habituation effect is illustrated by the exposure-effect relationships reported by Pearsons *et al*[48]. The effects studied by this team were the probability of EEG awakening and the probability of change from a deeper stage of sleep to a less deep stage. Their findings, as obtained from field and laboratory research, are illustrated in Figure 7. From the figure, it will be clear that, for a given *LAmax_i*, the probability of EEG awakening or sleep stage change is much greater among laboratory subjects than among people accustomed to experiencing night-time noise at home.

Therefore, to obtain insight into the effects of noise on sleep and health, it is particularly important to carry out field research involving people who are exposed to a given noise source over a longer period of time. Mechanisms can be studied in the laboratory, but the strength of an effect observed in the laboratory is not representative of the 'real' world.

The Committee has divided the effects of exposure to night-time noise on sleep into two groups: biological effects (acute responses to noise and effects over a night (before, while and after sleeping)) and effects on health and well-being resulting from chronic exposure to noise when sleeping (for details, see Tables 12 to 14 in Annex D).

3.2 Acute biological effects

The position as described in an earlier Health Council report

In 1991, the Health Council published an advisory report on aviation noise and sleep[6]. In the associated background study document, Hofman summarised the results of laboratory and field research published up to 1991[6]. Her findings are presented in Table 3. She grouped the results of the reviewed studies into five categories: significant change in the anticipated direction (significance level: 2.5 per cent), trend in the anticipated direction, no change, trend not in the anticipated direction, significant effect not in the anticipated direction. In the table, the latter four categories are unified under the heading 'no statistically significant change in the anticipated direction'. The results relate mainly to noise from road and air traffic, although one or two of the reviewed studies were concerned with the effects of noise from rail traffic or industrial activities.

Table 3 The results of research published up to 1991 relating to acute changes induced by exposure to night-time noise events[17].

Detection method	Effect	Number of studies in which significant change was observed	Number of studies in which no significant change was observed
EEG	Prolongation of sleep latency period	17	15
	EEG awakening	38	9
	Change from a deeper stage of sleep to a less deep stage	35	20
	Transition from REM sleep to another sleep stage and change in sleep structure	27	11
ECG	Increased heart rate	16	7

3.2.1 *Comparison of five acute effects of exposure to aviation noise*

A great deal of laboratory research has been carried out into acute responses to noise. The precise temporal correlation between noise and response observed in these studies leaves no doubt that the responses in question were brought about and strengthened *by noise*.

Figure 8 illustrates the relationship between each of several acute responses to a noise event (the passage of an aeroplane) and exposure to the event in question. The relationships were determined by the Committee using data from field research into the consequences of (almost exclusive) exposure to aviation noise. The graph shows the probability of a noise-induced response in a time window of five minutes spanning the occurrence of a noise event (from approximately one minute before to four minutes after the *LAmax_i* of the noise event). Notably, most responses occurred in the interval from one minute before to one minute after the moment of maximum noise exposure (*LAmax_i*). As indicated by the graph, noise increases the probability of the following:

- Change from a deeper stage of sleep to a less deep stage, as detected by EEG (stage_EEG)
- Motility (motor unrest) in one of the ten thirty-second intervals making up the five-minute observation window (motility)
- Motility onset (onset of motor unrest) in one of the ten thirty-second intervals making up the five-minute observation window (motility_onset)
- EEG awakening (awake_EEG)

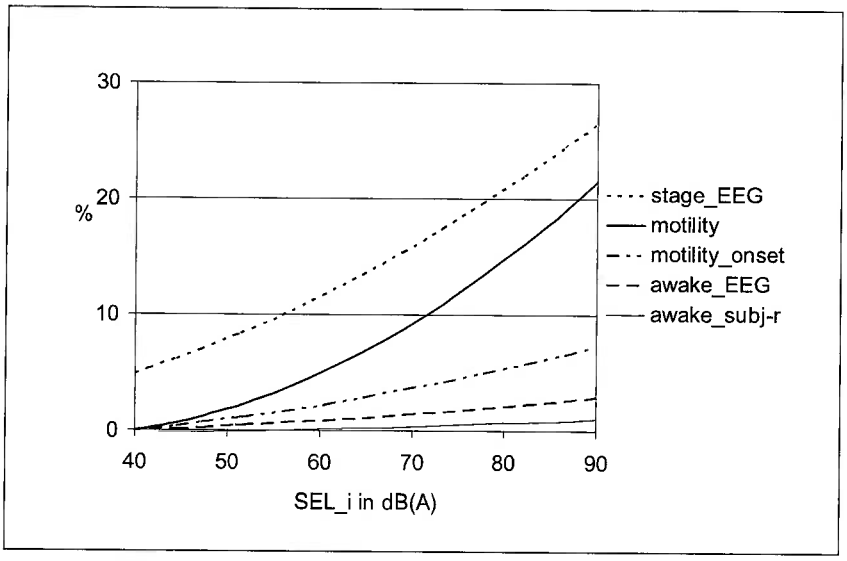


Figure 8 Comparison of the probability of various acute responses exhibited by a subject exposed to noise events while sleeping. The responses concerned are those occurring within a five-minute time interval, from one minute before to four minutes after the *LAmax_i* of the noise event. For an explanation of the response curve labels, see the main text. The relationships have been determined almost exclusively from research into aviation noise. Because of the assumptions upon which they are based, the curves for EEG awakening (awake_EEG) and sleep stage change (stage_EEG) should be regarded as, respectively, tentative and very tentative[48-53].

- Subject-registered (or behavioural) awakening (awake_subj-r). Subject-registered awakening is generally awakening that the subject registers by pressing a button. In other words, it is an event that entails the subject not only waking up, but also being aware of his/her circumstances to the extent necessary to recall that his/her wakefulness should be registered by performing the prescribed action. Subject-registered awakening therefore implies a higher level of consciousness than EEG awakening.

The relationships shown in Figure 8 are given for the range of *SEL_i* values from 40 to 90 dB(A). At night, an aeroplane passage with an *SEL_i* of 40 dB(A) is normally readily discernible indoors. An *SEL_i* of 90 dB(A) equates to a very loud noise event.

Not all the relationships illustrated in Figure 8 are equally reliable. The Committee believes that the relationships between exposure and subject-registered awakening, motility and motility onset have been defined on the basis of sound evidence. However, definition of the relationships based on EEG data (EEG awakening and sleep stage change) required an assumption, namely that the probability of noise-induced EEG awakening is 40 per cent of the probability of motility being triggered by noise[**]. However, it is not certain that this assumption is valid in the particular context of exposure to

aviation noise. Furthermore, in order to estimate the probability of a sleep stage change, the Committee has drawn upon the relationships between exposure and the probability of EEG awakening and the probability of sleep stage change illustrated in Figure 7. The Committee therefore regards the curve for EEG awakening as tentative and the curve for sleep stage change as very tentative.

From Figure 8, it is apparent that the observation threshold for the acute effects 'motility', 'motility onset', and 'EEG awakening' is an *SEL_i* of 40 dB(A) (*LAmax_i* of 32 dB(A)), while the observation threshold for subject-registered awakening is an *SEL_i* of 54 dB(A). On the basis of the tentatively plotted curves, the observation threshold for sleep stage change appears to be lower than an *SEL_i* of 40 dB(A).

3.2.2 *Extrapolation from aviation noise to road and rail traffic noise*

Figure 8 is based almost entirely on the results of research into aviation noise. Road and rail traffic noise also increase the probability of motility onset and of EEG-registered changes (EEG awakening and sleep stage change), and the observation thresholds for these noise sources are similar to those for aviation noise (see Annex D). It should be pointed out that this observation is based primarily on outdoor sound pressure level data; the use of accurate indoor data might yield a different result. In consideration of these matters, while there is insufficient evidence that road and rail traffic noise can cause subject-registered awakening, the Committee anticipates that road and rail traffic noise events are indeed capable of triggering such a response, although the threshold might not be an *SEL_i* of 54 dB(A).

3.2.3 *Motility and motility onset*

Over the last ten years, three large-scale field studies on aircraft noise have been carried out. Using data from these studies, it is possible to define the relationship between *LAmax_i* or *SEL_i* and the probability of acute motility being induced by the noise of a passing aeroplane[12,49,51]. The probability of acute motility increases as *LAmax_i* or *SEL_i* increases. From the Dutch research, it also appears that, at a given *LAmax_i* or *SEL_i*, the probability depends to a considerable degree on the indoor equivalent sound pressure level from the plane (*Li*): people who are ordinarily exposed to high levels of aviation noise while sleeping respond less to an individual aeroplane passage than people who only experience such exposure from time to time. The study findings also indicated that the type of aircraft manoeuvre (landing or taking off) did not affect the

* The figure of 40 per cent was calculated by Ollerhead by comparing all thirty-second intervals during which EEG arousals were observed in his subjects and all thirty-second intervals during which motility onset was observed[51].

probability of aviation noise-induced motility. The researchers also asked subjects about their attitude to air traffic and to the expansion of Schiphol Airport. Attitude was found to have no influence on the probability of acute motility induced by aviation noise.

The Dutch research findings are consistent with the findings of the study carried out in the USA[49]. However, the relationship between motility onset and the *LAmax* of an aeroplane passage defined on the basis of the British data is quite different from the relationship between motility and *LAmax_i* deduced from the Dutch research. The British researchers came to the conclusion that the threshold for motility onset by the noise of an aeroplane passage was an *LAmax* of 82 dB(A)[51]. If this outdoor value is reduced by 25 dB(A) (the figure given by the researchers[54] as the difference between outdoor and indoor sound pressure levels), one arrives at a threshold *LAmax_i* value of 57 dB(A). This is 25 dB(A) higher than the observation threshold determined by the Dutch researchers for motility and motility onset. In view of the pioneering nature of the British research and the significance that has long been attached to its results, the Committee considers the difference between the British and Dutch studies at more length in Annex D. Its conclusion is that the British research had certain shortcomings that the more recent Dutch research did not share.

3.2.4 *Subject-registered awakening and EEG awakening*

The relationship between the probability of subject-registered awakening and *SEL_i* illustrated in Figure 8 has been defined on the basis of a secondary analysis by Passchier-Vermeer[52]. According to this analysis, the observation threshold for aviation noise-induced subject-registered awakening is an *SEL_i* of 54 dB(A), corresponding to an *LAmax_i* of 42 dB(A).

The Committee puts the typical frequency of 'spontaneous' EEG awakenings, including short duration arousals, at ten to twelve occurrences per night and the typical frequency of 'spontaneous' subject-registered awakenings at 1.5 to two occurrences per night (in periods without noise events). If someone has woken up 'spontaneously', they will be able to hear a car, aeroplane or train that passes while they are awake. The *more frequent* and *longer in duration* the passages are, the greater the chance of hearing one after waking up spontaneously. In an extreme case, therefore, it is theoretically possible that someone could hear a passing car, plane or train ten times in the night without the associated noise being the cause of the person waking up.

The Committee found three reports on the effects of night-time noise on children's EEGs. These related to laboratory studies involving twenty-four, eight and six children, respectively[55-57] and one study of five children in their home environments[56]. Busby[55] found that children in the final third of their sleep (which mainly involves REM sleep) exhibited EEG awakening in response to noise in nearly 60 per cent of cases – albeit

involving noises of up to 95 dB(A). However, partly because of the lack of additional information regarding the cortical responses of children to night-time noise, the Committee is not able to make any definitive statement about the possibility of children being more sensitive to night-time noise than adults.

3.2.5 *Heart rate acceleration and stress hormone concentrations in the blood*

From the field research data published by Hofman *et al*[58], the Committee has calculated that peaks in the noise from a motorway (e.g. when a lorry passes) have approximately a 60 per cent chance of inducing heart rate acceleration, irrespective of the *LAmax_i*. The *LAmax_i* values recorded by the Hofman team were mostly between 30 and 70 dB(A). The average increase in heart rate worked out at four beats per minute, irrespective of the subject's sleep stage. The Committee regards heart rate acceleration as one form of acute cardiovascular change. Other acute changes directly associable with heart rate acceleration, such as acute changes in systolic blood pressure and vasoconstriction, follow the same pattern[59-63] (see Annex D). On the basis of laboratory research findings, it seems likely that the noise of a passing lorry or aeroplane with a similar *SEL_i* would have a broadly similar effect on heart rate[64-67] (see Annex D). Laboratory research has also indicated that, if one uses *SEL_i* as one's index of noise, noise events that quickly become louder at the start (such as the noise of a low-flying military jet or gunshot noise) have a greater effect on heart rate than noise events characterised by a more gradual increase in level (such as the noise of a lorry or a civil aircraft)[64,65,67]. However, it is not possible to quantify the extent of the effect.

The Committee is not aware of any field studies that have looked at acute noise-induced changes in the (stress) hormone balance. It is not surprising that no such research has apparently been carried out, since it would necessarily be highly invasive and therefore inappropriate for large-scale studies of subjects in a domestic setting.

Only one study was traced that focused on the impact that exposure to night-time noise has on children's physiological functions. In 1967, Semczuk investigated the effects of exposure to noise when sleeping, by using thoraxgraphy to monitor breathing in a study group of fifty children (five to seven years old) and a hundred adults[68]. The trigger level for respiratory changes associated with an aural stimulus (sound of a particular pitch) was 10 to 15 dB(A) lower in children than in adults. The researcher accordingly concluded that a child's autonomous nervous system is more readily activated by noise when sleeping than an adult's, and that children are therefore physiologically more sensitive to night-time noise than adults. The Committee supports this conclusion.

3.2.6 *Acute annoyance*

None of the studies reviewed by the Committee entailed the recording of acute noise-related annoyance during the sleep period, but some did involve subjects subsequently being asked questions on the topic. It is likely that making journal entries during the night would have a distorting effect by interfering with the sleep process. The Committee assumes that people do feel inconvenienced by noise during the night, even though such feelings have not actually been recorded. The subsequent logging of inconvenience by subjects serves as an indirect indicator of the existence of acute annoyance.

3.2.7 *Ranking of acute responses*

In Figure 9, the acute responses to noise are ranked in order of decreasing probability of induction by noise. Although no research into acute annoyance has been reported, the Committee considers it reasonable to suppose that inconvenience can only be experienced by a person who actually is awake.

3.2.8 *Groups with heightened sensitivity to acute effects*

The Committee has also sought to identify any evidence in the available research data that might indicate whether certain personal characteristics might be associated with heightened sensitivity to acute noise-induced effects on sleep. Although the strength of the evidence found by the Committee is limited, it does appear that people with cardio-

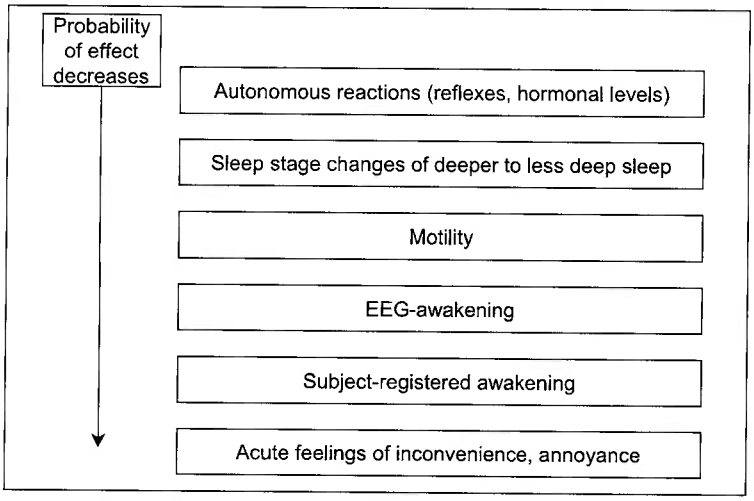


Figure 9 Acute effects of exposure to noise when sleeping.

vascular problems, people who regard themselves as particularly sensitive to noise, and children may all be particularly sensitive to acute cardiovascular effects. Because of the shortage of research data on children, it is not possible to say with confidence whether children are more sensitive than adults to other acute biological responses.

3.2.9 Conclusions

On the basis of the considerations set out above, the Committee draws the following conclusions:

- Road, rail and air traffic noises can induce acute responses in people who are sleeping. Response induction begins at a fairly low threshold level and becomes more likely as the intensity of the noise increases. The observation threshold for EEG awakening, motility, and motility onset associated with traffic noise is an *SEL_i* of approximately 40 dB(A); the corresponding figure for heart beat acceleration is less than 40 dB(A),), and that for subject-registered awakening (due to aviation noise) is approximately 54 dB(A). The observation threshold for EEG-detected sleep stage change is probably lower than 40 dB(A) (the relationship cannot be defined with confidence. Although there is insufficient data to provide direct evidence that road and rail traffic noise can induce subject-registered awakening, the Committee believes that peaks in road and rail traffic noise probably have the same effect as aircraft noise, although the associated observation threshold may not be an *SEL_i* of 54 dB(A). The induction of acute changes by industrial noise has not been the subject of scientific study. Nevertheless, the Committee expects that exposure to industrial noise is capable of inducing similar responses. It seems quite possible that the observation thresholds for industrial noise may be broadly similar to those for traffic noise, but the Committee draws no conclusions on this point.
- Almost no research data is available regarding the acute effects of night-time noise on children. The results of the one study that looked at children's respiratory response to noise exposure indicate that the threshold for response induction in children is 10 to 15 dB(A) lower than in adults. Because of the shortage of data, the Committee cannot exclude the possibility that children are also more sensitive than adults to acute cortical effects when sleeping; however, if so, this may only be the case during REM sleep, rather than during deep sleep.
- Laboratory research indicates that, if *SEL_i* is used as the index of noise, noise events involving a very rapid rise in intensity to their peak level have a greater effect on heart rate than events characterised by a more gradual early rise in intensity. However, the Committee is not able to quantify the effects concerned. Although what is known about the relationship between noise from military jets and subject-registered awakening is based purely on data collected from people living in the

vicinity of one airbase, and therefore needs to be verified by other pertinent scientific data, it would appear that, at high noise exposures, subject-registered awakening is much more likely to be induced by military jets than by civil aircraft. The Committee suspects that the increased probability of subject-registered awakening in response to louder military jet passages is linked to the great speed with which the noise from the approaching jet increases in intensity, thus inducing feelings of anxiety.

- The *more* noise events a person is exposed to per night, the greater the chance is that he or she will happen to hear one of the noises after waking up 'spontaneously'. The Committee believes it is reasonable to assume that, broadly speaking, between 1,5 to two times and ten to twelve times per night, a person is sufficiently conscious to coincidentally hear a noise event that has not actually awoken him or her. This may help to explain the extent of night-time noise-related annoyance. At a given *Lnight* value, the probability of coincidentally hearing a noise event after waking up will often be considerably greater with road traffic than with air and rail traffic, since road traffic noise involves frequent lower-level noise events, in addition to the peaks.
- Although the strength of the evidence is limited, it may well be the case that (as indicated above) people with cardiovascular problems, people who regard themselves as particularly sensitive to noise, and children are particularly sensitive to the acute cardiovascular effects of noise.

The results of the research into the acute effects of exposure to night-time noise when sleeping are summarised in Figure 10. The upper element of the diagram illustrates the general principle: night-time noise influences sleep in a way that can be measured by

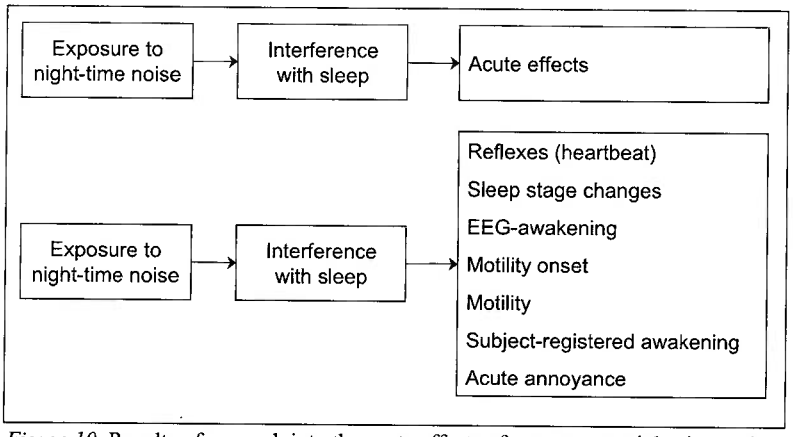


Figure 10 Results of research into the acute effects of exposure to night-time noise when sleeping.

reference to acute effect parameters. The lower element of the diagram indicates which parameters appear to be affected by noise during the sleep period. All acute responses to noise are regarded as biological effects by the Committee.

3.2.10 *Strength of the evidence*

Exposure to noise during the sleep period induces immediate physical responses. Table 4 lists the effects concerning which there is sufficiently strong evidence to conclude that they occur as a direct result of noise events during the night-time sleep period.

Table 4 Acute biological effects.

Cardiovascular changes[a]
Sleep stage change, from deeper to less deep sleep
EEG awakening
Motility onset
Motility
Subject-registered awakening

[a] The advisory report focuses mainly on heart beat acceleration, but there is also sufficient evidence of the induction of vasoconstriction and acute blood pressure rises.

There is no evidence that an acute change in (stress) hormone levels can be induced by exposure to night-time noise when sleeping, but one may assume that this is the case. It is not, however, possible to investigate the possibility in a field study, since such research would involve the use of invasive monitoring techniques. Also, there is only limited indirect evidence that noise events can induce acute annoyance.

3.3 Biological effects before, while and after sleeping

The scientific situation as described in an earlier Health Council report

To support preparation of the previously mentioned Health Council report on aviation noise and sleep, Hofman[17] also summarised the results of research published up to 1991 that had looked into the effects that exposure to night-time noise has during the course of a night or a day and over a longer timescale. Hofman's findings are presented in Table 5. As was the case with research into acute effects, the research referred to in the table related mainly to noise from road and air traffic, but one or two of the reviewed studies were concerned with the effects of noise from rail traffic or industrial activities.

Table 5 The results of research published up to 1991 relating to the effects of exposure to night-time noise events, as reviewed by Hofman[17].

Effect registration method	Effect	Number of studies in which significant change was observed	Number of studies in which no significant change was observed
Journal/cognitive testing	Over a night or day		
	- Diminished sleep quality	15	10
	- Daytime irritability and impaired cognitive performance	8	6
	- Sleep disturbance	25	2
Questionnaire	Over the longer term		
	- Night-time noise-related annoyance	42	2
	- Seeking healthcare	6	5
	- Increase in self-reported health problems	10	8
	- Increase in the use of somnifacient drugs and sedatives	8	5

As indicated in the table, almost all studies found that increasing night-time noise exposure was associated with statistically significant rises, particularly in long-term annoyance and sleep disturbance (difficulty getting to sleep, waking up in the night, waking up too early in the morning and not being able to get off to sleep again). In line with Hofman's review[17], the table provides no information about exposure levels or study quality.

Only a small number of field studies looked specifically at the relationship between noise exposure and its effect on sleep latency time, sleeping time, or the post-sleep period. Furthermore, research data on acute noise-induced changes (see section 3.3) has not in most cases been aggregated to provide full-night data. Most of the information presented below relates to research into the effects of road traffic and aviation noise. In most of the reviewed studies, several effect parameters were studied at once, which has made it difficult for the Committee to present a summary structured along the lines of the previous section (on acute effects). This section begins with a discussion of the results of research into the influence of road traffic noise, which is followed by a subsection on the effects of aviation noise. Consideration is then given to the findings of field research into the effects of night-time noise on motility and on stress hormone concentrations. Finally, the Committee addresses the possibility that immune functions might be influenced by exposure to night-time noise, which has been investigated only in the context of laboratory research.

3.3.1 *Road traffic noise: various effect parameters*

In 2003, the RIVM published a review of field research concerned specifically with the effects of night-time *road traffic* noise on sleep[69]. In the eleven reviewed studies that involved the use of sleep EEGs, ECGs or actimetric measurements and sometimes of journals, noise was also measured (in the bedroom) during the study nights.

Five of the eleven studies were deemed unsuitable for inclusion in the detailed analysis for various reasons (too small, no usable findings). Useful data was produced by four intervention studies carried out on behalf of the European Commission between 1980 and 1983. By increasing the acoustic insulating properties of bedroom windows, using personal hearing protection gear and sleeping on the quiet side of the house, the road traffic-related noise exposure was reduced by approximately 10 dB(A) on half of the subject-nights[58,59,70-73]. The studies in question involved a total of seventy people and 922 subject-nights. Jurriëns drew the following conclusions regarding the effects observed in relatively noisy situations (compared with quieter situations after intervention)[70]:

- The average duration of REM sleep is 6.5 minutes shorter (in adults, REM sleep normally lasts for approximately two hours).
- In reaction time tests, the average reaction time is twelve milliseconds (12 ms) longer than the overall average reaction time of 350 ms, and more mistakes are made (8 per cent)
- Self-reported quality of sleep is less (7 per cent)
- The W (waking) time recorded by EEG is 7 minutes longer (determined in two of the four studies)
- The average heart rate when sleeping is higher. In the Dutch research, the rate was 3.2 beats per minute higher (71.5 bpm, compared with 68.3 bpm)[58].

A study by Öhrström[74], which involved the use of journals only, was also included in the RIVM review. In this study, it was found that in situations characterised by higher levels of road traffic noise at night, people had more difficulty getting to sleep, were more likely to be woken up in the night by traffic noise, had poorer sleep quality and were more likely to be tired and irritable in the morning. The same research team recently completed a small longitudinal intervention study with adults and a cross-sectional study with children and adults, neither of which found that exposure to night-time road traffic noise had any statistically significant effect on the studied effect parameters[75,76].

The RIVM review additionally took in a German study of road and rail traffic noise[77,78]. This study's findings regarding average motility are discussed in more detail later.

3.3.2 *Aviation noise: various effect parameters*

A report by Passchier-Vermeer[12] defines the relationship between *aviation noise* and each of several effects on sleep over one sleep period (see Figure 23 in Annex D). The effect variables were: high motility for the subject's age; recalled aviation noise-induced awakening; subject-registered awakening three or more times per night; use of somnifacient drugs. The use of somnifacient drugs and sedatives rose markedly with increasing night-time noise exposure mainly among older subjects. Night-time aviation noise did not appear to have any effect on subjects' performance in reaction time tests taken at the end of the evening. The degree of subject-reported morning drowsiness (as indicated at 10am) did, however, appear to be related to night-time noise exposure: the greater the overnight exposure, the sleepier subjects felt in the morning.

The Passchier-Vermeer study also indicated that increased aviation noise exposure (equivalent sound pressure level) during the sleep latency period was associated with prolonged sleep latency and greater difficulty getting to sleep. People who when they went to bed were concerned about the possibility of being disturbed by aviation noise took an average of a quarter of an hour longer to go to sleep than the people that were not concerned.

3.3.3 *Road, rail and air traffic noise and motility*

In the above-mentioned German research into *road and rail traffic noise*[77,78] 188 subjects were exposed mainly to road traffic noise and a similar number mainly to noise from passing trains. The number of subject-nights with results on motility was 1710 in the road traffic group and 1581 in the rail traffic group. A recent analysis[79] of the data indicated that, among people exposed to *rail traffic noise*, average motility for a single sleep period was unrelated to the equivalent indoor or outdoor traffic sound pressure level during the period in question, whereas an increase in such levels was associated with a statistically significant rise in motility among people exposed to *road traffic noise*. The Dutch sleep disturbance study also found that average motility rose with increasing *aviation noise* exposure during the sleep period[12]. With road traffic noise, the increase in average motility per dB(A) rise in noise exposure was approximately 30 per cent greater than with aviation noise.

The Bristol-based team of Smith *et al*[80] made a phased investigation of the interrelationships between aviation noise, sleep disturbance and health. In the final phase, the motility of ninety people (forty-five couples) was monitored using actimeters for three nights, during which sound pressure levels were measured in the subjects' bedrooms. The sources of the noises audible in the subjects' bedrooms were not objectively deter-

mined, nor were any outdoor sound pressure levels measured. The study revealed no relationship between noise exposure and average actimetric level over the course of a night. The researchers took the view that this was mainly because the noise exposures experienced by the subjects were low.

3.3.4 *Road traffic and aviation noise: stress hormone concentrations*

Babisch[81] produced a survey of research into the effects of *road traffic and aviation noise* on hormone concentrations (adrenaline, noradrenaline, cortisol) determined from urine samples collected over the course of a night and in one study from saliva samples taken after awakening (for the measurement of cortisol only). In all, the survey took in eight field studies (see Tables 16 and 17 in Annex D).

In seven of the studies, the subjects were children, who in five studies were exposed to aviation noise and in two studies to road traffic noise. No link was found between exposure to aviation noise and cortisol concentrations, but higher road traffic noise exposures were associated with statistically significant rises in levels of this hormone. Adrenaline and noradrenaline concentrations exhibited statistically significant rises at higher aviation noise exposures in two of the four studies, but could not be linked to road traffic sound pressure levels in one study (not all of the studies involved monitoring concentrations of all three of the hormones referred to). Whether the increased hormone concentrations were brought about by exposure to night-time road traffic noise is unclear; they could also have been an after-effect of daytime noise exposure.

The only field study involving adult subjects focused on the effect of road traffic noise on the quantity of adrenaline and noradrenaline in the night-time urine of 234 women ages thirty to forty-five[82]. Among the women whose bedrooms were on the street side of their homes, increasing traffic volume (and therefore increasing equivalent sound pressure level) was associated with a statistically significant increase in noradrenaline concentration, but no link was established between traffic volume and adrenaline concentration. Among women whose homes had the living room on the street side, traffic volumes had no apparent influence on either adrenaline or noradrenaline concentrations. The fact that it was mainly noradrenaline concentrations that were raised is consistent with Ising's model, which predicts that noradrenaline concentration is particularly likely to increase in response to noise to which a person has in part habituated[83]. The effect of road traffic noise on noradrenaline concentration was particularly pronounced in women who indicated that they slept with the bedroom window closed to prevent their sleep being disturbed by road traffic noise. Among women who experienced no noise-related annoyance when their windows were closed, no statistically significant increase in noradrenaline concentrations was observed.

The modest amount of data available on this subject prevents the Committee drawing any firm conclusions. It does nevertheless appear that, under certain circumstances, exposure to noise can lead to raised stress hormone levels in sleeping adults; the possibility that noise can have a similar effect on children cannot be excluded. More definite conclusions must await the availability of further research data.

3.3.5 *Various noises in laboratory research: immune function*

Between 1968 and 1974, Osada *et al*[84-87] investigated immunological parameter changes associated with exposure to noise. However, the major changes observed in the four laboratory experiments with twenty-one subjects were almost certainly attributable to shortcomings in the study design*. The Committee is not aware of any other research into the influence of night-time noise exposure on immune functions.

In their survey article *The Neuroendocrine Recovery Function of Sleep*, Born and Fehm devoted a section to the possibility that night-time exposure to noise might affect the immune system[88]. On the basis of two experiments in which subjects were either deprived of sleep or allowed to sleep 'normally', the two authors postulate that night-time noise exposure may have a negative influence on the immune system. They add, however, that a great deal more research would be necessary to confirm such a hypothesis.

3.3.6 *Conclusions*

On the basis of the considerations set out above, the Committee draws the following conclusions regarding biological effects over the course of a night (before, while and after sleeping):

- There is sufficient evidence that, above a given threshold noise exposure, exposure to road and air traffic noise while sleeping is associated with the following:
  - Increased difficulty getting to sleep
  - Increased sleep latency period
  - Use of somnifacient drugs and sedatives, particularly among older people
  - Reduced REM sleep and increased time in a conscious state, as determined by EEG
  - Raised average heart rate
  - Raised average level of motility
  - More frequent subject-registered awakening
  - More frequent recalled noise-induced awakening

* Marth, personal communication.

- Reduced self-reported quality of sleep
- Increased drowsiness, tiredness and irritability

- There is limited evidence that exposure to road and air traffic noise while sleeping is associated with the following:
  - Increased daytime irritability
  - Impaired daytime cognitive performance
- Exposure to rail traffic noise has been investigated on an incidental basis only: no statistically significant rise in average motility during a sleep period was detected in response to exposure. No research has been carried out into the consequences of exposure to industrial noise.
- Little is known about the influence of exposure to night-time noise on immune functions.
- There is limited direct evidence that, under certain circumstances, exposure to night-time noise can influence (stress) hormone levels when sleeping; this effect was observed in women who were troubled by noise during the night and were unable to relieve the problem. The Committee suspects that noise has no consistent effect on stress hormone levels, and that any effects depend partly on personal and situational factors. More definite conclusions must await the availability of further research data.

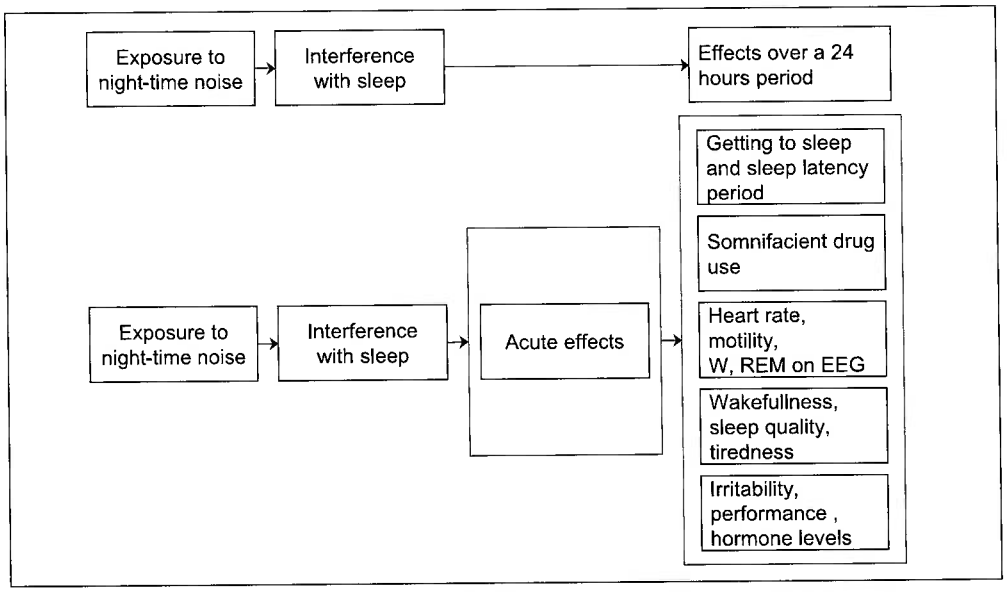


Figure 11 Results of research into the effects of night-time road and aviation noise on biological parameters over the course of a night (before, while and after sleeping). There is sufficient evidence of a causal relationship between exposure and the parameters in the upper four effect boxes, but only limited evidence of such a relationship with the parameters in the bottom effect box.

Figure 11 indicates which parameters appear to be affected by road and aviation noise during the sleep period. There is sufficient evidence of a causal relationship between exposure and the parameters in the upper four effect boxes, but only limited evidence of such a relationship with the parameters in the bottom effect box.

3.3.7 *Biological effects as predictors of impact on health and well-being*

In some cases, there is empirical data indicating that a biological effect of night-time noise can, after repeated exposure and under certain circumstances, ultimately have consequences for a person's health and well-being. Where certain other biological effects are concerned, although no such data is available, it is plausible that chronic exposure to night-time noise when sleeping may lead to physical responses indicative of a negative influence on health and well-being. The Committee's assessment regarding each of the effects concerned is given in Table 6.

Table 6 Biological (physiological and psycho-physiological) effects for which there is either sufficient or limited evidence of a causal relationship with night-time noise and which could plausibly have a negative influence on health and well-being in the event of chronic exposure[a].

Variable	Probability of negative implications for health and well-being
Difficulty getting to sleep, difficulty staying asleep	Empirical data
Change in cardiovascular activity	Plausible
Increased motility	Plausible
Changes in duration of various stages of sleep and in sleep structure, fragmentation of sleep	Empirical data
Changes in (stress) hormone concentrations	Plausible
Waking during the sleep period and/or prematurely in the morning	Empirical data
Drowsiness/Tiredness during the day and evening	Plausible
Impaired cognitive performance	Plausible
Increased irritability	Plausible

[a] The effects in question can occur after a single night's exposure. However, there is no empirical evidence that the occurrence of any of the effects in the context of a single night's exposure can influence health or well-being, nor is it plausible that this might be the case. The effects after a single night's exposure all come under the heading 'no empirical evidence of implications for health and well-being and no plausible causal association'.

3.4 The effects of long-term exposure

In field research with subjects who are exposed to noise on a nightly basis, it is not easy to distinguish the effects of a single disturbed night from the effects of long-term exposure. In many cases, the way relationships are defined depends on what is known about the noise exposure. Where the available data concerns sound pressure levels on particular nights, observed effects are typically related to such data. However, if the only available data consists of estimates of longer-term noise exposure, observed effect parameters are considered representative of the consequences of prolonged exposure. Making a distinction is therefore important mainly in the context of research data structuring. The consequences of noise-related sleep disturbance described in section 3.3 can therefore also be seen as the effects of long-term exposure, since our knowledge of them comes from data concerning people who have experienced chronic exposure to environmental noise. Hence, the Committee also considers the effects listed in Figure 11 to be the effects of long-term exposure.

3.4.1 Insomnia

A group of Japanese researchers carried out a questionnaire-based survey of 3600 adult Japanese women (aged between twenty and eighty) to gather information about the factors that contribute to insomnia[89]. Some 11 per cent of subjects were found to be affected by insomnia (as defined on the basis of the WHO's ICD10 classification system[90]). Analysis of the survey data took account of various distorting variables, such as age, number of (small) children in the family, social status, receipt of medical treatment, regularity of bedtimes, apnoea-like problems and serious unpleasant experiences in the six months prior to completing the questionnaire. When the percentage of insomniacs in each of the three areas with the highest exposures was compared with the percentage in the low-exposure areas, the ratios worked out at, respectively, 1.4 (2100 vehicles per hour, *Lnight* of around 65 dB(A)), 2.1 (2400 vehicles per hour, *Lnight* of around 67 dB(A)) and 2.8 (6000 vehicles per hour, *Lnight* of around 70 dB(A)). The most frequently reported problem was difficulty getting to sleep.

The seriousness of the problems caused by insomnia is illustrated by the quality-of-life weighting system developed by Stolk and Van Busschbach[91,92], under which insomnia has a quality-of-life weighting of 0.83. This means that a year affected by insomnia results in the loss of 0.17 DALYs.

Research into the effects of exposure to air and road traffic noise has shown that increases in night-time noise exposure or in noise exposure during the sleep latency

period have a statistically significant adverse impact on subjects' ability to get off to sleep and on sleep inception periods[12,13,93,94].

3.4.2 *Hypertension*

In the context of a longitudinal study (Spandauer Gesundheits Survey)[95], the health of adults in Berlin's Spandau district has been surveyed every two years since 1982. The ninth survey round involved 2015 subjects. In addition to going through the usual tests and questionnaires, 1718 (85 per cent) of these subjects were asked about noise-related annoyance from road, rail and air traffic, as well as from industrial sources (see Annex D). Hence, the noise research element of the study took the form of a cross-sectional study. The noise exposure was estimated using noise calculation models. The estimates made for aviation noise are not regarded as reliable by the Committee, but those for road traffic noise do not suffer from the same shortcomings. Furthermore, the road traffic noise exposure was measured on an incidental basis both during the day and at night. Details of the subjects' medical treatment histories over the two years since the previous survey and over the entire research period were gathered in interviews with the subjects. The findings showed that, after taking account of other factors that could explain any association between medical condition and noise exposure, the prevalence of hypertension was higher (by a statistically significant margin) among people for whom the *road traffic*-related *Lnight* was more than 55 dB(A) than among people for whom the road traffic-related *Lnight* was less than 50 dB(A) (odds-ratio 1.9). Prevalence among people for whom the road traffic-related *Lnight* was between 50 and 55 dB(A) was at an intermediate level. However, no statistically significant association was found between the prevalence of hypertension and road traffic noise *during the day*. The researchers suggest that hypertension is associated with nigh-time exposure but not with daytime exposure partly because people are often elsewhere during the day and partly because people are more sensitive to noise at night than during the day.

The Committee considers the following points to be relevant to the assessment of the research findings outlined above:

- The investigated outcomes: use of personal statements as the only means of determining whether subjects were receiving medical treatment for conditions such as hypertension. This may have led to considerable distortion of the research results. Although the report speaks at length about tests such as blood pressure measurement and the registration of medicine use, the resulting data is not used in the analyses. The Committee takes the view that if the measured data had been used for the analyses or – even better – if the analyses had been based entirely on measured data, the findings would have carried more weight.

- The researchers point out that the study population was made up largely of people who were very conscious of their health. In other words, the subjects were self-selected and this may also have led to distortion. The point being that, if night-time noise does have an effect on health and well-being, making people feel uneasy about their health, they may well be inclined to report all sorts of other problems that they don't really have. This could have resulted in the prevalence of hypertension among the most heavily exposed group being overestimated.
- The researchers do not report the raw data, i.e. the data in its original form, uncorrected for other factors capable of distorting the relationship between night-time noise exposure and the probability of developing a condition (confounding). It is therefore difficult to estimate how important these factors were and how plausible their supposed influence on the relationship between probability of hypertension and night-time noise exposure was.

On the basis of the considerations outlined above, the Committee has concluded that, although a link between night-time noise and increased risk of hypertension is plausible, the Spandau survey does not provide sufficient evidence of a causal association.

In this context, the Committee would point out that, in the 1994 report *Noise and Health*[2], an international Health Council Committee concluded on the basis of data from various, mostly German, studies that a causal relationship did probably exist between daytime noise exposure and hypertension risk. It was suggested that the observation threshold was an equivalent sound pressure level of 70 dB(A) over the course of a day. Consideration was not given to the possibility that night-time noise exposure might be at least partly responsible for the increased probability of hypertension associated with what it should be said are very high noise exposures. The Committee would like to see the possibility explored of re-analysing the data in a way that takes night-time noise exposures into account.

3.4.3 *Motility*

British research into the effect of aviation noise on sleep has revealed that the average probability of motility (motor unrest) during the course of a sleep period rises with increasing exposure to air traffic noise[96]. Horne reported that there was a strong relationship between average motility and perceived quality of sleep. Dutch field research into the effect of aviation noise on sleep and German research regarding the effect of road traffic noise has also found that average motility (motor unrest) increased with noise exposure when sleeping[12,13,79]. However, no association has been detected between motor unrest and rail traffic noise. Furthermore, the increase in motility with the *Li* of air and road traffic noise proved to be much greater than might be expected on the basis of

accumulated acute noise-related motility. The Committee believes that this phenomenon can be explained if one assumes that chronic exposure increases the physiological arousal level when sleeping, not only in the periods of the night when vehicles or aeroplanes are passing, but also when there is no traffic. In the Dutch field research into the effects of aviation noise on sleep, it was observed that overnight average motility was strongly associated with the number of occasions that a subject recalled waking during his or her sleeping time, with the number of subject-registered awakenings during this time, and with a series of variables determined from the questionnaire completed by the subject at the beginning of the study. The variables in question were: whether the subject used somnifacient drugs; quality of sleep; number of sleeping problems; number of times awoken by aviation noise; number of times per week aviation noise had a negative effect on sleep; health problems included on the abbreviated Health Perceptions Questionnaire ('VOEG-lijst').

The observed relationship between average motility and various negative consequences of exposure to night-time noise is regarded by the Committee as a strong indication that increase in average motility should also be seen as a negative consequence of exposure to noise when sleeping.

3.4.4 *Self-reported sleep disturbance*

On the basis of TNO's Disturbance Knowledge Base, exposure-response relationships have been defined for self-reported sleep disturbance by road, rail and air traffic[97,98]. The associated TNO reports contain various assessments of factors that influence sleep quality (problems caused by waking up in the night, waking up too early in the morning, night-time noise-related annoyance). Just as 'annoyance' is covered by an international definition, so high sleep disturbance is defined as a score of 72 or more on a scale of 0 (no sleep disturbance at all) to 100 (extreme sleep disturbance). The relationships between the various kinds of traffic noise and self-reported high sleep disturbance are illustrated in Figure 12.

From Figure 12, it will be apparent that, at a given *Lnight* value, aviation noise is linked to slightly more self-reported high sleep disturbance than road traffic noise, while rail traffic noise is associated with less disturbance than either of the other sources. The illustrated relationships are closely consistent with the provisional curves presented in 1997 in the Health Council's advisory report *Assessing Noise Exposure for Public Health Purposes*[8].

However, when one looks at the relationships between *Lnight* and the percentages of people experiencing 'at least sleep disturbance' and 'at least slight sleep disturbance', one finds that the relative positions of road traffic noise and aviation noise are reversed. It is worth noting that less certainty exists regarding the relationships between distur-

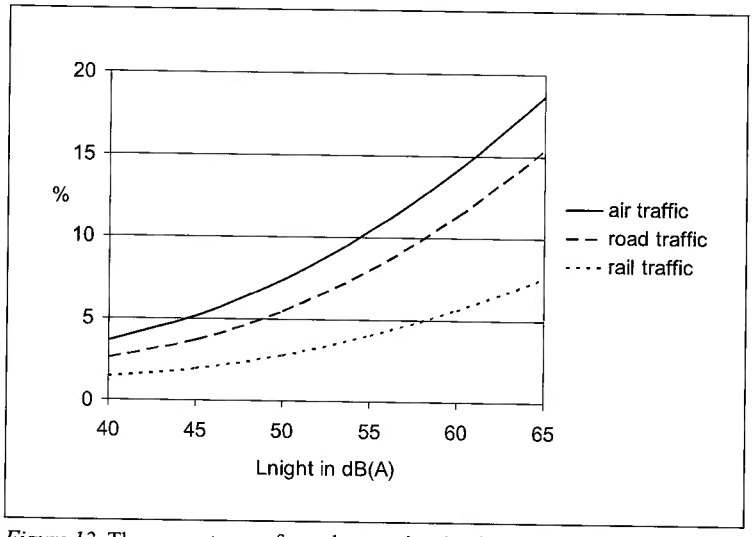


Figure 12 The percentages of people experiencing high noise-related sleep disturbance attributable to air, road and rail traffic, as a function of *Lnight*[97,98].

bance and aviation noise than regarding the relationships involving road and rail traffic noise.

The RIVM report[69] mentioned in 3.3.1 considers the question of whether a quantitative meta-analysis could be made of the results of questionnaire-based research into the influence of road traffic noise on perceived quality of sleep and on awakening. Although the RIVM describes several studies as being good quality, the researchers decided that it was not possible to perform a meta-analysis because of discrepancies in the studies' nomenclature, methods, exposure determination techniques and approaches to adjustment for distorting variables. Their ultimate conclusion was therefore that there were indications that road traffic was associated with reduced perceived quality of sleep and more frequent (or more prolonged) night-time awakening.

Leidelmeier and Marsman[99] carried out an interview-based study of 1242 households in the Netherlands, in which subjects were asked about daytime and night-time noise from neighbours and any associated annoyance. Distinction was made on the basis of the part of the house in which the noises were audible and any associated annoyance was experienced. Subjects proved least tolerant of noise from their neighbours that was audible in the master bedroom. The researchers distinguished five types of noise, which are listed below, along with the percentage of subjects who indicated hearing the relevant type of noise from a neighbouring dwelling at night in the master bedroom:

- Contact noise 22 per cent
- Noise from sanitary fittings, central heating, etc 19 per cent
- Noise from radio, TV and hi-fi 12 per cent
- Do-it-yourself (DIY) noises 8 per cent
- Pets 6 per cent

Where each of the five investigated types of noise were concerned, roughly 10 to 15 per cent of subjects indicated that they felt it was unacceptable for the noise to be audible during the day. Overall, nearly 30 per cent of subjects said that sanitary fittings should not be audible at night, while approximately 50 per cent felt each of the other four types of noise were unacceptable by night.

In 1993, Kranendonk *et al* produced a synthesis of the research conducted up to that point in time into the annoyance associated with noise from neighbours[100]. Subsequently, in 1998, Van Dongen *et al*[101] published a report on the relationship between noise from neighbouring dwellings and the airborne and contact noise attenuating indices I_{lu}, $I_{lu;k}$, and I_{co}, drawing on data from a questionnaire-based survey of the residents of six hundred dwellings, whose acoustic quality was determined in 202 cases. The results of the two studies are reasonably consistent (see Annex D). Both found that the chief causes of annoyance were loud radios, hi-fis and TVs, audible and sometimes intelligible voices, the slamming of doors and footsteps on floors and staircases. In both cases, it proved that, when I_{lu} had a value of 0 (the minimum requirement for new homes), 10 per cent of subjects reported high annoyance and 15 per cent reported annoyance caused by noise from neighbouring dwellings. These figures are not specific to night-time noise, but apply to annoyance over a twenty-four-hour period.

On the basis of the findings outlined above, the Committee concludes that the standard of inter-dwelling sound attenuation presently required does not provide sufficient protection to prevent annoyance caused by noise from neighbours. Since people are less tolerant of the noise their neighbours make at night-time than of their neighbours' evening or daytime noise, it may be assumed that much of the annoyance associated with noise from neighbours relates to the influence of such noise on sleep. The Committee returns to this point when addressing the State Secretary's questions.

3.4.5 *Health problems*

The Dutch field research into the effects of aviation noise on sleep established a relationship between personal noise exposure when sleeping (*Li*) and the frequency of health problems included on the abbreviated Health Perceptions Questionnaire[12,13]. Compiled on the basis of stress research, the Health Perceptions Questionnaire identifies

thirteen health-related problems, such as headache, stomachache, tiredness and digestive problems. It will be apparent that these are not life-threatening conditions. A rise in aviation noise-related *Li* from 0 to 35 dB(A) is associated with a two-fold increase in the frequency of problems. Various factors that might be expected to influence the relationship between noise and problem frequency, such as what time a person wakes up and whether they sleep with their bedroom window open, prove not to be influential in practice. The Committee interprets these findings as a strong indication that exposure to night-time aviation noise causes a rise in the incidence of health problems.

3.4.6 *Complaints about night-time noise*

The Committee believes that the submission of a complaint about noise is symptomatic of reduced well-being. Numerous factors influence a person's inclination in a given situation to make an 'official' complaint about a noise-related problem. It is not therefore possible to draw any general conclusions on the basis of what happens in a given situation. In the Netherlands, people can make complaints about, for example, the annoyance caused by noise from aircraft using Schiphol Airport, by noise road, rail and air traffic in the Rijnmond area, by events, and by industrial sources. Analysis of these complaints shows that, relatively speaking, night-time noise generates more complaints than daytime noise (see Annex D).

3.4.7 *Conclusions*

The Committee draws the following conclusions:

- Above a certain observation threshold, exposure to road and air traffic noise while sleeping has the following chronic consequences (where the strength of the evidence for a causal relationship is indicated between brackets):
  - Insomnia (sufficient evidence)
  - Increase in average motility (sufficient evidence)
  - Self-reported sleep disturbance (sufficient evidence)
  - Increase in self-reported health problems (sufficient evidence)
  - Submission of complaints (sufficient evidence)
  - Reduced sleep quality (sufficient evidence)
  - Increased use of somnifacient drugs and sedatives and increased reference to healthcare professionals (sufficient evidence)
  - Increased daytime irritability (limited evidence)
  - Impaired cognitive performance (limited evidence)
  - Impaired social contacts (limited evidence)

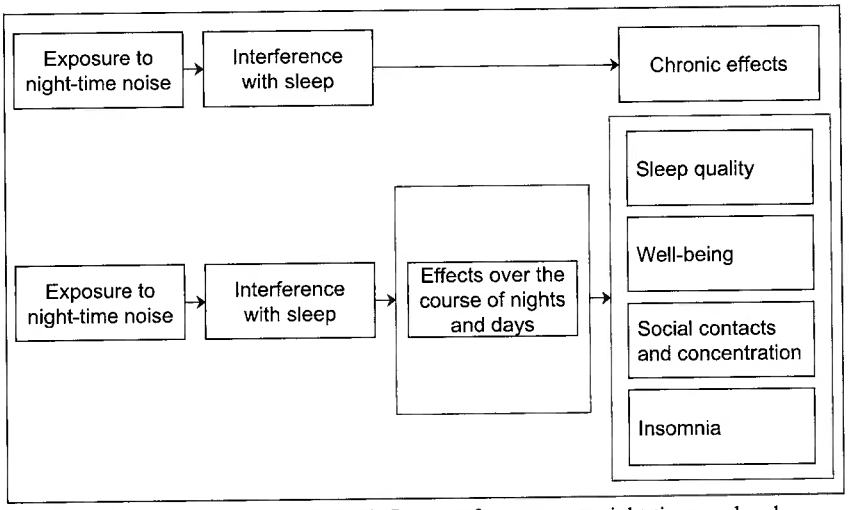


Figure 13 Results of research into the influence of exposure to night-time road and aviation noise on long-term health and well-being parameters.

- The effects of exposure to rail traffic noise have been studied only on an incidental basis. Average motility when sleeping was not found to be discernibly increased by such exposure. At a given *Lnight* value, the percentages of people experiencing self-reported slight sleep disturbance, sleep disturbance and high sleep disturbance due to rail traffic noise were slightly lower than the percentages experiencing such problems in connection with road traffic noise and aviation noise
- No information is available about the consequences of chronic exposure to industrial noise.

The findings outlined above are summarised in Figure 13. The effects referred to in the figure should be interpreted as follows:

- Social contacts and concentration: impaired social contacts and impaired performance of cognitive tasks
- Well-being: self-reported sleep disturbance, self-reported health problems, use of somnifacient drugs and sedatives, increased daytime irritability
- Sleep quality: reduced perceived sleep quality, difficulty getting to sleep and staying asleep, awakening, reduced sleeping time, increased average motility when sleeping.

3.4.8 *The influence of noise on sleep: correlations with sleep disorders and sleeping problems*

Thus far, consideration has been given to studies into the relationship between night-time noise exposure and the characteristics of sleep, health and well-being. Such charac-

teristics may be objectively measured (sleep latency period; EEG parameters; average motility; physiological and endocrine functions; reduced cognitive performance) or self-reported (difficulty getting to sleep; difficulty staying asleep; reduced sleep quality; waking up in the night; tiredness/drowsiness during the day; night-time noise-related annoyance; health problems; insomnia). A number of these characteristics are closely related to the characteristics of sleeping problems and insomnia generally observed in the population at large (see Annex E). Clearly, the disturbance of sleep by night-time noise is a matter of influence by an external factor, whereas the occurrence of insomnia and other sleeping problems in the population at large is probably attributable largely to personal characteristics; nevertheless, the Committee believes it is reasonable to assume that the same physical and mental processes are involved. Hence, it is plausible that sleep disturbance by environmental noise might contribute to the development or occurrence of female depression, hypertension, cardiovascular disease and occupational accidents, since all these phenomena are known to be associated with sleeping problems and insomnia. The size of any such contribution cannot be estimated. The evidence for such a link is indirect and limited in its strength.

It is plausible that people who suffer from insomnia or other sleep disorders that cause them to wake up frequently at night are more likely to be troubled by night-time noise. Insomnia is particularly prevalent among people with physical pain, dementia, depression, hypertension, heart and respiratory illness, and among older people and women who are pregnant or have been pregnant in the last twelve months. Age is not in itself a determining factor in the occurrence of insomnia or sleeping problems, which are attributable more to the accumulation of various age-related phenomena, such as lack of physical activity, changed eating and drinking patterns, dissatisfaction with one's social environment, illness and other medical conditions (see also Annex E).

People who work night shifts have to sleep by day, at least some of the time. Since in the daytime it is generally much noisier both indoors and outdoors than at night, people with variable working hours often have to sleep under less favourable circumstances than most of the population. Furthermore, such people tend to suffer some degree of disturbance to their sleeping-waking rhythm, as a result of which they frequently experience reduced-quality sleep even on the nights when they can go to bed at a 'normal' time. Consequently, night-shift workers are particularly sensitive to effects of night-time noise.

The Committee believes that certain groups of people are more likely to suffer adverse effects if exposed to night-time noise, and that this should be taken into account. The groups in question are as follows: older people; women who are pregnant or have been pregnant in the last twelve months; people who work night shifts; and people who

suffer from sleep disorders, physical pain, dementia, depression, hypertension, cardio-vascular disease or respiratory illness.

3.4.9 *Strength of the evidence*

The Committee's conclusions regarding the associations between exposure to night-time noise when sleeping and changes in health and well-being are summarised in Table 7. The effect parameters which the Committee has grouped under the five categories are specified individually and in each case an indication is given of the strength of the evidence for a causal relationship between the effect parameter in question and night-time exposure to noise when sleeping. With regard to the long-term health and well-being implications of exposure to night-time noise during the sleep period, the Committee's overall conclusion is that there is sufficient evidence that such exposure leads to reduced sleep quality and reduced general well-being, and limited evidence that it leads to impaired social contacts and concentration, increased probability of developing medical conditions and reduced life expectancy.

Table 7 Effects on health and well-being of prolonged exposure to noise during the sleep period.

	Effect parameter	Evidence
Sleep quality	Reduced perceived sleep quality	Sufficient evidence
	Difficulty getting to sleep, difficulty staying asleep	Sufficient evidence
	Sleep fragmentation, reduced sleeping time	Sufficient evidence
	Increased average motility when sleeping	Sufficient evidence
Well-being	Sleep disturbance	Sufficient evidence
	Health problems	Sufficient evidence
	Use of somnifacient drugs and sedatives	Sufficient evidence
	Increased daytime irritability	Limited evidence, plausible
Social contacts and concentration	Impaired social contacts	Limited evidence, plausible
	Impaired cognitive performance	Limited evidence, plausible
Medical conditions	Insomnia	Sufficient evidence
	Hypertension	Limited, indirect evidence, plausible
	Depression (in women)	Limited, indirect evidence, plausible
	Cardiovascular disease	Limited, indirect evidence, plausible
Reduction in life expectancy (premature mortality)	Occupational accidents	Limited, indirect evidence, plausible

3.5 Prevalence and disease burden

3.5.1 *Quantification*

The prevalence of an effect that is attributable to night-time noise in an exposed population is the difference between the number (or percentage) of people in the exposed population who experience the effect in question and the corresponding number (or percentage) of people in an unexposed population with otherwise similar personal and demographic characteristics.

By taking account of the extent and seriousness of an effect, the associated disease burden on a population can be calculated. The disease burden of an effect is an index of the reduction that the effect in question causes within a population in the number of healthy years of life, expressed in units such as DALYs (*Disability Adjusted Life Years*)[33,102].

In order to make a very rough estimate of the prevalence of the effects of night-time traffic noise on sleep, and thus on health and well-being, one first requires data on the distribution of exposure to night-time noise in the Dutch population. Such data is available, albeit in the form of rough estimates, within the RIVM; see Annex G. By linking this data to the exposure-effect relationships described in this advisory report, it is then possible to estimate the prevalence of an effect in the Dutch population.

In order to determine the disease burden of an effect, it is necessary to know the weighting factor for the calculation of the associated DALYs[91,92]. However, scientific consensus is as yet lacking with regard to the weighting factors for certain effects[32].

3.5.2 *Biological effects*

The Committee has divided biological effects into two groups: acute effects and effects over the course of a night (before, while and after sleeping).

In order to estimate the prevalence of acute effects, such as being woken by night-time noise, it is necessary to have nationwide data on the distributions of traffic noise *SEL* or *LAmax* values. Because sleeping times should preferably be included in the calculations, but are subject to considerable inter-personal variation, one ought to additionally know how *SEL* or *LAmax* values are distributed in various periods e.g. in each hour covering the overall spread of sleeping times, rather than simply between 11pm and 7am. To arrive at a reasonably reliable estimate, one should also have national data on distribution in the acoustic insulating properties of dwelling walls, taking bedroom window status (open/closed) into account. However, using a simplified model, one could generate point estimates of the prevalence of an effect using average sleeping time and

attenuation values, plus point estimates of the prevalence at above-average and below-average sleeping time and attenuation values, thus providing some insight into the spread of results associated with variations in these factors. Although such an exercise is in principle viable, the Committee is not in a position to perform the calculations itself.

3.5.3 *Health and well-being*

The Committee has concluded that there is sufficient evidence that exposure to night-time noise during the sleep period reduces sleep quality and general well-being. Furthermore, there is limited evidence of a causal association between exposure and impaired social contacts and concentration, increased risk of developing certain medical conditions, and premature mortality due to fatal occupational accidents. This conclusion is based upon assessment of research data regarding various effect parameters. The parameters in question are interrelated; for example, difficulty getting to sleep and staying asleep is closely related to diminished perceived sleep quality (all three effect parameters for sleep quality). Consequently, if one calculated the prevalence of each effect parameter separately (supposing that were possible), and aggregated the figures, one would arrive at an overestimate of the consequences of exposure to night-time noise. The Committee has therefore chosen to base its estimates of the prevalence of diminished sleep quality and general well-being on self-reported high sleep disturbance data. Where this parameter is concerned, exposure-effect relationships have been established for noise from road, rail and air traffic. Since there is only limited evidence that night-time noise can lead to impaired social contacts and concentration, hypertension and premature mortality due to fatal occupational accidents, and little is known about the possible exposure-effect relationships, no estimate can be made of the prevalence of these effects. The Committee has, however, worked out a figure for the prevalence of insomnia, but would emphasise that this figure, like that for self-reported high sleep disturbance, is merely an indicative estimate. For this reason, the estimate is couched in very general terms.

The estimates have been made using *Lnight* values for the year 2003 provided by the RIVM; see Annex G. The data used reflects the annual burdens on dwellings, as associated with road, rail and air traffic collectively (cumulative noise exposure). By combining this information with what is known about the exposure-effect relationships for self-reported high sleep disturbance by road traffic noise[97,98] (see Figure 12)* and insomnia[103], the Committee has been able to estimate the increase in the prevalence

* The estimates are based on road traffic, as in the Netherlands night-time noise exposure to road traffic noise is much higher than that to air and rail traffic noise. Furthermore, using the separate noise sources would lead to overestimating the total self-reported high sleep disturbance.

within the Dutch population of the two effects that was attributable to night-time traffic noise in 2003. The results are presented in Table 8.

Table 8 Rough estimate of the prevalence within the adult Dutch population (12.5 million people) of high sleep disturbance and insomnia attributable to night-time traffic noise in 2003.

Effect	Prevalence band[a]
Self-reported high sleep disturbance	6
Insomnia	5
Ratio between insomnia and self-reported high sleep disturbance	2%

[a] Prevalence bands: band 0: 0-1 person, band 1: 1-10 people, band 2: 10-100 people, band 3: 100-1000 people, band 4: 1000-10 000 people, band 5: 10 000-100 000 people, band 6: 100 000- 1 000 000 people.

As indicated in Table 8, the prevalence of noise-related self-reported high sleep disturbance among adults in the Netherlands falls in band 6 (100,000 to a million adults). The prevalence of noise-related insomnia is estimated to be significantly lower.

For the year 2000, the RIVM estimated separate *Lnight* values for the noise exposures associated with road, rail and air traffic noise in the Netherlands[15]. On the basis of these figures, it has been estimated that, in 2000, the prevalence of noise-related self-reported high sleep disturbance among adults in the Netherlands, as attributable to each of these three sources, fell in band 6 (more than 100,000 adults). The number of adults with high sleep disturbance by road traffic noise will have been between two and four times higher than the numbers able to report such disturbance by rail or air traffic noise. The prevalence of insomnia attributable to either road or rail traffic noise was in each case estimated to have been in band 4 (between one thousand and ten thousand people), while that attributable to aviation noise (calculated on the basis of data on the noise exposure in the general vicinity of Schiphol) was estimated to have been in band 3 (between a hundred and a thousand people).

3.5.4 *Disease burden*

In recent years, there has been considerable focus on quantifying the collective disease burden attributable to environmental factors. One initiative in this area has been the introduction of the *disability adjusted life year* (DALY)[32,34] as a unit of measurement; see also subsection 2.3.3. In response to questions posed by the State Secretary for Housing Spatial Planning and the Environment, the Health Council is to prepare a separate advisory report on the issues associated with the use of DALYs[38].

In order to quantify a disease burden, it is necessary to know how many people experience a given effect and for how long, as well as how serious the effect is. In its prevalence calculations described above, the Committee concentrated on self-reported high sleep disturbance and insomnia, in relation to which estimates were made of the numbers of people affected and for how long. To calculate the associated disease burdens, the Committee has adopted the weighting score of 0.17 ascribed to insomnia by Stolk *et al*[37], even though this figure was defined for a different purpose. In the context of seeking to put a figure on the disease burden associated with sleep disturbance[32], De Hollander suggested weighting factors of between 0.01 and 0.1, but indicated that further study was desirable.

On the basis of the available data, the Committee has concluded that the best estimate of the disease burden associated with high sleep disturbance by night-time traffic noise in the Netherlands is several tens of thousands of DALYs. The corresponding figure for insomnia is certainly considerably lower. These estimates suggest that, through its influence on sleep, night-time traffic noise accounts for an important part of the overall effect that the physical environment has on public health[32,104].

By means of disease burden calculations of this kind, the effects of night-time traffic noise on health and well-being can be compared with the effects of other factors. However, the Committee wishes to emphasise that a cautious approach should be taken, since there is considerable uncertainly about many of the estimates.

Chapter 4

Acoustic considerations

Night-time noise in the domestic environment almost always consists of a combination of separate noise events, with the exception of certain forms of industrial noise. In section 4.1, the Committee considers how such noise events combine to create an overall noise exposure over the course of a night.

Noises come in many different forms, from a low rumble to a soprano's top C, from a steady whisper to a sudden bang, from a murmur to a squeak or a grating sound. It seems reasonable to assume that the nature of a noise influences its effect. The question is, is it possible to define an exposure-effect relationship in a way that takes account of the influential characteristics of a noise, for example by applying adjustment factors to the exposure or noise data. In the 1997 advisory report *Assessing Noise Exposure for Public Health Purposes*, a Health Council Committee looked at this issue in detail (see Annex F for a summary)[8]. In section 4.2, the Committee considers the content of that report.

In the chapter's final section (4.3), the efficiency and effectiveness of domestic insulation as a means of reducing the influence of noise on sleep are examined.

4.1 The combination of noise events and acute effects

4.1.1 *The combination of noise events*

Where night-time environmental noise involving individually distinguishable noise events is concerned, *Lnight* is a so-called 'exponential summation of the *SEL* values of the constituent events*.

A given *Lnight* value is a unique specification of the number of noise events with a certain *SEL value*. Since, where a particular type of noise (such as a train or aeroplane passage) is concerned, there is a very high correlation is between the *SEL* and the maximum level of a noise event (*LAmax*), a given *Lnight* value also specifies the number of noise events with a certain *LAmax* value. For example, an *Lnight* of 35 dB(A) is the result of *one noise event per year* with an *SEL* of approximately 105 dB(A), *one noise event per night* (every night) with an *SEL* of approximately 80 dB(A) or *hundred noise events per night* (every night) with an *SEL* of approximately 60 dB(A).

4.1.2 *Lnight and effects*

It follows that, at least where the above-mentioned acute effects of exposure to night-time noise are concerned, the consequences associated with a given *Lnight* value may vary. Generally speaking, the sum of all acute effects (over a year, since *Lnight* is an annual average) in a situation characterised by a small number of high-intensity events is less than in a situation characterised by numerous events whose intensity is above the effect threshold but nevertheless comparatively low. The least favourable situation (that involving the most acute effects per year) would be a series of events with *SEL* values 4 to 5 dB(A) above the observation threshold for the effect in question[97,105,106]. For subject-registered awakening by aircraft noise, for example, the worst-case scenario involves all aircraft passages having an *indoor SEL* (*SEL_i*) of approximately 60 dB(A), whereas the worst situation for increased probability of acute motility would involve all passages having an *SEL_i* of approximately 45 dB(A). In other words, at a given *Lnight_i*, the characteristics of the least favourable situation depend on which acute effect one is concerned with.

In Table 6, the Committee indicated that long-term exposure to night-time noise when sleeping leads to an accumulation of acute effects indicative of a negative influence on health and well-being. Where the overall effect of exposure to night-time noise

* The points made in this section relate to both outdoor and indoor noise levels (although the latter are expressed in units that have the suffix '_i'). For definitions of the acoustic variables, see table 1.

is not the sum of the acute effects, such as frequently being awoken by noise, sleep disturbance and self-reported sleep quality, the Committee considers it plausible that a series consisting of numerous relatively low-intensity noise events has a greater effect than a series with the same overall *Lnight* value, consisting of a smaller number of higher-intensity noise events.

4.1.3 Consequences for regulation

The 1997 Health Council report *Assessing Noise Exposure for Public Health Purposes* proposes the use of *Lnight* for the regulation of exposure to night-time environmental noise. Of course, the extent to which controls based on *Lnight* can protect against the effects of exposure to night-time noise when sleeping depends on the level at which the limit is set. However, it follows from the considerations set out above that various situations might arise in which, although the prescribed *Lnight* value was not exceeded, the exposure levels were undesirable from a health and well-being perspective, due to the occurrence of relatively frequent low-intensity noise events.

If one concludes that exposure should be limited further, but that that cannot be achieved by applying stricter *Lnight* exposure limits, the best way forward would be to place a limit on the number of noise events. The reason being that setting *SEL* or *LAmax* exposure limits for noise events would allow for less favourable situations, unless the exposure limits were set at impracticably low levels. Hence limitation of the number of events is preferable.

There is another reason for specifying a maximum permissible number of noise events. The *greater the nightly number* of noise events (above the observation threshold), the greater the chance is that one will coincidentally hear such a noise after 'spontaneously' waking up in the night, possibly leading to annoyance and problems going back to sleep. As indicated in section 3.2, if all intervals of 'spontaneous' wakefulness were to coincide with a noise event audible in the bedroom, a person might under extreme circumstances hear a noise that had not woken him or her up approximately ten times in the course of a night.

4.1.4 Conclusion

At a given *Lnight,* (or *Lnight_i*) an acute effect of exposure to night-time noise is most influential if the *LAmax_i* or *SEL_i* values of the separate noise events are approximately 5 dB(A) above the observation threshold for the effect in question. In order to prevent the occurrence of the worst-case scenario associable with a given *Lnight* value, consideration should be given to regulating not only *Lnight*, but also the number of noise events. One consequence of setting a ceiling on the number of noise events would be

that one was less likely to hear a noise event after 'spontaneously' waking in the night, and therefore less likely to suffer sleep disturbance.

Whether it is necessary or desirable to set an exposure limit on the number of noise events, in addition to limiting *Lnight* values, depends on the level of the *Lnight* limit and the level of protection one wishes to provide. The relationships between acute effects and *SEL_i* values defined in this report, make this method of regulation a viable option.

4.2 Noise characteristics

The Committee has identified a number of forms of noise that may have a particularly pronounced effect on people exposed to them:

- Noise characterised by low-pitch components (buzzing)
- Noise consisting entirely of one or more low buzzing sounds (low-frequency noise)
- Tonal noise
- Noise events characterised by a rapid increase in intensity at the beginning (impulse noise)
- Industrial noise
- Noise characterised by sporadic high *LAmax* or *SEL* values.

4.2.1 Noise characterised by low-pitch components

As indicated in chapter 2, noise exposure is generally measured using a so-called A-weighting, which takes account of the frequency sensitivity of the human ear. However, there is evidence to suggest that this method may place insufficient emphasis on low-frequency noise components in particular. This possible drawback does not apply if use is made of the so-called C-weighting, which affords nearly as much weight to low-pitch components as to high-pitch components.

In the Netherlands, a study is in progress aimed at determining the differences between outdoor A-weighted and C-weighted equivalent sound pressure levels measured in situations that frequently arise in practice[107]. The measured average differences so far determined for aircraft, lorries, freight trains, shipping and industrial activities are, respectively, 9, 7, 5, 14, and 13 dB and the ranges of the measured differences are, respectively, 2-13, 2-15, 1-15, 9-21, and 6-24 dB. From these figures, it is apparent that noise from shipping and from industrial activities contains more low-frequency components than noise from road, rail or air traffic. The researchers believe that the differences are much greater indoors than out, because the fabric of residential buildings attenuates some frequencies more than others.

The extent to which the presence of lower-frequency components increases noise-related annoyance or sleep disturbance is still under investigation. The Committee antic-

ipates that the results of the research currently in progress will enhance understanding of the contribution that lower-frequency noise components make to annoyance and sleep disturbance, but does not expect that it will be necessary to revise the exposure-response relationships that have so far been defined, since these definitions already take account of any extra influence of lower-frequency components. However, the tendency to use higher powered equipment may mean that in the future the noise from certain sources will contain much more low-frequency noise components, possibly necessitating modification of the exposure-effect relationships as presently defined.

4.2.2 *Low-frequency noise*

After considerable deliberation, the authors of the 1997 Health Council report decided that low-frequency noise should not be incorporated into the assessment framework, since there was no reliable means of defining the necessary low-frequency noise adjustment factor. The present Committee sees no reason to revise this view, as no relevant new data has become available since. However, it does follow that the conclusions set out in this advisory report do not necessarily apply to low-frequency night-time noise. It should nevertheless be pointed out that low-frequency noise is relatively unusual in the domestic environment; at least, the Committee is unaware of any commonplace sources of such noise. Where sources of low-frequency noise are present, however, annoyance is most likely to occur at night, when such noise is not masked by higher-frequency noises in the domestic environment.

4.2.3 *Tonal noise*

Nor has any new data relating to tonal noise become available since 1997. The Committee accordingly endorses the recommendation contained in the earlier Health Council report, namely that the equivalent sound pressure level should be increased by between 0 and 5 dB(A) in cases that involve exposure to tonal noise when sleeping (see Annex F). It is worth noting that, like low-frequency noise, tonal noise is rare in the domestic environment.

4.2.4 *Impulse noise*

An impulse noise is a noise that increases very quickly, so that, as far as the listener is concerned, it seems to reach its maximum intensity almost immediately. Examples include gunshots and low-flying military jets. The international standard ISO1996/01[108], published in 2002, sets out a method for the assessment of impulse noises that is consistent with the thinking of the Health Council's 1997 report. This system indicates that

adjustment factors of 5 and 12 dB(A), respectively, should be used for certain specified impulse noises (low-flying military jets, car doors slamming, church bells chiming) and certain specified very impulse-like noises (gunshots, metal beating, pneumatic hammering, shunting of rail rolling stock). ISO 1996/01[108] lists the impulse noises and very impulse-like noises in respect of which adjustment factors should be applied, because, according to the working group that developed the standard, ISO 1996/01 there was too little readily interpretable research data available to enable quantification of the adjustment factors in audiological, physical or acoustic terms. The ISO standard did not adopt the assessment method prescribed in the 1996 US standard ANSI S12.9[109], which was based on the speed with which the sound pressure level rose at the beginning of an impulse noise. In the Netherlands, however, a provisional assessment method was introduced for railway yards, which followed the US method in working on the basis of the speed with which the sound pressure level rises at the beginning of a noise event[110].The maximal adjustment factor that can be used is the 12 dB(A) applicable in relation to very impulse-like noise.

The adjustment factors of 5 and 12 dB(A) are derived from research into noise-related annoyance. People probably find impulse noises more annoying because of the startle responses they tend to induce[105]. Research by Griefahn[65] (into the effects of gunshot noise in the laboratory), Vos[111] (into the effects of gunshot noise in the field, see Figure 24 in Annex D) and Fidell[49] (the effects of noise from military jets in the field, as analysed by Passchier-Vermeer[52], see Figure 22 in Annex D) have all shown that noise events characterised by a rapid initial rise in sound pressure level also cause considerably more sleep disturbance than 'ordinary' environmental noises.

4.2.5 *Industrial noise*

While attaching certain caveats, the 1997 Health Council report suggests that, in situations characterised by lower noise exposures, the equivalent sound pressure levels associated with industrial noise should be corrected by between 0 and 10 dB(A). This proposal was based on considerations regarding noise-related annoyance relative to noise exposure over the full twenty-four-hour period. Recent research has since shown that there is no scientific basis for making such an adjustment[112].

Figure 14 illustrates the relationships between industrial noise and annoyance, as defined using data from recent research by Miedema *et al*[112], and the relationships between road traffic noise and annoyance[113]. The figure shows the percentages of people experiencing high annoyance attributable to road traffic noise and industrial noise, the percentages of people experiencing at least moderate annoyance, and the percentages of people experiencing at least slight annoyance, as functions of *Lden*. It will be seen that the curves for industrial and road traffic noise are almost identical, and certainly do no

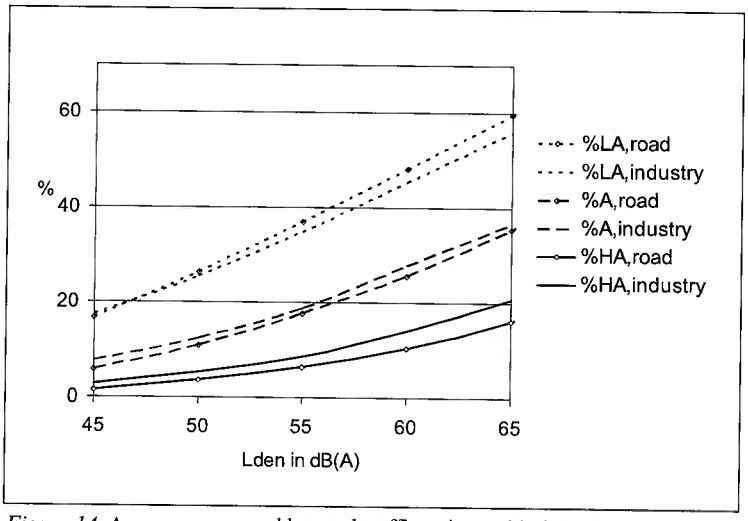


Figure 14 Annoyance caused by road traffic noise and industrial noise as a function of *Lden*[112,113]. %HA is the percentage of people experiencing high annoyance, %A is the percentage of people experiencing at least moderate annoyance, and %LA is the percentage of people experiencing at least slight annoyance.

justify the conclusion that, at *Lden* values of between 40 and 60 dB(A), industrial noise causes more annoyance than road traffic noise. The Committee consequently believes that there is no longer any justification for correcting the equivalent sound pressure levels associated with *night-time* industrial noise either.

4.2.6 *Sporadic high LAmax or SEL values*

The exposure-response relationships described above have been defined on the basis of data from situations where night-time noise events occurred regularly. It is therefore pertinent to ask whether these relationships remain valid in situations characterised by sporadic noise events with comparatively high *SEL* and *LAmax* values. The Committee anticipates that, in such a situation, the probability of an *acute* effect (of whatever kind) will be greater than the defined relationships suggest, since the hearer will necessarily be unused to noise events of the kind involved, and anxiety is very likely to play a role. Anxiety is particularly likely to play a role where the hearer associates a noise with a previously experienced threat to him/herself or others. A single event of this kind can also have consequences for the hearer's quality of sleep for the rest of the night and on subsequent nights. However, the Committee does not have sufficient research data at its disposal to develop these assumptions more fully.

In questionnaire-based studies of self-reported long-term effects (such as diminished sleep quality and night-time noise-related annoyance), a one-year assessment period is typically used. The Committee is not aware of any research that has looked at the specific effects that noise events with relatively very high *SEL* or *LAmax* values have on such self-reported parameters. The Committee cannot therefore make any scientifically justified statement about such effects.

4.2.7 *Conclusion*

Although little is known about how sleep is affected by exposure to noises with unusual characteristics, the Committee believes that it is reasonable to assume that the effects of exposure to some 'special' types of noise are greater than the effects of exposure to 'ordinary' traffic noise. The Committee is of the opinion that the conclusions of the 1997 Health Council report *Assessing Noise Exposure for Public Health Purposes* remain valid in relation to noise with low-frequency components, low-frequency noise, tonal noise and impulse noise. The adjustment factors that need to be applied to the exposure indexes are given in Annex F. However, where noise from industrial activities is concerned, data published since 1997 indicates that the application of an adjustment factor is no longer justified. The Committee is unable to make any definitive statement regarding the possibility that occasional, very loud noise events may have more far-reaching consequences.

4.3 Efficiency and effectiveness of the acoustic insulation of homes

4.3.1 *Data*

In the Netherlands, there have only been a number of isolated studies into the efficiency and effectiveness of acoustic insulation in the reduction of perceived road and aviation noise levels, or into people's views regarding such insulation[114-119].

Bitter *et al* looked at the effects of fitting additional acoustic insulation to flats beside busy motorways in Dordrecht[114] and Amsterdam[115]. A survey of residents 2.5 years after the modifications were made revealed that half the people living in the flats were no longer annoyed by night-time road traffic noise.

Van Dongen *et al*[116] carried out an exploratory study into sleep quality in homes fitted with additional acoustic insulation in the vicinity of Amsterdam's Schiphol Airport. Comparative analysis revealed that self-reported sleep disturbance and self-reported high sleep disturbance were slightly lower in the better-insulated dwellings than in 'ordinary' dwellings. However, the design of the study precluded the drawing of definitive conclusions.

Three reports were published between 1994 and 1999[117-119] regarding people's general views concerning modifications made to homes near Schiphol with a view to reducing aircraft noise-related problems. Some 85 per cent of subjects reported that the insulation had reduced noise-related annoyance indoors. Nevertheless, people in more than 55 per cent of the homes continued to experience at least slight noise-related annoyance, and people in 15 per cent of the homes reported experiencing high annoyance since the modifications were made. The distribution patterns of both overall and night-time levels of aircraft noise-related annoyance were clearly seasonal: on (cold) winter nights, 10 per cent of subjects often or always experienced annoyance during the sleep period, compared with 40 per cent on (warm) summer nights. The differences were closely related to the use of windows: only 25 per cent of respondents said they slept with the bedroom window at least slightly ajar in the winter, whereas 70 per cent did so in the summer.

Almost no research into the efficiency of domestic acoustic insulation has been done in other countries either, the exceptions being studies by Fidell and Silvati[120], Utley[121] and Minoura[122].

Fidell and Silvati[120] investigated what effect the fitting of insulation to attenuate aviation noise had on levels of annoyance. However, they did not look specifically at annoyance during the sleep period.

In the UK, an extensive study was done to establish how effective extra acoustic insulation was in reducing exposure to road traffic noise[121]. In the specially insulated homes, approximately a quarter of subjects whose bedrooms faced the street reported being very highly or highly annoyed by night-time road traffic noise; a similar number had difficulty getting to sleep because of the noise, and more than a quarter of respondents said they were woken up at night by road traffic noise. The results proved to be influenced to a considerable extent by whether the subject felt that, without the window open, his or her bedroom was too hot in the summer: 37 per cent of those who felt unable to sleep with the window closed in warm weather were very highly or highly annoyed by night-time road traffic noise, whereas only 15 per cent of those who didn't mind having the window closed experienced similar problems.

Minouri investigated the situation in the vicinity of a US air base on a Japanese island, with a view to determining how effective additional acoustic insulation was in an area with a very high aircraft noise exposure. Because the circumstances on the island are quite unlike any in the Netherlands, the findings – which indicated that the insulation was disappointingly ineffective – are not transferable to the Dutch situation.

In an interview-based study of 1242 households in the Netherlands, Leidelmeijer and Marsman[99] investigated the audibility of and annoyance associated with noise from neighbours during the day and at night. The researchers distinguished between five types of noise: noise from sanitary fittings, contact noise, noise from audio equipment,

do-it-yourself (DIY) noise and noise from pets. Further distinctions were made according to the part of the house where the noise was audible or caused annoyance, and the time of the day or night. Subjects proved least tolerant of noise from their neighbours that was audible in the master bedroom. Where each of the five investigated types of noise were concerned, roughly 10 to 15 per cent of subjects indicated that they felt it was unacceptable for the noise to be audible during the day. In each case, a higher percentage said the noise should not be audible in the evening, and a still higher percentage did not want to hear the noise at night (between 11pm and 7am). Overall, nearly 30 per cent of subjects said that sanitary fittings should not be audible at night, while approximately 50 per cent felt each of the other four types of noise were unacceptable by night.

Subjects were also asked whether they could hear voices from neighbouring homes. While the percentage of affirmative answers varied according to the type of dwelling, ordinary speech was to some extent audible in an average of 35 per cent of dwellings, and partially or readily comprehensible in approximately 8 per cent of dwellings. Raised voices could be heard, at least some to extent, in approximately 65 per cent of dwellings; they were at least partially comprehensible in 27 per cent of homes and readily comprehensible in approximately 10 per cent.

In 1993, Kranendonk *et al* produced a synthesis of the research conducted up to that point in time into the annoyance associated with noise from neighbours[100]. TNO later produced a report[101] on neighbour-noise and acoustic insulation based on the findings of a questionnaire-based survey of the residents of six hundred homes. They established that nearly half of the respondents heard at least some noise from neighbouring dwellings every day. Approximately 10 per cent of subjects found their neighbours' noise highly annoying. The chief causes of annoyance were loud radios, hi-fis and TVs, the slamming of doors and footsteps on floors and staircases.

The authors of both studies concluded that, given the minimum level of acoustic insulation required in new dwellings under the Building Decree[14] (an $I_{lu,k}$ value of 0 dB(A)), noise from neighbours caused high annoyance for 10 per cent of subjects and at least moderate annoyance for 25 per cent.

4.3.2 *Conclusion*

From the little data available, the Committee concludes that fitting additional acoustic insulation to homes can reduce the annoyance associated with night-time traffic noise to some extent. It is not presently possible to quantify the benefit, however. One thing that is clear, is that if steps are not also taken to enable householders to keep their bedrooms cool in hot weather, the benefit of additional acoustic insulation is liable to be offset in the summer by people opening their windows.

In addition, the Committee considers the following points to be important for assessment of the effectiveness and efficiency of domestic acoustic insulation and therefore important in the context of research in this field:

- There is a danger that fitting high-grade acoustic insulation to exterior walls in an effort to deal with a form of noise that is much louder than other noises in the environment will have the effect of cutting out all noises except the one that is causing problems.
- High-grade acoustic insulation against noises from external sources has implications for inter-dwelling acoustic insulation. If the latter is only of a moderate standard, as is frequently the case in the Netherlands, noises from neighbouring dwellings (sanitary facilities, TV, radio, kitchen noises, people going up and down stairs, parties, rows, voices) becomes much more apparent, potentially leading to social tensions.
- Many people like to sleep with their windows at least partially open, which negates the effect of acoustic insulation on the exterior walls to some extent. Although there are technical solutions for this problem, such as variable ventilation systems, that adjust the ventilation opening in line with rising or approaching noises from outside, they are not in widespread use.
- Very high levels of insulation can cause 'acoustic isolation': cutting the householder off from 'pleasant' outside noises, such as birdsong and children at play. However, the Committee anticipates that acoustic isolation is less likely to be a problem at night than during the day.

The Committee believes that the standard of inter-dwelling acoustic insulation presently required is not sufficient to provide protection against annoyance attributable to noise from neighbours. Since people are a lot less tolerant of the noise their neighbours make at night-time than of their neighbours' evening or daytime noise, it may be assumed that much of the annoyance associated with noise from neighbours relates to the influence of such noise on sleep.

Chapter 5

Answers to the State Secretary's questions

In this chapter, the Committee presents its answers to the specific questions posed by the State Secretary and summarises its conclusions. The answers to the State Secretary's questions are based upon the information provided in chapters 3 and 4. First, however, the Committee explains how the answers fit into the environment and health context described in section 2.4.

5.1 General principles

5.1.1 *Effects of exposure to noise when sleeping*

In its evaluation of the consequences of exposure to noise when sleeping, the Committee has applied the model illustrated in Figure 3. In this model, biological phenomena occur in response to environmental noise because, even when sleeping, an individual still needs to assess and process 'stimuli' from the environment. The biological responses that are liable to occur include waking up, difficulties getting off to sleep and increased average motility while sleeping. To some extent, these responses involve acute changes during exposure to a noise, and to some extent they involve changes that manifest themselves over the course of a night (before, while and after sleeping). Such effects can be predictors of long-term decline in health and well-being, which may or may not depend upon the nature and duration of the exposure. It is not therefore possible to say in advance whether a biological response to night-time noise will lead to a decline in health or well-being.

5.1.2 *Strength of the evidence*

In order to define the degree of certainty concerning the relationship between exposure to night-time noise and a particular effect, the Committee has defined three categories of evidence: sufficient, limited and insufficient evidence; see Table 2. The category 'limited evidence' is subdivided into two forms:

- A causal relationship is plausible, and has been observed to a limited extent in epidemiological research.
- No direct link has been epidemiologically established between exposure and effect, but there is good quality indirect empirical evidence for such a link, and the presence of a link is plausible. Indirect evidence may be said to exist if it has been observed that exposure has an intermediary effect, which is known from other research to lead to the ultimate effect under consideration.

5.2 Effects of noise when sleeping

Question 1: What are the effects (expressed in quantitative terms as far as possible) of exposure to noise when sleeping?

Environmental noise can be divided into noise from traffic (such as air, road and rail traffic), noise from stationary sources (such as factories and shunting yards), neighbourhood noise (noise from, for example, sports stadiums, racing circuit or open air events) and noise from neighbours (contact noise, noise of audio equipment, voices). Research into the relationship between, on the one hand, sleep characteristics and health and, on the other, exposure to night-time noise has tended to focus mainly on road and air traffic noise. In the following subsections, the Committee accordingly first addresses noise from these sources, before moving on to consider noise from rail traffic and stationary environmental sources, the neighbourhood noise and noise from neighbours.

The Committee distinguishes between biological effects and the accumulated effects on health and well-being of exposure resulting from sleeping in an environment affected by night-time noise. The Committee has divided effects of the latter kind into five categories: diminished sleep quality, diminished general well-being, impaired social contacts and concentration, medical conditions and reduction in life expectancy.

5.2.1 *Biological effects of road traffic noise and aviation noise*

Biological effects can be divided into acute (immediate) effects and effects that occur over the course of a night (before, while and after sleeping).

Acute biological effects

Noise during the sleep period induces an immediate response from the body. The effects that have been observed to take place in direct response on noise events that occur while the subject is sleeping are listed in Table 9. There is sufficient evidence for a causal relationship between each of these effects and night-time noise events.

Table 9 Acute biological effects for which there is sufficient evidence of a causal relationship with night-time noise (see Table 1 for terminological definitions).

Effect
Cardiovascular change[a]
Sleep stage change, from deeper to less deep sleep
EEG awakening
Motility
Onset of motility
Subject-registered awakening

[a] The advisory report focuses mainly on heart rate acceleration, but there is also sufficient evidence of the induction of vasoconstriction and acute blood pressure rises.

Most of these effects have been sufficiently well studied to enable exposure-effect relationships to be defined. Hence, it appears that effects such as EEG awakening and increased motility first manifest themselves at indoor *SEL* values of approximately 40 dB(A). Noise-related subject-registered awakening is liable to occur at *SEL* values of 55 dB(A) and above. These values are valid for adults; insufficient data is available to enable the definition of relationships for children. It is assumed that night-time noises can induce acute changes in the (stress) hormone concentrations in a sleeping subject's blood, but this has not been proven. Such changes cannot easily be studied in a field situation, because it would involve the use of invasive test techniques.

Effects before, while and after sleeping

Numerous biological effects over a night (before, while and after sleeping) have been observed in epidemiological research. Some of these relate directly to the acute responses: raised average heart rate, increased motility, more frequent subject-registered awakening, and longer waking intervals (as registered on a sleep EEG). The level of average motility observed in people who are exposed to night-time road and air traffic noise appears to be greater than might be expected on the basis of the acute responses alone. Average motility is closely related to waking up more frequently, diminished perceived sleep quality and increased drowsiness during the day. Furthermore, people who

when trying to get to sleep are exposed to road or air traffic noise, or are worried about the possibility of being disturbed by noise in the night ahead, have more difficulty getting to sleep. The effects that manifest themselves after a sleep period are reduced perceived sleep quality, increased irritability and rise of drowsiness and tiredness during the day. There is therefore sufficient evidence of a causal relationship between noise and all these effects.

There is limited direct evidence that under certain circumstances exposure to night-time noise can influence (stress) hormones levels in sleeping subjects: this effect was observed in women who were troubled by noise in the night and unable to take corrective action. However, more definitive conclusions regarding the influence of noise on (stress) hormone levels must await the availability of further research data.

The exposure-related biological effects over the course of a night are listed in Table 10. For each effect, the table indicates the strength of the evidence for the existence of a causal relationship between exposure and effect, and the plausibility of the effect being indicative of a negative influence on health and well-being.

Table 10 Biological (physiological and psycho-physiological) effects observed after chronic exposure over numerous nights, indicating the strength of the evidence for a causal relationship with exposure to road and air traffic noise and the plausibility of the effect being indicative of an influence on health and well-being.

Variable	Strength of the evidence	Plausibility of influence on health and well-being
Change in cardiovascular activity	Sufficient evidence	Plausible
Increased average motility (motility)	Sufficient evidence	Plausible
Changes in duration of various stages of sleep, in sleep structure, fragmentation of sleep	Sufficient evidence	Empirical data
Prolongation of the sleep inception period, difficulty getting to sleep	Sufficient evidence	Plausible
Changes in (stress) hormone levels	Limited evidence, plausible	Plausible
Immune functions	Insufficient evidence	-
Waking up in the night and/or too early in the morning	Sufficient evidence	Empirical data
Drowsiness/Tiredness during the day and evening	Sufficient evidence	Empirical data
Impaired cognitive performance	Limited evidence, plausible	Plausible
Increased irritability	Limited evidence, plausible	Plausible
Annoyance	Limited evidence, plausible	Plausible

5.2.2 *Consequences for health and well-being*

Road and air traffic noise

The Committee's conclusions regarding the relationships between exposure to night-time road and air traffic noise when sleeping and changes in health and well-being are

summarised in Table 11. The effect parameters which the Committee has grouped under the five categories listed in the first column are specified individually and in each case an indication is given of the strength of the evidence for a causal relationship between the effect parameter in question and night-time exposure to noise when sleeping.

Table 11 Effects on health and well-being of prolonged exposure to noise during the sleep period.

	Effect parameter	Evidence
Sleep quality	Reduced perceived sleep quality	Sufficient evidence
	Difficulty getting to sleep, difficulty staying asleep	Sufficient evidence
	Sleep fragmentation, reduced sleeping time	Sufficient evidence
	Increased average motility when sleeping	Sufficient evidence
Well-being	Sleep disturbance	Sufficient evidence
	Health problems	Sufficient evidence
	Use of somnifacient drugs and sedatives	Sufficient evidence
	Increased daytime irritability	Limited evidence, plausible
Social contacts and concentration	Impaired social contacts	Limited evidence, plausible
	Impaired cognitive performance	Limited evidence, plausible
Medical conditions	Insomnia	Sufficient evidence
	Hypertension	Limited, indirect evidence, plausible
	Depression (in women)	Limited, indirect evidence, plausible
	Cardiovascular disease	Limited, indirect evidence, plausible
Reduction in life expectancy (premature mortality)[a]	Occupational accidents	Limited, indirect evidence, plausible

[a] Cardiovascular disease also involves the loss of healthy life expectancy. However, no account has been taken here of the lost life-years, since there is only limited evidence for a causal association between cardiovascular disease and exposure to night-time noise.

With regard to the long-term health and well-being implications of exposure to night-time noise during the sleep period, the Committee's overall conclusion is that there is sufficient evidence that such exposure leads to reduced sleep quality and reduced general well-being, and limited evidence that it leads to impaired social contacts and concentration, increased probability of developing medical conditions and reduced life expectancy due to fatal occupational accidents.

Rail traffic and stationary environmental sources

Epidemiological research into the effects of rail traffic noise has been confined to self-reported sleep disturbance, changes in sleep EEG and motility. At a given noise expo-

sure, rail traffic noise is slightly less likely to induce these effects than road traffic noise or aviation noise. Although there is no direct evidence that rail traffic noise has any other effects, the Committee considers it plausible that other effects can occur, although the relationship between noise exposure and observation thresholds may not be the same as where road or air traffic noise are concerned.

No epidemiological research has been carried out into the consequences of exposure to night-time noise from stationary environmental sources. However, laboratory research has indicated that the effects of individual noise events associated with stationary environmental sources are essentially similar to the effects of events associated with road and air traffic noise. Lack of epidemiological research data prevents the Committee from drawing any definitive conclusions regarding the effects of continuous noise from stationary environmental sources.

Neighbourhood noise and noise from neighbours

Inventory research in the Netherlands indicates that sleep disturbance attributable to the most annoying forms of neighbourhood noise and noise from neighbours (contact noise and human noises in the environment) is on a similar scale to disturbance attributable to the most annoying sources of road traffic noise (mopeds and passenger cars). It is reasonable to assume that chronic sleep disturbance is in the long term liable to have consequences for health and well-being. The sound pressure level and other noise characteristics are liable to determine the nature of the influence to some extent, but certain other factors play a more prominent role than is the case with traffic noise. These factors include appreciation of the noise and of the party responsible for the noise, as well as the hearer's personal circumstances. However, scientific understanding of the relative importance of and interaction between acoustic and non-acoustic factors is not sufficient for the Committee to draw any definitive conclusions regarding the relationship between, on the one hand, exposure to night-time neighbourhood noise and noise from neighbours and, on the other, health and well-being.

5.3 Public health perspective

Question 2: How do such effects compare with other effects on health, in terms of seriousness and magnitude?

The Committee assumes that what the State Secretary is interested in is the magnitude of the effects within the Dutch population. The Committee has estimated the consequences of exposure to night-time traffic noise on the health and well-being of the Dutch population in terms of self-reported high sleep disturbance and insomnia. The Committee's

estimates are based on the cumulative noise exposure associated with road, rail and air traffic in 2003 (Annex G). Because the calculations inevitably involve considerable uncertainty, the results should be regarded as merely indicative.

The number of adults in the Netherlands experiencing high sleep disturbance due to traffic noise in 2003 has been estimated at between a hundred thousand and a million. The increase in the number of adults suffering from insomnia attributable to exposure to night-time traffic noise is put at between ten thousand and a hundred thousand. The number of people suffering from insomnia caused by traffic noise is 2 per cent of the people with high sleep disturbance.

For the year 2000, the RIVM has estimated separate *Lnight* values for the noise exposures attributable to road traffic, rail traffic and air traffic[15]. On the basis of this data, the Committee has calculated that the number of adults in the Netherlands experiencing self-reported high sleep disturbance due to noise from each of these three types of traffic in that year was between a hundred thousand and one million. The increase in the number of people suffering insomnia attributable to road traffic noise and rail traffic noise is in each case estimated at between one thousand and ten thousand, while the corresponding figure for aviation noise in the general vicinity of Schiphol Airport is between a hundred and a thousand people.

In recent years, there has been considerable focus on quantifying the collective disease burden attributable to environmental factors. One initiative in this area has been the introduction of the DALY as a unit of measurement. In response to questions posed by the State Secretary for Housing, Spatial Planning and the Environment, the Health Council is to prepare a separate advisory report on the issues associated with the use of DALYs. On the basis of information from a thesis by De Hollander and noise data provided by the RIVM (see Annex G), the Committee concludes that the disease burden associated with high sleep disturbance by night-time traffic noise in the Netherlands is several tens of thousands of DALYs. The corresponding figure for insomnia is certainly considerably lower. Although these estimates involve considerable uncertainty, they would appear to indicate that, through its influence on sleep, night-time traffic noise accounts for an important part of the overall effect that the physical environment has on public health.

By quantifying effects in DALYs, the effects of night-time traffic noise on health and well-being can be compared with the effects of other physical environmental factors. However, the Committee wishes to emphasise that a cautious approach should be taken, since there is considerable uncertainly about many of the estimates.

5.4 Risk groups

Question 3: Is it necessary to take special account of any population groups that are at particular risk?

As indicated in Chapter 3, the consequences of a given level of exposure to night-time noise when sleeping vary from person to person. The question therefore arises, is it possible to identify certain groups that are at increased risk? The Committee believes that there are some population groups whose health and well-being are more likely to be affected than others. This belief is based on extrapolations from what is known about sleep disorders and sleeping problems as they generally occur in the population at large, since very little of the research that has been done into the effects of night-time noise exposure has shed light on the risk factors affecting particular groups.

Although the strength of the evidence found by the Committee is limited, it does appear that people with cardiovascular problems, people who regard themselves as particularly sensitive to noise, and children may all be particularly sensitive to the acute cardiovascular effects of exposure to night-time noise. Because of the shortage of research data on children, it is not possible to say with confidence whether children are more sensitive than adults to other acute biological effects.

Where effects over the course of a night are concerned, people who suffer from insomnia constitute a risk group. People who during the sleep latency period worry about environmental noise need longer to get to sleep and perceive the quality of their sleep to be diminished.

Although there is no direct evidence, the Committee believes that adults who suffer from insomnia or another sleep disorder or who have another sleeping problem that causes them to wake up frequently in the night are more likely than 'sound sleepers' to suffer annoyance due to night-time noise reaching their bedrooms. The Committee also considers it plausible that there is an increased risk that the health and well-being of the following groups of adults will be adversely affected by exposure to night-time noise: older people; pregnant women and women who were pregnant within roughly the last year; people who work night shifts; people affected by physical pain, dementia, depression, hypertension, cardiovascular disease or respiratory illness. No research has been carried out into the relative risk of exposure to night-time noise having adverse consequences for the health and well-being of children.

5.5 Protection against night-time noise

Question 4: In view of the effects referred to, would it be advisable to introduce special rules, similar to those contained in Directive 2002/49 and the Aviation Act, for night-time noise from sources other than air traffic?

The Committee has been able, in its answers questions 1 to 3 (concerning the influence of noise on health), to comment regarding the influence of road and air traffic noise, and to a very limited extent regarding the influence of rail traffic noise and industrial noise, but has not been able to comment regarding the influence of noise from stationary sources, neighbourhood noise or noise from neighbours. Nothing can be said regarding noise from the latter group of sources in answer to question 4 either.

5.5.1 *Two noise indexes*

There is no decisive medical reason why road traffic, rail traffic or industrial activities should be treated differently to air traffic in the context of night-time noise regulation. In its 1997 advisory report *Assessing Noise Exposure for Public Health Purposes*, the Health Council put forward a system of two noise indexes for use in protection of the general public against traffic noise and industrial noise in the domestic environment[8]. The Committee sees no reason to depart from its predecessor's recommendations. As indicated in the 1997 report, an index of exposure to noise over a twenty-four-hour period needs to reflect general noise-related annoyance, while an index of exposure to night-time noise should be related to sleep disturbance. The desirability of a two-index system is emphasised by the summary given in Chapter 3: the effect mechanisms of and consequences of exposure to night-time noise differ at least in part from those associated with general noise-related annoyance.

The approach currently recommended by the European Union involves the application of the noise indexes *Lden* and *Lnight* (see section 2.2). In essence, this approach closely matches that put forward by the Health Council's 1997 report[8]. Again, one might ask whether it would not be sufficient to work with a single index, *Lden*, for all sources of noise. After all, *Lden* does make allowance for night-time noise, even attaching an additional weighting factor to nocturnal values. Furthermore, the regulation of sound pressure levels on the basis of *Lden* would imply limiting *Lnight* to a value 5 dB(A) or

more lower than *Lden*[*]. However, by using a two-index system, one can apply separate criteria to general noise-related annoyance and sleep disturbance, each tailored to the effects in question. This makes for more transparent regulation and, particularly in situations where high values of *Lden* are permitted, to more effective protection[**].

5.5.2 *Shortcomings of the index for night-time noise*

Although, as indicated above, the Committee favours *Lnight* as the index for night-time noise, this expression does have certain shortcomings.

If exposure to noise is the decisive factor influencing sleep, then the noise exposure in a person's bedroom is the variable that is most closely related to the effects of exposure. A number of examples are given in Chapter 3 to illustrate this point. Although the Building Decree[14] makes certain requirements regarding the noise-attenuating properties of the walls of new dwellings, and thus makes indirect requirements regarding the indoor noise exposure, the rules do not apply to existing homes. Consequently, at a given outdoor noise exposure, there is considerable variation in the noise exposures that people actually experience in their bedrooms. The picture is further complicated by differences in people's attitude to bedroom ventilation. Hence, the actual noise exposure and the magnitude and seriousness of the associated effects can vary substantially at a given *Lnight* value.

It is also important to recognise that the nation's sleeping times vary sharply, and that most people – especially younger people – have a different sleeping pattern at the weekend from the one they follow during the week. It is estimated that approximately 15 per cent of adults in the Netherlands go to sleep before 11pm, and 50 per cent sleep beyond 7am. Therefore, because *Lnight* relates to the period from 11pm to 7am, it by no means covers the sleeping times of the entire population. Hence, no requirement based on *Lnight* can ever provide full protection against sleep disturbance.

Despite the shortcomings highlighted here, the Committee does not advocate the use of an alternative index, because it is unrealistic to suppose that any regulatory method could address every conceivable factor. Furthermore, it is the Committee's view that a regulatory system based on the use of *Lnight* (in addition to *Lden*) can provide considerable protection against exposure to noise when sleeping. Just how effective such a regulatory system actually is will obviously depend on the *Lnight*-based standards and limits that are defined.

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* In the most extreme case, where all noise occurs between 11pm and 7am, *Lden* would be 5 dB(A) higher than *Lnight*. (*Lnight* = x, *Lden* = 10*lg[8/24*10**((x+10)/10)] = x + 5 (dB(A)). Under all other circumstances, *Lnight* would be more than 5 dB(A) lower than *Lden*.

** Such as additional acoustic insulation for bedrooms.

5.6 Indexes for night-time noise

Question 5: If so, is it sufficient for such rules to be based on *Lnight*, or are additional indexes of exposure required, with a view to regulating impulse-like noises and situations involving relatively infrequent but high-intensity noise events?

This question may be divided into the following two elements:

- Is *Lnight* an adequate sole exposure index for noise with no special characteristics?
- Should any additional indexes be used for the regulation of noise with special characteristics or in special situations?

The Committee's answers are based upon the deliberations set out in, respectively, section 4.1 and section 4.2.

5.6.1 *Lnight as an index of exposure*

The question is, would protection be enhanced by regulating not only *Lnight* values, but also individual noise events? One might, for example, impose a *maximum sound pressure level* for a noise event or limit the *number of* noise events per night. As indicated in section 4.1, the Committee considers it inappropriate to impose a maximum sound pressure level. The reason being that, for a given *Lnight* value, situations characterised by numerous events with relatively low *SEL* or *LAmax* values are generally more likely to be problematic than situations involving smaller numbers of events with higher *SEL* or *LAmax* values.

The *more* noise events a person is exposed to per night, the greater the chance is that he or she will happen to hear one of the noises after waking up 'spontaneously', and then have trouble getting back to sleep. This may help to explain the prevalence of sleep disturbance, and could justify limiting the number of noise events per night. As indicated in subsection 3.2.4, in an extreme case it is theoretically possible that someone could hear a passing car, plane or train car ten times in the night without the associated noise being the cause of the person waking up. The Committee believes that calculations could theoretically be made regarding these matters, but does not believe that there is presently enough detailed data available for anything better than rough estimates.

5.6.2 *Adjustment of Lnight for special noises*

As indicated in section 4.2, the Committee considers the following 'special' noises to be of particular relevance for the night-time domestic environment:

- Noise with lower-frequency components (such as engine noises with deep components)
- Low-frequency noise (such as noise from transformers)
- Tonal noise (such as sirens)
- Impulse noise (such as the noise from low-flying military jets or gunshot noises)
- Industrial noise
- Noise involving sporadic high *LAmax* or *SEL* values.

Little information is available regarding the influence on sleep of exposure to noise with special characteristics. Nevertheless, the Committee believes that in some cases the effects of exposure to such noise are greater than the effects of exposure to 'ordinary' traffic noise. With regard to noise with low-frequency components, low-frequency noise, tonal noise, and impulse noise, the Committee endorses the conclusions set out in the Health Council's 1997 report *Assessing Noise Exposure for Public Health Purposes*. Hence, adjustment factors are proposed for use in the regulation of noise with low-frequency components, tonal noise and impulse noise, but it has not been possible to define an *Lnight* adjustment factor for low-frequency noise. The values of the proposed factors are given in Annex F. Where noise from industrial activities is concerned, the Committee takes the view that research published since 1997 has demonstrated that no adjustment factors other than those referred to above are required.

It is not clear whether very loud sporadic noise events have any special implications for sleep. The Committee anticipates that the probability of such events having an *acute* effect (of whatever kind) is greater than the defined relationships might suggest, since the hearer will necessarily be unused to noise events of the kind involved, and anxiety is very likely to play a role. A single event of this kind can also have consequences for the hearer's quality of sleep for the rest of the night and on subsequent nights. However, the Committee does not have sufficient research data at its disposal to develop these assumptions more fully. In questionnaire-based studies of self-reported long-term effects (such as awakening, diminished sleep quality and night-time noise-related annoyance), a one-year assessment period is typically used. The Committee is not aware of any research that has looked at the specific effects that noise events with relatively very high *SEL* or *LAmax* values have on such self-reported parameters. The Committee cannot therefore make any scientifically justified statement about such effects.

5.7 Protection measures

Question 6: Could the public be protected by the use of a. performance-related or design requirements for residential buildings, b. personal protective gear, c. rules regarding sound pressure levels outside buildings, d. rules relating to vehicles and machinery, or e. a combination of these measures?

The Committee notes that the State Secretary does not mention publicity and dialogue as means of achieving protection. The Committee has nevertheless included publicity and dialogue in the response below, along with the measures that are referred to in the question. In its response, the Committee adheres to the standard strategy used in environmental management and occupational health and safety. This strategy involves first seeking to address a problem at source (which may entail reducing the number of sources), then exploring ways of intervening in the transfer from source to 'recipient', and considering recipient-oriented measures only as a final resort.

5.7.1 *Source-oriented measures*

The regulation of noise emissions from transport and industrial sources is a matter that has received increasing international attention. The ICAO* convention, for example, makes various provisions regarding noise production by aircraft[123,124]. Newer aircraft that meet the requirements of Chapter 3 are significantly quieter than those that merely comply with Chapter 2**. Measures designed to reduce noise from cars and aircraft can sometimes be undesirable in the context of reducing exhaust-related atmospheric pollution[38]. Furthermore, it is not sufficient to merely impose design requirements on vehicles and other machinery: maintenance and monitoring are also necessary in order to ensure that noise emissions are kept down in practice (buses are liable to become noisy with age, for example, while mopeds and scooters are sometimes 'hotted up' by their owners). In some cases, much more is technically possible than the regulations require, and social preferences (such as 4-wheel drive vehicles and wide tyres) often negate the 'gains' achievable through technological advancement.

5.7.2 *Intervention in the transfer from source to recipient*

Possible ways of controlling the transfer of noise from source to sleeper come under a number of headings: town planning measures (orientation of buildings and bedrooms, separation distances between noise sources and dwellings), acoustic screens and embankments, covers (tunnels) and domestic acoustic insulation. The Committee has restricted its detailed response to consideration of the last option.

An overview of published research into the effectiveness of domestic acoustic insulation as a means of controlling the influence of night-time noise is presented in section 4.3. Considering the large sums spent on fitting extra acoustic insulation to homes, the Committee finds it surprising that so little research has been done into the

* ICAO stands for International Civil Aviation Organization.

** Chapters 2 and 3 of Annex 16, Volume I of the ICAO convention.

effectiveness and efficiency of such modifications. As things stand, it is not possible to say more than that fitting acoustic insulation reduces sleep disturbance by night-time noise. It is clear that if steps are not also taken to enable householders to keep their bedrooms cool in hot weather, the benefit of acoustically efficient glazing is liable to be offset in the summer by people opening their windows.

Inventory research has revealed that many Dutch people are bothered by noise from their neighbours. The Committee regards this as indicative of shortcomings in the existing standards of inter-dwelling acoustic insulation. Since people are less tolerant of the noise their neighbours make at night-time than of their neighbours' evening or daytime noise, it may be assumed that much of the annoyance associated with noise from neighbours relates to the influence of such noise on sleep.

5.7.3 *Recipient-oriented measures*

It is possible for people to protect themselves against the effects of night-time noise by inserting ear plugs* of various kinds (plastic foam, moulded plugs, preformed and presized plugs and mouldable plugs) into the auditory duct. Properly fitted, ear plugs can reduce lower-frequency traffic noises by 15 dB(A) or more. Some types of plug are soft and therefore not at all uncomfortable to use while sleeping.

Personal hearing protection can provide a solution only in specific cases. The Committee does not consider hearing protection appropriate for the general prevention of noise-related problems in the population at large. Not only would it be impossible to make sure that people actually used their ear plugs in the privacy of their own homes, but wearers would in many cases be unable or less readily able to hear important sounds, such their partners, children, alarm clocks, intruders or sirens.

5.7.4 *Publicity and dialogue*

Where environmental factors that have a demonstrable adverse effect on the quality of the human environment are concerned, it is certainly the case that publicity and dialogue are necessary to ensure that effective and efficient action is taken to keep such effects within acceptable bounds. Publicity involves the unilateral provision of information to the private citizen by the government or the party responsible for the environmental factor concerned. Dialogue is a bilateral communication process that often begins with listening to the private citizen[38,125,126].

In relation to the effects of noise on sleep, publicity and dialogue have two important aspects: the provision of information about the consequences of exposure to noise

* Headphone-style hearing protectors are not practical for use at night, and ordinary cotton wool offer no protection[123].

and the two-way exchange of information aimed at the reconciliation of scientific data with the experiences of the private citizen, as well as information about the advantages and disadvantages of source-oriented, transfer-oriented and recipient-oriented measures.

5.7.5 *The combination of various types of measures*

From what has been said in the preceding subsections, it will be clear that there is very little research data available on the effectiveness and efficiency of protection measures. It is not therefore possible to give evidence-based guidance on the form that any protection regime should take. Nevertheless, the Committee considers it inevitable that the control of noise-related problems will necessitate the combination of source-oriented, transfer-oriented and in some cases recipient-oriented measures. This is because measures of all types are difficult to realise, irrespective of how effective or efficient they may be. In practice, cost issues come into play as well ('Who pays?' and 'Who is best able to afford the cost?'), as do questions regarding the quality of the planning of the human environment. Also of relevance in this context is increasing mobility, which tends to negate the benefits of technological advancement to some extent.

Finally, the Committee wishes to highlight the fact that noise-related sleep disturbance is not an isolated issue. Night-time noise almost always occurs in tandem with daytime noise. Not only do some people sleep during the day (by choice or out of necessity), but also exposure to noise has health implications at any time. The environmental noise issue is part of the wider debate on the quality of the human environment. The quality of the human environment and its (positive and negative) influences on health and well-being are determined by numerous factors (see Chapter 2), some being characteristics of the physical environment and some being of a social or behavioural nature. However complicated it may be to do so, this wider context should be taken into account. This underlines once more the importance of dialogue.

5.8 Recommendations for further research

In his letter, the State Secretary did not enquire regarding problems relating to research into sleep, health and noise. While the Committee does not therefore see the definition of a research programme as part of its remit, it is felt appropriate that this advisory report should be concluded with a summary of the most important gaps in knowledge previously highlighted.

The *2002 Actieprogramma gezondheid en milieu, uitwerking van een beleidsversterking* (Environment and Health Action Programme, the Practical Reinforcement of Policy) concluded that, in the Netherlands as elsewhere, research into the relationship

between environment and health needed fresh impetus. The Action Programme identified a number of themes concerning which more scientific knowledge was required, and placed the themes in a general order of priority. In this context, the Health Council was asked to advise on an environment and health research programme[38]. In the resulting advisory report, the Council highlighted the main gaps in knowledge regarding the influence that environmental factors have on health, and made recommendations regarding the research and reporting activities necessary to close those gaps. One of the themes addressed was exposure to noise. The report concluded that, in terms of their health implications, the themes exterior atmosphere, noise and indoor environment were of particular importance. The gaps identified in knowledge regarding the consequences of exposure to night-time noise were the effect that the level of insulation and the position of a person's bedroom have on the relationship between night-time noise exposure and consequences for health and well-being, the effectiveness of acoustic insulation on noise exposure and sleep disturbance, and the relationship between night-time road traffic noise exposure and effects on sleep and health. The present Committee feels it appropriate to elaborate on the research requirements referred to in the earlier report by highlighting the need for the following:

- Research into the long-term consequences of exposure for health and well-being, distinguishing between the effects associated with the noise exposure when sleeping, and those associated with the noise exposure during the daytime and evening. Most studies into effects such as hypertension, ischemic cardiovascular disease in adults and reduced cognitive performance by children have concentrated on relationships with daytime (and evening) noise exposure. However, recent research suggests that night-time noise and its effects on sleep and when sleeping play a much more significant role[82,95,128]. Knowledge regarding such matters is particularly important for the formulation of intervention policies.
- Research into the effects of night-time noise on children. Almost nothing is known about this subject. In the near future (summer 2004), the findings of the European research project *Road traffic and Aircraft Noise exposure and children's Cognition and Health* (RANCH) are to be published. RANCH is a field study looking at the relationship between, on the one hand, exposure to road and air traffic noise in the domestic environment and at school and, on the other, cognitive performance, blood pressure, general health, annoyance and sleep disturbance. It is not designed to shed light on the biological consequences in children of exposure to noise when sleeping. It is, however, expected to yield information about children's self-reported responses to night-time exposure to noise.
- Questionnaire-based or field research into insomnia caused by exposure to night-time noise, making use of clinical concepts. Such research would serve to bring together medical and environmental health expertise relating to insomnia.

- Research into the efficiency and effectiveness of acoustic insulation between dwellings and on or in exterior walls. Also of importance in this context is the position of the bedroom relative to the noise source and the influence of people's behaviour on the efficiency and effectiveness of insulation.
- Research into the effects of neighbourhood noise and noise from neighbours. Such research should be placed within the wider setting of research into the quality of the human environment.

Where the initiation of research is concerned, it is desirable to seek international alignment, as recommended by the Health Council in its report *Environmental Health: Research for Policy.*

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Annexes

Annex **A**

The State Secretary's letter

The President of the Health Council received the following request from the State Secretary for Housing, Spatial Planning and the Environment in a letter dated 3 February 2003, reference no. LMV 2003003076.

I am writing to ask the Health Council to prepare an advisory report on exposure to night-time noise. The background to this request is outlined below. Following the outline, you will find a list of the specific questions that I would like the Council's advisory report to address.

Background

In several earlier reports, the Council has directly or indirectly addressed the issue of night-time noise. To a certain extent, therefore, this request is prompted by the possibility that new information may have become available, which can confirm or shed new light on advice given in the past.

In its first report on this topic in 1972, the Council unequivocally stated that 'Sufficient undisturbed sleep is extremely important to health.' In 1991, the Council turned its focus specifically to the question of aircraft noise in a report that was prompted by the heated debate then in progress concerning the proposed expansion of Maastricht Airport (*Airplane noise and sleep. Sleep disturbance by airplane noise at night*). The conclusion of the latter report was that 'Although not all the results lend themselves to clear interpretation, the indications are that the regular disturbance of sleep by noise has an adverse effect on health and well-being.'

The very thorough advisory report *Noise and Health* (1994) stated that there was sufficient evidence to attribute a number of phenomena to exposure to noise when sleeping. The phenomena in question were

changes in heart rate, changes in sleeping pattern, awakening, sleep stage changes and changes in the subjective quality of sleep. In relation to each of these effects, 'observed effects levels' – exposure levels at and above which effects were demonstrable – were calculated from the published data. Where other phenomena were concerned, the evidence for a causal relationship was less convincing, or an observation threshold could not be calculated.

The advisory report *Assessing Noise Exposure for Public Health Purposes* (1997) recommended that the index *LAeq* (covering an eight-hour overnight period) should be used when assessing (the seriousness of) night-time exposure to noise. However, the exposure-response relationships presented in the report for sleep disturbance and awakening attributable to traffic noise and noise from stationary sources were qualified as 'provisional'.

Finally, the advisory report entitled *Public Health Impact of Large Airports* (1999) devoted considerable attention to the question of sleep disturbance. Although on the basis of recent research the Council described the evidence for a causal relationship between exposure to night-time noise and changes in stress hormone levels as limited, it was felt that there was sufficient reason to view sleep disturbance as a 'moderately serious' effect on health, similar to increasing respiratory illness. It was estimated that 'a considerable proportion of exposed individuals' were affected.

Recent developments

This request leads on directly or indirectly from a number of recent developments.

- The European Directive 2002/49 relating to the assessment and management of environmental noise (2002)* has been published, defining a separate index of night-time exposure to noise: the *LAeq* for an eight-hour period (*Lnight*). This index should at least be used for the compulsory strategic noise maps.
- The European Commission has completed a study of dose-effect relationships for the *Lnight*.
- The question of how best to quantify night-time exposure has also become topical in the context of the modernisation of the noise regulation policy tool set.
- In November 2002, the results of the field study of aviation noise-related sleep disturbance in the vicinity of Schiphol Airport** was published; a number of relevant studies have been reported by researchers in other countries***,****.

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* Position paper on dose response relationships between transportation noise and annoyance, Luxembourg: Office for Official Publications of the European Communities, 2002, ISBN 92-894-3894-0, European Communities, 2002

** Sleep disturbance by aviation noise, TNO/RIVM, 2002

*** Epidemiological research on stress caused by road traffic noise and its effects on health 1 - Results for hypertension, Maschke, UBA, 2002

**** Nachffluglarmwirkungen, Forschungsbericht 26, DLR, Grezner, 2001

Specific questions

In view of the matters outlined above, I would like the Council to respond to the following questions in its advisory report:

1 What are the effects (expressed in quantitative terms as far as possible) of exposure to noise when sleeping?
2 How do such effects compare with other effects on health, in terms of seriousness and magnitude?
3 Is it necessary to take special account of any population groups that are at particular risk?
4 In view of the effects referred to, would it be advisable to introduce special rules, similar to those contained in Directive 2002/49 and the Aviation Act, for night-time noise from sources other than air traffic?
5 If so, is it sufficient for such rules to be based on *Lnight*, or are additional indexes of exposure required, with a view to regulating impulse-like noises and situations involving relatively infrequent but high-intensity noise events.
6 Could the public be protected by the use of a. performance-related or design requirements for residential buildings, b. personal protective gear, c. rules regarding sound pressure levels outside buildings, d. rules relating to vehicles and machinery, or e. a combination of these measures?

Timetable

I would be very grateful if the Council could present its advisory report in autumn 2003 or thereabouts.

Yours sincerely,

(signed)
PLBA van Geel,
State Secretary for Housing, Spatial Planning and the Environment

Annex **B**

The Committee

- Professor JJ Heimans, *Chairman*
  Department of Neurology, VU University Medical Center, Amsterdam
- M van den Berg, *consultant*
  Ministry of Housing, Spatial Planning and the Environment, Directorate General for Environmental Management, The Hague
- Dr JJ van Busschbach
  Erasmus Medical Centre, Institute for Medical Psychology and Psychotherapy, Rotterdam
- JH Granneman
  Peutz bv, Zoetermeer
- Dr HME Miedema
  TNO Inro, Department of Environment and Health, Delft
- Professor FJN Nijhuis
  Occupational Perspective Centre, Hoensbroek
- Professor WF Passchier, *consultant*
  Health Council, The Hague
- Dr H Tiemeier
  Erasmus Medical Centre, Institute for Epidemiology and Biostatistics, Rotterdam
- Professor AJJM Vingerhoets
  University of Tilburg, Psychology and Health, Tilburg

- Dr AW de Weerd
  Haaglanden Medical Centre, Westeinde Hospital Site, Centre for Sleep and Waking Disorders, The Hague
- W Passchier-Vermeer, *Secretary*
  TNO Inro, Department of Environment and Health, Delft, and Health Council, The Hague

Administrative support: M Bakker, Health Council, The Hague.
Layout: M Javarmardi/J van Kan, Health Council, The Hague.

Annex **C**

Individuals and bodies who responded to the request for information

A letter was sent to more than fifty bodies with an interest in the subject matter concerning which the State Secretary had asked for advice. In addition, an advertisement was placed in the Government Gazette of 22 July 2003, inviting interested parties to submit any information that might be of value in the compilation of the advisory report.

Written responses were received from the following individuals and bodies:

- Greater Rotterdam Regional Health Service, General Healthcare Sector, Environment & Hygiene Department, R van Doorn
- Achterhoek Regional Health Service, CH Capel
- JJM Veraart, in a private capacity
- Kop van Noord-Holland Regional Health Service, JE de Leeuw den Bouter
- Noord-Kennemerland Regional Health Service, J Paulisse, enclosing a report entitled *Geluidhinder en slaapverstoring in Noord-Kennemerland* (*Noise-Related Annoyance and Sleep Disturbance in Noord-Kennemerland*); OMNIBUSONDERZOEK 2000
- Amsterdam Airport Schiphol, Business Unit Airlines, M Bouwmeester, enclosing a final draft report entitled *Non-auditory Health Effects of Aircraft Noise With Special Reference to Sleep Disturbance.*

E-mail responses were received from the following bodies:

- Northern South Holland Regional Health Service, M Mooij
- IPO BOAG, J Witteman

- Groningen Municipal Health Service, M Denekamp
- DCMR Rijnmond Environmental Service, Noise Bureau, RG de Jong
- ANWB, Department for Members' General Interests, P Clausing, enclosing a report entitled *Geluidbelasting in het Centraal Veluws gebied* (*Noise Exposure in the Central Veluwe Area*)

Annex **D**

Research into the consequences of night-time exposure to environmental noise when sleeping

D.1 Introduction

This annex contains a more in-depth review of studies that have been conducted into the effects of night-time noise. The annex's division into sections reflects the structure of the main body of the report. Thus, the annex deals in turn with research into the acute biological effects of exposure to noise when sleeping, research into biological effects over the course of a night (before, while and after sleeping) and research into the consequences for health and well-being of chronic exposure to night-time noise.

Tables 12 to 14 list the effect parameters, the technique used to measure them and a selection of references to research reports. Where biological effects are concerned, distinction is made in the references between field research and laboratory research.

Table 12 Acute biological effect parameters, the technique used to measure them and a selection of references to field and laboratory research.

Variable	Measurement technique	Field research references	Laboratory research references (selection)
Probability of acute cardiovascular changes	ECG, plethysmography	58,129	64-67
Probability of acute changes in stress hormone concentrations in the blood	Immediate blood sampling		
Probability of sleep stage change, from deeper to less deep sleep, including EEG awakening	Polygraphy (EEG, EMG, EOG)	48,130-132	48
Probability of motility (onset)	Actimetry	12,50,51,77-79,96,133	
Probability of subject-registered awakening	Pressing a button	12,49,50,52,134,135	

Table 13 Biological effect parameters relating to the course of a night (before, while and after sleeping), the technique used to measure them and a selection of references to field and laboratory research.

Variable	Measurement technique	Field research references	Laboratory research references (selection)
Prolongation of the sleep inception period, difficulty getting to sleep	Polygraphy (EEG, EMG, EOG), actimetry, journal	12,75	136,137
Changes in cardiovascular activity	ECG, plethysmography	70,131,132,138-140	141-146
Change in average motility during the sleep period	Actimetry	12,66,75,79	
Changes in the duration of the various stages of sleep, in sleep structure, fragmentation of sleep	Polygraphy (EEG, EMG, EOG), actimetry	59,70-73	136,137,140,141
Changes in (stress) hormone concentrations	Blood, saliva and urine sampling	82,82,140,147-154 153, 155-163	
Changes in immunological parameters			84-87
Recalled frequency of awakening and premature awakening by noise	Journal and actimetry	12,51,66,75,96	74,129,164-166,75,76,167, 47,150
Self-reported sleep quality, self-reported sleep disturbance	Journal	12,17,147,168,169	
Drowsiness/tiredness during the day and evening	Test, journal	12,17,147	
Cognitive performance	Test	12,17,147,70	
Irritability	Test, journal	17,147	
Annoyance	Journal	12,17,147	

Table 14 Parameters studied in field and questionnaire-based research into the influence of chronic exposure to night-time noise on health and well-being.

Variable	Measurement technique	Field research references
Sleep quality: reduced perceived sleep quality, difficulty getting to sleep, difficulty staying asleep, sleep fragmentation, reduced sleeping time, increased motility when sleeping	Questionnaire, journals, actimetry	12,12,17,51,66,75,79,80,96,96,147, 168,169
Well-being: self-reported sleep disturbance, self-reported health problems, use of somnifacient drugs and sedatives, daytime irritability	Questionnaire, test	12,51,66,75,97,168,169
Social contacts and concentration: impaired social contacts, impaired cognitive performance	Questionnaire, test	12,17,66,70,147
Medical conditions: insomnia, other investigated illnesses and medical conditions	Medical examination, questionnaire	12,89,170

D.2 Acute biological effects

D.2.1 Autonomous cardiovascular responses to noise events

Acute cardiovascular responses include raised (systolic) blood pressure, constriction of the blood vessels in the limbs and elsewhere, and accelerated heart rate. In this review, the Committee has restricted itself to acute heart rate accelerations in response to noise. The Committee is aware of only two field studies, both relating to road traffic noise[58,129]. Laboratory research has been taken into consideration because it sheds light on:

- Possible differences between the effects of exposure to road traffic noise and the effects of exposure to noise from other sources
- Possible differences between the effects of exposure when sleeping and exposure during the day
- Personal characteristics that influence the effects

Field research

The Dutch researchers Hofman *et al* carried out a field study with twelve subjects who lived beside a motorway[58]. They studied each subject in two situations, each for ten nights. The two situations differed in terms of the acoustic insulation provided by the fabric of the building, relating to the presence of double glazing, which provided an average attenuation of 9 dB(A). On each of the twenty nights that each subject was monitored, an EEG, two EOGs and an ECG were made and respiration was monitored. The

noise situation was described by the researchers as a gradually varying background level with superimposed noise peaks (when particularly noisy vehicles passed). A noise peak was defined as a noise event with an *LAmax_i* of at least 10 dB(A) above the prevailing background level over a ten-minute interval (*L90*). In each of the two study situations, there were approximately ninety-three noise peaks per night. *LAmax_i* was generally between 30 and 65 dB(A). The variation in ECG-determined heart rate over time was compared with the distribution of noise peaks over time. For each noise peak, the maximum change in the heart rate (ECR: Event-related Cardiac Response) was determined from eight heart rate figures (four before and four after occurrence of the noise peak). For the purposes of comparison, a 'pseudo-ECR' was calculated for a peak-free interval immediately prior to the noise peak. In 80 per cent of cases, the ECR was greater than the pseudo-ECR. Analysis revealed that the ECR was not dependent on *LAmax_i*, on the subject's sleep stage at the time of the noise peak, or on whether the bedroom had double glazing. However, the speed at which the noise increased in intensity did influence the ECR: faster rises in intensity were associated with higher ECRs. Figure 15 shows the results relating to noise peaks occurring while the subject was in sleep stage 3 or 4 (SWS). If it is assumed that, of the 80 per cent of ECRs that exceeded the associated pseudo-ECR, 20 per cent were higher purely by chance, just as 20 per cent of all ECRs were lower purely by chance, it follows that subjects' heart rates rose in response to 60 per cent of noise peaks, irrespective of sleep stage or *LAmax_i* value.

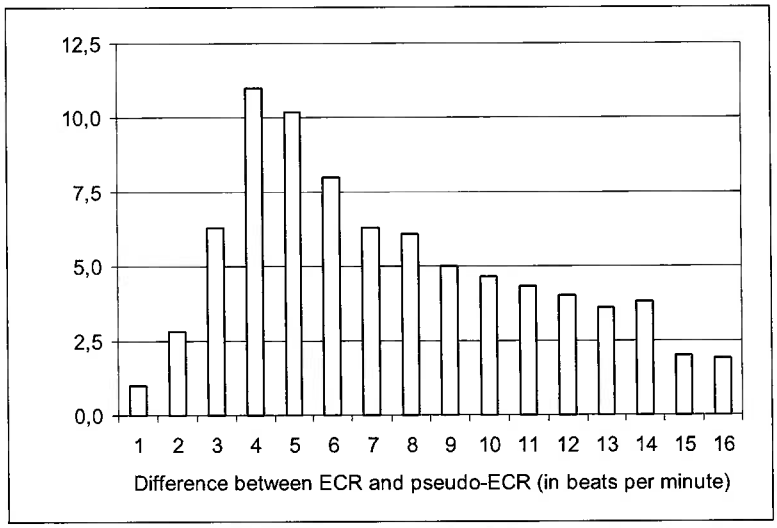

Figure 15 Percentage of cases in which the ECR (ECR: Event-related Cardiac Response) was higher than the pseudo-ECR, as a function of heart rate change involved in the ECR and pseudo-ECR. The various columns add up to a total of 80 per cent; in 20 per cent of cases, the difference between the ECR and pseudo-ECR was zero or negative.

A team led by Carter, a leading researcher in the field of the effects of noise on sleep, studied the effect of road traffic noise on seven older men, four of whom suffered from slight arrhythmia (simple premature ventricular contractions)[129]. They observed that in two of the four men with arrhythmia, noise peaks (*LAmax_i* of more than 70 dB(A) associated with lorries) induced a premature contraction 20 to 40 seconds later, especially if the men were in sleep stage 4 at the time. However, the researchers were unable to replicate this effect in a laboratory study involving road traffic noise[151]. In this context, Carter recounted an incident in which the sound of an alarm clock consistently induced ventricular fibrillation in a patient with a heart condition[171]. Carter argued that it was important that more research was done into the effects of noise on people with heart problems, since he anticipated that they were likely to be more than averagely sensitive to noise.

Laboratory research

Öhrström *et al* studied the acute effects of road traffic noise on heart rate in twenty-four subjects. Fifty-seven times a night for nine nights, subjects were exposed to the noise of a passing car or lorry with an *LAmax_i* of between 58 and 60 dB(A)[66]. The average increase in heart rate during the noise events was 1.5 beats per minute; among subjects who considered themselves sensitive to noise, the average increase was 1.8 beats per minute, while among subjects with no such self-perception, the increase was 1.1 beats per minute.

A French research team led by Muzet carried out a study in which twenty subjects were monitored for three nights, on one of which they were exposed to aircraft, lorry, moped and train noises[64]. The *LAmax_i* and noise event duration values were, for aircraft, 71 dB(A) and 21 seconds; for lorries, 66 dB(A) and 20 seconds; for mopeds, 56 dB(A) and 10 seconds; and, for trains, 62 dB(A) and 17 seconds. Over the course of the night, the noises were introduced randomly eight times per hour. In addition, subjects were exposed to similar noises of 15 dB(A) louder during the day. The results are presented in Figure 16. The increase in heart rate was not calculated in the same way as in the other publications referred to in this annex. Di Nisi *et al* worked on the basis of the difference between the highest heart rate and the slowest subsequent heart rate during a noise event (the latter rate generally being much lower than the average rate over an interval before or after the noise event).

The conclusion drawn by the researchers, which is illustrated by Figure 16, was that the response at night was much greater than that during the day. Furthermore, the daytime effects barely differed from one source to another. In addition to monitoring heart rate, the French team also used a finger plethysmograph to measure blood flow through subjects' finger tips. The plethysmography data also indicated that the most common

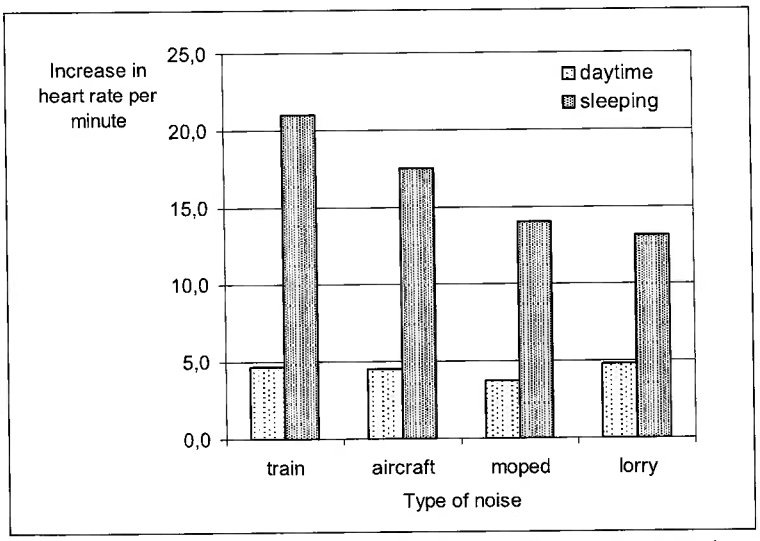

Figure 16 Comparison of the increase in heart rate during exposure to a noise event during the daytime and when sleeping. The daytime noises were 15 dB(A) louder than the noises subjects were exposed to when sleeping. From laboratory research by a French team[64].

response to the noise events, namely vasoconstriction, was also much more pronounced when sleeping than during the day. The subjects' score on a noise sensitivity scale did not appear to influence the magnitude of the heart rate response either during the day or at night, but was related to the degree of vasoconstriction during the day.

According to the data presented in Figure 16, aviation noise and lorry noise had a broadly similar effect on the sleeping heart rate. Because the train noise quickly rose to a maximum and remained at this level for almost the entire time until the train had passed, the *SEL_i* of the train noise was probably about the same as that of the aircraft and lorry noises. It is therefore plausible that, at a given *SEL_i*, the effect of train noise characterised by a rapid initial increase in intensity is slightly greater than the effect of noise from a passing aircraft or lorry. Comparing the data for the different sources, it is striking that mopeds – with a maximal level 10 to 15 dB(A) lower than the maximums of the other noise sources, and with the shortest duration – register quite high scores. The researchers did not investigate whether this was due, for example, to the faster rise in the intensity of moped noise or to aversion to the noise on the part of the subjects.

In view of the much greater heart rate responses observed in subjects exposed to noise when sleeping than in the same subjects during the day, the researchers suggested that more attention should be paid to protecting the general public against higher night-time noise exposures.

An Australian research team exposed nine subjects over three sleep periods to various types of noise: low-flying military jets, lorries, landing civil aircraft, and a five-sec-

ond 1000-Hz sound[67]. Each type of noise event was generated at *LAmax_i* values of 55, 65 and 75 dB(A). The time taken to reach maximum intensity (build-up period) and the overall duration of the noise event varied with the *LAmax_i* value; the values are given in Table 15.

Table 15 Details of the noise events featured in the research by Carter *et al*[67].

Noise event	Build-up period (seconds)			Overall duration (seconds)		
LAmax_i (dB(A))	55	65	75	55	65	75
Low-flying military jet	1	1	1	1	2	6
Civil aircraft	13	14	16	8	18	25
Lorry	10	19	20	10	20	27
1000-Hz sound	0	0	0	5	5	5

Subjects were exposed to a total of approximately 1300 noise events. The increase in heart rate during the noise events did not appear to be related to the type of noise involved. At *LAmax_i* values of 55 and 65 dB(A), the increase in heart rate averaged 1.5 beats per minute, while at 75 dB(A) it was approximately three beats per minute. Expressed in the form of *SEL_i* values, the civil aircraft and lorry noise events were very similar at each of the three exposure levels. At the lowest exposure, the *SEL_i* value of the 1000 Hz sound was approximately the same as those of the lorry and civil aircraft noises, while at the higher exposures it was roughly 3 to 5 dB(A) lower. Because of its short duration, the military jet noise had an *SEL_i* value approximately 10 dB(A) lower than those of the lorry and civil aircraft noises at the two lower exposures, and about 5 dB(A) lower at the highest exposure. It follows that, at a given *SEL_i*, the increase in heart rate induced by the 1000 Hz sound and the military jet noise is greater than that induced by the lorry and civil aircraft noises.

In Germany, Griefahn carried out an experiment in which twenty subjects were exposed in their sleep to a reproduction of the noise of shots from a tank, with an *LAmax_i* value of between 78 and 82 dB(A)[65]. A total of 1209 impulses were distributed over sixty-eight person-nights. On average, subjects' heart rates rose from 66 to 77 beats per minute, measured three seconds after the 'shot'. This increase by an average of eleven beats per minute was greater than the increase induced by road traffic noise under similar circumstances, albeit at lower *LAmax_i* values.

In 1967, Semczuk investigated the effects of exposure to noise when sleeping, by using thoraxgraphy to monitor breathing in a study group of fifty children (five to seven years old) and a hundred adults[68]. The trigger level for respiratory changes associated with an aural stimulus (sound of a particular pitch) was 10 to 15 dB(A) lower in children than in adults. The researcher accordingly concluded that a child's autonomous nervous

system is more readily activated by noise when sleeping than an adult's, and that children are therefore physiologically more sensitive to night-time noise than adults.

D.2.2 *Acute changes in hormone levels*

The lower four graphs in Figure 17 show how the concentrations in the blood of the stress hormones cortisol, adrenaline and noradrenaline and of the growth hormone (GH)[88] normally change in the course of a night.

The Committee is not aware of any field or laboratory research into acute changes in hormone concentrations in response to exposure to noise.

D.2.3 *Sleep stage change, including EEG awakening*

Reference has already been made in the main body of the report to the meta-analysis performed by Pearsons *et al*[48]; see Figure 7. The relationships between exposure and the probability of EEG awakening and the probability of sleep stage change were presented by Pearsons, using both *LAmax_i* and *SEL_i* as indexes of exposure. Only three of the five field studies reviewed by Pearsons involved EEG scans[130-132]; one other was a questionnaire-based study[66] and another involved monitoring movements of the bed in which subjects slept[172]. The latter two studies entailed very few observations, so their results had only a marginal influence on the outcome of Pearsons' meta-analysis. The relationships defined from the field study data were based on a total of 213 subject-nights with EEG scans and 2770 noise events. The noise sources in the three studies were civil air traffic and rail traffic.

The Committee traced only three reported studies that had looked at the effect of night-time noise on children's sleep EEG[55-57]. Lukas exposed twenty-two people, six of them children (five to seven years old) to aviation noise and sonic booms once they had entered stage 3 or 4, as registered on an EEG. He observed that the children's EEGs showed less response to noise while in deep sleep than the adults' EEGs. Eberhardt[56] reported the effects of exposure to road traffic noise on thirteen children. Eight of the children lived on a quiet street, but on several nights during the study period were exposed to recorded lorry noise (sixty-eight times per night); the other five children slept beside a busy road. EEG awakening occurred in the first group of eight children in response to 0.2, 0.8 and 2.1 per cent of noises with *LAmax_i* values of, respectively, 45, 55 and 65 dB(A). The only other statistically significant difference revealed by the EEG analysis was a six-minute increase in the time spent in a waking state (W) on the nights with the higher noise exposures. The children also reported that, on the nights when they were exposed to road traffic noise, they found it harder to go to sleep, found that they woke up more often, recalled being awoken more often by road traffic noise, felt less

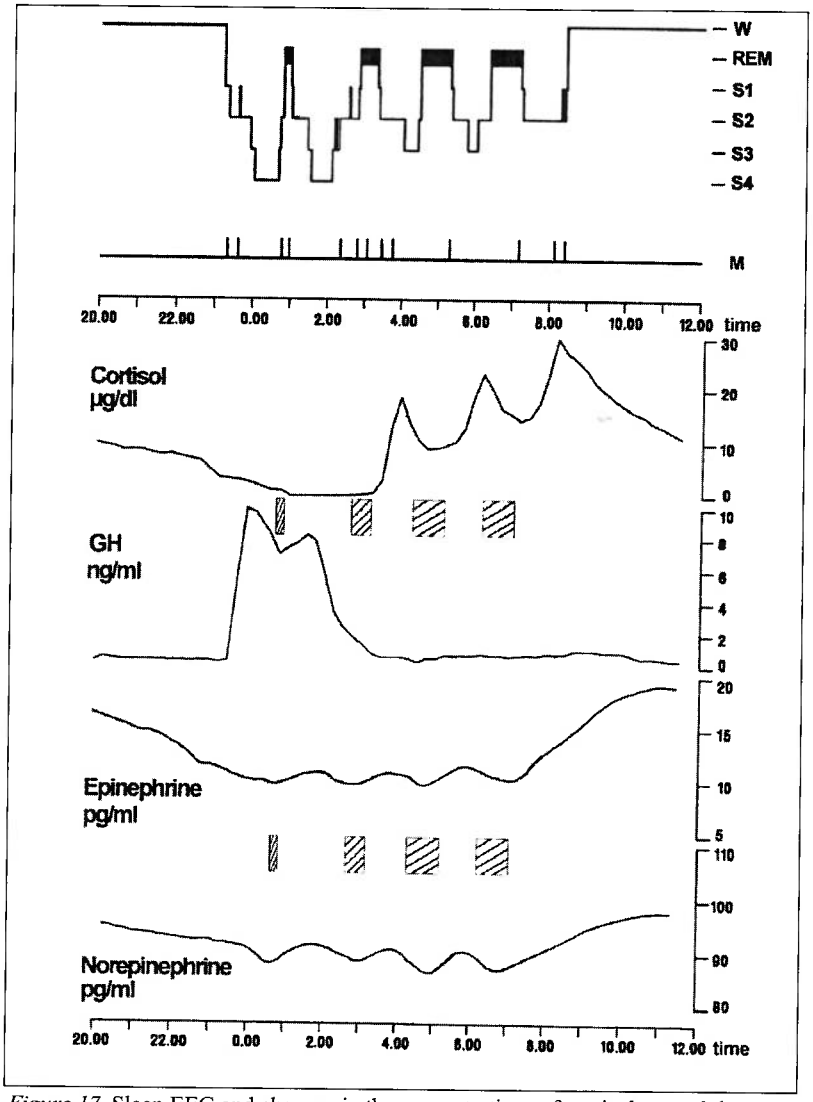

Figure 17 Sleep EEG and changes in the concentrations of cortisol, growth hormone (GH), epinephrine (adrenaline), and norepinephrine (noradrenaline) in the blood, as a function of the time from 8pm to 12 noon; typical patterns for healthy young adults[88].

well rested the following day and perceived the quality of their sleep to have been diminished.

Double glazing was fitted to the bedroom windows of the second group of just five children, thus attenuating the noise by an average of 10 dB(A). The only statistically significant effect of this intervention observed in the very small study group was a seven-minute reduction in sleep inception period. Eberhardt concluded that children exhibited

less strong responses to noise when sleeping than adults; he estimated that a noise needed to be about 10 dB(A) louder to induce a given EEG response in a child than was necessary to induce the same response in an adult.

In a laboratory study, Busby exposed twenty-four boys (eight to eleven years old) to three-second bursts of sound of successively increased volume (each sound being 2 to 5 dB(A) louder than the last), until EEG awakening occurred. It was possible to reproduce the sound at up to approximately 95 dB(A) above the perception threshold. The percentages of EEG awakenings from SWS, stage 2 and REM sleep were, respectively, 4.5, 34 and 50 per cent. When the night was divided into three phases, the percentages of awakenings and arousals in the first phase (characterised mainly by SWS) were 12 and 14 per cent, respectively; the corresponding figures in the second phase of the night were 30 and 20 per cent, respectively, and in the third phase (mainly REM sleep) 50 and 8 per cent, respectively. Comparing responses to noise in hyperactive children, hyperactive children on medication and non-hyperactive children, Busby observed no differences. From the findings, Busby concluded that, in the latter phase of the sleep period, children were very sensitive to noise, and that much more research was needed to build up a full picture of how children responded to noise when sleeping.

D.2.4 *Motility*

Over the last ten years, various large-scale field studies have been carried out, in which subjects wore actimeters when sleeping in order to record motility. In the USA, there have been two studies focusing on aviation noise[49,50,134], in Germany there has been one study concerned with road and rail traffic noise[60,77,78], in the UK there have been a further three studies on aviation noise[51,54,80,173], and finally one study into aviation noise has taken place in the Netherlands[12,13,174]. In several studies, noise events were linked over time with motility, as indicated by the actimeter data, in order to shed light on the acute motility responses. In four of the studies, it was thus possible to define the relationship between *LAmax_i* or *SEL_i* and acute motility during and attributable to aircraft noise events[12,49,51,134]. In the other studies, the researchers focused on average motility during the sleep period.

Figure 18 shows the increase in the probability of acute motility attributable to aviation noise in the fifteen-second interval with *LAmax_i* as deduced from the Dutch research. Acute motility was induced by the noise of a passing aeroplane from an

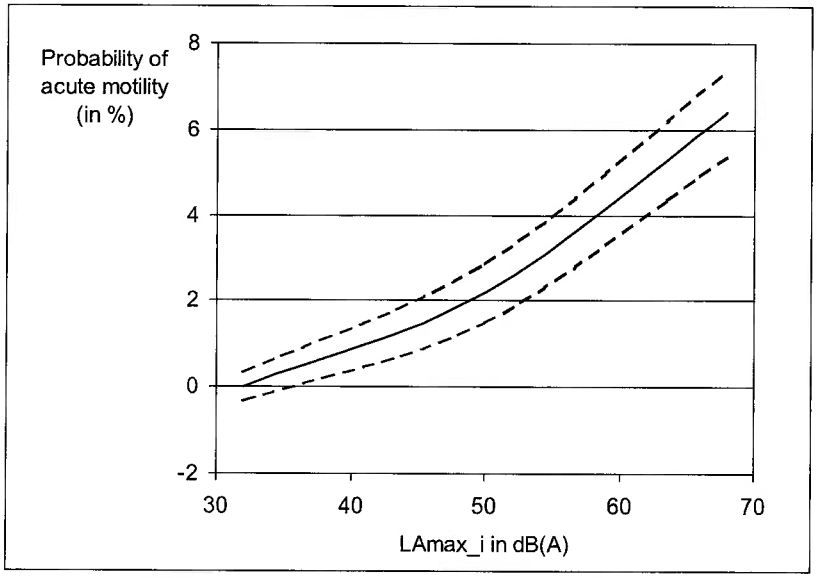

Figure 18 Probability (as a percentage) of acute motility being induced by an aircraft passage, as function a of the *LAmax_i* of the noise event, in the fifteen-second interval with *LAmax_i*. The figure also shows the so-called '95 per cent prediction-intervals'[12,13].

LAmax_i of 32 dB(A)*; an *LAmax_i* of 32 dB(A) is therefore the observation threshold for motility.

The curve shown in Figure 18 represents the average effect. The effect is strongly dependent on *Li*; as *Li* increases, so the probability of acute motility being induced by aviation noise decreases. In other words, people who are exposed to the sound of a passing aircraft numerous times while sleeping respond less to a single passage than people who are exposed to the sound only occasionally. The relationship between the probability of aviation noise-induced motility and exposure to aviation noise was not found to be gender-dependent and was barely age-dependent. The study findings also indicated that, at a given *LAmax_i* value, the type of aircraft manoeuvre (landing or taking off) did not affect the probability of aviation noise-induced motility. The researchers also asked subjects about their attitude to air traffic and to the expansion of Schiphol Airport. Attitude was found to have no influence on the probability of acute motility induced by aviation noise.

* In the Dutch study, *LAmax_i* was not measured in fast mode; rather *LAmax_i* was the maximum indoor equivalent sound pressure level measured over a one-second interval during an aircraft passage. Theory suggests that *LAmax_i* measured in fast mode should be 0.2 to 1 dB(A) higher than the maximum indoor equivalent sound pressure level measured over a one-second interval. The observation threshold for acute motility is therefore an *LAmax_i* in fast mode of 33 dB(A)

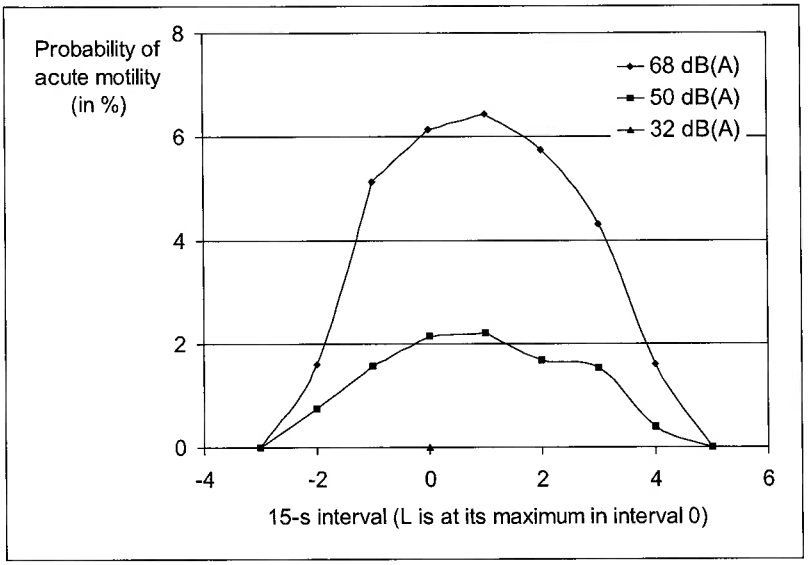

Figure 19 Probability of acute motility in three fifteen-second intervals before the interval in which the *LAmax_i* of an aircraft noise event occurred, in the interval of occurrence, and in five fifteen-second intervals after the interval of occurrence, for three flight passages with *LAmax_i* values of 68, 50, and 32 dB(A).

The Dutch researchers additionally calculated the probability of motility onset by aviation noise in the fifteen-second interval with *LAmax_i*. The observation threshold for motility onset also worked out at an *LAmax_i* for the aircraft passage of 32 dB(A).

Increased probability of acute aircraft noise-induced motility was sometimes observed both before and after the fifteen-second interval in which the *LAmax_i* occurred. This phenomenon is illustrated in Figure 19. Overall, the probability of motility being induced by aviation noise in any fifteen-second interval was more than four times that probability in the fifteen-second interval in which *LAmax_i* occurred.

The Dutch study's findings are consistent with those of the first US study, by Fidell *et al*[49]. This study focused exclusively on people exposed to (very) high night-time aircraft noise exposures. The researchers established that the observation threshold for motility was 45 dB(A). This figure is broadly in line with the 42 dB(A) calculated by the Dutch research team for subjects with an *Li* of 40 dB(A). In the second US study, which was much smaller-scale than the one just referred to, no statistically significant relationship could be demonstrated[134].

The relationship established in the British study between motility onset in a thirty-second interval and the *LAmax* of an aircraft passage differed considerably from the pattern illustrated in Figure 18, even allowing for the facts that the British researchers looked at the probability of motility in thirty-second intervals and that the relationship

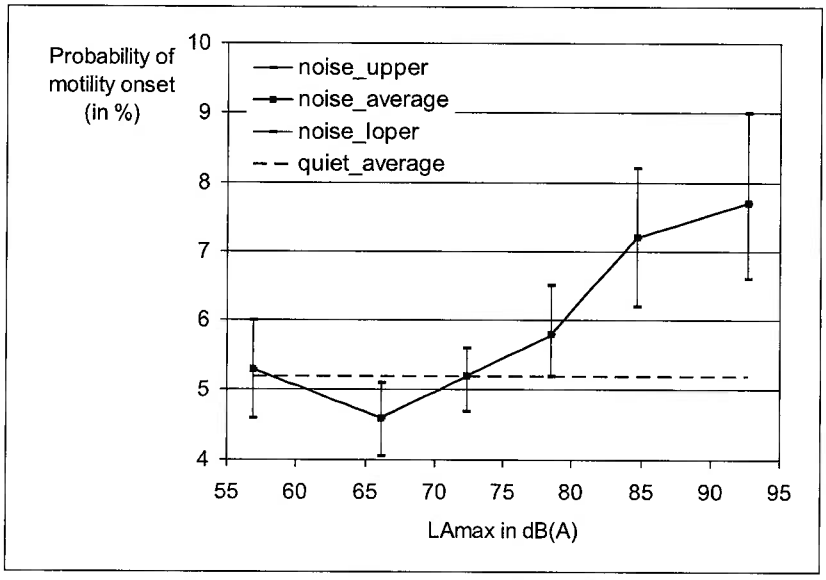

Figure 20 The relationship between the average probability of motility onset in the thirty-second interval in which *LAmax* occurs during an aircraft passage (noise_average) and *LAmax* of the passage, with the probability of motility onset outside these intervals also illustrated (quiet_average), based on research by Ollerhead[51]. The 95 per cent prediction interval (from noise_lower to noise_upper) is also given for each value.

between the probability of motility onset and the *LAmax* of an aircraft noise event is based on outdoor *LAmax* values. Various TNO reports have looked at this point[12,13,79,97]. Some of the findings of the British study are illustrated in Figure 20.

The British researchers came to the conclusion that the probability of motility onset by an aircraft passage began to increase from an *LAmax* of 82 dB(A)[51]. Even when this outdoor value is reduced by 25 dB(A) (the figure quoted by the researchers[54] as the difference between outdoor and indoor levels), the observation threshold works out at an *LAmax_i* of 57 dB(A). This is 25 dB(A) higher than the corresponding figure established by the Dutch team for the probability of aircraft noise-induced motility or motility onset. The Committee believes that the main reasons for the differences in the findings of the British and Dutch studies are as follows:

- In the British study, aviation noise levels were established on the basis of *outdoor* measurements only. The instruments used by the researchers were positioned so as to be directly exposed to the noise. The outdoor sound pressure levels on subjects' bedroom walls will in some cases have been (much) lower, particularly where there was screening in the form of objects such as other buildings and trees. The outdoor measurements therefore represent the upper values in a wide range of outdoor sound pressure levels on subjects' bedroom walls. The distribution of indoor values will

have been even greater, since the sound attenuating characteristics of each room will have differed, and the window aperture status will have varied from subject to subject and from night to night. In the Dutch research, where the average difference between the outdoor and indoor *LAmax* values of more than 63,000 aircraft passages during subjects' sleep periods was 21 dB(A), outdoor values as high as 82 dB(A) were sometimes associated with indoor values as low as 32 dB(A). These considerations suggest that, at a measured outdoor noise exposure of 82 dB(A), the actual exposure inside the subject's bedroom may well have been much lower. The Dutch study, by contrast, made use of measured indoor values.

- The British researchers considered whether there had been an aircraft passage only if their instruments registered a noise event with a sound pressure level of more than 60 dB(A) lasting for at least two seconds. If the timing of such a noise event coincided with the timing of a registered aircraft passage, the event was 'recognised' as an aircraft passage. Furthermore, any aircraft passage occurring within five minutes of the previous passage was excluded from the analyses. Then 'noise' was defined as any thirty-second interval in which the *LAmax* of a recognised aircraft passage occurred, and 'quiet' as all other thirty-second intervals. In other words, quiet included all intervals in which relatively quiet aircraft passages occurred, all intervals in which there were aircraft passages within five minutes of a previous passage, and all intervals during an aircraft passage before and after the interval of *LAmax* occurrence. The researchers then worked out the average probability of motility onset during 'quiet' periods (see Figure 20). However, this average will have been higher than a typical value for a genuinely quiet thirty-second interval, since all non-aircraft noises, all aviation noise outside the thirty-second intervals of *LAmax* occurrence and all 'quieter' and 'non-recognised' passages, plus the associated motility, were ignored. In consequence, the probability of motility onset *by aviation noise* (*noise – quiet*) was underestimated. In the Dutch study, an aircraft passage was included if its *LAmax* was 40 dB(A) or higher. Furthermore, distinction was made in the analyses between intervals characterised by the background sound pressure level only and intervals characterised by the presence of the background sound pressure level plus a non-aviation noise. Hence, the model takes account of the additional chance of motility or motility onset attributable to non-aviation noise, so that only the probability of extra motility caused by aviation noise is attributed to this source.
- The British study focused exclusively on *motility onset*, whereas monitoring *motility* would also have taken account of the duration of the effect. Furthermore, the Dutch researchers found that the probability of motility was more closely related to the *LAmax_i* (and *SEL_i*) than the probability of motility onset was.
- The British study looked only at *the thirty-second intervals of LAmax occurrence.* However, it was found in the Dutch study that, overall, motility onset was more

likely *in the intervals before and after the interval of LAmax occurrence* than in the interval of occurrence itself.

- The scope for performing calculations was more limited in 1992, with the result that not all the British team's data could be analysed. Hence, the relationship between the probability of motility onset and exposure to aviation noise was defined on the basis of data concerning the period 11.30pm to 5.30am only. However, it is apparent from both the Dutch study and the British study that aircraft passages became more likely to be associated with motility or motility onset as the subject's sleeping time progressed and the absolute time got later. Furthermore, limited calculation capacity obliged the British team to group aircraft passages into noise categories (see Figure 20). For each category, the average probability of motility onset was then calculated and a check made to establish whether there was a statistically significant difference between the calculated value and the average probability of motility onset during quiet. An analysis method involving the processing of all the data at once would undoubtedly have led to the definition of a much lower threshold value.

The original purpose of the German study was not to establish acute-level exposure-effect relationships, so the data from the study has recently been re-analysed with a view to defining such relationships for road and rail traffic noise[79]. Where rail traffic noise is concerned, the relationship has been defined between the probability of acute concurrent motility or motility onset and the *SEL* of a rail traffic noise event lasting up to two minutes. From these calculations, it appears that exposure to rail traffic noise does not increase the probability of acute motility or motility onset as much as exposure to aviation noise. After conversion of the German data to fifteen-second interval values, a 40 dB(A) *SEL* increase, from 60 to 100 dB(A), was estimated to be associated with roughly a 2.5 per cent increase in the probability of motility or motility onset. The corresponding figure for aviation noise is approximately 7 per cent.

In the German study, the exposure patterns for road traffic noise proved to be very different from the patterns for rail and aviation noise. Rail and aviation noise both involve distinct noise events, with an aircraft passage rarely lasting more than one minute and a train passage rarely longer than three minutes. (In the German study, 2,6 per cent of the nearly 69,000 train passages lasted longer than three minutes.) In total, nearly 17 per cent of thirty-second intervals included train noise. By contrast, nearly 53 per cent of thirty-second intervals involved road traffic noise in excess of the background level (i.e. three times the percentage for rail traffic noise).

In addition, a relationship has been established between acute motility during a thirty-second interval featuring road traffic noise and the equivalent sound pressure level during the interval[79]. The probability of acute motility during a given thirty-second interval featuring road traffic noise was found to rise to a small but statistically signifi-

cant extent as noise exposure increased: a rise in the equivalent sound pressure level over a thirty-second interval from 40 to 70 dB(A) was associated with a 0.3 per cent increase in the probability of motility. However, in the first thirty-second interval of a period featuring road traffic noise, the probability of acute motility was on average 3.4 per cent higher than in a given thirty-second interval without road traffic noise or in another thirty-second interval during a period featuring road traffic noise. In other words, the effect at the start of a period featuring road traffic noise is quite strong.

D.2.5 *Subject-registered awakening*

Over the last ten years, various meta-analyses have been performed on data from eight or nine field studies, with a view to establishing the relationship between the probability of noise-induced subject-registered awakening (awakening recorded by the subject during his/her sleep period, by pressing a button) and a noise index (*SEL_i*) for the event[52,134,135,175-179]. Two of the analyses – those by Finegold and Elias[135] in 2002 and Passchier-Vermeer[52] in 2003 – used the same database, previously assembled by Fidell[134]. Passchier-Vermeer additionally included in her analysis data from the Dutch study into sleep disturbance caused by aviation noise. In contrast to Finegold's meta-analysis, the secondary analysis performed by Passchier-Vermeer took account of the following:

- The type of noise source: civil aircraft, military jets, trains, other environmental noise events
- Differences in the probability of subject-registered awakening, EEG awakening and motility onset associated with a noise event of a given intensity (see Figure 8)
- The number of observations per subgroup (with subgroups formed on the basis of the *SEL_i* of the noise event, with the result thatthe number of observations per subgroup varied by a factor of 100)
- Differences in the time windows around a noise event within which the different researchers looked for evidence of awakening or arousal
- The probability of waking up in a period without noise events.

From the analyses, it was apparent that a statistically significant relationship was demonstrable between civil and military aircraft noise and subject-registered awakening, but not between rail traffic noise or 'other environmental noise events' and such awakening[52]. Since the relationship between the noise of military jets and subject-registered awakening is based purely on data relating to people living near to a single military air base, the findings should be verified by further research.

The database used by Passchier-Vermeer for her analyses included data on noise levels in the bedrooms of more than a thousand subjects in seven studies into the effects

of civil air traffic noise, involving more than 170,000 aircraft passages. In these seven field studies, the data on the aircraft passages was aggregated to seventy-eight points on the basis of *SEL_i*. This data was cross-referenced to the data on subject-registered awakenings to determine whether subjects recorded waking up within a five-minute window around an aircraft passage (from one minute before to four minutes after the *LAmax_i* of the passage).

The frequency of subject-registered awakening was also established for five-minute time windows in which no aircraft noise events occurred. The probability of *subject-registered awakening attributable to a noise event* was then calculated by subtracting the probability of *awakening in time windows without noise events* from the probability of *awakening in time windows with noise events.*

The average probability of subject-registered awakening in a five-minute interval without aviation noise was 1.73 per cent. Figure 21 shows the probability (as a percentage) of subject-registered awakening *attributable to aviation noise* during a five-minute interval, as well as the probability of *awakening for any reason during* a five-minute interval in which an aviation noise event occurred. The observation threshold for aviation noise-induced subject-registered awakening is an *SEL_i* of 54 dB(A). This is estimated to correspond with an *LAmax_i* (measured in fast mode) of 42 dB(A).

In principle, a noise with a very low sound pressure level can be audible in a very quiet environment. In young people, the binaural perception threshold (the threshold for the perception of a sound using both ears) is close to 0 dB(A). Although the threshold

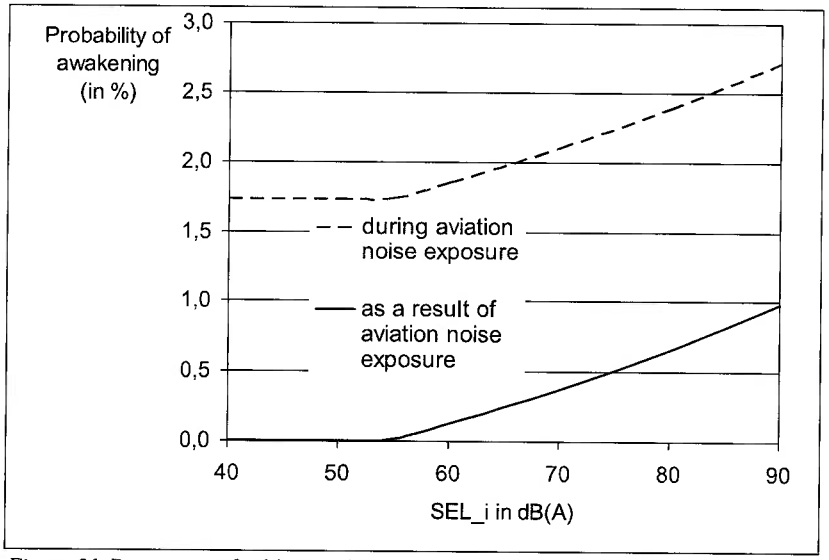

Figure 21 Percentage of subject-registered awakenings during a five-minute interval in which aviation noise occurs and percentage of subject-registered awakenings due to aviation noise as a function of indoor measured *SEL* (in dB(A))[52].

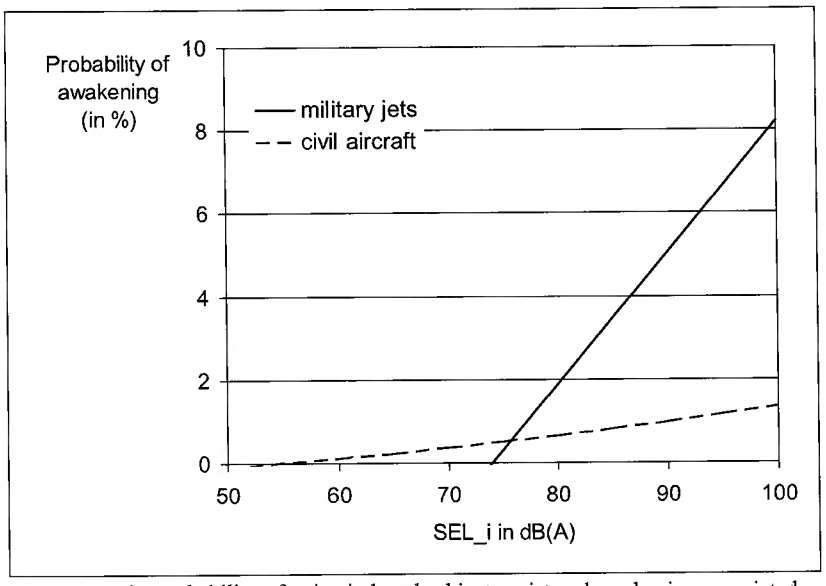

Figure 22 The probability of noise-induced subject-registered awakening associated with civil aircraft and military jets, as a function of indoor measured *SEL* (in dB(A))[52].

for the perception of higher frequencies (above about 2000 Hz) increases as a result of normal age-related loss of acuity, the ability to hear lower-frequency noises, such as aircraft noise, does not decline nearly as much. Not many people's bedrooms are so quiet that special sounds of close to 0 dB(A) can be heard, since such sounds are liable to be masked by normal background noise. Generally speaking, aviation noise can be heard in an otherwise quite bedroom when the level is more than about 15 to 20 dB(A). This implies that, when a person is awake, the noise of a distant aircraft can be heard if it is more than about 15 to 20 dB(A), provided that the bedroom is otherwise sufficiently quiet.

Figure 22 shows the probability of noise-induced subject-registered awakening associated with civil aircraft and military jets[52]. Although the data relating to military jets requires further verification, the Committee considers it appropriate to highlight the data, as it illustrates the effect of exposure to noise events characterised by a rapid initial rise in intensity.

D.2.6 *Acute annoyance and inconvenience*

The Committee is not aware of any field or laboratory research into acute annoyance or other acute problems due to night-time noise.

D.2.7 Summary

The results of the research into the acute biological effects of exposure to night-time noise described in this section can be summarised as follows:

Acute heart rate change

- In people who had been exposed to road traffic noise for years, heart rate accelerations occurred in response to road traffic noise peaks with *LAmax_i* values typically in excess of 30 dB(A). The observation threshold for heart rate acceleration is therefore likely to be below an *LAmax_i* of 30 dB(A).
- Noise is much more likely to induce heart rate acceleration at night than during the day.
- People with cardiovascular problems and people who consider themselves to be particularly sensitive to noise may well be more liable to experience noise-induced heart rate accelerations.
- The field research carried out by Hofman indicates that there is a cardiac response to roughly 60 per cent of motorway traffic noise peaks, irrespective of the hearer's sleep stage or the *LAmax_i* value of the noise peak (lorry).
- From laboratory research data, one can deduce that lorry passages and aircraft passages with similar *SEL_i* values have approximately the same effect on the heart rate.
- The results of the various laboratory studies referred to all indicate that, at a given *SEL_i*, a noise event characterised by a (very) rapid initial rise in intensity is more likely to affect the heart rate than a noise event that rises in intensity more gradually at the beginning. It is not possible to quantify this effect, however.
- Data from the only study involving children (five to seven years old) suggests that children's physiological responses to noise events during sleep are indicative of a 10 dB(A) higher sensitivity to noise.

Acute changes in hormone levels

Not investigated.

Sleep stage change, including EEG awakening

- Among people who are accustomed to exposure to night-time aviation noise, the observation threshold for EEG awakening is an *SEL_i* of 40 dB(A); the observation threshold for sleep stage change is probably slightly lower.

- The effect of night-time noise on children has been studied in the context of laboratory studies with just twenty-four, eight and six children, and in one field study of five children in a domestic setting. One of the researchers indicated that children were less sensitive than adults to the onset of acute EEG-registered changes by night-time noise. Another researcher who studied children observed that, in the last phase of the sleep period, noise peaks of up to 95 dB(A) induced EEG awakenings (excluding EEG arousals) 50 per cent of the time and EEG arousals 8 per cent of the time.

Motility, motility onset

- Among people who are accustomed to exposure to night-time aviation noise, the average observation threshold is an *LAmax_i* of 32 dB(A) both for acute motility and for acute motility onset (with *SEL_i* values of 38 and 40 dB(A)). The observation threshold for acute motility is comparatively high among people who have been habitually exposed to higher levels of night-time aviation noise, and comparatively low among people who are only occasionally exposed to the sound of night-time aircraft passages. People in the latter group consequently respond more to a single aircraft passage.
- Among people who are accustomed to exposure to night-time rail traffic noise, the observation threshold for motility is estimated to be an *LAmax_i* of about 30 to 35 dB(A).
- Among people who are accustomed to exposure to night-time road traffic noise, the probability of acute motility during a given thirty-second interval featuring road traffic noise barely increases at higher noise exposures. However, in the first thirty-second interval of a period featuring road traffic noise, the probability of acute motility is higher than in a given thirty-second interval without road traffic noise or in another thirty-second interval during a period featuring road traffic noise.

Subject-registered awakening

- Among people who are accustomed to exposure to night-time aviation noise, the average observation threshold for subject-registered awakening is an *SEL_i* of 54 dB(A), which corresponds to an *LAmax_i* of 42 dB(A).
- The one study into the relationship between noise from military jets and subject-registered awakening indicated that, at higher exposures, military jets are much more likely to induce subject-registered awakening than civil aircraft.

Acute annoyance and other acute problems

Not investigated.

D.3 Biological effects over the course of a night (before, while and after sleeping)

D.3.1 Introduction

Very few field studies have looked specifically at the relationship between an effect measured over the course of or following a sleep period and the noise exposure during sleep. Furthermore, data from the studies of acute noise-induced changes described in D.2 has not in most cases been aggregated to provide full-night figures.

In 2003, the RIVM performed a review of field studies that had specifically sought to shed light on the effects of night-time road traffic noise on sleep[69]. For this review, the researchers collated literature published since 1970. The reviewers found thirty-four field studies, of which twenty-three focused entirely on self-reported effects over an extended assessment period (e.g. a year). The results of these twenty-three studies are considered in the section of this annex devoted to the chronic consequences of exposure to (road traffic) noise. Ten of the other eleven studies used EEG, ECG or actimetry monitoring of sleeping subjects, sometimes supported by journal entries, to investigate effects over the course of a single night. In most cases, noise levels were also measured (in subjects' bedrooms) during the study nights. The eleventh study monitored effects on the basis of journal entries only. These eleven studies are considered in D.3.2.

In an article published in 2003, Babisch[81] provided an overview of research into stress hormone levels associated with exposure to noise, both in a domestic setting and in an occupational setting. He referred to approximately a hundred studies, twenty-three of them epidemiological. Eleven of these twenty-three studies were concerned with the effects of occupational exposure to noise, while twelve studies looked at the effects of exposure to noise in the domestic environment. In eight of these studies (three of which focused on exposure to road traffic noise and five on exposure to aviation noise), stress hormone levels were determined by analysing urine samples collected during and after sleep, or saliva samples collected after the subjects had waken up. These eight studies are considered below.

The eleven field studies mentioned in the RIVM report[69] and the eight field studies referred to in Babisch's article[81] were concerned almost exclusively with road and air traffic noise; just one of the studies looked at both road and rail traffic. No similar studies into the effects of stationary noise sources were traced. In the following subsections, first the results of the field studies of road traffic noise are dealt with, then the field studies of aviation noise, and finally the one field study of rail traffic noise. The subsection on aviation noise field studies also takes account of data from one quasi-field study.

This section concludes with an inventory of (laboratory) studies into the influence of night-time noise on the immunological properties of blood cells.

D.3.2 *Road traffic noise*

The eleven field studies referred to in the RIVM report[69] were as follows:

1 Four very small studies. These studies involved very small numbers of subjects and subject-nights (three, six, seven, and twelve people) and do not lend themselves to generalisation. They are not therefore considered further here[129,164-166].
2 Research from the USA[49]. This research into the effects of aviation noise included only a control group that was exposed to road traffic noise. The report does not include any aggregated single-night data relating to this group.
3 Four European studies conducted around 1980 in the Netherlands, Germany, France and the UK on behalf of the European Commission[58,59,70-73].
4 Research carried out in Germany[77,78]. This is the same research referred to in D.2.4, into the differences between the effects of night-time exposure to road traffic noise and rail traffic noise.
5 Research undertaken in Sweden by Öhrström[74], which monitored effects purely on the basis of journal entries.

The four European studies mentioned in list item 3 were intervention studies, in which road traffic-related noise exposure was reduced by approximately 10 dB(A) by various means: double glazing of bedroom windows, gap sealing, use of personal hearing protection, and temporary bedroom relocation to the quiet side of the house[58,59,70-73]. The four studies involved a total of seventy subjects and 922 subject-nights. Jurriëns drew the following conclusions regarding the effects observed in relatively noisy situations (compared with quieter situations):

- The average duration of REM sleep is 6.5 minutes shorter
- In reaction time tests, the average reaction time is twelve milliseconds (12 ms) longer than the overall average reaction time of 350 ms, and more mistakes are made (8 per cent)
- Self-reported quality of sleep is less (7 per cent)
- The W (waking) time recorded by EEG is 7 minutes longer (determined in two of the four studies)
- The average heart rate when sleeping is higher. In the Dutch research, the rate was 3.2 beats per minute higher (71.5 bpm, compared with 68.3 bpm)[58].

In the German research referred to in list item 4[77,78], 188 subjects were exposed mainly to road traffic noise and a similar number mainly to noise from passing trains. The num-

ber of subject-nights with usable data on motility was 1710 in the road traffic group. A recent analysis[79] of the data indicated that, among people exposed to road traffic noise, average motility for a single sleep period increased as the equivalent indoor or outdoor traffic sound pressure level was higher during the period in question.

The research by Öhrström referred to in list item 5[74] involved 106 subjects. Analysis of their journal entries revealed the following: 37 per cent of subjects in noisy environments had difficulty getting to sleep, compared with 8 per cent in quiet environments; the percentages of subjects in the two types of environment who were woken in the night by road traffic noise were 57 per cent and 4 per cent; average sleep quality, as rated on an eleven-point scale (where 0 equals very poor and 10 equals very good) was 6.2 for the noisy environments and 8.2 for the quiet ones; morning fatigue/alertness scores, as rated on an eleven-point scale (where 0 equals very tired and 10 equals not at all tired) were 5.0 and 7.0; the average scores for morning irritability, as rated on an eleven-point scale (where 0 equals very irritable and 10 equals not at all irritable) were 6.5 and 7.8. In other words, all parameters values were less favourable in the noisy situation.

Swedish researcher Öhrström recently made a longitudinal study of the change in noise-induced effects on sleep following realisation of a scheme designed to reduce road traffic noise by the enclosure of a road in a tunnel. The report on elements of the study[75] will shortly be followed by two publications in *J Sound Vib*[**]. The forthcoming data will show that the study was modest in scale: at each of two locations (one noisy, one quieter), thirteen subjects were monitored on each of three occasions, once before and twice after completion of the tunnel, which has reduced indoor noise by 10 dB(A) at the noisy location. Within the exposed group, no statistically significant change was detected in various parameters monitored before and after completion of the tunnel, the parameters in question being average motility, minutes spent in bed, sleep inception period, sleep duration, number of 'awakenings', and number of waking intervals of more than five minutes. Not surprisingly, in view of the small number of subjects, there is considerable variation in the average values; notably, there was an increase in the values of various parameters – i.e. a deterioration – in the third monitoring round, a year after completion of the tunnel, relative to the values measured in the second phase.

At the ICBEN2003 Congress, Öhrström presented a provisional report on those results from the international RANCH study that related to night-time noise. The subjects were seventy-nine children between the ages of nine and twelve, plus one parent of each child[76]. The equivalent sound pressure level of the road traffic over a twenty-four-hour period, as determined on the outside of the most heavily exposed wall , varied from less than 55 to more than 64 dB(A). The sleep parameters monitored were sleep quality

* Öhrström, personal communication.

(overall quality, as established by questionnaire, and nightly quality, as recorded in a journal), sleep inception period and average motility. Although marked differences were observed between the parameter changes in children and those in their parents, the changes did not appear to be dependent on the noise exposure.

Table 16 summarises data from the three field studies[82,152-154] referred to by Babisch[81], which sought to establish the effects of noise on hormone concentrations, as determined from urine samples collected over the course of a night or blood samples collected after awakening. Strictly speaking, the results do not show whether the observed changes are the result of exposure to road traffic noise during the night in question, or (at least partly) the result of exposure the previous day, or the result of chronic daytime or night-time exposure.

Table 16 Summary of data from the three field studies referred to by Babisch[81], which sought to establish the effects of noise on hormone concentrations[a].

Publication	Outdoor noise exposure (*LAeq* in dB(A))	Subjects	Adrenaline	Noradrenaline	Cortisol	Monitoring method
Babisch, 2001[82]	45-75, during the night	234 women	=	+	x	Urine collection during the night
Evans, 2001[153]	Less than 50 or more than 60 over the twenty-four-hour period	115 children	=	=	+	Urine collection during the night
Ising, 2002[154]	Lmax= 40 or Lmax= 66 dB(C) over the twenty-four-hour period	56 children	x	x	+	Urine collection during the night

[a] Relative effect at higher noise exposure: + statistically significant change in the anticipated direction, = no significant change in the anticipated direction, x not monitored.

Working at the Berlin Environmental Department, Babisch *et al* studied the effect of road traffic noise on the excretion of adrenaline and noradrenaline in the night-time urine of 234 women (thirty to forty-five years old)[82], some of whom lived in homes with the bedroom on the street side, and some in homes with the living room on the street side. The volume of passing road traffic varied considerably from dwelling to dwelling. The analyses took account of numerous distorting variables. Among the women with bedrooms on the street side, a statistically significant increase was observed in noradrenaline levels as the logarithm of the traffic volume rose. (The logarithm of the traffic volume is approximately proportional to the equivalent sound pressure level.) Changes in adrenaline level were not associated with changes in traffic volume, however. Among women with the living room on the street side, no effect was observed on either adrenaline or noradrenaline levels. The fact that it was mainly noradrenaline concentrations that were raised is consistent with Ising's model, which predicts that the noradrenaline

concentration is particularly likely to increase in response to noises to which a person is exposed for a long time[83]. The effect of road traffic noise on noradrenaline concentration was particularly pronounced in women who indicated that they slept with the bedroom window closed to prevent their sleep being disturbed by road traffic noise, and nevertheless experienced noise-related annoyance. Among women who experienced no noise-related annoyance when their windows were closed, no statistically significant increase in noradrenaline concentrations was observed. The researchers explain these findings as the result of a coping mechanism: among women who are able to prevent noise-related sleep disturbance by closing their windows, noradrenaline levels are not affected, but among women who are not able to cope in this way, they rise. If a raised noradrenaline level may be regarded as predictive of cardiovascular problems, the authors argue that only those people who are highly sleep disturbed due to environmental noise and are not able to take corrective action are at increased risk of developing cardiovascular problems. However, the research results do not exclude the possibility that the observed effect is a reversible change.

Evans and Lercher studied 115 children around the age of seven who were exposed to road and rail traffic in Austria[153,155]. Half of the children lived in an environment with relatively little road and rail traffic noise *(Lden* less than 50 dB(A), average 46 dB(A)), while the other half lived in an environment where noise levels were typically more than 60 dB(A) (average 62 dB(A)). The researchers compared various endocrine and cardiovascular functions: daytime diastolic and systolic blood pressure and heart rate, plus adrenaline, noradrenaline, cortisol and 20A-dihydrocortisol levels, as determined from urine samples collected in the course of the night. A statistically significant difference of more than 25 per cent was observed between the cortisol and 20A-dihydrocortisol concentrations of the two groups. In a test that involved asking the children to solve impossible puzzles, girls exposed to higher noise exposures performed less well than girls in the low-exposure group.

D.3.3 Aviation noise

D.3.3.1 Field research

Table 17 summarises the findings of Babisch's review[81] of research into aviation noise-related changes in hormone levels over the course of a night.

Table 17 The findings of research[156-159,162,163] into changes and differences[a] in stress hormone levels, as reviewed by Babisch[81].

Publication	Noise exposure (*LAeq* in dB(A))	Subjects	Adrenaline	Noradrenaline	Cortisol	Monitoring technique
Evans, 1995[156]	59-65 24 hours	135 children	+	+	=	Urine collection during the night
Evans, 1998[157]	53-62 24 hours	217 children	+	+	=	Urine collection during the night
Ising, 1999[163]	56-70 over the day	40 children	=	=	=	Urine collection during the night
Haines, 2001[158,159]	53-62 24 hours	204 children	=	=	=	Urine collection during the night
Stansfeld, 2001[162]	<57->66 over the day	238 children	x	x	=	Saliva collected in the morning

[a] Relative effect at higher noise exposure: + statistically significant increase, = no significant change, x not monitored.

The Bristol-based team of Smith *et al*[80] made a phased investigation of the interrelationships between aviation noise, sleep disturbance and health.

In the final phase, the motility of ninety people (forty-five couples) was monitored using actimeters for three nights, during which sound pressure levels were measured in the subjects' bedrooms. The sources of the noises audible in the subjects' bedrooms were not determined using an external identification system, nor were any outdoor sound pressure levels measured. Noise events were divided into two groups: prolonged noise events (more than one minute above the background level) and brief noise events (less than one minute above the background level, with an equivalent sound pressure level of more than 50 dB(A) over at least one five-second interval). The number of brief noise events averaged 8.2 per night (with an average *SEL_i* of 59 dB(A)), and the number of prolonged noise events averaged 6.4 per night (with an average *SEL_i* of 65 dB(A)). No association was found between noise exposure and actimetric activity. The researchers suggested that this was due to the low noise exposures that subjects were exposed to, even though there was considerable inter-individual variation in exposure values. The team reported having nevertheless observed statistically significant associations between noise exposure and motility among subjects on board a ship. The observed associations were:

- between the *number of* noise events and an index of sleep disturbance derived from several variables;
- between higher sound pressure levels during the sleep latency period and difficulty getting to sleep; and
- between higher sound pressure levels towards the end of a subject's sleeping time and premature awakening.

However, because the sources of the noises were not identified, the researchers could not exclude the possibility that the increased disturbance levels were related to the subjects' waking activities and were not therefore the effects of noise on sleeping patterns.

Passchier-Vermeer[12] identified four functions that she considered indicative of the effect of aviation noise over the course of a sleep period. The functions in question (see Figure 23) were:

- High average motility during sleep. This was quantified as follows. The data was analysed to calculate a level of motility as a function of age, which was exceeded by 5 per cent of subjects when not exposed to aviation noise when sleeping. A figure was then worked out for the percentage of subjects who on a given research night exhibited higher average motility than the 'normal' value for their age; the percentage of people who would exceed the normal motility value in the absence of aviation noise (approximately 5 per cent) was then deducted from the percentage for the night. The analysis revealed that average motility increased with rising night-time noise exposure, but this is not illustrated in Figure 23.
- Recalled awakening. In the journal that they were asked to write each morning, subjects indicated whether they had been woken by aviation noise in the night.
- Subject-registered awakening at least three times a night. Awakenings were recorded by the subject pressing a button on his or her actimeter.
- Use of somnifacient drugs. In their journals, subjects indicated each morning whether they had taken any sleeping pills the night before. The use of somnifacient drugs proved to be strongly age-related. Up to the age of about sixty, the use of somnifacient drugs was quite modest; above that age, use increased sharply with rising exposure to aviation noise.

Figure 23 shows night-time noise exposures in the form of *Lnight* values. The noise exposure was originally expressed as the equivalent sound pressure level during the sleep period. However, the equivalent sound pressure level data was converted to *Lnight* values on the basis of what is known about the relationship between equivalent sound pressure and *Lnight*. Because some effects are age-related, Figure 23 is based on the age profile of the adult Dutch population.

D.3.3.2 Quasi-field research

A research team at Berlin's Robert Koch Institute performed a quasi-field study with sixteen subjects living in the vicinity of Fuhlsbüttel Airport near Hamburg. The study involved observation of the effects induced by aviation noise reproduced in subjects' bedrooms using loudspeakers[47,150]. There were almost no night flights into or out of the

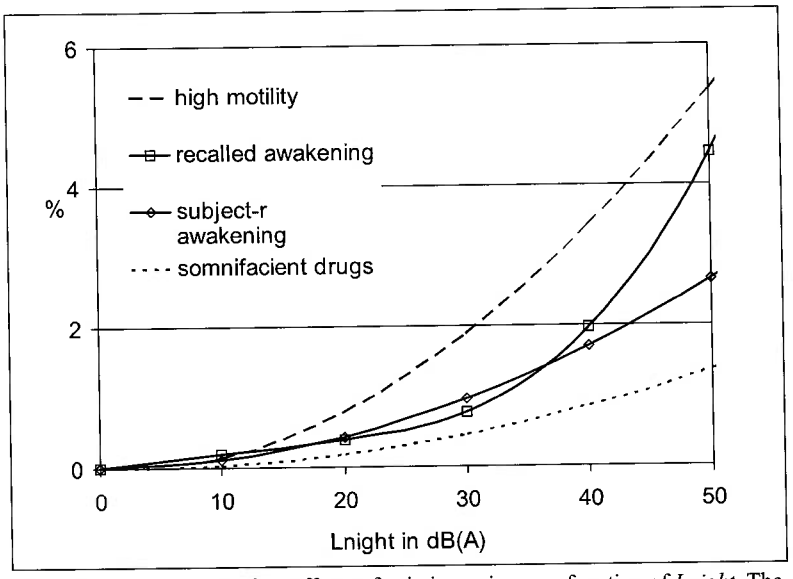

Figure 23 Prevalence of four effects of aviation noise, as a function of *Lnight*. The prevalence values express the following percentages, as attributable to exposure to aviation noise: percentage of people exhibiting high motility levels for their age, percentage of people who recalled being awoken by aviation noise, percentage of people who registered awakening at least three times in a night, and percentage of people who recorded using somnifacient drugs[12].

airport, so at the start of the study the subjects were unused to night-time aviation noise. After two nights without the introduction of any artificial noise, the subjects were exposed to recorded aviation noise with an *LAmax_i* of 65 dB(A) thirty-two times a night for thirty-eight nights. During and immediately after each sleep period, urine samples were collected and the total amount of cortisol present was determined. The researchers distinguished three adaptation patterns over the thirty-eight nights: total cortisol almost stable (observed mainly in female subjects); a large initial rise in total cortisol to a peak on the third night of exposure, followed by a gradual decline for the remainder of the study period; an initial decline in total cortisol, followed by a large peak on the third night of exposure, followed by a gradual increase for the remainder of the study period. With all three patterns, there were fluctuations in the course of the week, which were more pronounced in men than in women.

It is particularly interesting to note how the well-being scores recorded each day using a questionnaire changed over the study period. The scores were given using a scale, designed so that the average for the general population is zero, and 95 per cent of recorded values are between –3 and +3. Before the recorded aviation noise was introduced, in each of the three subsequent adaptation pattern groups the average well-being score was approximately 0.5 (i.e. a little better than normal for the population as a

whole). In the stable cortisol group, well-being during the first fourteen days of the study fell from 0.5 to zero, where it remained. In both the other groups, well-being scores continued to fall, from 0.5 to –1 in the increasing cortisol group and from 0.5 to –2.5 in the declining cortisol group.

D.3.4 *Field research rail traffic noise*

In the above-mentioned German research[77,78] 188 subjects were exposed mainly to noise from passing trains. The number of subject-nights characterised by motility was 1581. A recent analysis[79] of the data indicated that, among people exposed to *rail traffic noise*, average motility for a single sleep period was unrelated to the equivalent indoor or outdoor traffic sound pressure level during the period in question.

Evans[153] reported that the noise measurements included not only the road traffic noise exposure experienced by children, but also the train noise exposure. Although the article does not indicate the breakdown between road and rail traffic noise, it seems reasonable to assume that road traffic noise was predominant.

D.3.5 *Laboratory research into changes in immunological parameters*

Between 1968 and 1974, Osada *et al*[84-87] investigated the relationship between exposure to noise and changes in the *number* of cells in the blood with roles in the body's immune functions. They performed four laboratory experiments with twenty-one subjects, in which they monitored changes in leukocyte and (eosinophilic and basophilic) granulocyte levels in the blood associated with exposure to various types of noise (road, air and rail traffic noise, industrial noise, white noise and pink noise). When data from noise exposure nights was compared with data from non-exposure nights, major differences were observed in the average values and wide distributions around the average changes. However, shortcomings have subsequently been highlighted in the study design**, which almost certainly explains the observed changes.

In their survey article *The Neuroendocrine Recovery Function of Sleep*, Born and Fehm devoted a section to the possibility that night-time exposure to noise might affect the immune system[88]. In two experiments, subjects were either deprived of sleep or allowed to sleep 'normally', then certain blood cells (monocytes) were examined to determine whether they exhibited an immune response to a particular stimulus (production of interleukin-1 and TNF-α (tumour necrosis factor α), which affect the production of T-cells, which in turn are important for the production of interleukin-2). Contrary to what had been expected, the immune response of the monocytes was much stronger

* Marth, personal communication.

after a sleepless night than after an ordinary night. On the other hand, there were far fewer monocytes present in the blood after a sleepless night, and production of interleukin-2 by T-cells was much more vigorous after a normal night than after a night of sleep deprivation. On the basis of their findings, the two authors postulate that night-time noise exposure may have a negative influence on the immune system. They add, however, that a great deal more research would be necessary to confirm such a hypothesis.

D.4 Effects on health and well-being

The first two subsections below (D.4.1 and D.4.2) deal with research into the association between chronic exposure to *traffic noise* and medical conditions, sleep quality and well-being. Subsection D.4.3 is concerned with data on (the effects of) *noise from neighbours* and the associated topic of acoustic insulation between dwellings. Finally, subsection D.4.4 described a Dutch inventory study of traffic noise, industrial noise, neighbourhood noise and noise from neighbours.

D.4.1 Medical conditions

D.4.1.1 Insomnia

A group of Japanese researchers carried out a questionnaire-based survey of 3600 adult Japanese women (aged between twenty and eighty) living on eight study sites to gather information about the factors that contribute to insomnia[89]. Some 11 per cent of subjects were found to be affected by insomnia. (The researchers adopted a definition of insomnia based on the *ICD-10 classification of mental and behavioral disorders: clinical description and diagnostic guidelines* [90].) One of the factors whose relationship with insomnia was investigated was the volume of traffic on the road where the subject lived. It was found that a high traffic volume (a nightly average of more than two thousand vehicles per hour, with a lorry counting as ten vehicles) was an insomnia risk factor. Women living on busy roads were considerably more likely to suffer from insomnia than the other women. Analysis of the survey data took account of various distorting variables, such as age, number of (small) children in the family, social status, receipt of medical treatment, regularity of bedtimes, apnoea-like problems and serious unpleasant experiences in the six months prior to completing the questionnaire. When the percentage of insomniacs in each of the three areas with the highest exposures was compared with the percentage in the low-exposure areas, the ratios worked out at, respectively, 1.4 (2100 vehicles per hour, *Lnight* of around 65 dB(A)), 2.1 (2400 vehicles per hour,

Lnight of around 67 dB(A)) and 2.8 (6000 vehicles per hour, *Lnight* of around 70 dB(A)). The most frequently reported problem was difficulty getting to sleep.

D.4.1.2 Health diminution

A research team at Berlin's Robert-Koch Institute produced a 400-page report on the findings of the Spandauer Gesundheits Survey[95]: a longitudinal study, in the context of which the health of adults in Berlin's Spandau district has been surveyed every two years since 1982. The ninth survey round involved 2015 subjects, of whom 1714 were participating for at least the fifth time. In addition to going through the usual tests and questionnaires, these subjects were asked about noise-related annoyance from road, rail and air traffic, as well as from industrial sources. Noise maps were also produced showing the road traffic-related noise exposure on the homes of the 1718 people subjects who chose to complete the questionnaire on noise-related annoyance. The research into the effects of traffic noise was therefore essentially a cross-sectional cohort study. Furthermore, outdoor sound pressure levels were measured in front of ninety-six homes. However, it was not possible to take recent aircraft noise exposures into account. Most aircraft flying over the area were going to or from Tegel Airfield, which is closed at night (from 10pm for takeoffs and 11pm for landings, to 5am for both takeoffs and landings).

The analyses took account of twelve variables with the potential to distort the results. The presence and treatment of illnesses and medical conditions in the two years since the previous survey round (period prevalence), and in the research period as a whole (total prevalence) were investigated. The probability of a subject receiving medical treatment for a given illness or condition was determined for subjects whose road traffic-related *Lnight* was less than 50 dB(A) and expressed as an odds ratio (OR); in addition, 95 per cent confidence intervals (CIs) were stated in the report. The statistically significant results for subjects with a road traffic-related *Lnight* of more than 55 dB(A) were as follows:

- *Treatment for hypertension*: OR = 1.9 (CI = 1.1 – 3.2) (period prevalence)
- Treatment for hypertension if bedroom window was normally open: OR = 6.1 (CI = 1.3 – 29.2) (period prevalence)
- *Treatment for hypertension*: OR = 1.8 (CI = 1.1 – 2.9) (total prevalence)
- *Asthmatic bronchitis*: OR = 1.5 (CI = 0.9 – 2.5) (total prevalence)

With regard to people who were annoyed by road traffic noise, the following statistically significant association was found with daytime road traffic noise:

- *Treatment for psychological problems*: OR = 2.7 (CI = 1.3 – 5.6) (period prevalence)

Comparison of people exposed to a high exposure of aviation noise over a twenty-four-hour period with people exposed to a lower exposure revealed the following statistically significant association:

- *Treatment for thyroid problems*: OR = 3.8 (CI = 1.3 – 11.3) (period prevalence)

The researchers warn that their findings regarding non-cardiovascular illnesses and medical conditions (asthmatic bronchitis, thyroid problems) are potentially liable to distortion by variables other than the twelve that have been taken into account. Hence, the only conclusion that may be drawn regarding medical conditions is that, within the studied population, night-time exposure to road traffic noise is associated with treatment for hypertension.

The researchers also point out that the study population was made up largely of people who were very conscious of their health. If this population was more or less than averagely prone to hypertension, or inclined towards a lifestyle that increased or decreased the probability of hypertension, the association between hypertension and night-time noise exposure might not be reflective of the population at large.

Where the above-mentioned findings regarding hypertension and night-time road traffic noise were concerned, the OR for people exposed to a noise exposure of between 50 and 55 dB(A) was calculated to be between 1.0 and the PR given in the summary for road traffic noise exposures of more than 55 dB(A).

The researchers were not surprised to find that hypertension was demonstrably associated with night-time noise, but not with daytime noise, partly because people are often elsewhere during the day and partly because people are more sensitive to noise at night than during the day.

A methodological assessment of the research is made in the main body of this report.

D.4.2 Sleep quality and well-being

D.4.2.1 Increased motility

The British field study into the effect of aviation noise on sleep found that, over a sleep period, average motility and motility onset increased with rising exposure to aviation noise[96]. Horne reported that there was a strong inverse relationship between average motility and perceived quality of sleep. The Dutch field study into the effect of aviation noise on sleep and the German study regarding the effect of road traffic noise also found that average motility increased with noise exposure when sleeping[12,13]. The researchers found that average motility over the course of a night was strongly associated with the number of times that a subject recalled waking during his/her sleeping time, with the

number of times that a subject registered awakening during his/her sleeping time, and with the following variables regarding which subjects provided information by completing a questionnaire at the beginning of the study: number of medicines used, sleep quality, number of sleeping problems, frequency of aviation noise-induced awakening, weekly frequency of aviation noise-induced averse effects on sleep, and number of health problems. The secondary analysis of the German research into road and rail traffic noise (involving 1710 subject-nights characterised by motility in the road traffic subject group and 1581 such subject-nights in the rail traffic group) also indicated that average motility increased with rising road traffic noise exposure[79]. Exposure to rail traffic noise had no demonstrable effect on average motility. Where both aviation noise and road traffic noise were concerned, the increase in motility with *Li* (the equivalent sound pressure level during sleeping time over an extended period) was much greater than would have been expected solely on the basis of the increase in the probability of noise-induced acute motility. The average increase in motility per dB(A) increase in noise exposure appeared to be between 1.3 and 1.5 times greater for road traffic noise than for aviation noise[79].

In the first main phase of a British study of aviation noise, sleep disturbance and health conducted by Smith *et al*[80], 543 subjects from Bristol were asked to answer a questionnaire. Due to lack of information about the noise exposure experienced by respondents, their subjective perceptions of the problems they had experienced getting to sleep were used to estimate levels of exposure to aviation noise when sleeping. Questions were posed regarding health (based on the abbreviated version of the General Health Questionnaire), self-reported health, sensitivity to noise, sleep disturbance and negative affectivity (utilising the Neuroticism Scale in Eysenck's Personality Inventory). Significant health differences and differences in sleep disturbance experience were detected between the subjectively defined high-exposure and low-exposure groups. However, once adjustment was made for the influence of age and degree of neuroticism on health and sleep disturbance, no statistically significant difference was found to exist between the two groups.

In a follow-up survey, some of the respondents from the first main phase completed a further questionnaire. When the findings from the second questionnaire were compared with information regarding the same subjects gathered from the first questionnaire, it was found that diminished health, increased sleep disturbance and increased sensitivity to noise were all associated with an increase between the survey dates in the noise exposure perceived by the respondents. However, no link was found between change in sleep disturbance and change in health. The researchers explained the findings of the follow-up study as follows. If the original effect measured during the first main phase is eliminated (by concentrating on the differences) and there is little situational

change between the time of the first survey and the time of the second, (minor) changes will not be correlated.

A further survey with an improved design was subsequently carried out. For this survey, a number of locations in the vicinity of four airports were selected, some with a relatively high aviation noise exposure, and some with a lower exposure. A total of 1121 subjects were questioned orally and a further 658 subjects completed a written postal questionnaire. Differences between the higher exposure and lower-exposure subjects were detected in relation to the following parameters: perceived level of aviation noise when trying to get to sleep, sleep disturbance, physical health and well-being, particularly in terms of irritability, anxiety, depression and sadness. Even after making allowance for other variables, sleep disturbance and health remained closely related. From their findings, the researchers concluded that they were unable to demonstrate a causal relationship between sleep disturbance and health. They added that it was also possible that sleep disturbance was symptomatic of poor health.

D.4.2.2 Self-reported sleep disturbance, self-reported sleep quality diminution, and other self-reported effects of exposure to noise

On the basis of TNO's Disturbance Knowledge Base, exposure-response relationships have been defined for self-reported sleep disturbance by road, rail and air traffic[97,98] for use in an EU position paper. The main body of this report gives details of the relationships involving self-reported high sleep disturbance and includes a discussion of the findings.

The RIVM produced a report[69] which considered the question of whether a quantitative meta-analysis could be made of the results of research into the influence of road traffic noise on perceived sleep quality and difficulty staying asleep. Although the RIVM described several studies as being good quality, the researchers decided that it was not possible to perform a meta-analysis because of discrepancies in the studies' nomenclature, methods, exposure determination techniques and approaches to adjustment for distorting variables. Nevertheless, the Dutch researchers were of the opinion that there were qualitative indications that road traffic noise was associated with diminished perceived sleep quality and more difficulty staying asleep.

At the ICBEN2003 congress, the Dutch researcher Vos presented data from a questionnaire-based study of effects of gunshot noise on sleep[111]. Some of the findings are illustrated in Figure 24. The graph shows the percentage of people who indicated they were woken by gunshot noise (as established in Germany by Buchta) as a function of the average *SEL* (in dB(C)) of the noise discernible in the domestic environment. Informa-

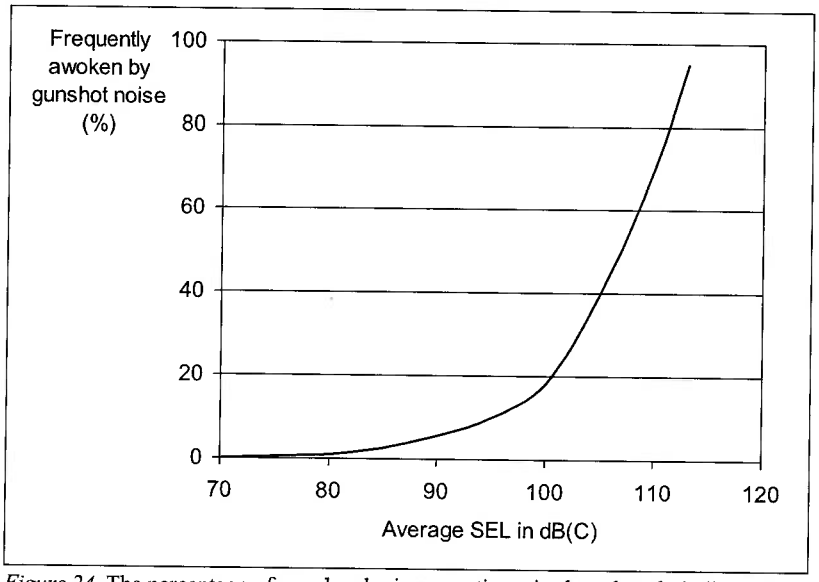

Figure 24 The percentage of people who in a questionnaire-based study indicated being frequently awoken by gunshot noise, as a function of the average *SEL* (in dB(C)) of the noise events[111].

tion about the number of noise events per night was not given, so it has not been possible to convert the data to *Lnight* values to enable comparison with the dose-response relationships for other noise sources. Vos was not (yet) able to make dose-response data other than that illustrated in Figure 24 available*.

D.4.2.3 Health problems

The Dutch field research into the effects of aviation noise on sleep established a relationship between personal noise exposure when sleeping (*Li*) and the frequency of health problems included on the abbreviated Health Perceptions Questionnaire[12,13]. Compiled on the basis of stress research, the Health Perceptions Questionnaire identifies thirteen health-related problems, such as headache, stomach-ache, tiredness and digestive problems. It will be apparent that these are not life-threatening conditions. A rise in aviation noise-related *Li* from 0 to 35 dB(A) is associated with a two-fold increase in the frequency of problems. The researchers considered whether a causal relationship existed, or merely a relationship. The latter might be the case if, for example, people with health problems were liable to get up later and were therefore exposed to the higher high aircraft noise exposures that occur in the morning, resulting in relatively high *Li*

* Vos, personal communication.

values. However, analysis revealed that neither the moment of awakening nor any of the other possible sources of bias investigated by the team had any influence on the relationship between the frequency of health problems and *Li*.

D.4.2.4 Making official complaints about noise

The submission of a complaint about noise may be regarded as symptomatic of reduced well-being. Numerous factors influence a person's inclination in a given situation to make an 'official' complaint about a noise-related problem. These factors include not only the level of annoyance or inconvenience experienced, but also to some extent whether the person knows who to complain to, how easy it is to make a complaint, whether the person believes his/her complaint is likely to be acted upon, and if it is known or suspected that other people are also making complaints. In the Netherlands, people who have experienced problems caused by the noise from aircraft on their way to or from Amsterdam's Schiphol Airport used to be able to complain to the Problem Desk at the Schiphol Airport Committee on Noise-Related Annoyance (now superseded by the Problem Desk at Cros, the Schiphol Airport Liaison Body). The RIVM performed an analysis of complaints to the Problem Desk[180,181] and linked the data to the findings of a questionnaire-based study[182]. Figure 25 illustrates the position between 1986 and 2001, showing the number of problems, the number of complainants and the number of aircraft movements. Approximately 15 per cent of problems were found to relate to noise during the night (11pm to 7am). From the data in Figure 25, it is also apparent that some people complain repeatedly in the course of a year; in 2001, for example, the average number of problems per complainant was thirty-seven.

The number of problems per thousand aircraft movements was 680 in 1997 and 410 in 2001. In 2003, the so-called 'Polder Runway' came into use, despite considerable opposition from people living near the airport. Provisional figures indicate that the number of problems in 2003 was double the number reported the previous year.

Approximately 15 per cent of all problems involved noise during the night (11pm to 7am). Night flights (11pm to 6am) accounted for 4 per cent of the total number of flights, and it is estimated that the number of aircraft movements occurring between 11pm and 7am was 8 per cent of the total[183]. It follows that night flights were linked to approximately twice as many problems as flights during the day and evening, even though the noisiest aircraft are not allowed to take off at night, so that night flights should on average be a little quieter than flights during the day and evening.

In Figure 26, the prevalence of problems is shown as a function of *Lden*. Notably, problems are less prevalent at the highest noise exposure than at a noise exposure of 61 to 62 dB(A). The researchers attribute this to the extra acoustic insulation fitted to homes in the most heavily exposed areas. Below an *Lden* of 50 dB(A), hardly any

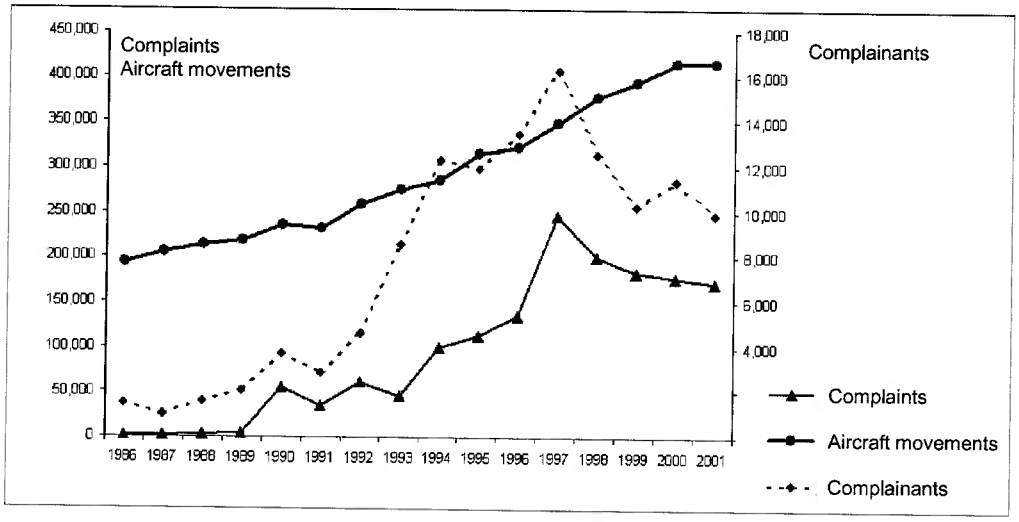

Figure 25 The number of people complaining about flights into and out of Schiphol Airport and the number of problems complained about, together with the number of aircraft movements between 1986 and 2001[180,181].

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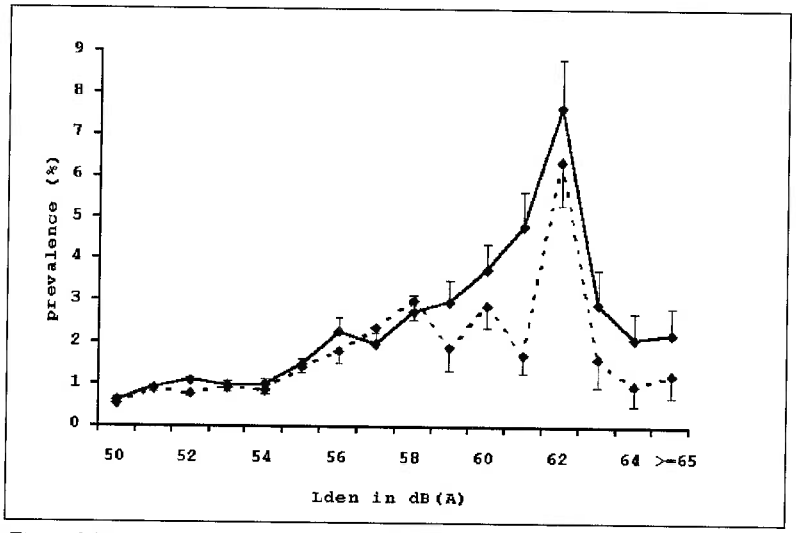


Figure 26 Prevalence of problems relating to particular flights into and out of Schiphol Airport in 1998 (solid line) and 1999 (dotted line), with an estimate of the uncertainty regarding the measurement points (half 95% prediction intervals), as a function of *Lden*[180,181].

omes have extra insulation; at an exposure of less than 60 dB(A), 20 per cent of homes have better insulation; at 61 and 62 dB(A), the figure rises to approximately 55 and 70 per cent, and from 63 dB(A), approximately 90 per cent of homes are well insulated.

A questionnaire-based study[182] has shown that the inclination to complain about aviation noise is linked to levels of annoyance, sleep disturbance, health worries and worry about air crashes.

DCMR runs a problem desk for people in the Rijnmond area who are experiencing problems due to environmental noise associated with industrial activities, road, rail and air traffic, etc[184]. Data on the complaints received in 2003 is presented in Table 18.

A total of 8303 problems were reported, of which 1265 (15 per cent) related to noise during the night (midnight to 7am). The heading 'Other noise' covers low and high-frequency machinery noise from unknown sources. Air traffic was the biggest cause of problems during the day and over a twenty-four-hour period. Although there were no regular scheduled night flights into or out of Rotterdam Airport, night-time aviation noise accounted for 25 per cent of all problems during the night. Industrial noise caused the fewest problems. It is interesting to note that relatively few complainants were concerned about (road and rail) traffic, but a lot of people complained about noise from events and bars, clubs and the like.

Table 18 Inventory of noise-related problems in the Rijnmond area reported to the DCMR Problem Desk in 2003[184].

	Number of problems per year			Percentage of problems		
	Day and evening: 7am to midnight	Night: midnight to 7am	Twenty-four-hour period	Day and evening: 7am to midnight	Night: midnight to 7am	Twenty-four-hour period
Traffic & transport	539	144	683	7,7	11,4	8,2
Air traffic	3423	316	3739	48,6	25,0	45,0
Industrial activities, etc	310	78	388	4,4	6,2	4,7
Bars, clubs, events, etc	892	342	1234	12,7	27,0	14,9
Other noise	1874	385	2259	26,6	30,4	27,2
Total	7038	1265	8303	100	100	100

D.4.3 Domestic acoustic insulation and influence on the effects of traffic noise

D.4.3.1 Domestic acoustic insulation

The Building Decree makes requirements regarding the sound attenuating characteristics of new homes and other noise-sensitive buildings[14]. For protection against industrial, road and rail traffic noise, each type of noise has to be limited to a twenty-four-hour value of 55 dB(A). This implies an outdoor night-time equivalent sound pressure

level of no more than 45 dB(A). If the characteristic attenuation provided by the building's outside wall is 20 dB(A), in relation to the spectrum of the noise source in question, this equates to an *Lnight_i* value of no more than 25 dB(A). If the outdoor twenty-four-hour value is higher than 55 dB(A), more stringent requirements apply. For protection against air traffic noise, requirements are made regarding the sound attenuating characteristics of new homes and other noise-sensitive buildings exposed to aviation noise exposures of more than 35 Ke*. The characteristic attenuation required depends on the 'sensitivity class' of the building and on the noise exposure in Ke, but is always at least 27 dB(A)**.

D.4.3.2 The influence of additional acoustic insulation on the effects of traffic noise

In the Netherlands, there have only been a small number of isolated studies into the efficiency and effectiveness of acoustic insulation in the reduction of perceived road and aviation noise levels, or into people's views regarding such insulation[114-119]. Bitter *et al* looked at the effects of fitting additional acoustic insulation to flats beside busy motorways carrying 70,000 vehicles per twenty-four-hours in Dordrecht[114] and Amsterdam[115]. In the Amsterdam study, 347 people completed an extensive questionnaire 2.5 years after extra insulation had been fitted to their homes to protect against road traffic noise (average additional attenuation 9 dB(A)). The questionnaire addressed matters such as the levels of noise-related annoyance being experienced at the time and previously experienced before the extra insulation was fitted. The findings confirmed that the insulation did reduce annoyance. Feedback regarding non-acoustic matters (humidity, ventilation and ease of cleaning) indicated dissatisfaction with the new insulation, however. Respondents were also asked about annoyance at different times during the twenty-four-hour period. The findings are illustrated in Figure 27. *

From Figure 27, it is clear that, while night-time noise-related annoyance was reduced by the fitting of extra acoustic insulation, the final outcome is less than ideal. A similar picture emerged from the Dordrecht study[114].

Van Dongen *et al*[116] carried out an exploratory study into sleep quality in homes fitted with additional acoustic insulation in the vicinity of Amsterdam's Schiphol Airport. The team determined the relationships between the percentages of people 'sleep disturbed' and 'highly sleep disturbed' and the outdoor noise exposure; the data was then compared with the provisional relationships[185] at the time for homes without special

* Ke stands for 'Kosteneenheid' (Kosten Unit, named after the Committee with professor Kosten as president), the standard unit of air traffic noise exposure in the Netherlands until recently.

** The Building Decree also makes requirements regarding acoustic insulation to protect against noise from installations in the same or adjoining premises, regarding resonant sounds, and regarding inter-dwellings sound attenuation, expressed in terms of *Ilu;k*, *Ilu*, and *Ico*.

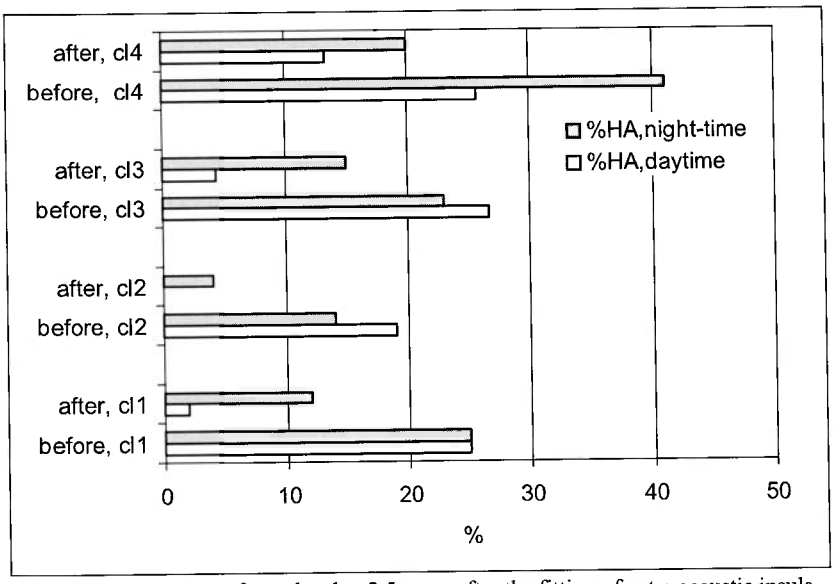


Figure 27 Percentage of people who, 2.5 years after the fitting of extra acoustic insulation to their homes, were highly annoyed by daytime and night-time noise at the time of questioning ('after') and prior to the fitting of extra acoustic insulation ('before'), attributable to four classes of noise (cl4 being the highest road traffic noise exposure, and cl1 the lowest).

insulation. From this comparison, it emerged that percentages sleep disturbed and highly sleep disturbed were slightly lower in the better-insulated dwellings than in 'ordinary' dwellings. However, the design of the study precluded the drawing of definitive conclusions.

Three reports were published between 1994 and 1999[117-119] regarding people's general views concerning modifications made to homes near Schiphol with a view to reducing aircraft noise-related problems. In 1994, opinion was gauged regarding the additional insulation fitted in the first phase of the Schiphol Insulation Plan. The modifications were made before introduction of the Building Decree of 01-10-1992[117]. In 1996, a comparison was made between satisfaction with the insulation packages installed before the Building Decree, and satisfaction with the 'scaled down' packages installed since the Building Decree[118]. The third report recounted the proceedings of an experts' workshop at which the problems associated with the insulation plan were addressed with a view to framing more flexible rules regarding acoustic insulation options[119]. General feelings about acoustic insulation fitted before and after implementation of the 1992 Building Decree were broadly similar: 75 per cent of subjects felt the insulation was good, 20 per cent rated it moderate, and 5 per cent thought it was poor. Some 85 per cent of subjects reported that the insulation had reduced noise-related annoyance indoors. Nevertheless, people in more than 55 per cent of the homes contin-

ued to experience at least slight noise-related annoyance, and people in 15 per cent of the homes reported to be highly annoyed since the modifications were made. The distribution patterns of both overall and night-time levels of aircraft noise-related annoyance were clearly seasonal: on (cold) winter nights, 10 per cent of subjects often or always experienced annoyance during the sleep period, compared with 40 per cent on (warm) summer nights. The differences were closely related to the use of windows: only 25 per cent of respondents said they slept with the bedroom window at least slightly ajar in the winter, whereas 70 per cent did so in the summer.

Fidell and Silvati[120] investigated what effect the fitting of insulation to attenuate aviation noise had on levels of being annoyed and being highly annoyed. However, they did not look specifically at annoyance during the sleep period.

In the UK, an extensive study was done to establish how effective extra acoustic insulation was in reducing exposure to road traffic noise[121]. The average sound attenuation achieved was 34 dB(A). Subjects whose bedrooms were adjacent to busy roads experienced night-time noise exposures with an *Lnight* value of between 57 and 77 dB(A). In the specially insulated homes, 23 per cent of subjects whose bedrooms faced the street reported being very highly or highly annoyed by night-time road traffic noise; 25 per cent had difficulty getting to sleep because of the noise, and 30 per cent said they were woken up at night by road traffic noise. The results proved to be influenced to a considerable extent by whether the subject felt that, without the window open, his or her bedroom was too hot in the summer: 37 per cent of those who felt unable to sleep with the window closed in warm weather were very highly or highly annoyed by night-time road traffic noise, whereas only 15 per cent of those who didn't mind having the window closed experienced similar problems. Some 85 per cent of subjects who said their bedrooms were too hot in the summer felt it necessary to sleep with the window open.

In Japan[122], people living in the vicinity of Kaneda Air Base and consequently exposed to very high night-time noise exposures caused by military jets were asked about the effectiveness of the additional acoustic insulation fitted to approximately 60 per cent of homes in the area, and about their satisfaction with the insulation. Scores for both effectiveness and satisfaction declined as noise exposures rose, from 80 and 60 per cent at a noise exposure with an estimated *Lden* of 65 dB(A), to 30 and 13 per cent at an estimated *Lden* of 85 dB(A). The seven investigated aspects of sleep disturbance (difficulty getting to sleep, waking up, difficulty getting to sleep after waking up, inconvenience caused by being woken too early in the morning, sense of having slept badly, and doubt about the prospects for a good following night's sleep) all proved to be related to outdoor noise exposure, but no difference was found between people living in specially insulated homes and people living in 'ordinary' homes. The researchers took the view that other forms of intervention, such as reducing night flying and switching to

alternative flight paths, were necessary to reduce the impact of noise on the sleep of people living in highly affected areas near the base.

D.4.4 Inter-dwelling acoustic insulation and noise from neighbours

D.4.4.1 Inter-dwelling acoustic insulation

The Building Decree makes requirements regarding the ability of new homes to attenuate sound from adjoining dwellings[14]. Sound attenuation between dwellings can be expressed using an index for the attenuation of airborne noise (I_{lu}); where account is taken of the volume of the reception room and the area of the common screening structure, it can be expressed using an index of characteristic sound attenuation ($I_{lu;k}$). The attenuation of contact noise between two dwellings is expressed using the contact noise index (I_{co}). For new homes, an airborne sound attenuation requirement ($I_{lu;k}$) of at least 0 dB applies. At an $I_{lu;k}$ of 0 dB, ordinary conversation in an adjoining home is audible, but incomprehensible. The quality of airborne sound attenuation is rated on a three-level scale:

- Minimum: $I_{lu;k}$ of 0 to +5 dB (normal conversation in an adjoining home is audible, but not comprehensible)
- Good: $I_{lu;k}$ of +5 to + 10 dB(A) (normal conversation in an adjoining home is not audible, the footsteps of a person in hard-soled shoes on a hard floor are readily audible and sometimes annoying)
- Very good: $I_{lu;k}$ greater than + 10 dB(A) (musical instruments, parties and the footsteps of a person in hard-soled shoes on a hard floor may be audible but are not annoying).

D.4.4.2 Noise from neighbours

Leidelmeijer and Marsman[99] published a report entitled *Geluid van buren: horen, hinder en sociale normen* (*Noise from Neighbours: Audibility, Annoyance and Social Norms*) regarding the findings of an interview-based study of 1242 households in the Netherlands, designed to shed light on the audibility of and annoyance associated with noise from neighbours during the day and at night. As a follow-up to the questionnaire, noise measurements were made in fifty homes. The researchers distinguished between five types of noise:

- Noise from sanitary and heating systems
- Contact noise
- Noise from audio equipment
- DIY (Do-It-Yourself) noise

- Noise from pets.

Distinction was also made according to the part of the house where the noise was audible or caused annoyance. The results are summarised in Table 19. 'Percentage for whom audible' is the percentage of respondents who reported hearing the type of noise in question. 'Percentage tolerant' is the percentage of the respondents for whom the given noise was audible who did not report being annoyed by it.

Table 19 Percentage of survey respondents able to hear and tolerant of each of five types of noise (where 'tolerant of' means able to hear but not annoyed by, i.e. 100 - percentage of hearers reporting annoyance)[99].

Part of house	Sanitary and central heating systems		Contact noise		Noise from audio equipment		DIY noise		Pets	
	% for whom audible	% tolerant	% for whom audible	% tolerant	% for whom audible	% tolerant	% for whom audible	% tolerant	% for whom audible	% tolerant
Living room	18	80	37	86	35	85	15	67	12	88
Kitchen	12	93	16	87	12	91	8	80	5	88
Master bedroom	19	76	22	73	12	74	8	65	6	76
Other bedrooms	5	88	8	75	3	57	2	75	2	70
Bathroom	13	97	6	83	3	100	2	89	1	100
Other rooms	4	95	3	87	1	80	1	100	0	100
Landing/hall/stairs	9	80	8	100	5	100	2	100	2	100
Throughout house	10	91	14	71	5	73	28	89	8	86

Clearly, respondents were least tolerant of noise from their neighbours that was audible in the master bedroom. Subjects were also asked whether they considered it acceptable for the various noises to be audible by day, by evening or by night. Where each of the five investigated types of noise were concerned, roughly 10 to 15 per cent of subjects indicated that they felt it was unacceptable for the noise to be audible during the day (for pets, the figure was 20 per cent; DIY noise was rated unacceptable on weekdays by 5 per cent of respondents and on Sundays by 17 per cent; for noise from audio equipment, the figure was 15 per cent). In each case, a higher percentage said the noise should not be audible in the evening, and a still higher percentage did not want to hear the noise at night (between 11pm and 7am). Overall, nearly 30 per cent of subjects said that sanitary fittings should not be audible at night, while approximately 50 per cent felt each of the other four types of noise was unacceptable by night.

The researchers concluded that audible noise from neighbours was by no means always perceived to be annoying. Whether annoyance is caused depends on the timing, the part of the house where the noise is audible, the volume, whether the noise is expected, how often the noise is audible, the duration of the noise, whether the noise is considered avoidable, and the number of sources.

Subjects were also asked whether they could hear voices in neighbouring homes. While the percentage of affirmative answers varied according to the type of dwelling, ordinary speech was to some extent audible in an average of 35 per cent of dwellings, and partially or readily comprehensible in approximately 8 per cent of dwellings. Raised voices could be heard, at least some to extent, in approximately 65 per cent of dwellings; they were at least partially comprehensible in 27 per cent of homes and readily comprehensible in approximately 10 per cent.

The results of the acoustic insulation tests in fifty homes indicated no statistically significant relationship between the airborne and contact sound attenuation indexes and the audibility of (airborne) noise from neighbouring dwellings.

In 1993, Kranendonk *et al* produced a synthesis of the research conducted up to that point in time into the annoyance associated with noise from neighbours[100]. Their synthesis covered four Dutch, one Swedish, one British and one French study. The various studies used a variety of effect indexes (annoyance scoring systems) and a variety of means of determining airborne and contact sound attenuation. Although it was, the researchers reported, difficult to assess all the data on the same basis, they were able to produce a table of synthesised findings (see Table 20). The average annoyance score was determined on a seven-point scale, where 7 equated to not annoyed and 1 to highly annoyed. As will be apparent from Table 20, an I_{lu} of zero corresponds to an average annoyance score of 5, to 10 per cent of people experiencing to be highly annoyed and to 25 per cent of people experiencing some annoyance.

Table 20 Acoustic performance of a dwelling (in terms of I_{lu} and *Ico*) and the corresponding average annoyance scores and percentages of people experiencing to be annoyed or highly annoyed[100].

I_{lu}	I_{co}	Average annoyance score	% people highly annoyed	% people annoyed (including highly annoyed)
-13	-5	3	50	75
-7	0	4	25	50
0	+6	5	10	25
+7	+11	6	2,5	10
+13	+17	7	0,5	2,5

TNO produced a report[101] on the relationship between noise from neighbouring dwellings and the airborne and contact noise attenuating indices I_{lu}, $I_{lu;k}$, and I_{co}, drawing on data from a questionnaire-based survey of the residents of six hundred dwellings, whose acoustic quality was determined in 202 cases. It was established that nearly half of the respondents heard at least some noise from neighbouring dwellings every day. Approximately 10 per cent of subjects found their neighbours' noise highly annoying.

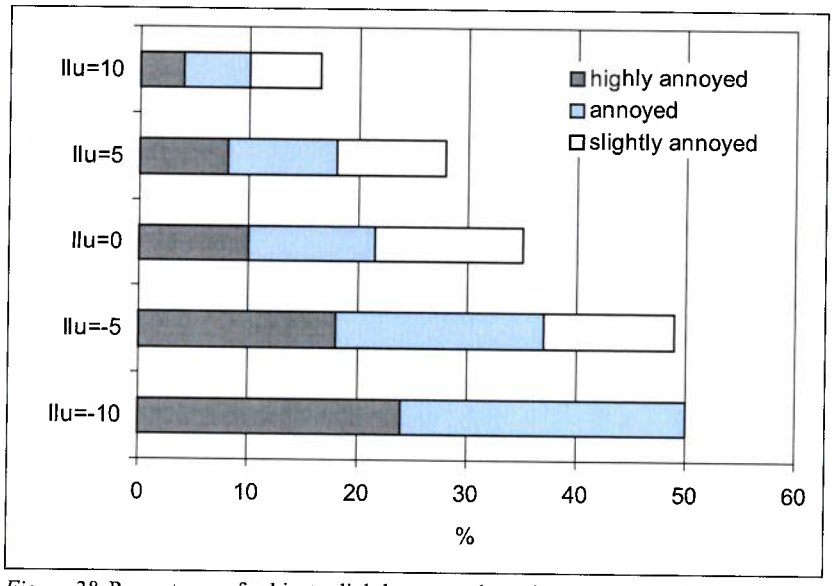


Figure 28 Percentages of subjects slightly annoyed, moderately annoyed and highly annoyed by noise from neighbouring dwellings[101].

The chief causes of annoyance were loud radios, hi-fis and TVs, the slamming of doors and footsteps on floors and staircases. Nearly all respondents said that in their own behaviour they were considerate of their neighbours and 80 per cent regarded themselves very tolerant of noise from their neighbours.

No relationship was established between contact noise-related annoyance and I_{co} value. This was not considered surprising by the researchers, because there was not a great deal of spread in the contact noise index values of the dwellings.

The correlation between the percentages of people experiencing annoyance and I_{lu} value is illustrated in Figure 28.

The percentages of people identified by the team as experiencing being annoyed and highly annoyed at a given I_{lu} value are broadly in line with the findings of Kranendonk *et al*[100]. At an I_{lu} of zero, the percentages are identical, while at higher and lower I_{lu} values there is a small difference.

D.4.5 *Data from inventory studies*

The national inventory study[9] carried out in 1998 asked respondents to indicate the extent to which their sleep was disturbed by noise from various sources, by giving a number between 0 and 10, where 0 = not disturbed at all and 10 = very highly disturbed. A standardised method was then used to calculate the percentage of respondents reporting sleep disturbance and high sleep disturbance. This involved transforming the eleven-

point scale into a continuous scale from 0 to 100. Respondents who scored 50 or more on this scale were deemed to suffer from self-reported sleep disturbance, and those who scored 72 or more to suffer from high self-reported sleep disturbance. This implies that the number of respondents affected by sleep disturbance includes the number affected by high sleep disturbance.

Table 21 gives the percentage of respondents reporting sleep disturbance and high sleep disturbance due to each noise source. It is not possible to make comparisons between source groups by simply aggregating the source group percentages, because it is not reasonable to assume that the percentage of people affected by (high) sleep disturbance due to a particular group of sources is the sum of the percentages for the individual sources within that group. Where road, rail and air traffic is concerned, passenger cars, lorries and mopeds are the sources to which most sleep disturbance is attributable (affecting, respectively, 7, 6, and 10 per cent of respondents). Where neighbour noise and neighbourhood noise are concerned, the predominant sources are contact noise (footsteps on stairs, slamming of doors), radio, hi-fi & TV, and the noise from other human activities, which were referred to by, respectively, 8, 6, and 8 per cent of respondents. Sleep disturbance due to noise from air or rail traffic, or to industrial noise is (much) less common than sleep disturbance due to the above-mentioned sources.

Table 21 Noise-related sleep disturbance associated with sources of various types[9].

Source group	Noise source	Percentage of respondents reporting sleep disturbance	Percentage of respondents reporting to be highly sleep disturbed
Road traffic	Passenger cars and taxis	7	2
	Delivery vans	3	1
	Lorries	6	3
	Buses	2	1
	Motor cycles and motocross cycles	5	2
	Mopeds	10	4
	Motor-assisted bicycles	4	2
	Military vehicles	0	0
Air traffic	Passenger and cargo aircraft	4	2
	Recreational, executive and advertising aircraft	0	0
	Military aircraft (other than helicopters)	2	1
	Helicopters	1	0
Rail traffic	Trains	2	1
	Trams	0	0
	Light rail vehicles	0	0

Shipping	Commercial shipping	0	0
	Pleasure craft	0	0
Commercial, industrial and professional activities	Retail areas	0	0
	Factories and business premises	1	0
	Loading/unloading sites, etc	1	1
	Lorry parks	1	0
	Shunting yards and rail yards	1	0
	Building and demolition sites	1	0
	Road building	1	0
	Agricultural tractors	1	0
	Civilian shooting ranges	0	0
	Military exercise areas, shooting ranges, etc	0	0
Recreational activities	Fairs, circuses, amusement parks, etc	3	1
	Discos, dance halls, etc	2	1
	Musical practice facilities	0	0
	Sports fields, stadiums, sports halls, swimming baths, tennis courts	1	0
	Racing, motocross and carting circuits	0	0
	Ultra-light aircraft	0	0
	Model aircraft	0	0
	Mass-participation open-air events	2	1
Noises from neighbouring dwellings	Noises from sanitary and heating systems	3	1
	Contact noise (footsteps on stairs, slamming of doors)	8	3
	Radio, hi-fi, TV	6	3
	DIY equipment	4	2
	Pets	5	2
Other noises in the residential environment	Neighbours gardening noises	1	0
	Noise from public spaces around one's home	1	0
	Noise from children playing outside	2	1
	Noise from street/public greenery maintenance	3	1
	Other human noises	8	3
	Noise from neighbours' pets/animals	5	2
	Church bells, mosques	3	2
	Bottle banks	1	0

In 2000, the Noord-Kennemerland Regional Health Authority carried out a written inventory study[186], in which 7728 people were invited to participate. The response rate was 68 per cent, meaning that approximately 5250 people completed the questionnaire. The subjects came from nine municipalities in the Noord-Kennemerland region (Akersloot, Alkmaar, Bergen, Egmond, Graft de Rijp, Heiloo, Limmen, Schermer and Schoorl). The questionnaire included a number of questions identical to those used for the national inventory study[9]. The levels of high sleep disturbance reported by the respondents are given in Table 22, along with the corresponding data from the national study (for comparison). The percentages of people reporting high sleep disturbance in Noord-Kennemerland are twice the corresponding national figures. Since no noise exposure data is available for Noord-Kennemerland, it is not possible to establish whether the high levels of disturbance are, at least to some extent, the result of noise exposures that are above the national averages. Data from 2000/2001 for the province of North Holland as a whole indicates that 6.3 per cent of homes in the province have an air traffic-related *Lnight* value of 40 dB(A) or higher, compared with 1.9 per cent nationwide[15]. Where noise from motorways, municipal roads and rail traffic is concerned, exposures in North Holland are close to the national averages. The correction of an RIVM report released on 24-05-2004 does not contain a provincial breakdown of urban road traffic levels (i.e. the largest source of noise exposure in the Netherlands)[15], so it is not possible to determine how the noise exposure due to urban road traffic in North Holland compares with that in the country as a whole.

Table 22 Noise-related sleep disturbance in Noord-Kennemerland[186].

Source	Percentage of Noord-Kennemerland respondents reporting to be highly sleep disturbed	Percentage of national survey respondents reporting to be highly sleep disturbed
Mopeds	10	4
Noise from neighbours	7	3
Motor cycles	6	2
Lorries	5	3
Passenger cars	5	2
Aircraft	4	2
Other	3	

Annex **E**

Sleep disorders and sleeping problems

In this annex, the Committee presents an overview of sleep disorders and sleeping problems. Particular attention is paid to insomnia, but other sleep disorders are also considered, albeit in less detail.

E.1 What is insomnia?

Insomnia can occur without being triggered by a particular illness or condition; such insomnia is known as primary insomnia. Secondary insomnia, on the other hand, is a consequence of some other illness or condition. Definitions of primary insomnia are given in the *ICD-10 classification of mental and behavioral disorders: clinical description and diagnostic guidelines* published by the WHO[90], in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) of the *American Psychiatric Association*[187], and in the *Beknopte handleiding bij de Diagnostische Criteria van de DSM-IV (Guidance Notes to Accompany the Diagnostic Criteria of the DSM-IV)* produced by the Netherlands Association for Psychiatry (NVvP)[188]. In the latter publication, primary psycho-physiological insomnia is defined as a condition that satisfies the following criteria:

- The principal complaint is difficulty getting to sleep or staying asleep, or not feeling refreshed after sleep, persisting for at least a month.
- The sleep disorder (or the associated daytime tiredness) causes significant suffering or impairment of the sufferer's social or occupational performance or ability to function in some other important field.

- The sleep disorder does not only occur in the context of narcolepsy, sleep-related respiratory disorder, circadian rhythm-related sleep disorder or parasomnia.
- The sleep disorder is not a consequence of the direct physiological effects of a substance (narcotic, medication) or a somatic condition.
- The sleep disorder does not only occur in the context of another psychological disorder (such as a depressive disorder, generalised anxiety disorder, or delirium).

The occurrence of chronic primary psycho-physiological insomnia is seen as the coincidence of endogenic causal factors, initiatory factors and sustaining factors[189]. Endogenic causal factors are physiological factors such as raised heart rate, increased muscle tension and raised body temperature, together constituting a raised physiological state of arousal, and psychological factors such as anxiety, nervousness and the inability to clear the mind[190].

Factors that sustain insomnia and therefore act as obstacles to recovery include poor (non-adapted) sleeping habits (inappropriate use of somnifacient drugs, staying in bed too long, keeping irregular hours, excessive napping during the day) and worrying about the possible consequences of not getting enough sleep (anxiety about failure during the day, anxiety about losing control over situations, acquired sense of helplessness).

According to Vgontzas *et al*[191], their epidemiological research supports the hypothesis that primary insomnia mainly involves chronic hyper-arousal, which is evident not merely at night, but around the clock. They take the view that relatively little research has been carried out into the effects of primary insomnia on the cardiovascular system. They argue that their results indicate that people with primary insomnia are not only more likely to suffer from psychological conditions, but also from physical conditions such as hypertension and obesity (plus the associated metabolic abnormalities). Accordingly, the researchers argue that the focus of treatment should be the hyper-arousal, rather than the insomnia, which is merely a consequence of the hyper-arousal.

According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV)[187] and the NVvP's *Guidance Notes*[188], secondary insomnia (insomnia in association with another psychological disorder) is a condition that satisfies the first four criteria given above for primary insomnia, is additionally associated with a so-called 'Axis I' or 'Axis II' disorder (such as a depressive disorder, a generalised anxiety disorder, or an adjustment disorder accompanied by anxiety), and is sufficiently serious to warrant separate medical attention.

Secondary insomnia can also be a consequence of other medical conditions, such as pain, depression, night-time restless legs syndrome and alcoholism. Working variable shifts, including night shifts, can induce or aggravate chronic insomnia.

E.2 How prevalent is insomnia?

Ohayon describes more than fifty studies concerned with the prevalence of insomnia in a broad sense (i.e. not merely primary psycho-physiological insomnia) in the population at large[192]. The reviewed studies include estimates of the prevalence of insomnia, based on four distinct criteria: difficulty getting to sleep or staying asleep, night-time manifestations of insomnia accompanied by daytime problems arising from lack of sleep, self-reported dissatisfaction with sleep quality, and insomnia diagnosed on the basis of DSM-IV. Approximately a third of the general populace is sometimes affected by insomnia satisfying the first criterion. When the second definition is applied, 9 to 15 per cent of the population are reckoned to be affected. Under the third definition, 8 to 18 per cent of the population suffer from insomnia. By application of the DSM-IV classification, one arrives at a figure of 6 per cent for the average prevalence of insomnia in the population at large. However insomnia is defined, it is more prevalent among women than among men. Furthermore, insomnia becomes more common with increasing age, except when defined on the basis of self-reported dissatisfaction with sleep quality.

Prevalence can also be expressed in terms of the estimated probability of suffering a significant sleep disorder at some time in one's life. On this basis, the prevalence of insomnia is put at roughly 30 per cent[193]; in other words, the average Dutch person has approximately a one-in-three chance of falling victim to a significant sleep disorder at some time or other.

A German study involving two thousand adult subjects looked at the possibility of a link between insomnia (as defined in the DSM-IV) and quality of life (as measured using the abbreviated SF-36 questionnaire)[194]. Some 22 per cent of insomniacs rated their quality of life as 'poor' and 28 per cent as 'good', while the corresponding figures for subjects without sleeping problems were, respectively, 3 and 68 per cent. These figures must be treated with caution, however, since subjects' quality of life will have been influenced not only by their insomnia, but also by other illnesses and conditions.

Sleeping problems are not confined to adults. Kim *et al*[195] asked 1365 Chinese youngsters aged twelve to eighteen about any sleeping problems they might have. Nearly 17 per cent reported symptoms of insomnia, including difficulty getting to sleep (11 per cent), waking up in the night (6 per cent) and waking up too early in the morning (2 per cent).

A great deal of research has been carried out into the prevalence of insomnia not only in the population at large, but also in particular groups. Hence, insomnia is known to be much more common among people affected by certain illnesses and medical conditions than in the overall population[196-221]. For example, women who are pregnant or have been pregnant in the last twelve months or so are at increased risk of insomnia[222].

E.3 The consequences of insomnia and sleeping problems; association with other illnesses and medical conditions

E.3.1 The direct consequences of insomnia

According to Stolk *et al*[91], insomnia has a substantial negative effect on quality of life. In the quality-of-life weighting system developed by this team, insomnia, as diagnosed by a GP, has a quality-of-life weighting of 0.83. In other words, a year suffering from insomnia 'costs' 0.17 years of healthy life. Various other authors have also reported negative effects of insomnia on quality of life[18,194,223-226]. People with chronic insomnia of any kind also tend to perform less well at work and suffer memory and concentration problems[227]. Insomniacs make disproportionately great use of healthcare facilities and medications, including somnifacient drugs and sedatives[18,194,223,224].

E.3.2 Association of insomnia with other medical conditions and illnesses

When considering the relationships between insomnia and other medical conditions and illnesses, it is important to distinguish between an association and a causal relationship. Many researchers have reported an association between certain abnormalities, but have failed to demonstrate the link between cause and effect. Schwartz *et al* made an extensive survey of insomnia, *cardiovascular disease* and *mortality risk* on the basis of epidemiological research data[228]. They consider it likely that insomnia and the associated daytime tiredness are part of a more general syndrome that is associated with chronic stress, causes autonomous dysfunction and brings an increased risk of cardiovascular disease. Shaver *et al*[201] drew a similar conclusion on the basis of a study of middle-aged women.

Age is not in itself a determining factor in the occurrence of insomnia[209,225,229-231], which is attributable more to age-related phenomena, such as increasing lack of physical activity, changes involving other lifestyle factors (obesity, use of alcohol), dissatisfaction with the social environment, and illnesses and abnormalities.

With a view to establishing whether chronic insomnia increased the risk of *hypertension*, Suka *et al* conducted a five-year longitudinal study involving 4,800 Japanese workers[232]. Their conclusion was that people who have difficulty getting to sleep and people who have difficulty staying asleep are more likely to develop hypertension (OR respectively 1.9 and 2.0).

Numerous studies have been carried out into the link between *depression* and (sometimes ill-defined) insomnia[196,198,201,206,207,211,212,215,218,220,221,224-226,233-246]. In most of these studies, a statistically significant association was found, but no causal relation-

ship demonstrated. One exception in this regard was the twelve-year longitudinal study by Mallon *et al*[220]. Among women, insomnia at the start of the research period proved to be a statistically significant predictor of the development of depression during the course of the study (*odds ratio* = 4.1). Insomnia was not found to be a predictor of subsequent depression in men, however.

E.3.3 *Association of sleep disorders and sleeping problems with road traffic accidents*

Ohayon *et al*, who have carried out a large number of epidemiological studies[192,213,214,231,244,247-255], take the view that *dissatisfaction with sleep quality* is much more closely related to sleep pathology than the phenomena of insomnia as such.

It is often assumed that sleeping problems play a role in *road traffic accidents* (RTAs). In this context, it is important to distinguish between sleeping problems and incidental sleep deprivation. Having analysed data from the 1985 *CARfile study*, Webb[256] concluded that drowsiness was the primary cause of 1.6 per cent of accidents.

Connor[257] produced an extensive survey of the significance of sleep disorders in RTAs. Analysis of data from the cross-sectional studies produced no evidence of an association between insomnia and the probability of involvement an RTA. However, the case-control study[258] did show up a statistically significant association between sleep apnoea and the probability of injury in an RTA.

E.3.4 *Association of sleep disorders and sleeping problems with occupational accidents*

A number of studies indicate that sleeping problems increase the probability of involvement in a (fatal) occupational accident[256,259-263]. Over a twenty-year period, Akerstedt *et al*[259] interviewed 47,860 people (men and women) by phone regarding sleep and health factors and regarding specific work-related factors. By studying a register of deaths (from which cases of suicide were excluded), the researchers identified 166 fatal occupational accidents. Analysis found the following to be statistically significant predictors of involvement: gender, sleeping problems in the two weeks prior to the interview (relative risk 1.6, 95 per cent confidence interval 1.2 to 2.9) and working outside normal daytime working hours (relative risk 1.9, 95 per cent confidence interval 1.1 to 2.5).

A study of 880 construction workers by Chau and Gauchard[260] revealed that sleeping problems increased the probability of involvement in an accident with a moving object on site (*odds ratio* 2.3, 95 per cent confidence interval 1.3 to 4.1).

The same researchers[261] made a comparison between 427 women who had taken sick leave as a result of falling at work after (physically) losing their balance, and a control group of 427 women. On the basis of interviews conducted by industrial doctors, it

was concluded that there was an association between sleeping problems and the risk of falling at work.

Lindberg *et al*[262] undertook a prospective study, in which 2,874 men completed a questionnaire at the outset, and 2,009 completed a follow-up questionnaire ten years later. Information about occupational accidents was obtained from a national register; it was found that 247 of the 2009 men who completed both questionnaires had been involved in a total of 345 accidents. Men who at the beginning of the study reported both 'napping' and feeling sleepy during the day proved to have been involved in more occupational accidents; the link was statistically significantly, even after correcting for numerous other factors capable of influencing the association between sleeping problems and occupational accidents (odds ratio 2.2, 95 per cent confidence interval 1.3 to 3.8). No statistically significant association was found involving men who napped but did not feel sleepy during the day, or involving men who felt drowsy during the day but did not take naps.

Melamed and Oksenberg[263] interviewed 532 industrial workers in order to gather information on the influence of drowsiness on the probability of involvement in an accident at work. By asking numerous questions, the number of accidents in the two years prior to the interviews was determined. Analysis of the responses revealed that the probability of involvement in an occupational accident was higher, to a statistically signifi-

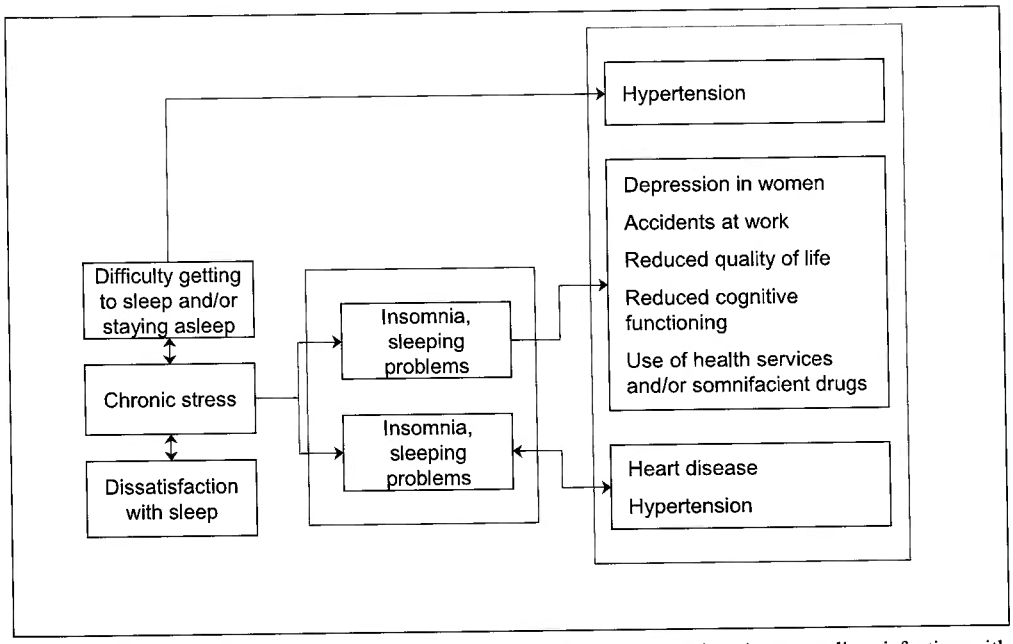


Figure 29 Causes and backgrounds of insomnia and sleeping problems (chronic stress, dissatisfaction with sleep, difficulty getting to sleep and/or staying asleep), consequences of insomnia/sleeping problems (indicated with s) and the associations between insomnia/sleeping problems and other illnesses and medical conditions (indicated with s).

cant extent, among workers who reported feeling drowsy at work than among those who did not (odds ratio 2.2, 95 per cent confidence interval 1.3 to 3.8).

Figure 29 illustrates the causes and consequences of insomnia and sleeping problems, as well as the association that insomnia and sleeping problems have with other illnesses and medical conditions.

E.3.5 *Conclusions*

On the basis of the foregoing, the following conclusions may be drawn:

- Insomnia has a negative effect on quality of life. People with chronic insomnia perform less well at work and experience memory and concentration problems. Insomnia increases usage of healthcare facilities and the consumption of medications, such as somnifacient drugs and sedatives. Insomnia and the associated daytime tiredness are part of a more general syndrome that is associated with chronic stress and causes autonomous dysfunction.
- People who are affected by insomnia are more likely to suffer depression (women), obesity (plus the associated metabolic abnormalities) and cardiovascular disease.
- People with sleeping problems are more likely to develop hypertension.
- People with general sleeping problems (difficulty getting to sleep, difficulty staying asleep, waking spells at night) are more likely to be involved in occupational accidents.

In addition, the following conclusions may be drawn regarding heightened sensitivity to insomnia:

- Because of the association of insomnia with depression, hypertension, obesity and cardiovascular disease, people with these conditions may be regarded as particularly sensitive to insomnia. Women who are pregnant or have been pregnant in the last twelve months or so are also more likely than the average person to experience a period of insomnia.
- Age is not in itself a determining factor in the occurrence of insomnia[209,225,229-231], which is attributable more to age-related phenomena, such as increasing lack of physical activity, changes involving other lifestyle factors (obesity, use of alcohol), dissatisfaction with the social environment, and illnesses and abnormalities. As a result, older people may also be regarded as particularly sensitive to insomnia and sleeping problems.

Annex **F**

Health Council Advisory Report *Assessing Noise Exposure for Public Health Purposes* (1997/23)

Methodology

In the Health Council's 1997 advisory report *Assessing Noise Exposure for Public Health Purposes*[8], the 'Uniform environmental noise exposure metric' Committee of the Health Council proposed a system for determining noise exposures representative of the twenty-four-hour daily cycle (EEL) and the overnight period (ENEL). This method involves five steps:

1 *Frequency weighting of acute sound pressure levels*
The Committee opted for A-weighting, i.e. sound pressure levels expressed in dB(A), for both the twenty-four-hour daily cycle and the overnight period

2 *Adjustment for special characteristics and combinations of sound pressure levels*
The Committee assigned adjustment factors, as described below, to noises and noise situations involving characteristics a, b, and c in a twenty-four-hour daily cycle or an overnight period:

a Exposure to low-level industrial noise without impulse components: adjustment factor above 60 dB(A), 0 dB(A); at 40 dB(A), 10 dB(A); in the range between, calculated by linear interpolation

b Situations in which the noise includes audible tones: adjustment factor between 0 and 5 dB(A), depending on the frequency of the tone and the difference

between the sound pressure level of the tone and the prevailing background sound pressure level

c Situations in which the noise includes (strong) impulse components: adjustment factor 5 dB(A) for impulse noise (such as the sound of a low-flying military jet, a car door slamming or church bells ringing) and 12 dB(A) for very impulse-like (such as gunshot noise, metal beating, pneumatic hammering, shunting of rail rolling stock).

The Committee attached certain qualifications to its proposal of the adjustment factors for use in the assessment of a situation over a twenty-four-hour daily cycle. With regard to assessment of the overnight period, the Committee indicated that consideration should be given to further adjustments to take account of the possibility of sleep disturbance. "Although scientific evidence is lacking, the Committee considers it likely that night-time exposure to noise with the characteristics listed above would result in increased sleep disturbance. It therefore considers it prudent to provisionally apply these adjustments also in deriving the *ENEL* metric, and recommends further research on this matter."

For the combination of sound pressure levels for parts of a day, including the application of adjustment factors for intervals in which sound with special characteristics occurs, the Committee recommended working on the basis of the equivalent sound pressure level over a given period.

3 The combination of (corrected) equivalent sound pressure levels for parts of a day to give a value that is representative for a twenty-four-hour daily cycle
The Committee recommends adjustment factors of 0 dB(A) for the daytime (7am to 7pm), 5 dB(A) for the evening (7pm to 11pm) and 10 dB(A) for the night (11pm to 7am). The corrected equivalent sound pressure levels are exponentially averaged. Step 3 is not necessary when calculating an ENEL, since the combination of day, evening and night values is clearly not relevant in relation to a night-only metric.

4 *The combination of daily exposure values to give a value that is representative for a year*
No seasonal or weekday/weekend adjustment factors are proposed. The equivalent sound pressure levels for each twenty-four-hour daily cycle of a year are exponentially averaged. This results in a Ladjusted,den value. For Ladjusted,night, the Committee also recommends the exponential averaging of equivalent sound pressure levels for the overnight period.

5 *Noise source-related adjustments*
The final step in the construction of uniform exposure metrics for the twenty-four-hour daily cycle and the night involves adjusting *Ladjusted,den* and *Ladjusted,night* so that the exposure-response relationships of the various noise sources are in line

with that of a selected source. The particular reference source selected by the Committee was road traffic. The proposed effect metric for the twenty-four-hour exposure was the percentage of people experiencing high annoyance. However, other effect metrics are generally used in other countries (in Germany for example, the percentage of people experiencing annoyance is used). Consequently, the Committee, being made up of experts from various countries, developed the EEL on the basis of the high annoyance percentage merely as an example. Using road traffic noise as the reference source, differences between EEL and Ladjusted,den are given for aviation and rail traffic noise. Depending on the noise exposure involved, these differences are between +3 and +5 dB(A) for air traffic, and between -1 and -8 dB(A) for rail traffic. The effect metrics given by the Committee for the night are the percentage of people reporting high sleep disturbance and the annual frequency of awakening due to a noise source. Because the information available at the time regarding the exposure-response relationships for road, rail and air traffic noise was not considered sufficiently reliable, the Committee decided against constructing an ENEL.

Applicability

The 'Uniform environmental noise exposure metric' Committee considered the methodology valid for the assessment of noise in most more or less stable situations, but not for the assessment of changes in noise situations over the short term. The Committee also pointed out that the method was not designed for use in relation to low-frequency noise, noise from incidental sources (such as rescue helicopters, ultra-light aircraft and advertising aircraft), neighbourhood noise or noise from neighbours.

Annex **G**

The distribution of traffic-related noise exposure in the Netherlands

Enclosure accompanying letter (reference 1034/04 LOK/HD/wh) from HSMA Diederen, Environmental and Nature Planning Office, RIVM, dated 23 June 2004 to the Health Council.

Updated distributions for *Lden* and *Lnight*

Table 1 Lden % of dwellings per noise category, cumulative distribution of road traffic, rail traffic and air traffic.

	0-50 dB	51-55 dB	56-60 dB	61-65 dB	66-100 dB
NBG2001[a]	32	31	25	9	3
2003[b]	37	31	22	8	2

[a] Corrected memorandum *Nachtelijke Blootstelling Geluid* (*Night-time Exposure to Noise*), dated 24-5-2004; noise maps 100 m resolution.

[b] Noise maps 25 m resolution.

Table 2 Lnight % of dwellings per noise category, cumulative distribution of road traffic, rail traffic and air traffic.

	0-40 dB	41-45 dB	46-50 dB	51-55 dB	56-60 dB	61-100 dB
NBG2001[a]	23	27	30	15	4	1
2003[b]	29	29	26	12	3	1

[a] Corrected memorandum *Nachtelijke Blootstelling Geluid* (*Night-time Exposure to Noise*), dated 24-5-2004; noise maps 100 m resolution.

[b] Noise maps 25 m resolution.

With regard to *Lden* and *Lnight*, we advise adhering to the distributions given for 2003 in Tables 1 and 2 above. These figures are the best estimate we can currently make, on the basis of the most recent information and modelling.

Explanatory notes

The distributions given above are based on noise maps plotted using RIVM's EMPARA model. This model makes use of data files with information on the positions of roads and railways, from which the associated noise exposures are calculated for grid squares using standard mathematics techniques. Noise maps for air traffic have also been obtained from NLR.

Since the start of 2004, noise exposures have been calculated for grid squares of 25 by 25 metres (as opposed to the old 100 by 100 metre squares). The finer resolution allows for more accurate reflection of the spatial variation in sound pressure levels actually occurring in the vicinity of roads and railways. The updated distribution data therefore differs from the distribution data published in 2001, but not to a particularly great extent.

The method we have used is described in the Ministry of Housing, Spatial Planning and the Environment's publication *Naar een Landelijk Beeld van Verstoring* (*Towards a National Picture of Disturbance*), publication no. 12, 1997. The accumulated noise exposure includes the values for road traffic, rail traffic and air traffic.

The reliability of the distribution data in Tables 1 and 2 depends not only on the scale used, but also on the current validity of the traffic data. Hence, it is worth stating that the data for motorways (obtained from AVV), railways (obtained from ASWIN), provincial roads (obtained from ERC) and air traffic (obtained from NLR) was updated for the 2004 Environmental Balance (based on the situation in 2003) and are therefore up to date.

The basic data that we used for municipal roads, however, was incomplete and somewhat out of date. To enable us to nevertheless obtain a full picture, we estimated the current traffic volumes on the majority of municipal roads using information about road types and a limited set of data from recent traffic counts. Because the municipal traffic data is to a large extent not based on recent volumetric figures and takes no account of features such as screens and quiet asphalt, the calculated noise exposures for a given location may differ considerably from the values that proper acoustic tests would return. However, it is assumed that any anomalies will, statistically speaking, balance one another out, so that the picture for the country as a whole and the associated distribution pattern constitute a reasonable approximation of the actual situation.

Address

Fingal County Council
Planning Enforcement
County Hall
Main Street
Swords
Co Dublin
K67 X8Y2

June 28th, 2023

Re: Dublin Airport Northern Runway

Dear Sir/Madam,

Please find attached a copy of a filled out complaint form regarding alleged Unauthorised Development with respect to the daa and the operation of Night time flights at Dublin Airport.

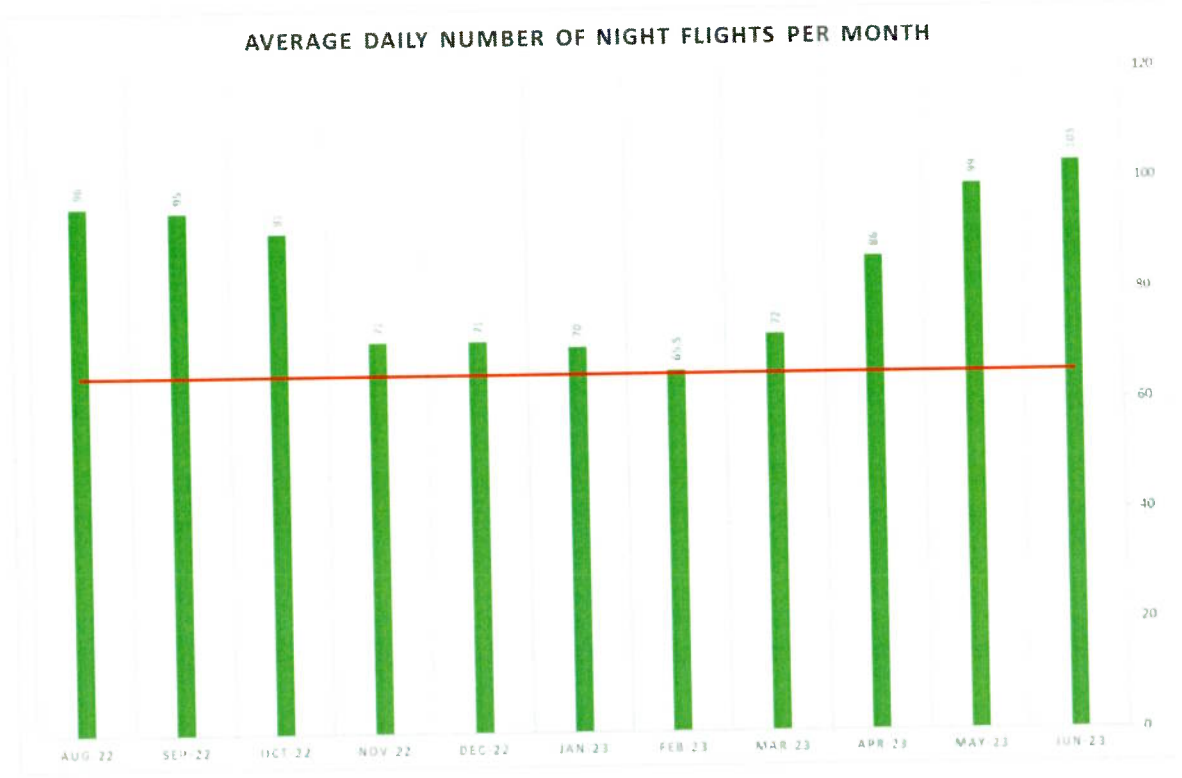
We note that the North Runway became operational on August 24th, 2022.

An Bord Pleanala granted planning permission for the North Runway in 2007 (F04A/1755). Condition 5 of the planning permission states that:

> 5. On completion of construction of the runway hereby permitted, the average number of night time aircraft movements at the airport shall not exceed 65/night (between 2300 hours and 0700 hours) when measured over the 92 day modelling period as set out in the reply to the further information request received by An Bord Pleanála on the 5th day of March, 2007.
>
> **Reason:** To control the frequency of night flights at the airport so as to protect residential amenity having regard to the information submitted concerning future night time use of the existing parallel runway.

92 day Summer period

Since August 2022, the average daily number of night-time flights per month has exceeded 65 for each month. From analysis of the further information requests during the Oral Hearing in 2006, it is clear that the intention of An Bord Pleanala was for a 65 movement limit once the North Runway had completed construction. The daa have stated that the fact that Condition 5 specifies the 92 day Summer average allows them flexibility not to enforce it until the Summer period. We are now in this Summer period and the daa continue to ignore Condition 5. Below is the average daily number of night-time flights per month since the North Runway opened.There are in excess of 100 flights since June 15th.



To avoid any confusion regarding the wording of Condition 5, below are flights that occurred over the night of June 25th/26th inside the 92 day summer period.
There were 106 aircraft movements between 23:00 and 07:00.

The list of aircraft and their arrival and departure times are as follows:

4CA280	EIN3LG	EI-DEJ	Airbus A320-214	6/25/2023 23:00
4CA4EB	RYR9SJ	EI-DPG	Boeing 737-8AS	6/25/2023 23:02
4CA217	EIN33W	EI-DEF	Airbus A320-214	6/25/2023 23:04
4CAFD2	RYR7BW	EI-GXM	Boeing 737-800WL	6/25/2023 23:09
4CA4F2	RYR3KR	EI-DPN	Boeing 737-8AS	6/25/2023 23:11
4CA214	EIN34Y	EI-DEE	Airbus A320-214	6/25/2023 23:13
4CA5C8	EIN40W	EI-DVE	Airbus A320-214	6/25/2023 23:15
4CA76A	RYR19YK	EI-EFO	Boeing 737-8AS	6/25/2023 23:18
4CA63B	EIN529	EI-FNJ	Airbus A320-216	6/25/2023 23:23
4A4D79	HYS252	YR-SKY	Airbus A320-232	6/25/2023 23:25
4A1921	FIA711	YR-FIA	Airbus A320-233	6/25/2023 23:26
4CA814	RYR60YF	EI-EKV	Boeing 737-8AS	6/25/2023 23:33
4CA245	RYR1CG	EI-DCO	Boeing 737-8AS	6/25/2023 23:40

4CADFE	RYR3JM			6/25/2023 23:42
4CAC55	RYR94GT	EI-HGR	Boeing 737-8200	6/25/2023 23:44
4CAC89	RYR47HR	EI-IFS	Boeing 737-8200	6/25/2023 23:46
4CA251	RYR4W	EI-DCR	Boeing 737-8AS	6/25/2023 23:50
4CA854	RYR2WK	EI-EML	Boeing 737-8AS	6/25/2023 23:57
4CA770	EIN4VM	EI-EDS	Airbus A320-214	6/26/2023 0:00
4CA2CB	EIN63K	EI-DER	Airbus A320-214	6/26/2023 0:10
4CA6C4	EIN24K	EI-DVI	Airbus A320-214	6/26/2023 0:13
4CAC1F	RYR5TC	EI-HEZ	Boeing 737-8200	6/26/2023 0:20
4CA215	EIN7VT	EI-DEG	Airbus A320-214	6/26/2023 0:22
4CA837	EIN497	EI-GAM	Airbus A320-214	6/26/2023 0:24
4CA27D	RYR80RR	EI-DHP	Boeing 737-8AS	6/26/2023 0:28
4CA4F8	RYR3TD	EI-DPV	Boeing 737-8AS	6/26/2023 0:31
4CAC88	RYR1443	EI-IFR	Boeing 737-8200	6/26/2023 0:33
4CADE4	RYR30UE	EI-IGY	Boeing 737-8200	6/26/2023 0:35
3412D3	EIN737	EC-HDS	Boeing 757-256	6/26/2023 0:38
4A1921	FIA712	YR-FIA	Airbus A320-233	6/26/2023 0:40
4D225B	RYR11YP	9H-QCU	Boeing 737-8AS	6/26/2023 0:40
4CAC53	RYR2BY	EI-HGO	Boeing 737-8200	6/26/2023 0:42
4CAD64	RYR9YZ	EI-HMY	Boeing 737-8200	6/26/2023 0:44
4CA92D	EIN56V	EI-DVM	Airbus A320-214	6/26/2023 0:46
49520F	TAP26T	CS-TPO	Embraer 190-100LR	6/26/2023 0:53
4CAA58	RYR733K	EI-EVS	Boeing 737-8AS	6/26/2023 0:59
4CA293	EIN4RL	EI-DEM	Airbus A320-214	6/26/2023 1:04
4D244D	TOM71D	9H-GKJ	Airbus A320-232	6/26/2023 1:14
4CAD10	RYR52CV	EI-HGX	Boeing 737-8200	6/26/2023 1:16
4CA75E	RYR45HY	EI-EFC	Boeing 737-8AS	6/26/2023 1:18
4CA2C3	RYR9PR	EI-DLF	Boeing 737-8AS	6/26/2023 1:22
4D23A4	TOM2YE	9H-GKK	Airbus A320-232	6/26/2023 1:24
4CA802	RYR930J	EI-EKD	Boeing 737-8AS	6/26/2023 1:27
4CA76A	RYR7AN	EI-EFO	Boeing 737-8AS	6/26/2023 1:32
4CA4F2	RYR7EH	EI-DPN	Boeing 737-8AS	6/26/2023 1:43
4CAC86	RYR275Y	EI-HMZ	Boeing 737-8200	6/26/2023 1:55
4CADA4	RYR3ZV	EI-IFV	Boeing 737-8200	6/26/2023 1:58
4CA2C9	EIN799	EI-DEP	Airbus A320-214	6/26/2023 2:00
4D2317	BCS5QC			6/26/2023 2:03

4CADBF	RYR8ZK	EI-IGM	Boeing 737-8200	6/26/2023 2:05
4CAFB3	RYR4QA	EI-GSG	Boeing 737-800WL	6/26/2023 2:07
4CA15D	EIN4GJ	EI-CVB	Airbus A320-214	6/26/2023 2:09
4CA27F	EIN499	EI-DEK	Airbus A320-214	6/26/2023 2:11
4CA281	EIN5HL	EI-DEI	Airbus A320-214	6/26/2023 2:20
4CADC4	RYR69SB	EI-IGG	Boeing 737-8	6/26/2023 2:28
440BC1	BCS2882	OE-LND	Boeing 757-223SF	6/26/2023 2:59
4CA15C	EIN58R	EI-CVC	Airbus A320-214	6/26/2023 3:02
451D99	BCS3TW	LZ-CGS	Boeing 737-4Q8F	6/26/2023 3:58
A48850	UPS248	N391UP	Boeing 767-304ERSF	6/26/2023 4:01
4CA614	EIN13K	EI-FNG	Airbus A330-302	6/26/2023 4:13
40087	ETH518	ET-ARE	Boeing 787-8	6/26/2023 4:23
4CAA4F	BCS2937	EI-STS	Boeing 737-48EF	6/26/2023 4:26
4CA5C7	EIN104	EI-DUZ	Airbus A330-302	6/26/2023 4:28
40102	ETH574	ET-ASH	Boeing 787-8	6/26/2023 4:45
4CABD2	EIN1TC	EI-LRF	Airbus A321-253NX\/LR	6/26/2023 4:48
4CA15E	EIN1MN	EI-DAA	Airbus A330-202	6/26/2023 4:50
4CABD4	EIN13R	EI-LRH	Airbus A321-253NX\/LR	6/26/2023 4:53
A48850	UPS248	N391UP	Boeing 767-304ERSF	6/26/2023 5:03
40106	ETH500	ET-ASL	Boeing 777-360ER	6/26/2023 5:09
4CA7D8	EIN122	EI-GAJ	Airbus A330-302	6/26/2023 5:11
40087	ETH518	ET-ARE	Boeing 787-8	6/26/2023 5:37
4CA9BB	EIN11P	EI-LRB	Airbus A321-253NX\/LR	6/26/2023 5:47
4CA2C3	RYR16UU	EI-DLF	Boeing 737-8AS	6/26/2023 5:55
40102	ETH574	ET-ASH	Boeing 787-8	6/26/2023 5:57
3C6483	DLH983	D-AIDC	Airbus A321-231	6/26/2023 6:06
AA7BA7	AAL724	N775AN	Boeing 777-223ER	6/26/2023 6:08
4CADBF	RYR4KU	EI-IGM	Boeing 737-8200	6/26/2023 6:10
4CA245	RYR1WZ	EI-DCO	Boeing 737-8AS	6/26/2023 6:12
4CA13D	EIN66V	EI-CVA	Airbus A320-214	6/26/2023 6:13
06A0B6	QTR56X	A7-BCR	Boeing 787-8	6/26/2023 6:15
39E68C	AFR47GQ	F-HZUM	Airbus A220-300	6/26/2023 6:17
4CADC4	RYR37GR	EI-IGG	Boeing 737-8	6/26/2023 6:19
4D23A4	TOM1TE	9H-GKK	Airbus A320-232	6/26/2023 6:21
4CADE4	RYR62ZZ	EI-IGY	Boeing 737-8200	6/26/2023 6:23
486493	KLM68T	PH-NXJ	Embraer 195-400STD-E2	6/26/2023 6:24

4CADA4	RYR36LU	EI-IFV	Boeing 737-8200	6/26/2023 6:26
4CAFB3	RYR6MR	EI-GSG	Boeing 737-800WL	6/26/2023 6:30
AB10CB	AAL722	N812AA	Boeing 787-8	6/26/2023 6:30
4CA7B5	RYR23XX	EI-EFZ	Boeing 737-8AS	6/26/2023 6:32
4CAC86	RYR12UY	EI-HMZ	Boeing 737-8200	6/26/2023 6:34
4CA251	RYR80CQ	EI-DCR	Boeing 737-8AS	6/26/2023 6:35
40106	ETH500	ET-ASL	Boeing 777-360ER	6/26/2023 6:37
4CA2CA	EIN23F	EI-DES	Airbus A320-214	6/26/2023 6:39
4CAC84	RYR22MD	EI-HGF	Boeing 737-8200	6/26/2023 6:41
4CA76A	RYR30QZ	EI-EFO	Boeing 737-8AS	6/26/2023 6:42
4CA15C	EIN59K	EI-CVC	Airbus A320-214	6/26/2023 6:44
4CA92D	EIN40G	EI-DVM	Airbus A320-214	6/26/2023 6:45
4CA217	EIN60H	EI-DEF	Airbus A320-214	6/26/2023 6:46
4CA640	EIN45F	EI-DVH	Airbus A320-214	6/26/2023 6:48
4CAA58	RYR952D	EI-EVS	Boeing 737-8AS	6/26/2023 6:49
4CAD7A	EIN1GT	EI-NSA	Airbus A320-251N	6/26/2023 6:51
4CAC1F	RYR30DG	EI-HEZ	Boeing 737-8200	6/26/2023 6:52
4CA280	EAI05ED	EI-DEJ	Airbus A320-214	6/26/2023 6:54
4CAC53	RYR3CL	EI-HGO	Boeing 737-8200	6/26/2023 6:55
4CABD4	EIN2MW	EI-LRH	Airbus A321-253NX\/LR	6/26/2023 6:56
4CA854	RYR7HF	EI-EML	Boeing 737-8AS	6/26/2023 6:58

Condition 5 is one of the two conditions that the daa are actively trying to amend via their planning application F20A/0668. This application is currently under appeal with An Bord Pleanala. The planning notice clearly states that it's the daa's intention to replace Condition 5 with a Noise Quota System (NQS).

The accompanying documentation for application F20A/0668 clearly states that the daa believe that flights will be lost once the North Runway commences operations. In

section 3.2.10 of the revised EIAR, the daa provide figures for 2022-2025 and the projected lost passengers:

3.2.10 Table 3-1 presents the assessed impact of the Permitted Scenario is a cumulative loss over the 4-year period 2022-2025 of 6.3m passengers when compared with the Proposed Scenario.

Table 3-1: Annual Traffic Impact Summary (millions of passengers)

	2022	2023	2024	2025	2022-2025 Total
Proposed	21.0	26.7	30.8	32.0	110.5
Permitted	19.6	24.9	29.3	30.4	104.2
Difference	-1.4	-1.8	-1.6	-1.6	-6.3[1]

Source: Quantification of Impacts on Future Growth, Update 2022 - 2025 Period (Mott MacDonald, 2021)

The daa predicted a loss of 1.4m passengers in 2022 due to Conditions 3(d) and 5. But the daa are currently ignoring Condition 5 and lost no passengers.

So the daa have used Condition 5 to mislead Fingal County Council into granting them permission for F20A/0668 and yet they are ignoring Condition 5.

The daa have stated in correspondence that they are relying on the advice of the Commission for Aviation Regulation (CAR) who is responsible for slots at Dublin Airport, for their non-compliance with Condition 5. The CAR is not the planning authority, Fingal County Council is.

In a document received via FOI titled '*20171017_388690 - DAA Operating Restrictions - 2017 report v1.2 Final_Redacted.pdf*', the daa's consultants Mott MacDonald, clearly state that '*Although the night restriction compliance is measured over the 92 day period,* ***spirit of the restrictions would require night period scheduling limits to be applied on a year-round basis'***.

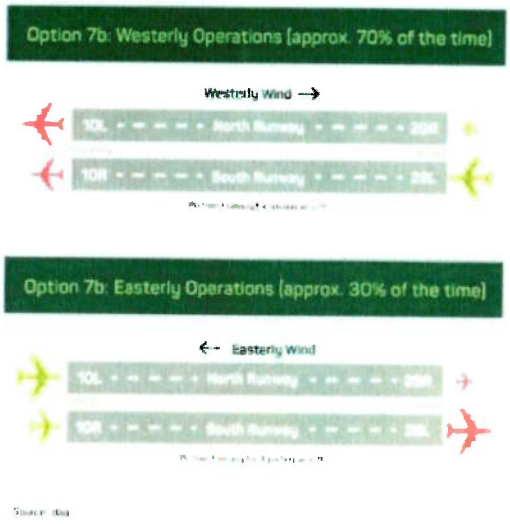
Background

Planning Conditions

- The North Runway planning permission (P1061 21742) contains the following conditions to take effect from completion of the new runway
  - Condition 3(d) states that *Runway 10L:28R shall not be used for take off or landing between 2300 hours and 0700 hours*[1]
  - Condition 5 states that *the average number of night time aircraft movements at the airport shall not exceed 65/night (between 2300 hours and 0700 hours) when measured over the 92 day modelling period*

- This study interprets Condition 5 as follows
  - Night movements are based on actual aircraft landing or taking-off times
  - The 65/night limit is based on the average over the 92 day modelling period (16 June to 15 September)
  - All night operations, including ad hoc operations and unplanned operations (e.g., delayed daytime flights), as well as regularly scheduled night flights are taken into account
  - Therefore, scheduling limits to ensure compliance must take account of aircraft taxi times and make reasonable allowances for delayed flights
  - Although the night restriction compliance is measured over the 92 day period, the spirit of the restrictions would require night period scheduling limits to be applied on a year round basis

Runway Modes of Operation



(1) except in cases of safety, maintenance considerations, exceptional air traffic conditions, adverse weather, technical faults in air traffic control systems or declared emergencies at other airports

As current operations at Dublin Airport breach Condition 5 of planning application F04A/1755 and of An Bord Pleanala permission PL 06F.217429, we request that Fingal County Council immediately enforce compliance with the limit of 65 flights at night. The current operations are putting the health of residents at risk.

Yours Sincerely

Signature

_____

Name

Planning Enforcement Fingal County Council

Complaint Form Regarding Alleged Unauthorised Development

(Please read the notes before completing this form)

1. Address of where the alleged unauthorised development is being carried out:	Dublin Airport and surrounding communities
2. Full description of the alleged unauthorised development:	daa operating greater than 65 flights at night contravening Condition 5 of the 2007 Planning Permission for the North Runway (F04A/1755)
3. Date work/use commenced:	2023
4. Name and Address of Property Owner/Occupier:	Dublin Airport Authority, Fingal, Co Dublin
5. Name and Address of person carrying out alleged unauthorised development:	Dublin Airport Authority and airlines arriving and departing from the airport
6. Name and Address of Developer:	Dublin Airport Authority, Fingal, Co Dublin
7. Any other relevant information regarding the location, previous use, etc.	Please see additional information above
8. Your name and address (this information will be kept confidential):	Name Address
9. Your telephone number:	Phone number
10. Your Email address:	email

I HAVE READ THE NOTES RELATING TO THIS FORM AND UNDERSTAND THE IMPLICATIONS OF SAME

Signature: **Signed:**

Date:

Note: Complaints will not be investigated unless name and address are given and the form is signed

PLEASE COMPLETE THIS FORM AND EMAIL TO: planning.enforcement@fingal.ie
OR POST TO ADDRESS BELOW

Fingal County Council
Planning Enforcement
County Hall
Main Street
Swords
Co Dublin
K67 X8Y2

ADDITIONAL INFORMATION

Site location map

ENF No:
23/100B

COMHAIRLE CONTAE FHINE GALL

FINGAL COUNTY COUNCIL

S153 CE 1
TO ISSUE

RECORD OF CHIEF EXECUTIVE'S ORDER

IN THE MATTER OF THE LOCAL GOVERNMENT ACT 2001 (AS AMENDED)

AND IN THE MATTER OF THE PLANNING AND DEVELOPMENT ACT 2000 (AS AMENDED)

SECTION 153 OF THE PLANNING AND DEVELOPMENT ACT 2000 (AS AMENDED)

Section 153 – Decision on Enforcement

SUBJECT
Whether to issue an Enforcement Notice

Lands:	Dublin Airport, Co. Dublin
Planning Permission:	Planning Authority Reg. Ref No: F04A/1755 ABP Ref. No: PL 06F.217429 North Runway Permission - Condition 5
Enforcement Complaint:	Unauthorised development comprising development carried out in non-conformity with Condition 5 of the North Runway Permission (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429)

WHEREAS Dublin Airport Authority plc ("*daa*") obtained a grant of planning permission, following an appeal to An Bord Pleanála, for development comprising, *inter alia*, the development of the North Runway ("*the North Runway Permission*" - Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429) – the said grant of permission was subject to 31 Conditions, including Condition 5 which provides:

> "On completion of construction of the runway hereby permitted, the average number of night time aircraft movements at the airport shall not exceed 65/night (between 2300 hours and 0700 hours) when measured over the 92 day modelling period as set out in the reply to the further information request received by An Bord Pleanála on the 5th day of March, 2007.
>
> **Reason:** To control the frequency of night flights at the airport so as to protect residential amenity having regard to the information submitted concerning future night time use of the existing parallel runway."

The application documentation, including the EIS and information provided by way of further information, the Inspector's Report and the Board Order provide the context to the imposition of Condition 5;

AND WHEREAS a complaint was received by Fingal County Council ("*the Council*"), on 24th March 2023, and subsequent complaints followed, in relation to alleged unauthorised development at the Lands – being non-compliance/non-conformity with Condition 5 of the North Runway Permission (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429) and including an alleged exceedance over the permitted number of night time aircraft movements;

AND WHEREAS pursuant to s.152(1)(a) of the Planning and Development Act 2000 (as amended) ("*the 2000 Act*"), having considered the said complaint, the Council issued a Warning Letter, dated 25th April 2023, to the daa in respect of the alleged unauthorised development – being alleged non-compliance/non-conformity with Condition 5 of the North Runway Permission (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429);

AND FURTHER WHERE the daa responded to the said Warning Letter, pursuant to s.152(4)(b) of the 2000 Act, setting out its response to the allegation of unauthorised development by way of correspondence, dated 23rd May 2023 – which included support documentation;

AND WHERE, as part of the Council's investigation into the matter, further information/clarification was sought from the daa by way of correspondence, dated 9th June 2023 and 15th June 2023, and the daa responded to same by way of correspondence, dated 14th June 2023 and 19th June 2023;

HAVING CONSIDERED, *inter alia*, the complaint received and the responses from the daa, including supporting documentation (including the aforesaid) and having considered the Council's Planning Report, dated 18th July 2023, together with the Appendices to same, prepared as part of the Council's investigation into the alleged unauthorised development and the recommendation therein;

AND NOTING that the Council's Planning Report, dated 18th July 2023, provides, *inter alia*: a summary of the relevant planning history to the Lands – including matters relevant to Condition 5; a summary of the complaint received per s.152; details on the Warning Letter issued pursuant to s.152; outlines and considers the various responses/arguments made by daa in response to the said Warning Letter; a response

to the said responses/arguments made by daa; outlines an interpretation of Condition 5 of the Planning Permission;

AND HAVING NOTED AND CONSIDERED the requirements of section 153 of the 2000 Act, including *inter alia* the following provisions which provide, *inter alia*:

> "(1) As soon as may be after the issue of a warning letter under section 152, the planning authority shall make such investigation as it considers necessary to enable it to make a decision on whether to issue an enforcement notice or make an application under section 160.
>
> ...
>
> (3) A planning authority, in deciding whether to issue an enforcement notice shall consider any representations made to it... and any other material considerations.
>
> ...
>
> (7) Where a planning authority establishes, following an investigation under this section that unauthorised development (other than development that is of a trivial or minor nature) has been or is being carried out and the person who has carried out or is carrying out the development has not proceeded to remedy the position, then the authority shall issue an enforcement notice under section 154 or make an application pursuant to section 160, or shall both issue such a notice and make such an application, unless there are compelling reasons for not doing so...";

RECOMMENDATION of the SENIOR EXECUTIVE PLANNER: Accordingly, in accordance with section 153(1) of the 2000 Act, having considered the proper planning and sustainable development of the administrative area of Fingal County Council including the preservation and improvement of the amenities thereof, and having carried out an investigation such as to enable it to make a decision in accordance with section 153(1) of the 2000 Act and having considered representations made to it under **section 152**(1)(a) and submissions or observations made under **section 152**(4)(b) and any other material considerations, I recommend that an enforcement notice issue pursuant to section 154 of the Planning and Development Act for the following reasons:

- The use of the airport for night-time aircraft movements was, for the reason of protecting residential amenity, limited by An Bord Pleanála in the consent of the North Runway. Night-time use of the airport was limited by Condition 5 to levels of activity submitted by the daa in the course of the application;

- Residential amenity is protected by Condition 5 by way of mitigation of an identified significant impact through the control of the frequency of that impact, to an intensity of use forecast by the daa at the time of the application to extend the airfield by construction of the North Runway. An Bord Pleanála confirmed and determined the magnitude of night-time flights acceptable in its consideration of proper planning and sustainable development. The night-time use was limited in this manner by An Bord Pleanála to address concerns regarding the cumulative impact of the proposal in combination with existing development;

- The North Runway has been constructed and became operational on the 24 August 2022. A scheduling and slot allocation process was undertaken and a summer 2023 operating schedule was determined and is currently in operation;

- The summer schedule which is being carried out is in breach of the limit applied in Condition 5;

- Taking account of the foregoing, it is therefore concluded that by virtue of the scheduled and actual operations reported, the frequency of night flights in Dublin Airport is not in conformity with Condition 5 of the North Runway permission and is for that reason unauthorised development. The 2000 Act, including s.154(5)(a)(ii) provides that the planning authority can issue an Enforcement notice to requirc the daa, to proceed with a development in conformity with Condition 5;

- Unauthorised development is occurring and will continue to occur in non-conformity with Condition 5 and that unauthorised development is occurring at the Lands and development is not being carried out in conformity with Condition 5 of the North Runway Permission (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429);

- The daa has not sought to remedy the said unauthorised development, there are no compelling reasons for not taking enforcement action, having regard to the nature of the unauthorised development at issue and the nature of Condition 5, including the reason/purpose of same;

- In circumstances where unauthorised development is occurring and will continue to occur at Dublin Airport, contrary to Condition 5 of the North Runway Permission (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429) comprising the continued and ongoing exceedance of the permitted average number of night-time (between 2300 hours and 0700 hours)

aircraft movements at the airport – being a permitted average of 65 aircraft movements per night when measured over the 92-day modelling period;

- Noting the nature of the unauthorised development and the evidence presented and matters discussed in the aforesaid Report dated 18th July 2023 and appendices thereto, it is considered that a period of 6 weeks for compliance with the terms of the Enforcement Notice is reasonable and appropriate in the circumstances.

Recommender Hugh O'Neill
P.P.
Senior Executive Planner

ORDER:

The report entitled *Informing a "decision on enforcement" under Section 153 of the Planning and Development Act 2000 (as amended)* from the Senior Executive Planner dated the 18th July 2023 and the appendices attached thereto have been considered. The findings and recommendations and the reasons set out therein **to issue** an enforcement notice pursuant to section 154 of the Planning and Development Act are hereby **ACCEPTED** and **ADOPTED** in this decision.

In accordance with section 153(1) of the 2000 Act, having considered the proper planning and sustainable development of the administrative area of Fingal County Council including the preservation and improvement of the amenities thereof, and having carried out an investigation such as to enable it to make a decision in accordance with section 153(1) of the 2000 Act and having considered representations made to it under **section 152**(1)(a) and submissions or observations made under **section 152**(4)(b) and any other material considerations the Planning Authority hereby **DECIDES** and **SO ORDERS** that an enforcement notice issue pursuant to section 154 of the Planning and Development Act for the following reasons:

- The use of the airport for night-time aircraft movements was, for the reason of protecting residential amenity, limited by An Bord Pleanála in the consent of the North Runway. Night-time use of the airport was limited by Condition 5 to levels of activity submitted by the daa in the course of the application;

- Residential amenity is protected by Condition 5 by way of mitigation of an identified significant impact through the control of the frequency of that impact, to an intensity of use forecast by the

daa at the time of the application to extend the airfield by construction of the North Runway. An Bord Pleanála confirmed and determined the magnitude of night-time flights acceptable in its consideration of proper planning and sustainable development. The night-time use was limited in this manner by An Bord Pleanála to address concerns regarding the cumulative impact of the proposal in combination with existing development;

- The North Runway has been constructed and became operational on the 24 August 2022. A scheduling and slot allocation process was undertaken and a summer 2023 operating schedule was determined and is currently in operation;

- The summer schedule which is being carried out is in breach of the limit applied in Condition 5;

- Taking account of the foregoing, it is therefore concluded that by virtue of the scheduled and actual operations reported, the frequency of night flights in Dublin Airport is not in conformity with Condition 5 of the North Runway permission and is for that reason unauthorised development. The 2000 Act, including s.154(5)(a)(ii) provides that the planning authority can issue an Enforcement notice to require the daa, to proceed with a development in conformity with Condition 5;

- Unauthorised development is occurring and will continue to occur in non-conformity with Condition 5 and that unauthorised development is occurring at the Lands and development is not being carried out in conformity with Condition 5 of the North Runway Permission (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429);

- The daa has not sought to remedy the said unauthorised development, there are no compelling reasons for not taking enforcement action, having regard to the nature of the unauthorised development at issue and the nature of Condition 5, including the reason/purpose of same;

- In circumstances where unauthorised development is occurring and will continue to occur at Dublin Airport, contrary to Condition 5 of the North Runway Permission (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429) comprising the continued and ongoing exceedance of the permitted average number of night-time (between 2300 hours and 0700 hours) aircraft movements at the airport – being a permitted average of 65 aircraft movements per night when measured over the 92-day modelling period;

- Noting the nature of the unauthorised development and the evidence presented and matters discussed in the aforesaid Report dated 18th July 2023 and appendices thereto, it is considered that a period of 6 weeks for compliance with the terms of the Enforcement Notice is reasonable and appropriate in the circumstances.

Approver Malachy Bradley

Senior Planner

28/07/2023 **Dated**

thereunto empowered by order of the Chief Executive, Fingal County Council C.E No **8539** delegating to me all powers, functions and duties in relation to the Council of the County of Fingal in respect of this matter.

Comhairle Contae Fhine Gall
Fingal County Council

An Roinn um Pleanáil agus
Infrastruchtúr Straitéiseach
Planning and Strategic
Infrastructure Department

ENF No:	23/100B
S.153 CE No:	PENF/0133/2023
S.154 CE No:	PENF/0134 /2023

COMHAIRLE CONTAE FHINE GALL

FINGAL COUNTY COUNCIL

IN THE MATTER OF THE LOCAL GOVERNMENT ACT 2001 (AS AMENDED)

AND IN THE MATTER OF THE PLANNING AND DEVELOPMENT ACT 2000 (AS AMENDED)

ENFORCEMENT NOTICE

Section 154 of the Planning and Development Act 2000 (as amended)

DEVELOPMENT CARRIED OUT IN NON-CONFORMITY WITH A GRANT OF PLANNING PERMISSION INCLUDING CONDITIONS

To: daa Public Limited Company,
Three, The Green,
Dublin Airport Central,
Dublin Airport,
Swords, Co. Dublin K67 X4X5

Re: **Lands at Dublin Airport, Co. Dublin ("*the Lands*").**
Planning Permission for the North Runway - Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429
Condition 5

WHEREAS Fingal County Council ("*the Council*"), being the Planning Authority for the functional area in which the above mentioned Lands are located, having considered only the proper planning and sustainable development of the administrative area of Fingal County Council, including the preservation and improvement of the amenities thereof, any representations made to the Council under section 152(1)(a) of the Planning and Development Act 2000 (as amended), any

submissions or observations made under section 152(4)(b) of the Planning and Development Act 2000 (as amended) and any other material considerations, and having investigated the matter, has, in accordance with section 153 of the Planning and Development Act 2000 (as amended) decided to issue this Enforcement Notice.

AND WHEREAS subsequent to the 1st day of October 1964 and within seven years immediately preceding the date of this Notice, the following development is being carried out, and will continue to be carried out, in non-conformity with Condition 5 of the Planning Permission for the North Runway (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429) being the continued and ongoing exceedance of the permitted average number of night time aircraft movements at the airport being 65 aircraft movements per night namely between 2300 hours and 0700 hours (when measured over the 92 day modelling period as set out in the reply to the further information request received by An Bord Pleanála on the 5th day of March, 2007).

AND WHEREAS the reason for Condition 5 was to control the frequency of night flights at the airport so as to protect residential amenity having regard to the information submitted concerning future night time use of the existing parallel runway.

YOU ARE HEREBY REQUIRED, pursuant to section 154 of the Planning and Development Act 2000 (as amended) within **6 weeks** of the date of the service this Notice to proceed with the development in conformity with Condition 5 of the Planning Permission for the North Runway (Planning Authority Reg. Ref No: F04A/1755 / ABP Ref. No: PL 06F.217429) so that the average number of night-time (between 2300 hours and 0700 hours) aircraft movements at the airport is 65 aircraft movements per night or less - when measured over the 92-day modelling period;

AND TAKE NOTICE that you are further required to refund the Council the sum of €350.00 being the sum of costs and expenses reasonably incurred by it in relation to the investigation, detection and issue of this Enforcement Notice and any Warning Letter issued under s.152 of the Planning and Development Act 2000, as amended, including costs incurred in respect of the renumeration and other expenses of its employees, consultants and/or advisors pursuant to s.154(5)(d) of the Planning and Development Act 2000, as amended.

AND TAKE NOTICE that, if within the period specified above, or within such extended period (not being more than 6 months) as the Council may allow, the steps specified in this Notice to be taken are not taken, the Council may, insofar as same is relevant/applicable to the unauthorised development complained of herein, enter on

the land and take such steps, including the removal, demolition or alteration of any structure, and may recover any expenses reasonably incurred by them in that behalf.

AND TAKE FURTHER NOTICE that if, within the said period above, or within such extended period as may be allowed by the Council (not being more than six months), the steps in this Notice to be taken by you, have not been so taken, you may be guilty of an offence.

If the Council decides to prosecute you for non-compliance with this Notice and you are found guilty of an offence by the Courts, you may be liable on summary conviction to a fine not exceeding €5,000 and/or imprisonment for a term not exceeding 6 months or both or on conviction following trial on indictment to a fine not exceeding €12,697,381 and/or a term of imprisonment not exceeding 2 years or both.

You will further be liable on conviction for the costs and expenses of such prosecution.

Dated: The 28th day of July 2023

Signed: _____
SENIOR PLANNER

To whom the appropriate powers have been delegated by Order of CE 8539 of the Chief Executive, Fingal County Council.

To be Served On: daa Public Limited Company,
Three, The Green,
Dublin Airport Central,
Dublin Airport,
Swords, Co. Dublin K67 X4X5

being the owner and person carrying out the unauthorised development.

Outlook

RE: ENF 24/263 - Dublin Airport 32 Million Cap

From Planning Enforcement <Planning.Enforcement@fingal.ie>
Date Mon 23/12/2024 05:14

Dear Sir/Madam,

I wish to inform you that a Warning Letter pursuant to Section 152 of the Planning and Development Act 2000, as amended, issued on the 17th December 2024, in relation to your complaint regarding the above.
The particulars of the Warning Letter are as follows:

This alleged unauthorised development comprises:

- **Exceedance/breach of the 32 million per annum passenger capacity restriction for the year 2024 (January 2024-December 2024) contrary to Condition No. 3 of Planning Permission F06A/1248 (PL 06F.220670) and Condition No. 2 of Planning Permission of F06A/1843 (PL 06F.223469).**

Please be advised that this matter is receiving the full attention of the Planning Enforcement section.
When an update is available to you, you will be notified in writing.

Kind regards,
E. H.

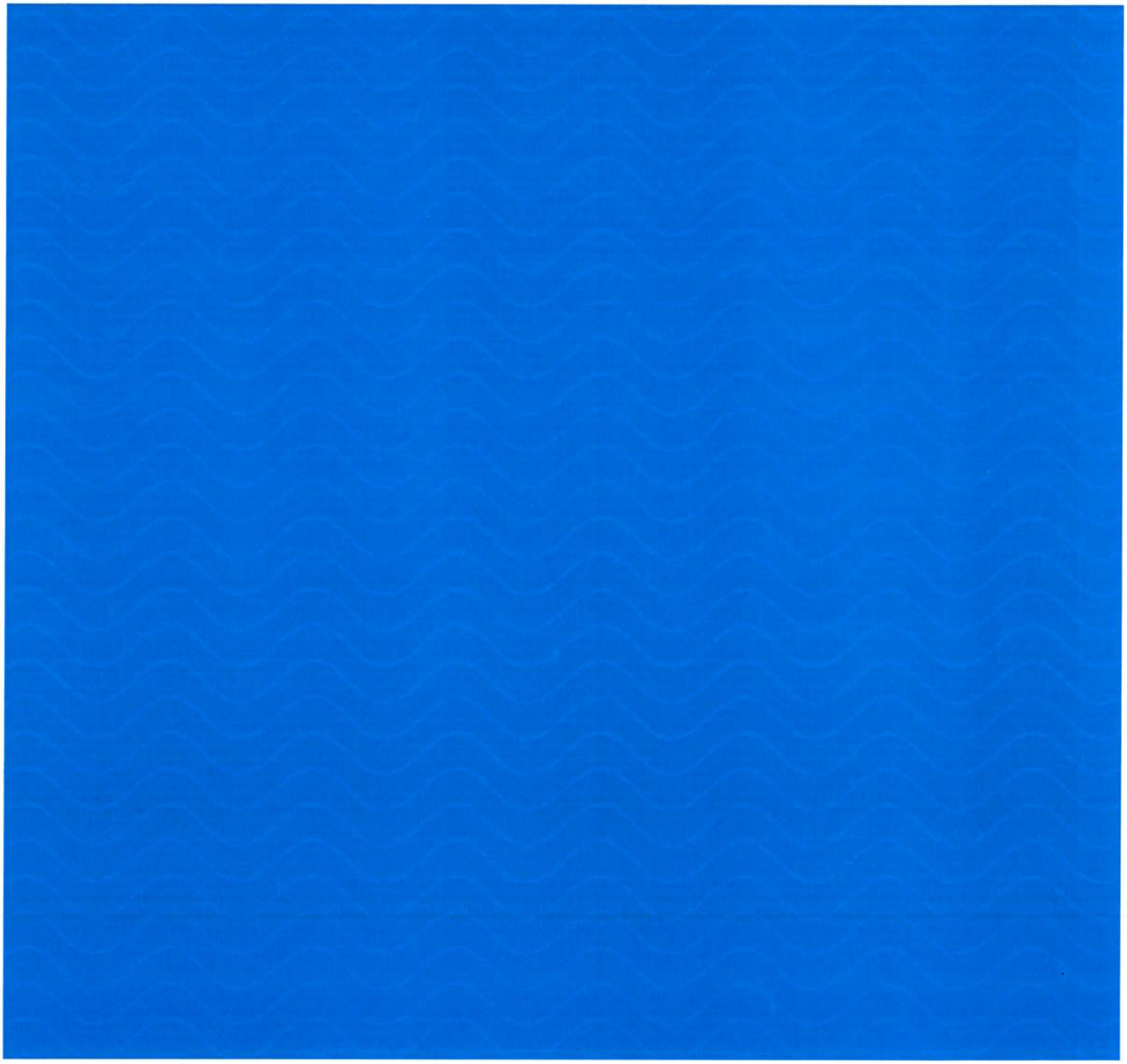
On behalf of
Fearghal McSweeney | Administrative Officer | Planning & Strategic Infrastructure Department | Fingal County Council | County Hall | Main Street | Swords | Co. Dublin | K67 X8Y2
Ph : (01) 8905000
Email: planning.enforcement@fingal.ie

Comhairle Contae Fhine Gall
Fingal County Council

bheith slán nó saor ó earráidí mar d'fhéadfadh faisnéis a bheith idircheaptha, truaillithe, caillte , scriosta, nó teacht déanach nó neamhiomlán . Dá bhrí sin , ní féidir linn glacadh le freagracht as aon earráidí nó easnaimh atá sa teachtaireacht seo , nó aon iatán , a tháinig chun cinn mar thoradh ar an tarchur ríomhphoist . Tá an teachtaireacht cuardaithe ag bogearraí Frithvíreas.

enHEALTH

The health effects of environmental noise



Title: The health effects of environmental noise
Publications Number: 12214

CONTENTS

SUMMARY

Overview

The potential health risks of environmental noise are gaining increasing attention.

With increasing urban populations and changes in urban development, a growing number of people in Australia are being exposed to environmental noise.

The research literature has grown substantially, providing new insights into how people are being exposed to noise and potential health risks.

This review intends to inform noise policy and regulation in Australia by evaluating the evidence of the health effects of environmental noise. It also highlights specific areas for further research.

The review concludes that although the evidence is still emerging, it is sufficient to show that noise adversely affects health. Actions to reduce environmental noise exposure should be considered where feasible.

Scope of this review

This review updates and revises a 2004 enHealth Australia report on the non–auditory effects of environmental noise. It evaluates more than 200 research papers, publications and policies from January 1994 to March 2014.

It includes a systematic review of international evidence on the influence of environmental noise on sleep, cardiovascular disease and cognitive outcomes.

For each outcome, the review considers evidence for the relationship between levels of environmental noise exposure and health outcomes, the influence of different noise sources, and impact on vulnerable populations.

It considers annoyance as a mediating factor between environmental noise exposure and health outcomes, rather than a separate factor. The auditory impacts of noise are excluded as most of these studies are in the context of occupational noise.

Chapter 1 in this document defines noise and common noise measurements, and introduces the effects of noise on health.

Chapter 2 identifies sources of environmental noise and reviews current Australian regulatory approaches to managing community exposures. It draws on the European Union's experience in implementing its environmental directive. This framework allows for reliable and strategic noise mapping and action planning and may prove useful in an Australian context.

Chapters 3, 4 and 5 systematically review studies on the effect of noise on sleep disturbance, cardiovascular disease and cognition.

Chapter 6 includes discussion on the highest quality studies examining these health effects. It aims to give further guidance to assist regulatory authorities and public health professionals by providing insight into causal probability, identifying threshold boundaries for health effects and the magnitude of these effects.

Chapter 7 details the review's recommendations for policy review and further research, and actions for state health, environment and planning authorities.

The objectives and methodology for this review are further defined in appendix A.

Summary of findings

There is sufficient evidence of a causal relationship between environmental noise and both sleep disturbance and cardiovascular disease to warrant health based limits for residential land uses:

- During the night-time, an evidence based limit of 55 dB(A) at the facade using the $L_{eq,night,}$ or similar metric and eight-hour night-time period is suggested.
- During the day-time, an evidence based limit of 60 dB(A) outside measured using the $L_{eq,day,}$ or similar metric and a 16 hour day-time period is suggested.

There is some evidence that environmental noise is associated with poorer cognitive performance. However findings were mixed and this relationship requires further investigation.

It is plausible that aircraft, rail and road traffic noise have differential effects on sleep quality and cardiovascular health, but the evidence is not conclusive.

It is possible that health impacts may be greater among certain vulnerable groups, but further investigation is needed before making conclusions.

Research on the health impacts of environmental noise in the Australian context should be a priority. There is a particular lack of research on environmental noise exposure and health impacts in rural areas. Intervention studies examining the effects of change in noise exposure on changes in population health are also needed.

Key recommendations of this review

This review makes four overarching recommendations for measures to address the health impacts of environmental noise.

Recommendation 1: Recognise that environmental noise is a health risk

Policy

- consider this review when developing national environmental noise goals
- include noise as an important environmental health issue for strategic and local planning at a state and national level
- review the adequacy of existing health guidelines in state and territory legislation

Interventions

- promote awareness of the impacts of environmental noise on health

Information

- inform communities and stakeholders of national and international standards and guidelines

Recommendation 2: Promote measures to reduce environmental noise and associated health impacts

Policy

- review consistency of existing legislation across all levels of government

Interventions

- review noise arising from transportation, including noise criteria for areas adjacent to transport infrastructure
- promote noise mitigation measures such as acoustic barriers or noise insulation in residential buildings and licensing controls to limit noise impacts

Information

- develop a national environmental noise reduction education program, which could be supplemented with additional state-specific campaigns

Recommendation 3: Address environmental noise in planning and development activities

Policy

- include environmental noise in the health impact assessment of proposed developments, where warranted
- determine baseline environmental noise levels to inform planning actions (noise mapping)
- review noise control practices and how to further integrate noise control into planning processes, for all levels of government (with attention to future noise research findings)
- foster national consistency on guidelines to minimise or prevent environmental noise from developments, limiting noise from major sources, and methods to set noise limits

Interventions

- carry out baseline monitoring of environmental noise levels to ascertain existing ambient levels across a broad range of populations and land use areas
- apply appropriate controls where noise is known to have an effect
- develop national and state action plans for both the long and short term to integrate planning and research at all levels of government
- develop guidelines for noise sensitive developments for layout, design and construction for planning authorities

Information

- develop state information strategies to keep communities informed of advances in measures to improve noise

Recommendation 4: Foster research to support policymaking and action

Policy

- identify factors giving rise to sensitivity to noise and vulnerability to non-auditory health effects to inform environmental, planning and health policies

Interventions

- conduct a rigorous evaluation of national, state and city population exposures to each major noise source
- support noise mapping projects to determine community noise exposures to each major noise source that could be used to inform land use planning or burden of disease studies
- conduct evaluations of noise reduction schemes on community health
- promote further research on the effects of noise on learning performance in children, sleep disturbance, annoyance and cardiovascular health and mental wellbeing to establish threshold levels

Information

- translate research findings into useful information for community and relevant stakeholders

Oversight of this review

An expert advisory group oversaw this review and endorsed the final document. The group comprised experts in acoustics, environmental health, epidemiology, sleep medicine, urban studies and noise exposure, public health medicine and environmental noise regulation.

The group provided technical advice on the review's scope, content, conclusions and recommendations. The group also oversaw the process for commissioning evidence reviews including the scope, search strategy and criteria for high quality research and the revision of high quality papers and grading of evidence.

ACKNOWLEDGEMENTS

This document was prepared for enHealth by the Environmental Health Branch of NSW Health, including staff members: Richard Broome, Stephen Conaty, Greer Dawson, Ben Scalley, Carlos Corvalan, Jo Cochrane, Stacey Wyett and Anna Bethmont. Staff at the University of Wollongong Australia provided input, including: Ross Gordon, Lance Barrie, Chris Magee and Karen Larsen-Truong.

An expert advisory group provided editorial comment and assistance through the preparation of the draft revisions. The group consisted of: Gordon Downey (NSW Environment Protection Authority), Lex Brown (Cities Research Institute, Griffith University, NSW), Lin Fritschi (Curtin University, WA), Nathaniel Marshall, (The University of Sydney, NSW) and Norm Broner (Broner Consulting Pty Ltd).

This document updates the original enHealth document, *the Health Effects of Environmental Noise – Other Than Hearing Loss*, published in 2004.

1 SOUND, NOISE, HEARING AND HEALTH

1.1 Noise, environmental noise and health

Noise can be defined as unwanted sound. Environmental noise, or community noise, is defined by the World Health Organisation (WHO) as 'noise emitted from all sources except noise at the industrial workplace' (Berglund et al., 1999).

The main sources of community noise include: transport (road, rail and air traffic), industries, construction, public works, and the neighbourhood.

The potential health risks of environmental noise are gaining increasing attention. WHO defines health as 'a state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity' (WHO, 1946). This broad definition enables us to consider not only the direct impacts environmental noise has on health, but also its impacts on sleep disturbance, cognitive effects and annoyance. In 2011, WHO quantified the burden of disease due to environmental noise exposure. Health end points included cardiovascular disease, cognitive impairment, sleep disturbance, tinnitus and annoyance. In one example of this, WHO estimates that at least 1 million healthy life years are lost every year from traffic-related noise in western Europe (WHO, 2011).

Table 1-1: Definitions and acronyms

Term	Definition
A-weighting i.e. dB(A)	A frequency weighting devised to attempt to take into account the fact that human response to sound is not equally sensitive to all frequencies
Amplitude	A measurement of the energy carried by a wave – the greater the amplitude of the wave, the higher the level of energy carried; for a sound wave, the greater the amplitude, the louder the sound
Audibility threshold	Also known as the absolute threshold of hearing, it is the minimum sound level across the frequency spectrum that an average ear with normal hearing can register with no other sound present
Broadband sound	When a sound is produced by a broad range of frequencies, it is generally called broadband (such as sound from a waterfall)
Decibel (dB)	A unit of measure used to express the level of sound, calculated as the logarithmic ratio of sound pressure level against a reference pressure
Environmental noise	A term to describe unwanted outdoor noise generated by human activity
Frequency (hertz, Hz)	The number of sound waves or cycles passing a given point per second; 1 cycle per second = 1 hertz (Hz)
Noise	Unwanted sound or an unwanted combination of sounds.
Presbycusis	Age-related hearing loss. The cumulative effect of ageing on hearing
Sound	An energy form that travels from a source in the form of waves or pressure fluctuations, transmitted through a medium and received by a receiver (e.g. human ear)
Sound frequency ranges	Infrasound <20 Hz Low-frequency sound 20 – 200 Hz Mid-frequency sound 200 – 2000 Hz High-frequency sound 2000 – 20,000 Hz
Sound intensity (I)	A measure of the sound power per unit area of a sound wave; alternatively, the product of the sound pressure and the particle velocity

Term	Definition
Sound power (watt, W)	A measure of the sonic energy per unit of time of a sound wave; alternatively called acoustic power; calculated by the sound intensity times the unit area of the wave; the total acoustic power emitted in all directions by the source
Sound pressure	A measure of the sound power at a given observer location; can be measured at the specific point by a single microphone or receiver
Sound pressure level (SPL)	A logarithmic measure of the sound pressure of a sound relative to a reference value, measured in decibels (dB) above a standard reference level using the formula SPL = $10\log_{10}[p^2/p_{ref}^2]$ where p_{ref} is the reference pressure or 'zero' reference for airborne sound (20×10^{-6} Pascals)
Syscusis	Lowering of the threshold of aural discomfort and pain
Unspecified noise	Noise for which study authors have not specified a frequency range or decibel level
Vibration	Vibration refers to the oscillating movement of any object and can be used to describe what a person feels
Tinnitus	The conscious perception of sound in the absence of an external sound
Tonal sound	Sound containing audible discrete frequencies

1.2 Basics of noise measurement

In scientific terms, sound is energy that travels from a source in the form of waves or pressure fluctuations. It is transmitted through a medium and picked up by the human ear or another receiver.

Sound has several important properties:

- level or amplitude (loudness) of sound – the sound pressure level (SPL) relative to a reference sound pressure level, which is measured in decibels (dB) using a logarithmic scale
- duration or time period – how sound is distributed over time (continuous, intermittent or impulsive)
- frequency (pitch) – the number of sound waves or cycles passing a given point per second; measured in cycles per second (1 cycle per second = 1 hertz (Hz)).

Humans can hear a wide range of sound frequencies, from 20 to 20 000 Hz and over a wide range of amplitudes, from a whisper to the point of pain.

Noise definitions vary slightly in different countries. In general, noise is classified in three broad frequency ranges:

- low frequency range: 20 – 200 Hz
- medium frequency range: 200 – 2,000 Hz
- high frequency range: 2,000 – 20,000 Hz.

Frequencies below 20 Hz are infrasonic. As the frequency below 200 Hz falls to about 16 Hz and less, the hearing sensation changes to a feeling of pressure.

Low frequency noise is part of urban background noise. Examples include noise from road vehicle and aircraft emissions, industrial and construction activities, ventilation and air-conditioning units, and compressors. Low frequency noise also occurs in nature. Examples include noise from wind or waves at a beach.

Very high frequencies (above 20,000 Hz) are ultrasonic and cannot be heard by the human ear.

Figure 1-1 gives examples of familiar sounds at their noise level dB(A). It shows that the risk of hearing loss depends on the noise level and length of exposure.

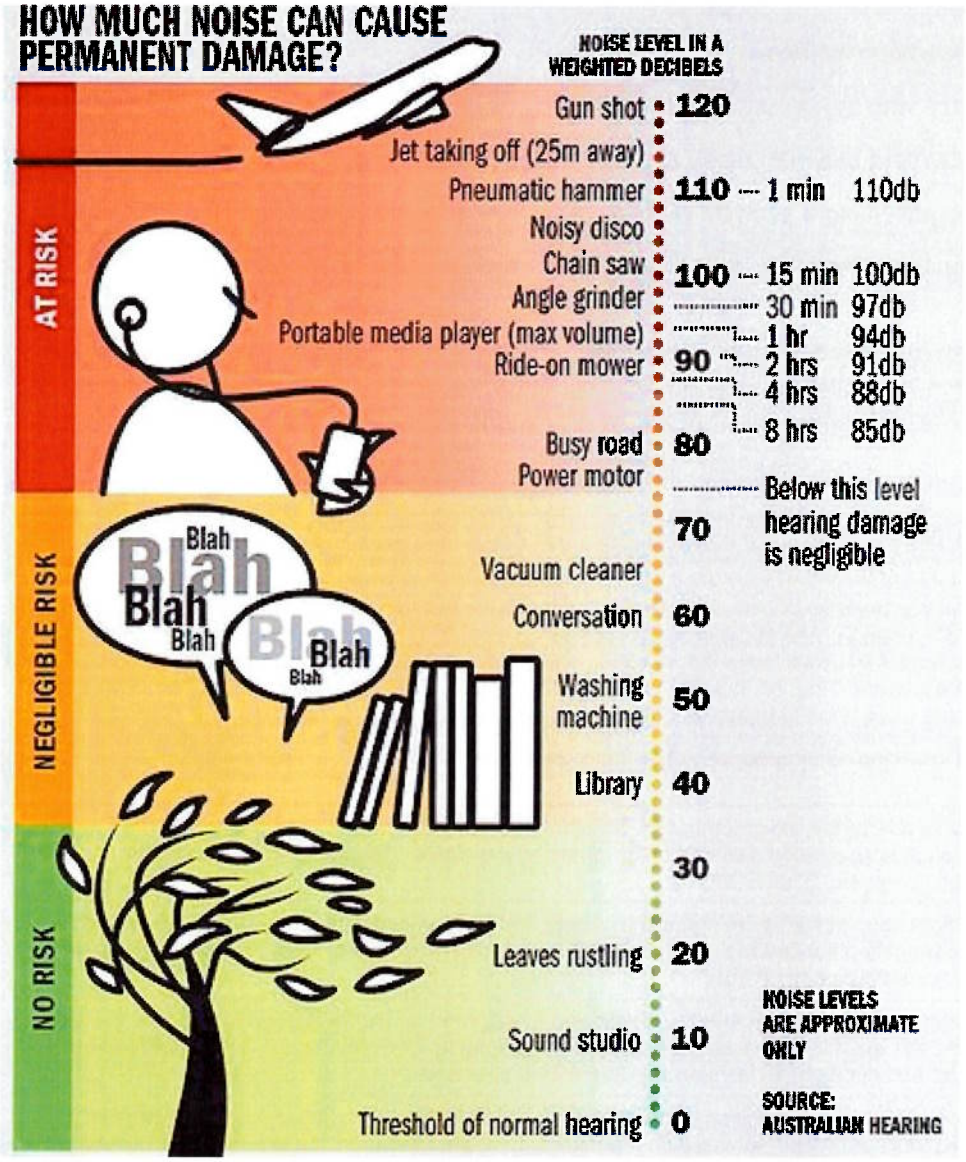


Figure 1-1: Noise levels of familiar sounds and the risk of hearing loss (Australian Hearing, 2014; image adapted by *The Sydney Morning Herald*, 2011).

Humans hear some frequencies more acutely than others and sound measurements are often filtered to reflect this sensitivity. The most common example is the 'A-weighting'. This focuses on the mid and high-range frequencies we hear and has less emphasis on low frequencies to which our hearing is less sensitive. However, it should be noted that although humans are less sensitive to low frequencies, that does not mean we should give less emphasis to low frequencies. Many complaints arise from low-frequency noise.

As sound is emitted from a source, it spreads in the air and its level decreases as it travels further. According to the WHO (1990) this attenuation is due to several factors:

- the distribution of acoustic energy over a geometrically expanding area with increasing distance
- sound absorption by the air
- interference with the ground surface
- physical barriers between noise sources and receivers
- meteorological factors such as wind, temperature gradients and humidity.

When interpreting acoustical data, different metrics are often used for different classifications or types of noise.

A knowledge of sound, noise and human response leads to a selection of noise descriptors, frequency and time weightings to describe and replicate human responses to sound and its impact. Table 1-2 lists common descriptors used to quantify the noise environment.

Table 1-2: Common noise descriptors

Descriptor	Definition
$L_{Aeq,T}$	The equivalent continuous A-weighted sound pressure level measured over a period T – that level of constant noise equivalent to the varying noise levels occurring over a measurement period T, often termed the energy-average noise level. It is often used to measure road and rail noise, industrial noise, noise from heating, ventilation and air conditioning and occupational noise exposure. Time periods can include $L_{Aeq,night}$ and $L_{Aeq,day}$. Similarly, periods can vary from 1 minute to 24 hours and are recorded as $L_{Aeq,1\ min}$ and $L_{Aeq,24\ hr}$*
L_{peak} (linear)	Used in setting hearing conservation limits for impulsive noise
$L_{Ar,T}$	The time average A-weighted sound pressure level of a sound source during a specified time interval, plus specified adjustments for tonal and impulsive character of the sound (time weighting may be 'F' or 'S' †)
L_{dn}	Day-night sound level is the equivalent A-weighted sound level during a 24-hour period with a 10 dB weighting applied to L_{Aeq} during the hours of 10pm to 7am to reflect greater annoyance experienced during night time
L_{den}	The day-evening-night level is the equivalent A-weighted sound level during a 24-hour period with a 5 dB weighting for evening and a 10 dB weighting for night. Day is 12 hours, the evening 4 hours and the night 8 hours and is determined over a year
L_{night}	The night-time noise indicator is the A-weighted long-term average sound level determined over all the nights of a year and in which the night is 8 hours. The definition of L_{night} does not include an addition of 10 dB
L_{ax}, L_{AE} or SEL	Sound exposure level of a discrete noise event is the instantaneous A-weighted sound pressure level integrated over the duration of the noise event and referenced to a duration of one second. SEL is used for measuring noise from individual pass-bys of transportation. A cumulative L_{Aeq} over a reference period can be determined from this. SEL is also sometimes used for sleep disturbance criteria
L_{Amax}	The maximum instantaneous sound pressure level measured on 'F' time weighting or 'S' time weighting

* Local regulatory requirements may define varying periods for $L_{Aeq,T}$.

† F and S are defined in relevant Australian Standards.

Descriptor	Definition
$L_{An,T}$	The A-weighted sound pressure level obtained by using 'F' or 'S' time weighting that is equalled or exceeded for a percentage of the time interval considered. Common examples are: • $L_{A10,T}$: the A-weighted sound pressure level which is exceeded 10% of the time; T, often used to represent the average of the maximum noise levels during a measurement period • $L_{A90,T}$: the A-weighted sound pressure level which is exceeded 90% of the time; T, often used to represent the average of minimum noise levels during a measurement period or the background noise level in the absence of the noise under investigation
N70	Other noise descriptors are used in some circumstances. This includes N70 (number of aircraft events >70 dB(A) over any specified period), which is used to describe over-flight noise exposures. The 70 dB(A) sound level is chosen because an aircraft noise event of this, or louder, magnitude is likely to disturb conversation or interfere with listening to the radio or television inside a house with an open window

1.3 Tranquillity, quiet areas and potential positive health effects of sound environments

The absence of unwanted sound (noise) is not necessarily quietness. In fact, natural background sounds in certain contexts can be seen as enjoyable or wanted. For example: wind rustling in trees, waves crashing on a beach, waterfalls and birds singing. Some human sounds may also be comforting, such as the burble of voices or the sound of children playing.

Tranquillity is a term used globally. It is defined as: 'the quality or state of being tranquil; calmness, serenity, a disposition free from stress or emotion and a state of peace and quiet'. It can also be defined as: 'a sense of calm or quietude'. It is often understood in terms of engagement with the natural environment (Jones, 2012).

Related concepts include soundscapes and quiet areas. Soundscape is a complementary concept to environmental noise management, where sound is seen as a resource to be managed. Soundscapes focus on sounds of preference rather than sounds that cause discomfort. The metric is listener-centred rather than an objective-based energy metric.

Quiet areas are referred to in the European Union's Environmental Noise Directive. These are defined for an urban agglomeration as 'an area which is not exposed to a value of L_{den}, or of another separate indicator greater than a certain value set by the member state, from any noise source' (European Union, 2002). This definition of quiet, put more simply, is 'not noisy'. The directive legislates for the identification and protection of quiet areas throughout the European Union.

The benefits of quiet or tranquil places are not usually considered in terms of health but rather in ideas of amenity, attractiveness, pleasantness, calmness, restfulness and restoration. While there are plausible grounds for considering some acoustic environments as conducive to health benefits, there is a lack of substantive evidence on the issue. This is an emerging field. Aiming to achieve tranquillity may encourage broader interest in managing the acoustic environment.

1.4 Theoretical models to account for how noise affects human response

Several theoretical models explain the complex relationship between noise and the human response to it. Some of these models are outlined below. However, a detailed discussion is outside the scope of this document.

1.4.1 The noise/stress concept and general stress model

The noise/stress concept (Babisch, 2002) considers noise in terms of its physiological response: a psychosocial stressor that stimulates the sympathetic and endocrine systems. Noise activates

the hypothalamo-pituitary-adrenal axis and the sympathetic-adrenal-medullary axis producing catecholamines and steroid hormones that affect metabolism. Changes in adrenalin, noradrenalin and cortisol levels are frequently observed in acute and chronic noise experiments.

According to the general stress model, neuroendocrine arousal suppresses the immune system, influences the metabolic state of the organism, and acts as a mediator along the pathway from the perceived sound to the stress-related disease. Some established risk factors may be affected. For example, risk factors for ischemic heart disease, including blood lipids, glucose level, haemodynamic and haemostatic factors, can be elevated by neuroendocrine arousal (Babisch, 2002).

1.4.2 Theory of the four primary interferences

In this theory, Miedema (2007) proposes four primary interferences caused by environmental noise, which may be accompanied by acute stress responses. These primary effects can lead to long-term effects, and chronic stress is proposed to play an important role.

Sound masking route (communication disturbance)

Sound masking reduces speech comprehension, which may limit speech and human interaction in noisier environments.

Attention route (concentration disturbance)

Attention involves selection of elements such as visual impressions, acoustical impressions or mental representations and selecting, ending or redirecting attention to each. Attention can be focused, or it may be divided over more elements. Noise can negatively affect processes requiring attention.

Arousal route (sleep disturbance)

Higher levels of arousal lower the probability of falling asleep or continuing sleep. Because of its arousal potential, sound can prevent a person falling asleep, affect sleep quality and cause awakening.

Affective–emotional route (fear and anger)

Many sounds are neutral. However, some types of noise can cause affective–emotional responses. Examples include fear and anger.

1.4.3 Effect modifiers

Other factors considered include social and psychological effect modifiers. There is a growing body of literature on the psychological and psychosocial modifiers of annoyance and dissatisfaction due to noise (Guski, 1999; Hatfield et al., 2001; Kroesen et al., 2010; Nitschke et al., 2014; Schreckenberg et al., 2010).

Annoyance

Annoyance is defined as 'a feeling of displeasure associated with any agent or condition, known or believed by any individual or group to adversely affect them' (Berglund et al. 1999). Noise annoyance is a feeling of resentment, displeasure, discomfort, dissatisfaction or offence caused by noise interference. It is a well-established construct in the study of environmental noise and is considered an important end point for measuring the impact of noise in exposed populations.

However, its relationship with health remains uncertain. In Australia annoyance is often considered an issue of amenity. But it forms an important part of the regulatory framework for noise.

It is not yet possible to predict noise annoyance on an individual level, given the many exogenous and endogenous factors that affect it. However, relationships between noise exposure and annoyance can be understood together with several effect-modifying factors. To assess noise-induced annoyance at the population level, a standardised questionnaire can be used. The percentage of respondents who report being highly annoyed can then be used as a prevalence indicator for annoyance in the population (WHO, 2011).

Several theoretical models, including those described above (Babisch, 2002; Miedema, 2007) consider annoyance on a causal pathway to health effects such as stress, cardiovascular effects and sleep disturbance.

1.5 Effects of environmental noise on health and related outcomes

Early research on the health effects of noise is from research into occupational health, and subsequently environmental health, in the 1960s and 1970s in Scandinavia, Europe and the US, as well as Australia. Environmental noise has become an increasingly important issue and many more studies on the health effects of noise have been done over the past few decades. The focus of these studies has shifted from the effect of noise on hearing and cardiovascular health to its broader effect on wellbeing, quality of life and amenity.

While environmental noise is generally recognised as a problem, the extent to which noise adversely affects health, particularly where subjective measures are used, is the subject of continued discussion. This section provides a brief overview of the effects of noise on health.

1.5.1 Effects on hearing

A person who is not able to hear as well as someone with normal hearing (hearing thresholds of greater than 25 dB in both ears) is said to have hearing loss. Around 2.1 million Australians are affected by complete or partial hearing loss (ABS, 2012).

Prevalence of hearing loss is age related: less than 1 per cent of people under the age of 15 are affected by hearing loss, while three in every four people over the age of 70 are affected. In about one-third of people with hearing loss, exposure to excessive noise was reported to be at least partially responsible.

The most common sources of noise injury are workplace noise and recreational noise (Wilson, 1998). Further consideration of exposure to occupational or recreational noise-induced hearing loss is outside the scope of this document.

1.5.2 Effects on health and human response other than hearing loss

Sleep

Sleep is essential for human function. A good night's sleep is also considered essential for quality of life. Sleep disturbance is a common complaint of noise-exposed populations and has the potential to affect health and quality of life.

Sleep parameters can be measured in terms of immediate effects, after-effects and long-term effects. Immediate effects include arousal, sleep stage changes, awakenings, body movements, total wake time and autonomic responses. After-effects include sleepiness, daytime performance and cognitive deterioration. Long-term effects include self-reported chronic sleep disturbance. Chapter 3 addresses noise and sleep disturbance.

Cardiovascular disease

Cardiovascular disease includes ischaemic heart disease, myocardial infarction, hypertension (high blood pressure) and stroke. The number of epidemiological studies on the association between exposure to road traffic and aircraft noise and hypertension and ischaemic heart disease has increased in recent years. Very few studies have investigated the cardiovascular effects of exposure to rail noise (WHO, 2011). Chapter 4 addresses noise and cardiovascular disease.

Cognitive performance

Most observational studies examining cognitive performance are done in children, with experimental studies often involving young adults. Few studies investigate the effects of environmental noise on older adults.

Outcomes investigated include attention, memory, reading comprehension and mathematical tasks.

Chapter 5 addresses noise and cognition.

1.5.3 Other reported health effects and outcomes

Mental health

Environmental noise is not believed to be a direct cause of mental illness, but it is thought to accelerate and intensify the development of latent mental disorders (Berglund et al., 1999).

The effect of noise is complicated. Research suggests that poor psychological health is associated with greater annoyance responses. Studies in adults have found that noise exposure relates to an increase in reported psychological symptoms such as anxiety and depression, rather than to clinically diagnosable psychiatric disorders.

Overall, evidence suggests that in adults and children, noise exposure is unlikely to be associated with serious psychological illness. However, there may be effects on wellbeing and quality of life (Clark and Stansfield, 2007).

Birth outcomes

Ristovska et al. (2014) conducted a systematic review looking at the association between exposure to noise and birth outcomes. The evidence suggests an adverse effect on birth weight. Only a small number of studies have looked at other reproductive outcomes, and no clear links have yet been established.

Vulnerable groups

Particular sub-groups of the population are more vulnerable to experiencing annoyance or adverse health effects from noise.

Vulnerable groups include people with particular diseases or medical problems; people in hospital or rehabilitating at home; people dealing with complex cognitive tasks; those who have a visual or hearing impairment; babies and children; and the elderly.

These groups should be considered when recommending noise regulation or protection, including types of noise effects and specific environment and lifestyle factors (Berglund et al., 1999).

2 NOISE EXPOSURE AND REGULATORY APPROACHES IN AUSTRALIA

This chapter examines the noise environment in Australia. While most of Europe has been able to build a picture of the types and extent of noise exposure across the continent, a lack of systematic data for the Australian context makes understanding and quantifying our noise environment difficult.

In the absence of information that reliably and systematically maps noise exposure and affected populations, researchers use modelled or measured information from significant sources such as aircraft and road traffic.

Complaints information and social surveys may provide some insight into the impact noise has on communities and individuals. These may or may not be typical of how the general population responds.

Both types of information are useful. Modelled and measured data provides an objective measure of noise levels. Complaints and social surveys provide further insight into people's subjective or physical responses to noise. However, complaints data does not always correspond to areas with the highest recorded noise levels. This underscores the subjective nature of noise and suggests other factors such as habituation are important.

The availability of different types of noise data varies, and information is available for some jurisdictions but not others. For example, Airservices Australia provides online summaries of noise monitoring data from major airports that are updated quarterly. Information on road and rail traffic may be available for major developments but obtaining this data is logistically difficult. This information and other data are needed if we are to build a picture of noise exposure across Australia (Airservices Australia, 2015a, b).

This chapter describes some of the common environmental noise sources and provides examples of the types of data available. It summarises the regulatory response to major sources and examines noise mapping in the European context under *European Noise Directive (END) 2002/49/EC relating to the assessment and management of environmental noise* (European Union, 2002).

A significant portion of this chapter focuses on road, aircraft and rail noise, which are characterised by lower, intermittent and higher frequencies respectively. Most research is done on road, aircraft and rail noise because their characteristics are similar to other noises.

2.1 Sources of noise exposure

2.1.1 Road traffic noise

Road traffic noise is mainly generated from the engine and from frictional contact between the wheels, the ground and the air. Road contact noise exceeds engine noise at speeds higher than 35km/hour. However, the physical principle responsible for generating noise from contact between the tyre and the road is less well understood (Berglund et al., 1999). It is estimated that more than 70 per cent of environmental noise (unwanted sound) in urban Australia is due to road traffic (Marquez et al., 2005).

Noise levels from traffic can be predicted from the traffic flow rate, the speed of the vehicle, the proportion of heavy vehicles, and the nature of the road surface. Vehicle noise is related to traffic speed. As speed-changing traffic is noisier than steady traffic, congestion may add to noise.

Congestion typically reduces traffic noise due to lower vehicle speeds. An indirect consequence of congestion is an increase in night-time freight as freight operators, encouraged by government agencies, try to avoid daytime congestion. Noise from heavy truck exhaust and gear changes as well as engine noise and braking, is a particular problem. Rising traffic levels and growing freight movements lead to increasing violations of transport noise level guidelines (Marquez et al., 2005).

In highly urbanised Australia, the population exposed to noise is mostly concentrated in metropolitan areas (Brown and Bullen, 2003). Most noise impacts of traffic occur when people are in their homes. Estimating community exposure requires estimating the levels of road traffic noise at the facades of dwellings in Australian cities.

A survey of road traffic noise in five capital cities by Brown and Bullen (2003) shows the proportion of dwellings affected by road traffic noise. The study was done in 1997–98. At the time, it provided the best available estimate of road traffic noise exposure in urban Australia. The study drew a random sample of dwellings from the urban centres in each capital and estimated road traffic noise exposure at each dwelling.

The results show that 8 to 20 per cent of dwellings are exposed to $L_{A10,18h}$ levels above 63 dB and 5 to 11 per cent above $L_{A10,18h}$ 68 dB. L_{A10} is the noise level exceeded for 10 per cent of the measurement period. $L_{A10,18h}$ is the average of L_{A10} noise levels from 6am to midnight.

Sydney was significantly different to the other cities with a higher proportion of dwellings subject to external noise between $L_{A10,18h}$, 60 and 70 dB. The study suggested this might be due to a different pattern of road use and Sydney's physical location.

Figure 2-1 shows an estimate of the proportion of dwellings in the urban centres of Sydney, Melbourne, Brisbane, Perth and Adelaide where calculated traffic noise exceeds values on the $L_{A10,18h}$ scale.

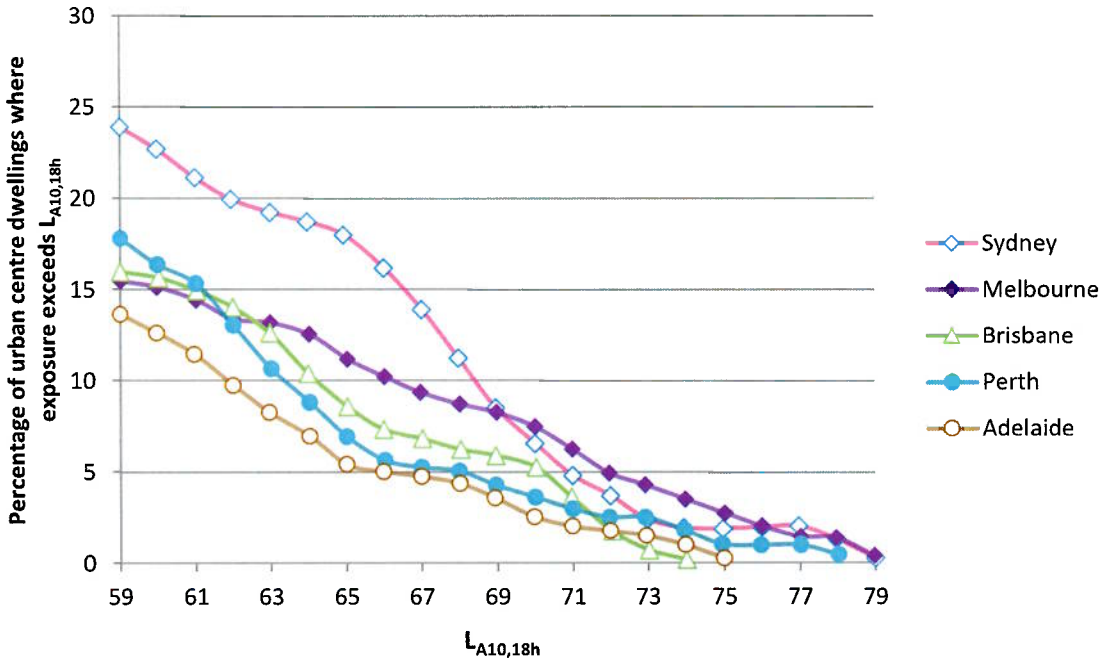


Figure 2-1: Cumulative noise exposure of dwellings in Australian capital cities, $L_{A10,18h}$ (Adapted from Brown and Bullen 2003)

Since that survey, vehicle fleet mix has changed. The ABS Motor Vehicle Census (2014) shows a slight decrease in the proportion of passenger vehicles in Australia with these accounting for about 75.4 per cent of all registered vehicles in 2014 as opposed to about 80 per cent in 1999. This has been offset by a rise in the proportion of light commercial vehicles, heavy rigid trucks, buses and motorcycles in each jurisdiction. The total number of vehicles increased from about 12.3 million in 1999 to about 17.6 million in 2014. The passenger vehicle fleet rose from about 9.7 million to about 13.3 million in the same period.

These changes will have an impact on the noise environment and the characteristics of the noise experienced. A noise measurement survey by Victoria's Environment Protection Authority (EPA) compared noise measurements in 2007 with data collected in 1978 (EPA Victoria, 2007). It measured noise levels at 50 sites across the inner, middle and outer suburbs of Melbourne and showed that despite the growth in traffic volumes, noise levels across Melbourne were similar to those in 1978 (Figure 2-2). This graph depicts noise levels in terms of the $L_{Aeq,1hour}$.

These results suggest that while traffic volumes have grown and the mix has changed, quieter vehicles and other factors may be offsetting any rise in noise. Examples include improvements in road surface and better policies for new and upgraded roads. However, increasing residential densities along major urban roads means a greater percentage of the population is likely to be exposed to higher traffic noise.

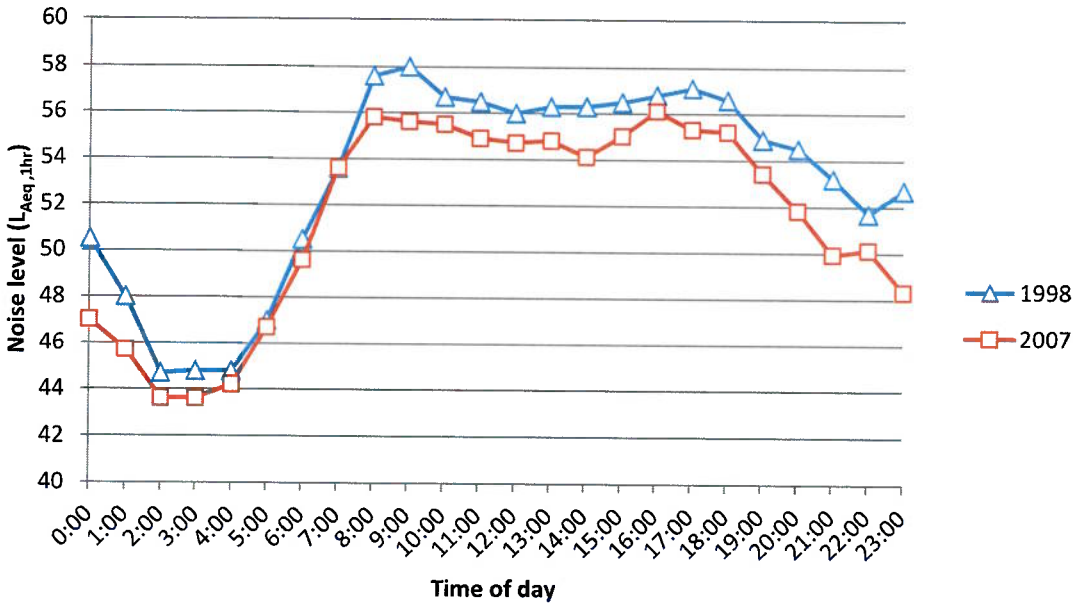


Figure 2-2: Average noise levels for each hour on weekdays for 1978 and 2007 in Melbourne (Adapted from EPA Victoria, 2007)

Following EPA Victoria's noise measurements in 2007, WSP Acoustics did environmental noise modelling for the authority on the greater Melbourne area in 2013. It provided estimates of the population exposed to a range of noise levels. Using Sound PLAN, it constructed a three dimensional representation of the environment of greater Melbourne. This provided noise maps to visualise noise exposure. These maps can inform EPA Victoria's input into activities such as land use planning, transport planning and design standards that change the community's exposure to noise. Modelling for each scenario considered ground contours, road and traffic data, locations of sensitive receptors, noise barriers and other inputs affecting the road traffic noise environment (WSP, 2014).

Mitigation of road traffic noise

Noise mitigation of road traffic tends to focus on controlling noise at the source, between the source and the receiver (noise pathway), and at the receiver location. Effective noise management may use a combination of mitigation techniques to reduce noise. Effectiveness is the degree of reduction achieved and perceptions of change in the noise environment. It also includes practical considerations such as feasibility of construction and if these measures are reasonable.

Noise mitigation techniques include vehicle noise control (Department of Infrastructure and Regional Development, Australian Design Rules) and controlling traffic (reducing volumes, controlling speed or decreasing flow).

Construction techniques include road alignments (vertical and horizontal), low noise road surfaces and noise barriers (NSW Environment Protection Authority Road Noise Policy, 2011).

Urban planning controls and acoustic insulation for new buildings next to busy roads are also used to reduce noise (Australian Building Codes Board and some state planning departments).

The results of these different options vary.

Controlling vehicle noise and traffic can reduce noise by 1 to 5 dB(A).

Noise barriers can cut up to a 10 dB(A) although effectiveness depends on barrier height, length, material density and distance from noise source. However, barriers can only be fitted along no-access roadways and many urban roadways have road frontages from properties. Extra height in barriers can reduce noise further, although these are restricted by structural elements and aesthetics. Retrofitting noise walls to existing roads is expensive (Austroads, 2005).

2.1.2 Aircraft noise

Aircraft operations generate substantial noise, exposure to which is concentrated around airports. Take-off produces intense noise, including vibration and rattle, while landings generate noise in long low-altitude flight corridors. For the most part, larger and heavier aircraft are responsible for more noise than lighter aircraft (Berglund et al., 1999).

In older, turbojet-powered aircraft, the main mechanism of noise generation was turbulence created by the jet exhaust mixing with surrounding air. In more modern aircraft this noise source is significantly reduced by using high by-pass ratio turbo-fan engines that surround the high velocity jet exhaust with lower velocity airflow generated by the fan. Noise can also be generated by the fan itself, particularly during landing and taxiing. Multi-bladed turbo prop engines can produce relatively high levels of tonal noise (Berglund et al., 1999).

The overall sound pressure levels from airports can be determined from the number and types of aircraft, their flight paths, the proportions of take-offs and landings, and the atmospheric conditions. Airports hosting helicopters or smaller aircraft used for private business, flight training and leisure purposes may also contribute to significant noise associated with flight paths.

Over the past three decades, Australia has seen a substantial increase in aircraft numbers and movements. Kingsford Smith airport in Sydney has experienced the greatest growth in flight movements (BITRE, 2014). This increase, seen in Figure 2-3, has resulted in continued exposure to aircraft noise, particularly on communities close to airports and underneath flight paths. This is despite reduced noise emissions from newer types of aircraft.

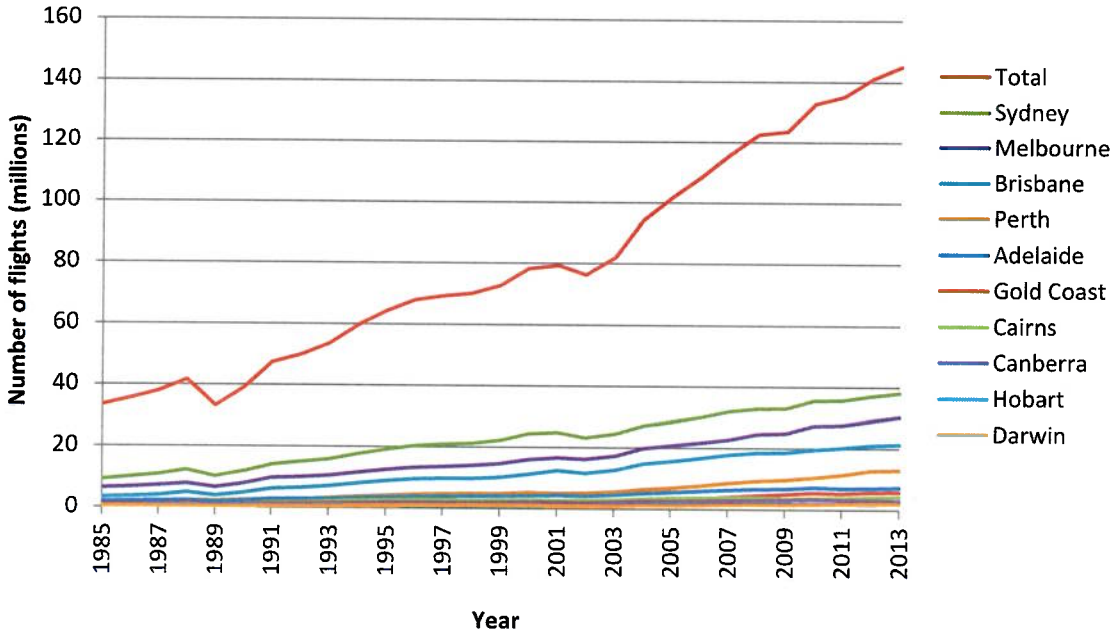


Figure 2-3: Aircraft movements at Australian airports 1985-2013 (Data sourced from Bureau of Infrastructure, Transport and Regional Economics (BITRE) 2014a)

Australian Noise Exposure Forecast

Information about aircraft noise in Australia is provided through the Australian Noise Exposure Forecast (ANEF). This forecast system is based on findings from a major socio-acoustic survey done near several Australian airports (Hede et al., 1982).

The study shows that a weighting period from 7pm to 7am gives the best correlation between noise dose and community reaction. The contours relate to the total noise energy received by locations on the ground near an airport on an annual average day. They show predicted future aircraft noise levels.

While ANEF is an effective land use planning tool, it does not convey information about the actual aircraft noise levels experienced at a given location. This means other noise descriptors are often used as supplements to ANEF contours.

ANEF is the officially endorsed chart for an aerodrome.

N contours

N contours are designed to supplement ANEF and better describe aircraft noise levels to the public. They were developed by the Commonwealth Department of Infrastructure and Transport in consultation with industry and the community. N contours measure the number of noise events per day exceeding 60, 65 or 70 dB (see Table 2-1) and show the expected noise levels in a particular area (Department of Transport and Regional Services, 2000).

Table 2-1: Description of N contours

N contour	Definition
N60	Number of events exceeding 60 decibels per day
N65	Number of events exceeding 65 decibels per day
N70	Number of events exceeding 70 decibels per day
Night contours	For example: 6 or more events exceeding 60 decibels per day

Australian Noise Exposure Index

The Australian Noise Exposure Index (ANEI) is similar to ANEF but based on historical data, where flight paths and aircraft movements are known rather than forecast. It uses an integrated noise model comprising data for the flight path, aircraft type, runway used and time of day (weighted for 7pm to 7am).

ANEI contours are plotted on a map using geographic information systems (GIS) software. The contours are consistent with flight tracks and aircraft operations for the period.

Figure 2-4 shows ANEI contours for Sydney airport. The population beneath the ANEI contours is estimated using the latest census data and suburb boundary information.

The Australian Noise Exposure Concept (ANEC) is an illustration of aircraft noise exposure at a site, using data that may bear no relationship to actual or future situations.

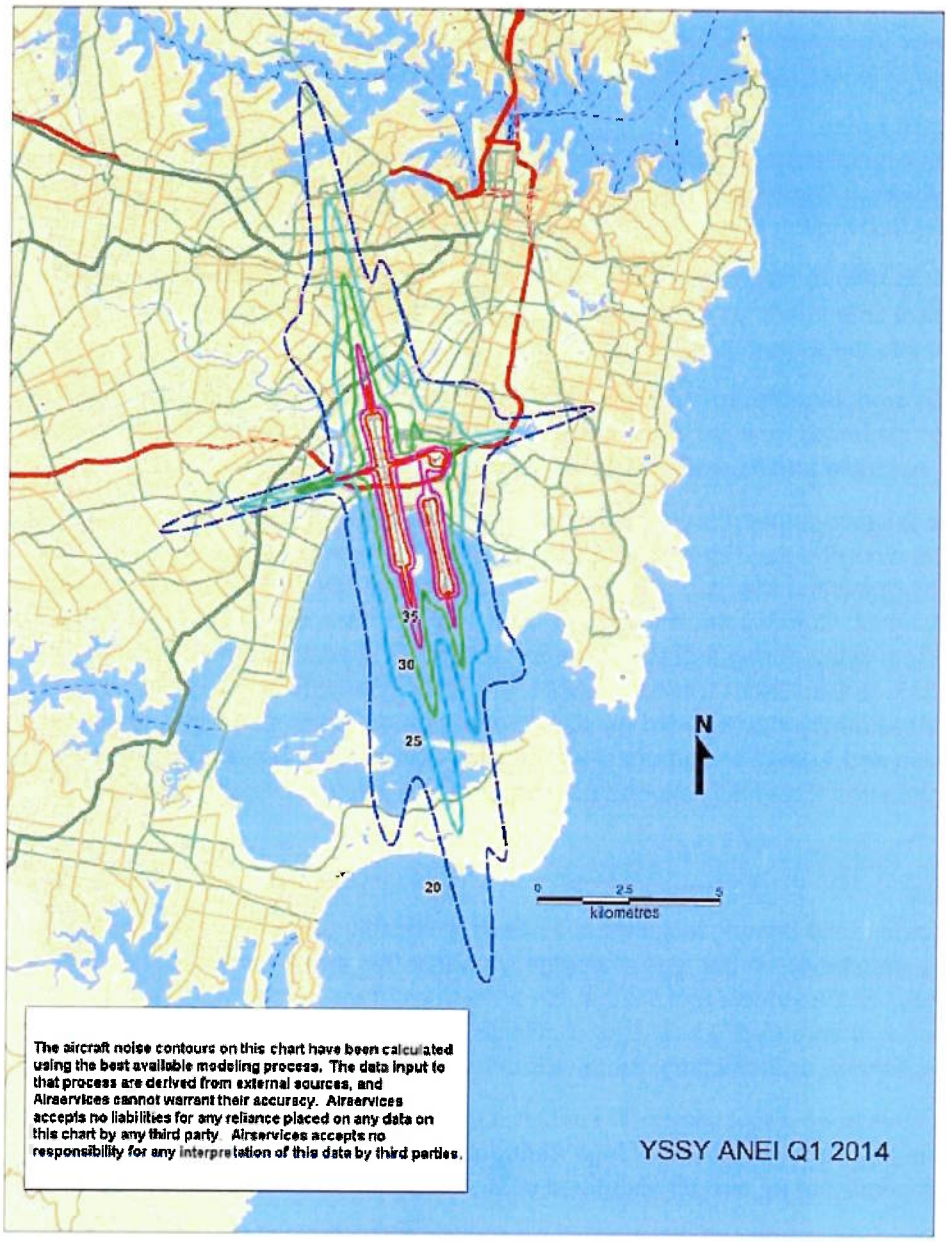


Figure 2-4: ANEI contours for Sydney Airport, January to March 2014 (Airservices Australia 2015)

Aircraft noise monitoring

Noise monitoring is done at major airports including Adelaide, Brisbane, Cairns, Canberra, Gold Coast, Melbourne, Perth and Sydney. Information includes the identity, flight path and altitude of each aircraft operating to and from the airport, and the noise levels produced by individual aircraft. The information is collected for each 24-hour period per week by fixed noise monitors or environmental monitoring units along the flight path.

This data can be used in several ways to show average noise during a period, background noise levels or the number of noise events over a certain threshold.

Airservices Australia provides online summaries of noise monitoring data from major airports that are updated quarterly (Airservices Australia, 2018a). It also displays historical and near real-time noise data from each monitoring unit in WebTrak (Airservices Australia, 2018b).

Mitigation of aircraft noise

Aircraft operating in Australia are required to adhere to noise standards set out by the International Civil Aviation Organisation in *Annex 16 — Environmental Protection, Volume I — Aircraft Noise* to the Convention on International Civil Aviation (ICAO, 2008).

Some airlines seek to reduce noise by buying quieter aircraft or organising their fleet so quieter aircraft fly at sensitive times. Airlines can also take a continuous descent approach, using technology to glide into the airport in one smooth descent.

Airports and airlines work together to minimise noise exposure during night hours. This includes procedures such as preferred runways and flight paths and reducing engine thrust when safe to do so (Airservices Australia and Australian Airports Association).

Curfews attempt to balance airport commercial operations and safety requirements with the need to reduce night-time aircraft noise. They do not stop all aircraft movements, but they limit take-offs and landings by restricting the type of aircraft that can operate, the runways they can use and the number of flights. Curfews usually operate from 11pm to 6am, with most commercial aircraft prohibited from flying during that time. The exceptions to this are shoulder movements, which occur from 5am to 6am and 11pm to midnight. These are permitted on a quota basis to account for differences during the northern hemisphere's summer, which affects flying schedules (Airservices Australia and Australian Airports Association). Curfews are in place at Sydney, Adelaide, Coolangatta and Essendon airports (Department of Infrastructure and Regional Development, 2016).

2.1.3 Rail noise

Rail noise depends on many factors, including the speed at which the train is travelling. Noise characteristics vary depending on the type of engine, wagons, the rails and their foundations, as well as the roughness of the wheels and the rail. Small radius curves in the track can lead to very high frequency sound, often called 'wheel squeal'. Noise is also generated by running engines, whistles and loudspeakers, and shunting operations in marshalling yards.

High-speed trains have been associated with sudden, but not impulsive, rises in noise. At speeds greater than 250 km/hour, the proportion of high frequency sound energy increases with the sound similar to an overflying jet aircraft (Berglund et al., 1999).

The Cooperative Research Centre (CRC) for Rail Innovation (CRC for Rail Innovation, 2011) classifies rail noise as:

1. Rolling noise: the vertical excitation of the rail and wheel generated by variations or roughness of the wheel or the rail surfaces
2. Impact noise: the result of discontinuities in the running surfaces of the rail and wheel
3. Traction noise: generated by power units of any kind including diesel or electrical power sources. It covers possible mechanisms associated with the function of converting the supply energy to mechanical work
4. Friction braking noise: generated by the interaction between the friction material and the rotating element. In some cases this is seen as a subset of traction noise
5. Curving noise: caused by friction induced self-excitation of the wheel and rail in the lateral direction on low radius curves, including flanging noise and curve squeal noise

6. Aerodynamic noise: caused by disturbance of air flow over the train, becoming significant at high speeds (greater than 200km/hour)
7. Other noise sources: including wagon 'bunching', coupler noise, warning signals, communication systems noise, stabling and yard noise, maintenance noise, and internal noise such as air conditioning and gangway noise.

Growth in rail sector

The use of rail freight (rolling stock or fleet) is expected to grow 1.9 times the 2010 level by 2030 (BITRE, 2014b) .

Rail is competitive for long distance non-bulk freight, such as from Sydney to Perth. This expanded use of rail for freight may increase noise in metropolitan areas and in rural areas that have not been previously affected.

Figure 2-5 shows the increase in sending freight by road and rail to 2013, with rail freight set to increase significantly (BITRE, 2014c).

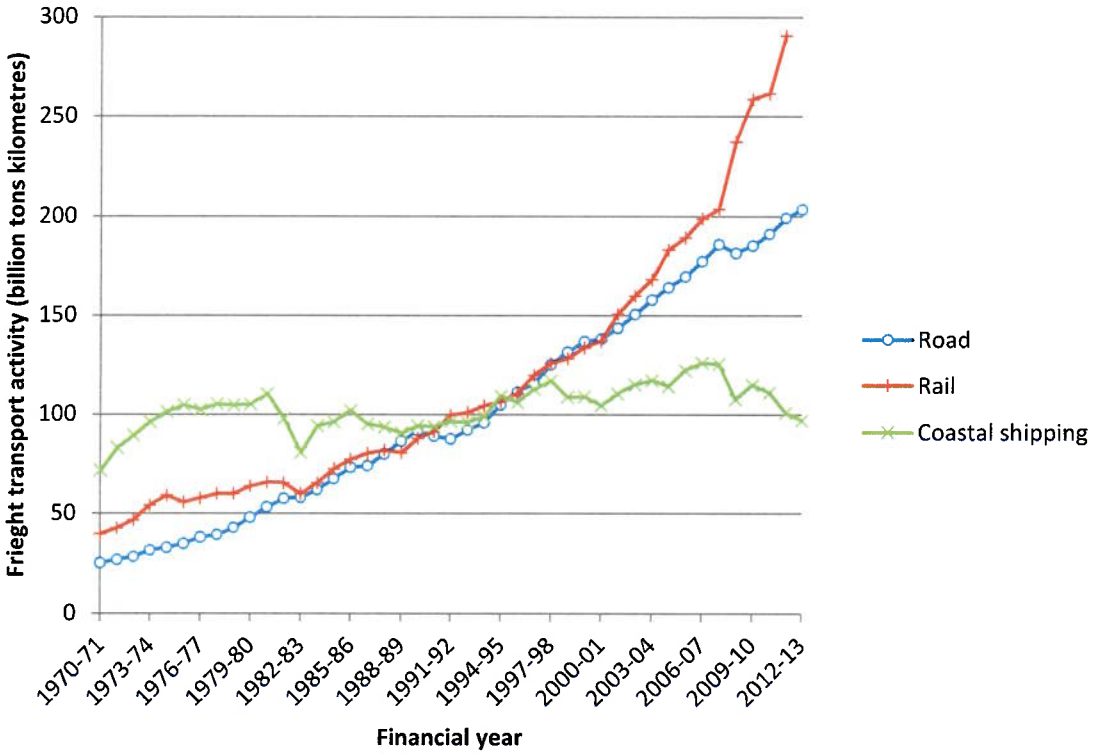


Figure 2-5: Domestic freight transport activity by mode (Adapted from BITRE 2014c)

Growth of passenger rail

Rail passenger transport is not expected to increase as much as freight, due to the dominance of private cars. Very high speed trains have been proposed to connect Brisbane, Sydney and Melbourne, with the first link between Sydney and Canberra operational by 2035 (Department of Infrastructure and Regional Development, 2013). If high-speed rail is a genuine possibility in Australia, its health impact should be considered now.

Proportion of the population exposed to rail noise

Estimates from Europe indicate the noise contribution from railways is around 10 per cent of the total noise burden from both roads and railways (EPA, 2014). There are no estimates for Australia, but an example of rail noise exposure is shown below.

In 2002 the former NSW Rail Infrastructure Corporation undertook modelling work on five priority lines in the Sydney metropolitan rail network. The percentage of receivers (people) exposed to different noise levels are shown for two of these train lines in Figure 2-6. With increasing urban density and the development of new passenger and freight lines, the number of people exposed will have steadily increased.

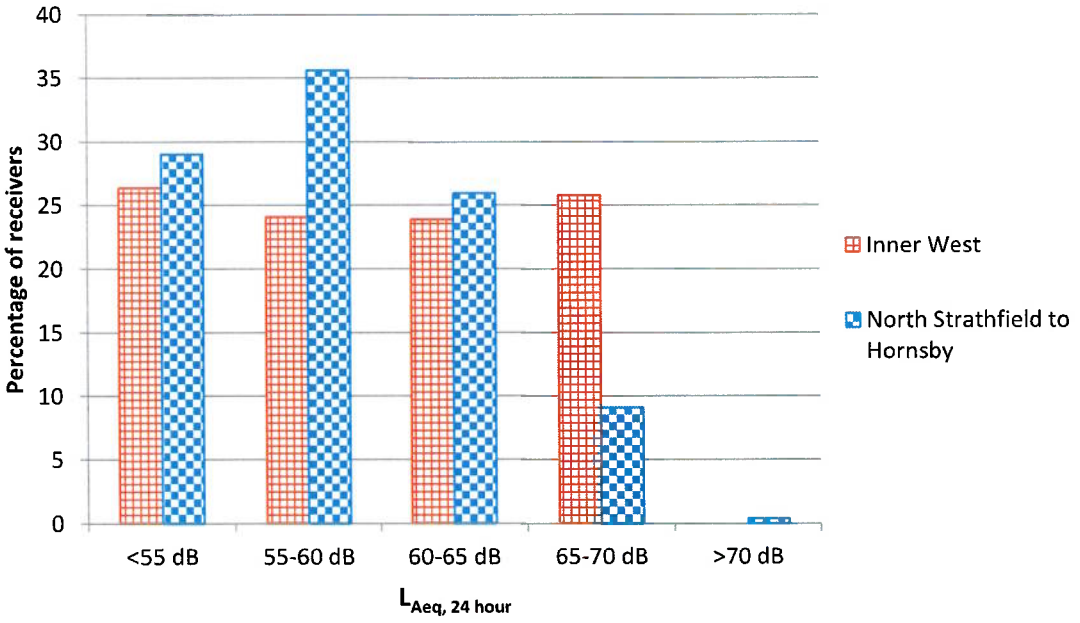


Figure 2-6: Percentage of receivers exposed to various noise categories along two major railway lines in Sydney

Mitigation of rail noise

Several European studies confirm that measures to reduce noise at the source are more cost effective than constructing noise barriers. Mitigation strategies tend to follow those outlined for road traffic noise. This may be problematic for rail upgrades, as source control measures usually provide only a small decrease in noise levels and may take significant time to be installed.

Examples of types of mitigation include: minimisation of wheel and rail roughness (for example regular wheel and rail grinding); reduction of wheel and rail acoustical radiation; track lubrication to reduce squeal on curves; and lessening of sound propagation using rail screens, barriers and vehicle skirts.

Appropriate combinations of measures applied to wheel and track design can reduce noise by more than 10 dB(A) L_{Aeq}. However, this requires a coordinated approach between rolling stock operators and infrastructure owners. This can prove challenging in many contexts, particularly where responsibility for vehicles and track are segregated (CRC for Rail Innovation, 2011).

2.1.4 Industrial noise and other fixed noise sources

Noise from mechanised industry creates problems both for indoor and outdoor settings. The noise is generally due to machinery and often increases with the power of the machines. The noise generated by machinery may contain mainly low or high frequencies, tonal components, be impulsive or have unpleasant and disruptive temporal sound patterns. Rotating and reciprocating machines produce sound that includes tonal components.

Air-moving equipment tends to create noise with a wide frequency range. Components or gas flows that move at high speed result in high sound pressure levels (Berglund et al., 1999). Examples include fans and steam pressure relief valves, as well as operations involving mechanical impacts, such as stamping, riveting and road breaking.

Fixed sources of industrial and other noise include: extractive industries – oil, gas and mining, manufacturing, construction, agriculture, military and power generation.

The National Health and Medical Research Council (NHMRC) has investigated the evidence on wind farms and human health and concluded there is no consistent evidence that wind farms cause adverse health effects in humans. Given the poor quality of current direct evidence and the concern expressed by some community members, high quality research into possible health effects of wind farms, particularly within 1500 metres, is warranted (NHMRC, 2015).

2.2 Social surveys of noise annoyance

South Australia noise perception and quality of life survey (2014)

In South Australia, a representative state-based survey interviewed 3015 people using a standardised noise annoyance survey tool (Nitschke et al., 2014). Noise from road transport was reported as a source of annoyance (little to extreme) by the highest proportion of respondents (27.7 per cent), followed by noise from neighbours (22 per cent), construction noise (10.0 per cent), air conditioner noise (5.8 per cent), rail transport noise (4.7 per cent) and industrial noise (3.9 per cent).

The survey indicated that 25.1 per cent of people surveyed lived less than 50 metres from a major road in South Australia. When the results were extrapolated to the state population, 6.9 per cent of people were estimated as being highly annoyed by noise from at least one source.

Perth community noise survey (2011)

The West Australian Department of Environment and Conservation (DEC) undertook a survey in 2011 to evaluate community attitudes to and experience of local noise. A stratified random sample of 410 respondents from the greater Perth area was surveyed. Of the respondents, 30.2 per cent considered noise a problem in their area, with 12.7 per cent considering noise a significant problem, and 5.6 per cent considering it to be a major problem (DEC, 2011).

Victoria noise survey (2007)

A social survey of 1213 respondents by Environment Protection Authority Victoria was done in 2006 to understand the impact of noise on the community. It found that almost half of all Victorians (49 per cent) had been disturbed or annoyed by environmental noise at some stage in the preceding 12 months (EPA Victoria, 2007).

2.3 Relevance of urban and built form, climate and behaviour to noise exposure

The urban population of Australia accounts for about 70 per cent of the total population (ABS, 2014). Concerns about the growth of larger cities have placed more focus on urban design and planning in the past five years, with most state governments producing strategic plans for their capital cities. These include policies to minimise outer suburban sprawl and encourage higher density residential development around major activity centres and routes served by public transport. Policies to abate the problem of increases in external noise have also been put forward by public and private sector agencies.

The main responses to reduce noise are through building design, public engineering works and land use planning. Examples of good architectural design of buildings to reduce noise include orientation of buildings and habitable rooms away from the noise source. Examples of public engineering works include barriers and landscaping close to roads and railways as well as quieter roads and railways. Examples of land use planning approaches include separating noisy transport routes from noise sensitive areas, managing traffic and reducing speed, and restricting the slope of roads and curves in railway tracks to decrease noise.

In NSW the State Environment Planning Policy (Infrastructure) 2007 sets out specific planning provisions and controls for developments in rail corridors and near busy roads.

Legislated planning mechanisms are important at the earliest stage of the development, such as at the zoning, subdivision or initial development design stages. This helps manage the potential for land use conflict around noise before construction starts.

For residential dwellings near noise sources, the effectiveness of exposed façades in attenuating noise is another important factor. The simplest types of facades reduce sound by about 15 dB(A) from outside to inside when the windows are closed. Double brick walls generally provide adequate noise reduction. Weatherboard walls can be upgraded with in-cavity insulation, although the effectiveness is relatively small. Insulation of roofs is also important, particularly in areas where aircraft noise is an issue.

Due to their lightweight construction, windows are generally the weakest point in the sound propagation path. Single and double window glazing can reduce noise by up to 30 and 35 dB(A) when closed. However, when windows are slightly open, outside sound levels are reduced only by 10 to 15 dB(A). This is particularly important as many Australians prefer their windows slightly open at night for ventilation. In Western Australia state planning policies recommend fans or air conditioning in conjunction with upgraded glazing to ensure adequate ventilation when windows are closed to exclude noise.

2.4 Regulatory approaches and mechanisms to limit exposure

2.4.1 Road traffic noise

The Australian Design Rules for motor vehicles are national standards for safety, anti-theft and emissions. They are generally performance based and cover issues such as occupant protection, structures, lighting, noise, engine exhaust emissions, braking and other items. Under the *Motor Vehicle Standards Act 1989*, four rules apply to noise from vehicles. These define the limits on external noise generated from cars, trucks, buses, motor cycles and mopeds (Department of Infrastructure and Regional Development, Australian Design Rules). Similarly, state-based road rules prohibit driving in a way that makes unnecessary noise. An example includes Victorian Road Safety Rule 291 that states: "a person must not start a vehicle, or drive a vehicle, in a way that makes unnecessary noise or smoke".

Noise from engine brakes is the greatest source of community complaint against the heavy vehicle industry. In November 2007, Australian transport ministers unanimously approved a regulatory proposal and model law for an in-service engine brake noise standard and testing procedure for heavy vehicles. The standard would provide an objective enforcement approach that defines a limit on the noise emitted from an engine brake. However, this has not yet been implemented across the states and territories due to technical and operational issues (National Transport Commission, 2013). State-based vehicle standards put limits on noise from in-service noise but these are often less stringent than Australian Design Rules.

Traffic restrictions and traffic calming measures have generally reduced traffic noise due to changes in: traffic volume and composition, road layout and surface, vehicle speed and driving style. The use of traffic calming and restrictions may need more attention to address urban noise in residential areas. Transportation and town planners may need to explore freight traffic patterns, particularly in areas with increasing urban density, and consider approaches such as special routing, freight traffic centres and ways to encourage more environmentally friendly freight traffic.

Efforts to reduce noise exposures through home insulation and construction of noise barriers in communities exposed to road traffic noise have also been made. Australian Standard 3671:1989, Acoustics – Road traffic noise intrusion – Building siting and construction, provides guidance on acoustic requirements for residential dwellings near roads. There are also statutory approval processes for new and redeveloped roads.

Examples of policies used in these approval processes in NSW include the Road Noise Policy (NSW EPA, 2011), which assigns acoustic design requirements. The NSW State Environment Planning Policy (Infrastructure) requires homes built alongside busy road and rail corridors to incorporate measures to achieve required internal noise levels. NSW Roads and Maritime Services has a specialised noise abatement program to address road traffic noise through a range of approaches.

2.4.2 Aircraft noise

Air Navigation (Aircraft Noise) Regulations (1984) require all aircraft operating in Australian airspace to comply with noise standards and recommended practices under the Chicago Convention (Convention on International Civil Aviation). These are set out in the International Civil Aviation Organisation (ICAO) document Annex 16, Environmental Protection – Volume I (ICAO, 2008). Aircraft found to be compliant are issued with a noise certificate. Aircraft without a noise certificate are not permitted to operate in Australia.

Flight activities and aircraft curfews are the responsibility of Airservices Australia, individual airport authorities and the Commonwealth government. The *Airports Act (1996)* was passed to cover environmental protection regulations. It governs noise and other environmental issues, but only 21 airports are covered by this act.

The Australia Standard AS 2021:2015 Acoustics – Aircraft noise intrusion – Building siting and construction (Standards Australia, 2015) provides guidance on the siting and construction of buildings near airports to minimise aircraft noise. The assessment of potential aircraft noise exposure at a given site is based on the Australian Noise Exposure Forecast (ANEF) system. The standard also provides guidelines for the type of building construction necessary to reduce noise to a given level. It is widely referred to in guiding strategic land use planning near airports. The AS 2021:2015 specifies that it is acceptable to build noise-sensitive developments in areas where ANEF is less than 20. Noise-sensitive developments are conditionally acceptable between ANEF 20 and 25 provided required internal sound levels are achieved through building design. However, some airport noise complaints come from areas beyond ANEF 20 contours.

Noise insulation programs were established around Sydney Airport in 1995 and Adelaide Airport in 2000. Residential properties with greater than ANEF 30 contour exposure and public buildings (schools, churches, day care centres and hospitals) with greater than ANEF 25 contour exposure were eligible for assistance in obtaining insulation. The programs aimed to achieve a 35 dB(A) lowering of noise levels for bedrooms, and 30 dB(A) for living rooms (Department of Infrastructure and Regional Development, 2014). Sydney airport also has a long-term operating plan to manage aircraft noise by directing flights over water and non-residential land and by spreading the noise across different communities (Airservices Australia, 2015a,b).

2.4.3 Rail noise

There has been a great deal of discussion at the national government level about rail infrastructure and ways to improve rail operations. Funds for improving track and rolling stock might be invested in equipment with reduced noise generation. Limited information is available on national efforts to reduce rail traffic noise in concert with rail improvements. However, a national initiative to develop rolling stock standards is being led by the Rail Industry Safety and Standards Board.

Individual states have developed rail noise initiatives, including standards, guidelines and noise abatement programs. These programs include methods for assessing and prioritising requests for mitigation from people particularly affected. Environmental planning guidelines for residential developments near rail corridors set acceptable internal noise levels and provide advice to developers on how to achieve them.

2.4.4 Industrial noise and other fixed noise sources

Control of industry noise affecting communities is the responsibility of planning and environment authorities in the states and territories. Local ordinances or operation restrictions may be needed if construction activities take place in an area with sensitive uses, such as schools or hospital zones, or outside standard construction hours. Reductions in industrial noise can be achieved by encouraging quieter equipment or by zoning controls to separate acoustically incompatible land uses, such as the contrast between residential and industrial zones. Noise emissions, like other environmental emissions, may also be licensed or regulated under relevant environmental legislation.

2.4.5 Other noise sources

Domestic equipment may have times-of-use restrictions, such as grass cutting machines, leaf blowers, chainsaws, domestic air conditioners, mobile air compressors, pavement breakers, and mobile garbage compacters. This includes the use of power tools on residential properties either under state and territory legislation or local government regulation. The former Standing Council on Environment and Water discussed a national policy on noise labelling for portable equipment but this has yet to come to fruition. Noise labelling is required in some states, for example NSW, under the revised *Protection of the Environment (Noise Control) Regulation* 2008.

Other noise sources of concern include that from fireworks and explosives during celebrations, and from children's toys. Australian Standard AS/NZS 8124.1:2002, Safety of toys, includes noise regulations.

2.4.6 Building requirements to protect against noise

The internal acoustic requirements for dwellings are determined by the National Construction Code (NCC, 2016) as well as local councils. The Australian Building Codes Board administers and maintains the code to encourage national consistency based on minimum safety and health requirements. The code is given legal effect by relevant legislation in each state and territory.

Australian Standard 2107:2016, Acoustics—Recommended design sound levels and reverberation times for building interiors, is the standard most commonly referred to in building acoustics. The standard, while not mandatory, sets out recommendations for design sound levels for building interiors. The Australian Association of Acoustical Consultants has also produced a *Guideline for Apartment and Townhouse Acoustic Rating* (AAAC, 2010), a performance-based guideline for insulation. The guideline contains a star rating corresponding to the intrusion of external noise into bedrooms and habitable rooms as shown in Table 2-2. This has been adopted by many in the industry.

Table 2-2: Guideline for acoustic rating of apartments (Adapted from Australian Association of Acoustical Consultants, 2010)

Apartment rooms	External noise intrusion	2 star	3 star	4 star	5 star	6 star
Bedrooms	Continuous noises	36 dB(A)	35 dB(A)	32 dB(A)	30 dB(A)	27 dB(A)
	Intermittent noises	50 dB(A)	50 dB(A)	45 dB(A)	40 dB(A)	35 dB(A)
Other habitable rooms including open kitchens	Continuous noises	41 dB(A)	40 dB(A)	37 dB(A)	35 dB(A)	32 dB(A)
	Intermittent noises	55 dB(A)	55 dB(A)	50 dB(A)	45 dB(A)	40 dB(A)

2.5 Best practice noise exposure information – noise mapping

Broadly defined, noise mapping is a means of presenting calculated and/or measured noise levels in a representative manner over a particular geographic area. The European experience may provide a basis for an Australian approach. The European Union Environmental Noise Directive (END) (2002) applies to noise to which humans are exposed. It focuses on built-up areas, public parks or other quiet areas in an agglomeration, quiet areas in open country, near schools, hospitals and other noise-sensitive buildings and areas (Article 2.1). The END is one of the main instruments to identify noise pollution levels and to trigger the necessary action at member state and European Union level.

In the context of the END, the European Commission has common noise assessment methods (CNOSSOS–EU) for road, railway, aircraft and industrial noise to improve the reliability and comparability of results across the European Union. This framework allows for coherent and reliable strategic noise mapping and action planning. Assessment of noise exposure is done using strategic noise maps with harmonised noise indicators L_{den} and L_{night} for major roads, railways, airports and agglomerations.

In the first phase (June 2007) strategic noise maps were compiled for EU member states. These covered agglomerations with more than 250,000 inhabitants, major roads with more than 6 million vehicle passages a year, railways with more 60,000 train passages a year and major airports with more than 50,000 movements a year.

The second phase (June 2012) produced strategic noise maps for agglomerations with a population of more than 100,000.

The END also determines levels of exposure to environmental noise using the above indicators. Estimates of the number of people living in dwellings exposed to values of L_{den} and L_{night} at the most exposed building façade are done separately for road, rail, air traffic and industrial noise. Where possible and available, information about people living in dwellings with special insulation against noise or with quiet façades is also reported.

Noise maps are only as accurate as the input data and techniques used to calculate sound levels. They may not always accurately depict sound level variations that occur locally. They can also be expensive to produce.

Despite these limitations, noise maps have significant uses for public health in providing estimates of exposure that can help quantify the burden of environmental noise. The European experience provides a useful insight into how similar work might be done in Australia.

3 NOISE AND SLEEP DISTURBANCE

3.1 Introduction and background

Sleep serves an important restorative purpose in promoting functioning and a sense of wellbeing. Obtaining sufficient duration and quality of sleep is important for overall health and wellbeing. Sleep problems are common in many countries, including Australia (Deloitte Access Economics, 2011).

Poor sleep has been linked to numerous adverse consequences, including health conditions such as cardiovascular disease, depression and obesity (Riemann et al., 2011), as well as accidents and disability due to fatigue (Horne and Reyner, 1999), and lost workplace productivity (Iverson et al., 2010; Rosekind et al., 2010). These translate into considerable social and economic costs, with three sleep disorders alone – obstructive sleep apnoea, primary insomnia and restless leg syndrome – estimated to cost the Australian economy $36 billion a year (Deloitte Access Economics, 2011). The economic costs of sleep problems more broadly (such as daytime sleepiness or short sleep) are estimated to be considerably higher (Deloitte Access Economics, 2011).

Many genetic, lifestyle, health and environmental factors have the potential to influence the quality and amount of sleep. Poor sleep can reflect lifestyle factors such as screen time, physical activity, alcohol consumption and caffeine consumption. Psychological characteristics such as stress, sensitivity and personality characteristics have also been linked to sleep quality.

Environmental noise has long been identified as a potential cause of poor sleep. Reviews conducted to help inform guidelines show a strong basis for believing that environmental noise during the night is a contributor to poor sleep (WHO, 2009). Many recent studies have suggested that exposure to road, rail and aircraft noise is linked to a range of sleep disturbances, including increased arousals (Tassi et al., 2010), insomnia symptoms (Halonen et al., 2012), and poorer self-reported sleep quality (Kim et al., 2014).

3.2 Systematic review of the literature: environmental noise and sleep disturbance

A systematic review of the literature was done for studies from January 1994 to March 2014 on the relationship between environmental noise and sleep. Appendix A details the review's objectives and methodology.

3.2.1 Search results

The results of the search process are summarised in the following PRISMA flow chart.

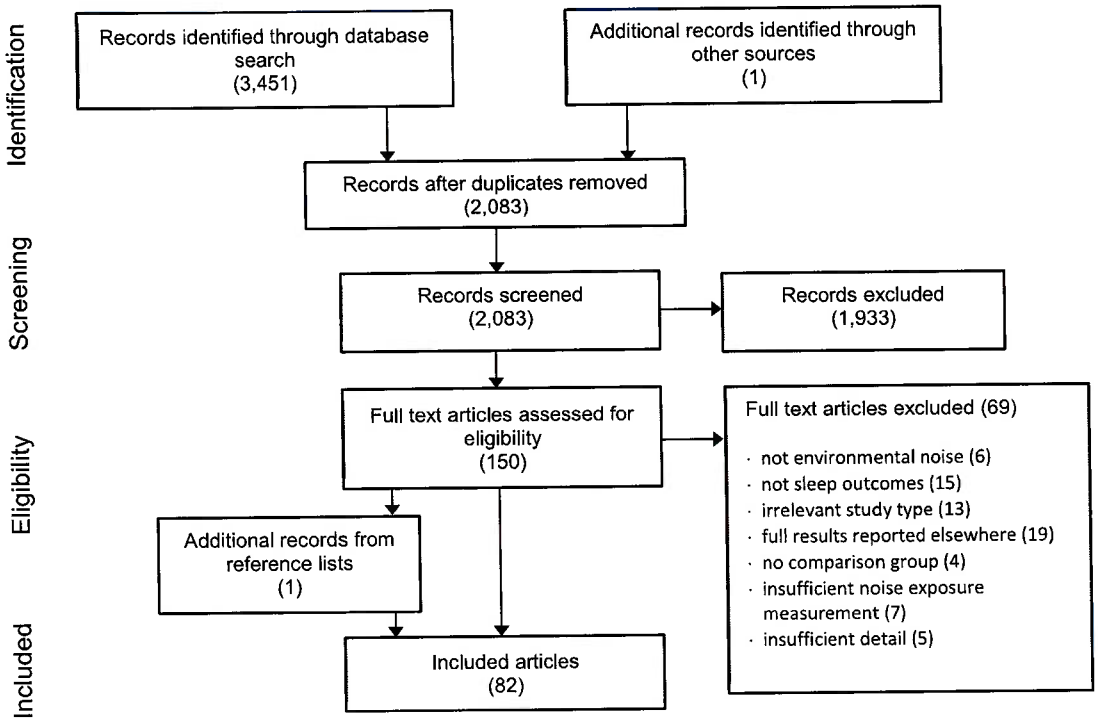


Figure 3-1: PRISMA flow chart – number of articles identified and reviewed during the systematic review (Moher et al. 2009)

3.2.2 Overview of included studies

Although outside the scope of this review, it is obvious that loud noises disrupt sleep. Loud noises are used throughout the world to disturb sleep as a method of studying its underlying functions.

Of the 82 articles identified, 79 were from distinct studies as some articles reported on the same data. Of these 79 studies, 43 were observational and 36 experimental. Most were observational studies (31 were cross-sectional studies (NHMRC level IV) and there was one prospective cohort study (NHMRC level II). There were eight field studies, where individuals had their sleep patterns and noise exposure monitored in their homes for several days. These were categorised as NHMRC level III-2 studies. Three studies included both a cross-sectional and field study component.

According to the NHMRC hierarchy of evidence (Table A-7), the experimental studies were either non-randomised experimental studies (31 studies, NHMRC level III-2) or pseudo-randomised studies (5 studies, NHMRC level III-1). Although many were non-randomised in design, several used counterbalancing to allocate participants to conditions and were thus rated as having a lower risk of bias (Table A-5). Most of these studies were done in temperature and sound-controlled sleep laboratory settings (32 studies). Some were done in the participant's home (7 studies) for some or all of the experimental period. Simulated noise was delivered via loudspeaker or personal music player with earphones.

3.2.3 Noise exposure and how it was measured

Observational studies explored: road traffic noise (29 studies), aircraft noise (8), railway noise (7), road work noise (1) and blast noise from a military base (1). Experimental studies simulated noise from: road traffic (21 studies), aircraft (9), railways (16) and road work (1).

For observational studies, noise exposure was measured by direct measurement with sound level meters in various locations (28 studies) or estimated using models or noise contour maps (17). In experimental studies, noise was delivered in such a way as to control the noise levels participants were exposed to.

The most common noise indicators used in the included studies were A-weighted equivalent sound levels (L_{Aeq}) for various periods. Maximum sound pressure levels (L_{Amax}) were also commonly used.

3.2.4 Types of outcomes reported

The included studies assessed a wide range of sleep outcomes. The most common were self-reported sleep disturbance outcomes (36 observational and 28 experimental studies). These included subjective assessments of problems falling and staying asleep, sleep duration, sleep quality/ disturbance ratings and feelings of tiredness/feeling well rested the next day.

Objective measures of sleep disturbance include activity trackers which can be referred to as actigraphy, actimetry or accelerometer (7 observational and 5 experimental studies) and polysomnography (4 observational and 22 experimental studies). These measure sleep parameters including arousals, gross bodily movement (motility) and sleep structure.

Other outcomes reported in these studies were the use of sleep medications (two observational studies) and prevalence or incidence of insomnia using International Statistical Classification of Diseases definitions (one observational study). One experimental study used an infrared pupillographic sleepiness test.

3.2.5 Quality ratings

Quality ratings according to GRADE criteria are shown in Table 3-1 to Table 3-3. These indicate that on aggregate, the quality of the evidence was rated as low.

All included studies are listed in section 8.3.

Table 3-1: GRADE evidence profile for environmental noise and sleep - Self-reported sleep disturbance (problems falling and staying asleep, sleep duration, quality/disturbance ratings, feelings of tiredness/or being well rested, and symptoms of insomnia)

No of studies (design)	Reasons for rating quality down	Reasons for rating quality up	Summary of key findings	Quality score
Thirty-two (cross-sectional)	Serious risk of bias	None	Exposure to road, rail and aircraft noise was associated with increased risk of sleep disturbance.	⊕⊕○○ Low
One (prospective cohort)	Serious risk of bias One small study	None	Self-reported sleep quality affected by road traffic noise, and significantly improved through noise abatement. Number of awakenings not affected by noise or noise abatement.	⊕⊕○○ Low

No of studies (design)	Reasons for rating quality down	Reasons for rating quality up	Summary of key findings	Quality score
Six (field studies)	Serious risk of bias Some inconsistency	None	Significant decreases in sleep quality and increased awakenings in participants exposed to high levels of night-time road traffic noise. Little to no effect of aircraft and rail.	⊕⊕○○ Low
Ten (experimental)	Some risk of bias Some inconsistency	None	Disruptions to sleep and poorer sleep quality are greater with increasing noise levels. Evidence was strongest for two aircraft noise studies.	⊕⊕⊕○ Moderate

Table 3-2: GRADE evidence profile - Objective sleep disturbance (actigraphy, polysomnography, accelerometer, Infrared pupillographic sleepiness test)

No of studies (design)	Reasons for rating quality down	Reasons for rating quality up	Summary of key findings	Quality score
Eleven (field studies)	Serious risk of bias Serious inconsistency	None	Increasing sleep stage changes and motility with maximum levels of aircraft and rail noise. Mixed results for road noise.	⊕⊕○○ Low
Twenty-six (experimental)	Some risk of bias Some inconsistency	None	Noise significantly changed sleep structure with less slow wave sleep, greater latency to slow wave sleep, more arousals and sleep stage changes.	⊕⊕⊕○ Moderate

Table 3-3: GRADE evidence profile for environmental noise and sleep - Use of sleep medication (self-report)

No of studies (design)	Reasons for rating quality down	Reasons for rating quality up	Summary of key findings	Quality score
Two (cross-sectional)	None	None	Increasing aircraft and railway noise levels associated with increased risk of sleep medication use.	⊕⊕○○ Low

3.3 Summary of findings from the systematic review

3.3.1 What is the evidence of a causal effect of environmental noise on sleep disturbance?

This systematic review identified 79 studies published between 1994 and 2014 examining the relationship between environmental noise exposure and sleep disturbance. A total of 43 of these studies were observational and 36 experimental.

A particular issue in sleep studies is the problem of blinding participants or outcomes assessors to the condition being tested. This is coupled with the problem of defining what constitutes disturbed sleep.

Subjective measures may provide a better indication of when sleep has been notably disturbed but suffers from bias because of the blinding issue.

Objective measures tend to derive from highly sensitive physiological measures such as collected by polysomnography and it remains unclear what sized effect, if any, perturbations in many of these measures means for people's health, or annoyance levels.

Many of the measurements of sleep may be too sensitive for a person to even notice and may be below their level of a just-noticeable difference.

Another issue is the heavy reliance on laboratory based experiments in sleep and noise research. These can be designed with better scientific rigour but this always comes at a cost to the external validity of the study as the participants are often heavily screened and do not represent the population as a whole. The participants are also not sleeping in their own environments, which may influence their response to noise either positively or negatively.

3.3.2 Observational studies

Several studies below examined more than one noise source.

Aircraft noise

Eight studies examined the associations between aircraft noise exposure and sleep disturbances. All indicated that aircraft noise was associated with poorer sleep.

Road traffic noise

A total of 28 studies examined the associations between exposure to road traffic noise and sleep disturbances. Most of these (21 of 28) indicated that higher noise levels were linked with poorer sleep. The rest found non-significant results.

Rail noise

Seven studies examined the relationship between railway noise and sleep disturbance. Six reported a significant relationship between rail noise and sleep disturbance, with one reporting non-significant results. Most assessed both freight and passenger rail noise in the study.

Other noise sources

Three studies examined other relevant environmental noise sources such as general community noise and noise from military areas. All found that higher levels of noise were linked with poorer sleep.

Study limitations

Despite the consistency of these findings, the quality of the evidence provided by these studies was determined to be low. This low quality rating reflects issues relating to study design (such as predominantly cross-sectional studies), and high risk of bias (primarily due to measurement of sleep and control of confounders). These issues are detailed below and limit our ability to draw definitive conclusions about the effects of environmental noise on sleep.

For the study design, most of the observational studies (34 out of 43) were cross-sectional (NHMRC level of evidence: IV). Although most of these reported significant relationships between environmental noise and sleep, they are not able to provide insight into the causal effect of noise on sleep. Further, 18 of the 34 cross-sectional studies had a high risk of bias and 13 had moderate risk of bias. Only two studies were rated as having a low risk of bias (Halonen et al., 2012; Kim et al., 2014). The large number of studies with moderate or high risk of bias was primarily due to self-reported measures of sleep (27 out of 34 studies) and inadequate control of relevant confounding variables (22 out of 34 cross-sectional studies). The Lundby tunnel study (Öhrström, 2004; Öhrström and Skanberg, 2004) was the only prospective cohort study in this review. It was rated as having a high risk of bias due to self-reporting measures of sleep and lack of control for potential confounders.

The eight field studies (NMHRC level of evidence: III-2) give better insight into the causal nature of the relationship between noise exposure and sleep disturbance. This is because these studies provide an indication of the concurrent relationships between noise exposure and sleep in the usual sleep environment. The immediate effects of noise exposure on sleep outcomes can therefore be assessed in these studies. However, only two of the eight studies had a low risk of bias. Of the remaining studies, three had a moderate risk of bias and three had a high risk of bias. The main issues underlying the moderate and high risk of bias were self-reported measures of sleep and inadequate control.

Some further issues in methodology require discussion. It was difficult to draw clear conclusions from these studies due to the large variation in the sleep outcomes assessed. For example, the types of sleep outcomes assessed included sleep disturbance, sleep quality, insomnia symptoms, night-time awakenings, daytime dysfunction, and use of sleep medication, sleep stages and sleep efficiency. This was further compounded because most sleep outcomes were based on self-reporting measures only, with a large number of studies using single-item measures of sleep quality. These measures lack validity compared with objective measures and have the potential to lead to imprecise estimates on the relationship between noise and sleep. These issues suggest that caution is needed when interpreting the results of the observational evidence base.

The noise exposure indicator is relatively consistent across studies (usually L_{Aeq} or L_{Amax}). However, studies varied considerably in how the noise exposed was estimated (such as direct measurement or contour maps) and the site at which it was measured (such as at building façade or the participant's ear). This complicates the synthesis of the evidence.

Similarly, within the studies it is important to distinguish between façade noise levels, often used in Australia and France, and the free field noise levels often used in other countries. Free field noise levels account only for noise coming from a source. Façade levels account for both noise coming from a source and noise reflected back from a façade. A façade level is typically 2.5 to 3.0 dB higher than the corresponding free field.

Studies with a low risk of bias

Only two cross-sectional (Halonen et al., 2012; Kim et al., 2014) and two longitudinal studies (Basner et al., 2006; Frei et al., 2014) had a low risk of bias. The results of these are briefly outlined below.

Halonen et al. (2012) conducted a cross-sectional study of 7019 adults and found that symptoms of insomnia were significantly higher when road traffic noise measured at a residential façade exceeded L_{night} 55 dB (odds ratio (OR) = 1.32 [1.05 – 1.65]). Kim et al. (2014) examined the relationship between exposure to aircraft noise (from a military airport) and sleep quality in a sample of 1982 adults. The results indicated that noise levels (Weighted Equivalent Continuous Perceived Noise Level measured externally) between 60 and 80 dB (OR = 2.61 [1.58 – 4.32]) and > 80 (OR = 3.52 [2.03 – 6.10]) were linked with disturbed sleep.

Basner et al. (2006) conducted an experimental field study of 64 adults. They found that aircraft noise events that were above 33 dB (measured at the ear) were associated with increased awakenings. Frei et al. (2014) conducted a study of 1122 adults comparing sleep disturbance using a standardised sleep disturbance score with modelled road traffic noise. This study found that road traffic noise levels > 55 dB L_{Aeq} (measured at the residential façade) were associated with a greater prevalence of sleep disturbance.

3.3.3 Experimental studies

There were 36 experimental studies examining the relationships between environmental noise exposure and sleep outcomes. Several studies examined multiple noise sources, such as road, rail and air.

Most studies indicated that exposure to environmental noise was significantly associated with sleep disturbances.

Aircraft noise

Nine studies examined the effects of aircraft noise exposure and sleep disturbances. All indicated that aircraft noise led to poorer sleep.

Road traffic noise

Twenty one studies examined the effect on sleep of exposure to road traffic noise. Most (15 out of 21) indicated that higher noise levels were linked with poorer sleep. The rest reported non-significant results.

Rail noise

Sixteen studies examined the effects of rail noise on sleep disturbance. Most (15 out of 16) reported significant deleterious effects of noise on sleep. Most assessed both freight and passenger rail noise within the study.

Other noise sources

Only one study investigated the effects of construction noise. It found that higher noise levels were associated with poorer sleep.

Study limitations

The experimental studies have higher level of evidence ratings (NHMRC), and thus provide an important insight into the effects of noise on sleep. In general, these studies had lower risk of bias compared with the observational studies. For example, nine studies had a low risk of bias and 14 had a moderate risk of bias. About one third of the experimental studies (13 studies) had a high risk of bias.

The main factors underlying moderate and high risk of bias reflect the lack of randomisation to conditions or the lack of counterbalancing. Several studies did not blind participants and outcome assessors to the condition allocation, which could also increase the risk of bias, noting that it is difficult to blind participants to noise conditions. Although many studies used objective measures of sleep, several relied on self-reported measures. In combination with the issues raised above, the often small sample sizes (such as those less than 10) contributed to an elevated risk of bias.

The wide variety of sleep outcomes examined also makes it difficult to draw clear conclusions about the effects of noise on sleep. The lack of prospective study registration in this field makes it impossible to gauge the extent of selective reporting of outcomes. Although most experimental studies used polysomnography, the specific sleep parameters varied. These parameters included sleep duration, sleep efficiency, sleep stages, sleep-stage transitions, sleep latency, time in rapid eye movement (REM) sleep and sleep spindles.

The implications of many of these outcomes (such as sleep spindles and sleep stage transitions) are yet to be determined. This means the implications of some findings for sleep disturbance are not clear.

Although the experimental studies generally had a lower risk of bias compared with the observational studies, many of them may lack external validity. This is particularly the case for those studies that assessed sleep in laboratory settings. The results of these studies may not provide a valid indication of the effects of noise on sleep in a real world setting.

3.3.4 Studies with a low risk of bias

All of the nine studies with a low risk of bias indicated that exposure to various sources of noise was linked with disturbed sleep. For example, Schapkin et al. (2006a) examined the effects of rail noise on sleep assessed via polysomnography and self-reporting in a sample of 22 adults. The results showed that increasing rail noise (from quiet to L_{Aeq} 50 dB(A)) measured at the ear was linearly associated with poorer subjective sleep.

Schapkin et al. (2006b) examined the effects of nocturnal aircraft noise measured at the ear on self-reported sleep. The results indicated that subjective sleep quality linearly worsened with increasing aircraft noise levels (from quiet to L_{Aeq} 50 dB(A)).

Basner and Samel (2005) examined sleep in 128 subjects (16 controls) across 13 consecutive nights. Their results indicated that exposure to aircraft noise measured at the ear was significantly associated with some indicators of disturbed sleep. This included increased awakenings and alterations to sleep architecture resulting in less slow wave sleep and more stage 1 light sleep.

Subsequent analysis suggested these associations became apparent only at maximum sound pressure level (SPL) at or above 50 dB(A) (awakenings), at or above 55 dB(A) (increased stage 1 light sleep), and at or above 65 dB(A) (decreased slow wave sleep). The analysis also suggested these associations were significant only when the number of aircraft noise events was greater than or equal to eight (increased awakenings), 16 (reduction in slow wave sleep), and 64 (increased stage 1 light sleep).

3.3.5 Summary of the evidence

The observational and experimental studies together indicate a significant relationship between exposures to higher levels of environmental noise and sleep disturbances. However, the issues in method noted above and variations in study design makes it difficult to draw definitive conclusions from the evidence base.

The quality of the evidence was rated as low for the observational studies given the large number of cross-sectional studies and the high risk of bias. The experimental studies generally provided better quality evidence.

Both observational and experimental studies assessed a wide range of sleep parameters using various measures.

Many studies used both objective and subjective measures of sleep disturbance. Noise was found to exert a larger effect on self-reported sleep compared with objectively assessed sleep. One mediating factor may be that annoyance caused by noise may cause sleep disturbance and extended awakening. Some individuals may therefore over-report the effects of noise on the quality of their sleep. Some studies using a combination of objective and subjective measures found effects for self-reported sleep but no or very weak effects for polysomnography-assessed sleep. Examples include the study by Schapkin et al., 2006a. This suggests that the effects of environmental noise are overestimated in those studies using self-reported sleep measures.

3.3.6 Is there a dose–response relationship between environmental noise and sleep disturbance?

Many observational studies demonstrated that sleep disturbances become more pronounced as noise level increases (e.g. Banerjee, 2013; Bluhm et al., 2004; Boes et al., 2013; de Kluizenaar et al., 2009; Franssen et al., 2004; Frei et al., 2014; Kim et al., 2014).

The precise measures of sleep varied considerably between studies, as did the quantification of noise exposure. For example, Boes et al. (2013) examined the effects of a 1 dB(A) increase in

noise, de Kluizenaar et al. (2009) broke noise exposure into 10 dB(A) categories, and Frei et al. (2014) assessed four noise exposure groups (< 30 dB(A), 30 – 40 dB(A), > 40 – 55 dB(A), and > 55 dB(A)). This lack of consistency means it is possible to conclude that observational studies show a dose–response relationship, but the precise nature of the relationship cannot be determined easily.

Several experimental studies also indicated a dose–response relationship between noise exposure and sleep disturbance (e.g. Basner and Samel, 2005; Kawada and Suzuki, 1995; Schapkin et al., 2006a). Again, major methodological differences between studies make it difficult to combine studies. Studies were also difficult to compare due to the varying noise metrics used.

As an example, L_{den} is a noise metric that describes a hybrid of noise over the day, evening and night. It could be argued that the day and evening parts are irrelevant to sleep (unless the subjects sleep during the day). A night-time level would be more helpful. Also, L_{Aeq} is a noise metric that effectively describes noise as an average over an extended period. Particularly in the case of aircraft and train noise, it depends on the number of noise events and their specific noise levels.

Reported thresholds are outlined below for each of the three main noise sources.

Road traffic noise

Seven observational studies examined the effects of road traffic noise and found significant impairments in sleep quality associated with noise levels measured at the exterior façade above 55 dB L_{night} (Banerjee, 2013; Halonen et al., 2012; Ristovska et al., 2009) and 55 dB L_{Aeq} (Frei et al., 2014; Kristiansen et al., 2011; Lercher and Kofler, 1996; Yoshida et al., 1997)

Several experimental studies also reported significant effects of peak or equivalent noise levels at or above 45 dB(A) (Kawada and Suzuki, 1999; Kuwano et al., 2002).

Rail noise

Two observational studies examining rail noise found significant relationships with sleep disturbances at noise levels measured at the exterior façade of ≥ 60 dB L_{Aeq} (Aasvang et al., 2008) and ≥ 60 dB L_{den} (Lercher et al., 2010).

Experimental studies indicated that the effects of rail noise on sleep were observed at lower levels, with several studies finding effects above 50 dB(A) (Kaku et al., 2004; Saremi et al., 2008; Bonnefond et al., 2008) and 54 dB(A) (Griefahn and Robens, 2010).

Aircraft noise

Two observational studies indicated that threshold effects for aircraft noise were comparatively low at 32 dB $L_{Aeq,night}$ (Passchier-Vermeer et al., 2002) and 33 dB L_{ASmax} (Basner et al., 2006).

Experimental studies indicated some effects of aircraft noise at 39 dB(A) (Schapkin et al., 2006b) and 45 dB(A) (Basner et al., 2008), but effects were reported to be most evident at higher levels (for example, > 50 dB(A) or ≥ 65 dB(A)).

3.3.7 Is there any evidence that certain populations are vulnerable to the effects of environmental noise on sleep disturbance?

Only a small number of studies formally investigated whether the relationships between environmental noise and sleep disturbance were more pronounced in certain populations.

Halonen et al. (2012) found the effects of road traffic noise on insomnia symptoms were more pronounced in individuals with higher levels of self-reported anxiety traits. Bjork et al. (2006) found the effects of road traffic noise on self-reported sleep disturbances were greater in individuals with higher levels of annoyance and in individuals born overseas.

This raises the possibility that some effects may be greater in certain populations, but there is not sufficient evidence to draw strong conclusions on this.

3.3.8 Does the association between environmental noise and sleep disturbance vary by noise source?

Few studies compared whether the influence of noise on sleep disturbance varied depending on the source. Studies tended to examine one source, such as aircraft or road traffic noise. With little consistency in methods, such as sample characteristics, noise levels and experimental conditions, it is not possible to meaningfully compare results.

However, a small number of studies did compare the effects of different sources of noise. Griefahn et al. (2006b) compared the effects of aircraft, rail and road noise. Their results indicated similar effects from the sources of noise, although the effects appeared greatest for rail noise. Aasvang et al. (2011) compared the effects of road traffic noise with railway noise. The results indicated that railway noise had a greater effect on rapid eye movement (REM) sleep compared with road traffic noise. This suggests that railway noise may have a larger effect on sleep outcomes.

Basner et al. (2011) provided further insight into the nature of these differences in an experimental study that compared the effects of rail, road and aircraft noise on sleep parameters. Interestingly, the nature of the differences between noise sources varied depending on whether sleep was assessed via polysomnography or self-reported.

When polysomnography was examined, road traffic noise had the largest effects on sleep structure and continuity. However, when self-reporting measures were used, aircraft and rail noise were found to have a larger effect on sleep compared with road traffic noise (Basner et al., 2011).

Basner et al. (2011) suggested that because road traffic noise events are relatively short they were perceived as having less effect on sleep. In other words, the events were not long enough for participants to consciously perceive their sleep was affected.

In contrast, rail and aircraft noise typically last longer and so may be more likely to be perceived as having affected sleep. Basner et al. (2011) attributed the greater effects of road traffic noise on polysomnography-assessed sleep parameters to the specific acoustic properties of road traffic noise, such as faster sound pressure level rise time and greater energy in the high-frequency octave bands compared with aircraft noise.

It is plausible that aircraft, rail and road traffic noise have differential effects on sleep quality. However, because available data is limited it is not possible to draw definitive conclusions on the nature and magnitude of these differences.

3.3.9 Is there any evidence that annoyance is a mediator linking environmental noise exposure to sleep disturbance?

Annoyance is discussed by a large number of studies as a likely mechanism linking environmental noise exposure with poor sleep, particularly self-reported sleep. Some studies examined both annoyance and sleep disturbance as an outcome, but there is no evidence that studies have formally examined whether annoyance is a mediator linking noise exposure with sleep disturbance.

Frei et al (2014) found that annoyance was strongly related to self-reported sleep measures; actigraphy and diaries were used to assess sleep in a nested sub-group of this study. It was reported that measured sleep efficiency was more strongly associated with modelled noise exposure than with self-reported annoyance. This suggests annoyance is a mediating factor for

subjective sleep complaints but not an objective measure for noise. It is possible that annoyance is a mechanism linking noise exposure with poor sleep. But it is not clear if these effects are limited to self-reported or objective assessment of sleep. Because of the lack of formal investigation, it is not possible to draw any definitive conclusion on the role of annoyance in the environmental noise-sleep disturbance literature.

3.4 Conclusions

Some studies suggest a dose–response relationship between noise and physiological effects on sleep. The systematic review identified 79 studies and sub-studies published between 1994 and 2014 that examined the associations between exposure to different forms of environmental noise and sleep disturbances. In general, the results of these studies are consistent in indicating that exposure to sources of environmental noise (mainly road traffic, rail and aircraft noise) are associated with sleep disturbances.

Overall the quality of the studies in this review was low, reflecting study design, risk of bias, and inconsistency in outcome measures. As a result, an NHMRC rating statement of C is applied to the overall body of evidence (see rating criteria in appendix A). The body of evidence from this systematic review has limitations and care should be taken in interpreting the findings.

4 NOISE AND CARDIOVASCULAR DISEASE

4.1 Introduction and background

Cardiovascular disease encompasses all conditions and diseases affecting the heart and blood vessels (AIHW, 2014a). In Australia, coronary heart disease, stroke and heart failure are the most common forms (AIHW, 2014a).

Although the incidence of cardiovascular disease has declined in Australia over the past two decades (AIHW, 2014a), it is estimated that 22 per cent of the adult population has some form of the disease. It remains the major cause of death in Australia, accounting for 31 per cent of all deaths, and second only to cancer as the largest contributor to total burden of disease (AIHW, 2014b). There are many risk factors for cardiovascular disease, including age, sex and genetics, as well as modifiable risk factors such as overweight/obesity, sedentary lifestyles, unhealthy diet, smoking and alcohol consumption (AIHW, 2009).

There has also been considerable interest in the role of environmental factors such as air pollution and noise in increased risk of cardiovascular disease. The World Health Organisation estimates that around 1.5 million ischemic heart disease deaths occur globally each year (based on 2012 estimates) due to ambient air pollution (WHO, 2014). Although there are no global estimates of the impacts of environmental noise on ischemic heart disease, regional estimates for Western Europe indicate that the burden is large at 61,000 Disability Adjusted Life Years (DALYs) a year. This is around 1.8 per cent of all ischemic heart disease DALYs attributable to transport noise (WHO, 2011).

Research since the late 1960s suggests that exposure to different forms of environmental noise is linked with a greater risk of cardiovascular disease and changes in indicators of cardiovascular health, such as heart rate and blood pressure (Babisch et al., 1990; Knipschild, 1977). Many subsequent studies have further examined these relationships and some reviews of the evidence have been conducted (Babisch, 2006).

4.2 Systematic review of the literature

A systematic review of the literature was conducted for studies investigating the relationship between environmental noise and cardiovascular disease for the period January 1994 to March 2014. This is further detailed in appendix A.

4.2.1 Search results

Details of the results of the search process are summarised in the following PRISMA flow chart.

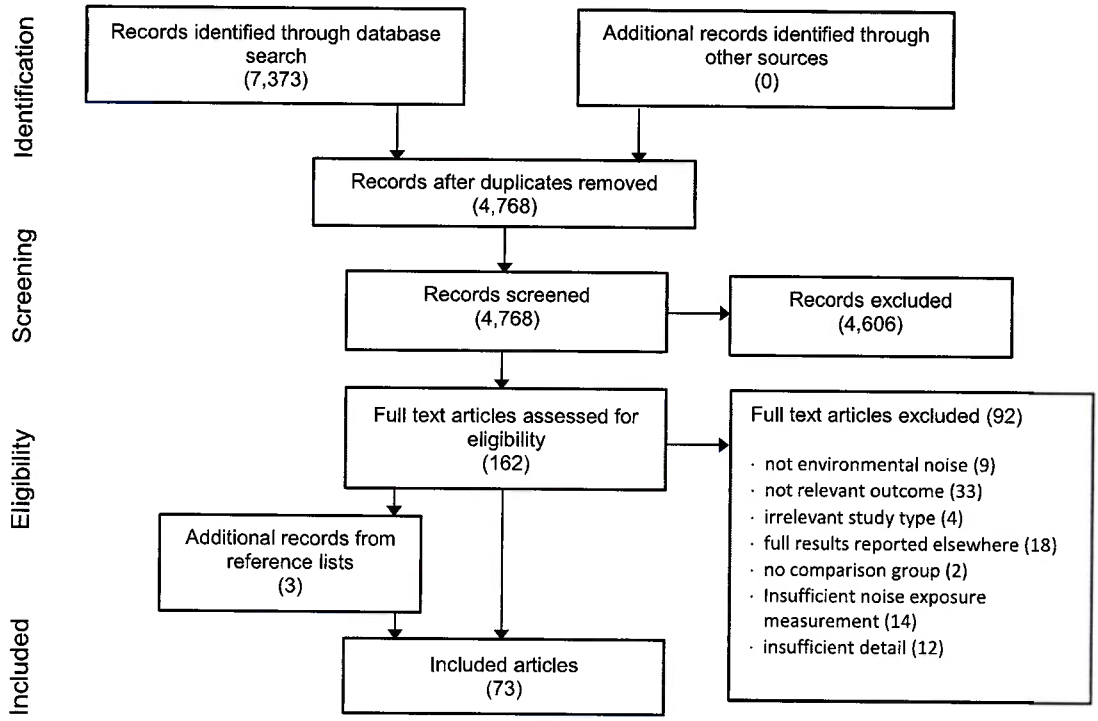


Figure 4-1: PRISMA flow chart. Number of articles identified and reviewed during the systematic review (Moher et al. 2009)

4.2.2 Overview of included studies

Of the 73 articles identified, 65 were from distinct studies (some reported on the same data); 62 were observational designs, while three were experimental. The majority of observational studies (40) were cross-sectional studies (NHMRC level IV). Some studies had multiple components with different methods, such as cross-sectional and prospective cohort components.

There was also a small number (10) of prospective cohort studies (NHMRC level II), ecological studies (five) (NHMRC level IV), case-control studies (four) (NHMRC level III-3) and field studies (three) (NHMRC level III-2).

All of the experimental studies were non-randomised experimental studies (three) (NHMRC level III-2). One was conducted in a sleep laboratory, one in a sound and temperature-controlled room and one in a park setting.

4.2.3 Noise exposure and how it was measured

Observational studies explored road traffic noise (42), aircraft noise (19), railway noise (seven), and general environmental noise (five). Experimental studies addressed the effects of road traffic (three), and aircraft noise (one) on cardiovascular disease. Several studies examined multiple sources of noise.

For observational studies, noise exposure was measured by direct measurement with sound level meters (17 studies) or estimated using models and contour maps (39 studies). Six studies used a combination of direct measurement and models/contour maps, while three did not clearly specify the measurement approach. Noise was measured using sound level meters in all three experimental studies.

The most common noise indicators used were A-weighted equivalent sound levels (L_{Aeq}) for various periods. Maximum sound pressure levels (L_{Amax}) were also commonly used.

4.2.4 Types of outcomes reported

A breakdown of the cardiovascular disease outcomes in these studies is:

- hypertension/blood pressure (45 studies)
- cardiovascular disease mortality (3 studies)
- ischemic heart disease and myocardial infarction (16 studies)
- stroke (6 studies)
- other relevant outcomes such as diabetes and aortic calcification (4 studies).

The measures used to assess these outcomes varied considerably. For example, a range of self-reported diagnoses and direct measurements of blood pressure were used across studies.

Note that several studies examined multiple cardiovascular disease outcomes.

4.2.5 Quality ratings

GRADE is a structured process for rating quality of evidence in systematic reviews. Quality ratings according to GRADE criteria are shown in Table 4-1. This indicates that on aggregate, the quality of the evidence was rated as low.

All included studies are listed section 8.4. GRADE criteria are detailed in appendix A.

Table 4-1: GRADE evidence profile for environmental noise and cardiovascular diseases (65 studies)

No of studies (design)	Reasons for rating quality down	Reasons for rating quality up	Key findings	Quality score
Cardiovascular disease mortality				
One (ecological)	None	None	Increased risk of death from myocardial infarction in people exposed to aircraft noise over 60 dB(A) especially those exposed >15 y	⊕⊕○○ Low
Three (prospective cohort)	None	None	High levels of transportation noise (≥ 65 dB(A)) associated with elevated risk of mortality.	⊕⊕⊕○ Moderate
Ischaemic heart disease and myocardial infarction (self-report)				
Four (cross-sectional)	Serious inconsistency	None	Road traffic noise may be associated with greater self-reported heart disease and stroke but confounding of air pollution may be an issue.	⊕○○○ Very low
Ischaemic heart disease and myocardial infarction (hospital record)				

No of studies (design)	Reasons for rating quality down	Reasons for rating quality up	Key findings	Quality score
Three (cross-sectional and ecological)	Serious risk of bias Serious inconsistency	None	Small association found between road traffic noise and hospitalisations for myocardial infarction. Aircraft noise may have small impact on hospitalisations for cerebrovascular disease, ischaemic heart disease and heart failure.	⊕⊕○○ Low
Three (prospective cohort)	Some inconsistency	None	Road traffic noise not significantly associated with ischaemic heart disease or cerebro-vascular disease. May have a small impact on myocardial infarction.	⊕⊕⊕○ Moderate
Four (case control)	Serious inconsistency	None	Mixed results for road traffic noise. May have small impact on hospitalisations for myocardial infarction, particularly in males at very high equivalent sound levels (>70 dB(A)).	⊕⊕⊕○ Moderate
Stroke (self-report)				
One (cross-sectional)	Serious risk of bias Serious inconsistency Stroke not analysed separately from other cardiovascular heart disease outcomes. One small study	None	No significant findings.	⊕○○○ Very low
Stroke (hospital records)				
One (ecological)	Some risk of bias Only one study	None	Aircraft noise at high equivalent sound levels may have a small effect on hospitalisations for stroke.	⊕○○○ Very low
One (prospective cohort)	Only one study	None	Road traffic noise (L_{den}) at very high levels may have small effect on hospitalisations for older people (≥ 64 y).	⊕⊕⊕○ Moderate
Hypertension (measured)				
Twelve (cross-sectional)	None	None	Road traffic noise not significantly associated with hypertension.	⊕⊕○○ Low
Two (prospective cohort)	Some risk of bias	None	Aircraft noise may be associated with increased hypertension in older males.	⊕⊕○○ Low
Hypertension (self-report)				
Sixteen (cross-sectional)	Serious risk of bias Serious inconsistency	None	Higher exposure to road traffic noise associated with increased self-reported hypertension.	⊕⊕○○ Low

No of studies (design)	Reasons for rating quality down	Reasons for rating quality up	Key findings	Quality score
Four (prospective cohort)	Serious risk of bias Serious inconsistency	None	Higher exposure to road traffic noise associated with increased self-reported hypertension.	⊕⊕○○ Low
Type 2 diabetes insulin levels (hospital records)				
One (prospective cohort)	Only one study	None	Road traffic noise may slightly increase risk of hospitalisation. No effect from rail noise.	⊕⊕⊕○ Moderate
One (experimental)	Serious risk of bias Only one small study	None	Insulin levels may be sensitive to road traffic noise.	⊕○○○ Very low
Blood pressure and heart rate				
Fifteen (cross-sectional)	Moderate risk of bias Some inconsistency	None	Road and aircraft noise significantly associated with increased systolic blood pressure, particularly in children.	⊕⊕○○ Low
Four (prospective cohort)	Serious risk of bias Serious inconsistency	None	Mixed results. Blood pressure is sensitive to changes in noise levels.	⊕⊕○○ Low
Two (field experimental)	None	None	During sleep aircraft noise events (L_{max}) had an effect on blood pressure and dipping in diastolic blood pressure. No effect on heart rate. Maximum noise level, not noise type (such as road or air) was most important.	⊕⊕○○ Low
One (experimental studies)	Serious risk of bias One small study	None	Walking through a noisy or quiet park made little difference to blood pressure and heart rate.	⊕⊕○○ Low
Cardiac arrhythmia				
One (experimental)	Serious risk of bias One small study	None	No effect of air and road traffic noise on cardiac arrhythmia.	⊕○○○ Very low
Coronary artery atherosclerosis and calcification				
Two (cross-sectional)	Serious risk of bias Serious inconsistency	None	Higher quality study suggests a small effect of road traffic noise on atherosclerosis.	⊕⊕○○ Low

4.3 Summary of findings from the systematic review

4.3.1 What is the evidence of a causal effect of environmental noise on cardiovascular health?

A total of 65 studies examining the relationship between environmental noise and cardiovascular outcomes were included in this review. Most of these studies were observational (62), with only three experimental studies identified. The findings for the observational and experimental studies are summarised below.

4.3.2 Observational studies

Aircraft noise

A total of 19 observational studies examined the associations between aircraft noise and various cardiovascular outcomes. Most studies (15) reported a significant relationship between exposure to aircraft noise and adverse cardiovascular outcomes in the total sample (14 studies) or in sub-groups (1 study). These studies indicated that exposure to aircraft noise was significantly associated with hypertension, increased blood pressure, hospitalisations for cardiovascular diseases, use of medications for hypertension and other cardiovascular disease and cardiovascular mortality. Only three studies reported no significant associations between aircraft noise exposure and cardiovascular health.

Road traffic noise

Forty-three observational studies examined the relationships between exposure to road traffic noise and cardiovascular outcomes. The evidence in these studies was mixed. A total of 21 studies reported that increased road traffic noise was significantly associated with adverse cardiovascular outcomes. One found a significant result in the opposite direction, with increased noise associated with lower systolic blood pressure in children (van Kempen et al., 2006). A further nine studies found no significant effect in the total sample, but evidence of associations in sub-groups such as certain age or gender groups. Twelve studies reported no significant associations between road traffic noise and cardiovascular outcomes.

Rail noise

The associations between rail noise and various cardiovascular outcomes were examined in seven studies. One of these studies indicated that greater railway noise was associated with hypertension (Dratva et al. 2012). One study indicated that railway noise was associated with hypertension but not stroke or diabetes (HYENA; Sørensen et al., 2011a, 2011b, 2013). One found that rail noise was associated with self-reported hypotension in females under the age of 42 (Lercher and Widmann, 2013). Another four reported no significant association between railway noise and cardiovascular outcomes.

General environmental noise

Five studies examined general environmental and community noise exposure. Except for one study (Lepore et al., 2010), all indicated that greater noise exposure was associated with poorer cardiovascular health.

Study limitations

There are some important limitations of the observational studies. A key limitation is that most of the observational studies were cross-sectional (NHMRC level of evidence: IV) and are unable to provide an indication of the direction of causation. Although there were several prospective, case-control, and field studies, the results were mixed. This limits conclusions on the temporal effect of environmental noise on cardiovascular health.

While the type of noise exposure indicator used was relatively consistent across the studies (usually L_{Aeq} or L_{Amax}), there was considerable variation in how the noise exposure was estimated, such as using direct measurement or contour maps. There was also variation in the location at which the measurements were taken, such as at the building façade or participant's ear, complicating the synthesis of evidence.

There were also considerable differences between studies in the types of cardiovascular outcomes examined and the measures used to assess them. Cardiovascular outcomes assessed included: incidence of hypertension, stroke, heart disease or diabetes; treatment of hypertension; hospital records; mortality data; and aortic calcification. This variation makes it difficult to draw clear conclusions about the effect of environmental noise on cardiovascular health. These issues are compounded because the observational studies differed in whether cardiovascular outcomes were assessed using self-reporting or objective measures. A large number of studies examined self-reported hypertension, which is less accurate than an objective measure of hypertension based on blood pressure measurements. Many middle and older-aged adults may have undiagnosed hypertension, which would not be reflected in these self-reported measures. Therefore, self-reporting measures can limit the validity of findings and contribute to risk of bias.

There is also considerable potential for residual confounding, given that many studies did not control for relevant covariates such as air pollution. This is important as some studies found that an association between noise exposure and cardiovascular outcomes became non-significant when air pollution was added as a covariate (for example, Babisch et al., 2014a). Failure to control for these covariates could lead to false positive associations between noise exposure and cardiovascular health.

Twenty studies were rated as having a low risk of bias, 22 a moderate risk, and 21 a high risk. The primary reasons for moderate and high risk related to the use of self-reported measures of cardiovascular health and lack of control for relevant confounding variables.

Studies with a low risk of bias

The 20 studies with low risk of bias generally indicated that environmental noise exposure was linked with poorer cardiovascular health, although some findings were mixed. For example, several of the studies with a low risk of bias found non-significant results. Babisch et al. (1994) conducted a prospective case-control study of 4035 male adults and found that day-time exposure to road traffic noise ($L_{Aeq,6\text{-}22hours}$, exposure range 40 – 65 dB(A)) was not significantly associated with myocardial infarction incidence. In a prospective study of 18,213 adults, de Kluizenaar et al. (2013) found that road traffic noise (L_{den} at most exposed façade, per 10 dB increase) was not associated with cardiovascular disease hospitalisations. Foraster et al. (2011) found that road traffic noise (L_{night} and $L_{Aeq,24h}$ measured at the most exposed façade, per 10 or 5 dB increase) was not associated with hypertension in a cross-sectional study of 3480 adults. De Kluizenaar et al. (2007) found that road traffic noise (L_{den} at most exposed façade, per 10 dB increase) was not associated with use of antihypertensive medication. However, a significant effect was observed in adults aged 45 to 55 (odds ratio (OR) = 1.39 [1.08, 1.77]) at higher noise exposure (L_{den} > 55 dB). Clark et al. (2012) found that daytime road traffic and aircraft noise ($L_{Aeq,16\,h}$) were not associated with measured blood pressure in a sample of 351 children.

Other studies with a low risk of bias suggest a relationship between environmental noise and adverse cardiovascular outcomes. For example, Babisch et al. (2014a) conducted a cross-sectional study of 4166 adults and found that noise (L_{den} at exposed façade, per 10 dB increase) was not associated with hypertension but was associated with higher systolic blood pressure (OR per 10 dB(A) increase in noise = 1.43 [1.10, 1.86]). Selander et al. (2009) conducted a case control study of 3666 adults. Road traffic noise ($L_{Aeq,24h}$ ≥50 dB(A)) was not associated with

myocardial infarction risk in the total sample but a significant effect was observed in participants without hearing loss and a history of exposure to other noise sources (OR = 1.38 [1.11, 1.71]). Gan et al. (2012) conducted a prospective study of 466,727 adults and found that combined rail, air and road noise (postcode level L_{den}, range) was associated with cardiovascular disease mortality (OR per 10 dB(A) = 1.09 [1.01, 1.18]). In the diet, cancer and health cohort study, a prospective study of 57,053 adults, road traffic noise (L_{den} at most exposed façade, range) was associated with stroke (OR = 1.14 [1.03, 1.25]) and diabetes (OR = 1.11 [1.05, 1.18]).

4.3.3 Experimental studies

The findings of three experimental studies were included in this systematic review. Carter et al. (1994) examined the effects of exposure to aircraft and road traffic noise under laboratory conditions. The results indicated noise was not significantly associated with cardiac arrhythmia. Tomei et al. (2000) examined the effects of exposure to road traffic noise on levels of insulin under laboratory conditions. The results indicated that higher noise levels were significantly associated with increases in insulin levels. Finally, Janssen et al. (2012) conducted a field-based study examining the effects of exposure to road traffic noise on heart rate and blood pressure and did not find any significant results. The risk of bias for these studies was high, which primarily reflected issues relating to lack of control groups.

This review identified a number of experimental studies examining cardiac-related outcomes that were not relevant to this review because they focused on cardiac responses to noise during sleep. Rather than indicating an adverse effect on cardiac health, these cardiac responses most likely reflect an arousal response during sleep, perhaps indicative of awakening. These outcomes were therefore not considered relevant to cardiovascular health. Several studies also examined the effects of noise exposure on levels of hormones related to cardiovascular health, such as cortisol. Although these hormones are important, they are not considered cardiovascular disease outcomes, but rather part of the causal pathways linking noise and cardiovascular health.

4.3.4 Summary of the evidence

As noted above, most studies examining the associations between environmental noise exposure and cardiovascular outcomes have been observational. These results suggest that exposure to environmental noise is associated with poorer cardiovascular outcomes. The most consistent findings were observed for aircraft noise, while several studies indicated an association between road traffic noise and cardiovascular health. Use of self-reporting measures of cardiovascular disease, along with lack of control for important confounders, contribute to the low quality ratings for the identified studies. The magnitude of the reported effects across studies is small.

4.3.5 Is there a dose–response relationship between environmental noise and cardiovascular health?

A small number of studies formally examined whether there was a dose–response relationship between noise exposure and cardiovascular outcomes. These studies suggested such a relationship. Many studies also reported that stronger relationships with cardiovascular outcomes were observed as noise levels increased (Babisch et al., 2012, 2014a, 2014b; Bluhm et al., 2007; Chang et al., 2012; Dratva et al., 2012; Eriksson et al., 2010a; Gan et al., 2012; Hansell et al., 2013; Jarup et al., 2008; Kälsch et al., 2014; Liu et al., 2013). These differed considerably in terms of how noise exposure was quantified. For example, some examined effects per 1 dB, 5 dB, or 10 dB increases, while others examined varying categories of noise exposure.

Very limited data was available regarding threshold effects. Given the variability in research designs and low study quality, summary threshold effects could not be determined from the studies in this review. Individual studies offer findings that indicate levels at which adverse outcomes are observed. These do not indicate clear thresholds but may inform future research that examines potential thresholds. These findings are outlined below for each of the three main noise sources.

Aircraft noise

Some studies indicate that average day-evening-night noise levels are associated with adverse cardiovascular outcomes: ≥ 50 dB L_{den} (Franssen et al., 2004), > 55 dB(A) L_{den} (Correia et al., 2013; Rosenlund et al., 2001), ≥ 55 dB(A) L_{Aeq} (Eriksson et al., 2007), ≥ 60 dB(A) L_{den} (Huss et al., 2010), or > 70 dB(A) L_{den} (Matsui et al., 2001). In terms of specific periods, daytime levels above 63 dB(A) have been linked with adverse cardiovascular outcomes (Hansell et al., 2013). Focusing specifically on the period from 3am to 5am, Greiser et al. (2007) found that noise levels ≥ 40 dB(A) were linked with adverse cardiovascular health. In addition to averaged noise events, Rosenlund et al. (2001) found that maximum noise levels > 72 dB(A) were linked with poor cardiovascular health.

Road traffic noise

Several studies found a significant relationship above 55 or 60 dB(A) $L_{Aeq.}$ (Bendokiene et al., 2011; Bluhm et al., 2007; Bodin et al., 2009; Regecova and Kellerova, 1995); Yoshida et al. (1997) found a significant effect at noise levels ≥ 65 dB(A) L_{Aeq}. Another study found a significant relationship at noise levels ≥ 60 dB(A) L_{den} (Banerjee et al., 2014). Two others indicate higher thresholds, with effects observed at > 70 dB(A) $L_{Aeq,6\text{-}22hours}$ (Babisch et al., 2005) and ≥ 80 dB(A) L_{Aeq} (Chang et al., 2011).

Rail noise

There was insufficient evidence to draw any conclusions on the relationship between rail noise and cardiovascular health.

4.3.6 Is there any evidence that certain populations are vulnerable to the effects of environmental noise on cardiovascular health?

Aircraft noise

Two studies indicated that the association between aircraft noise exposure and hypertension was stronger in older individuals (Eriksson et al., 2007; Rosenlund et al., 2001). Eriksson et al. (2010a) found that the association of aircraft noise with hypertension was evident in males (but not females). Babisch et al. (2013) and Eriksson et al. (2010a) found the effects of aircraft noise on cardiovascular outcomes were pronounced in individuals who reported high levels of noise annoyance.

Some studies also reported that the effects of noise exposure were most pronounced in individuals who had lived in noise-exposed areas for a longer period. For instance, Huss (2010) found that the association between aircraft noise and myocardial infarction mortality was greatest in individuals who had lived in the area for 15 years or more. This is consistent with the HYENA study (also see Floud et al., 2013) where an association between aircraft noise and self-reported cardiovascular disease was evident only in those who had lived in the area for more than 20 years.

Road traffic noise

The relationships with cardiovascular outcomes were found to vary by several factors. Several studies reported stronger associations between traffic noise exposure and outcomes such as hypertension (Bluhm et al., 2007; de Kluizenaar et al., 2007), coronary heart disease (Banerjee

et al., 2014), myocardial infarction (Grazuleviciene et al., 2004) in middle-aged adults (aged 55–64 years). Sørensen et al. (2011a) found the association between road traffic noise and stroke was evident only in individuals aged over 65. Two studies indicated that the association of road traffic noise with cardiovascular outcomes was evident in individuals who had lived in an area for a longer period (Babisch et al., 2005; Barregard et al., 2009). Five studies reported significant differences by gender. The associations of road traffic noise with coronary heart disease (Banerjee et al., 2014) and hypertension (Bendokiene et al., 2011; Bjork, 2006; Lercher and Widmann, 2013) were stronger in females. In contrast, Belojevic (2008b) found that the relationship between road traffic noise and hypertension was stronger in males.

The effects of road traffic noise on cardiovascular outcomes were also stronger in individuals with higher noise sensitivity (Lercher and Widmann, 2013) and in those without hearing loss (Selander et al., 2009).

4.3.7 Does the association between environmental noise and cardiovascular health vary by noise source?

Most studies in this review examined the effects of one noise source (see de Kluizenaar et al., 2013). Although many other studies examined multiple noise sources, direct comparisons of effects were not made. Some studies investigating the effects of both road traffic and aircraft noise found significant associations for aircraft noise but not for road traffic noise. This may suggest that the effects of aircraft noise are stronger, but this is a very tentative conclusion. It is possible that aircraft, rail and road traffic noise have differential effects on cardiovascular health, but existing evidence is not conclusive.

4.3.8 Is there any evidence that annoyance is a mediator linking environmental noise exposure to cardiovascular health?

Many studies discussed annoyance as a potential pathway by which environmental noise exposure could influence cardiovascular health. However, only a few studies tried to examine whether annoyance was a mediator (see Fyhri and Klaeboe, 2009) and the evidence was inconclusive.

4.4 Conclusion

Variation in research design, study quality, adjustment for confounders, and outcome reporting make construction of dose–response relationships difficult for environmental noise and cardiovascular health.

The systematic review identified 65 studies published between 1994 and 2014 investigating the relationships between exposure to environmental noise and cardiovascular health. In general, the results were mixed, particularly for road traffic noise; the effects of rail noise on cardiovascular disease outcomes were not conclusive. Findings for the effects of aircraft noise were generally more consistent. However, it is important to note that for all noise sources, the magnitudes of the associations with cardiovascular disease were small. Small effect sizes are not surprising given that environmental noise could be one of a multitude of risk factors for cardiovascular disease. Other factors such as cigarette smoking and heredity probably play a much larger role in influencing an individual's level of risk.

It is important to note that there are some important limitations of the evidence base. These limitations include a large number of studies using self-reported measures, variation in study designs, quantification of noise exposure, site at which noise exposure was measured, and differences in the scope of confounding variables controlled. These issues mean it is not possible to identify a clear threshold where the effects on cardiovascular health emerge or worsen. As a

result, an NHMRC rating statement of C is applied to the overall body of evidence: the body of evidence has limitations and care should be taken in the interpretation of findings. See appendix A for details on ratings.

Further research is needed using designs that can demonstrate causality, using objective outcome measures. Controlling for a broad range of potential confounders is important to rule out the possibility of residual confounding. This is particularly the case for air pollution, which may be an important confounder but is not controlled in many studies. Based on existing research, vulnerable groups may include older adults. There is an absence of studies investigating annoyance as a mediator.

5 NOISE AND COGNITION

5.1 Introduction and background

Cognition is the process of learning that includes thinking, understanding and remembering. A large number of studies have examined the relationships between exposure to different sources of environmental noise – road traffic, aircraft and rail – and cognition. Associations have important implications since good cognitive performance is linked to higher quality of life, improved mental health and better academic and job performance. However, many aspects of the relationship between environmental noise and cognition remain unclear.

5.2 Systematic review of the literature

A systematic review of the literature was conducted for studies investigating the relationship between environmental noise and cognition for the period January 1994 to March 2014. This is further detailed in appendix A.

5.2.1 Search results

The flow chart below details the results of the search process.

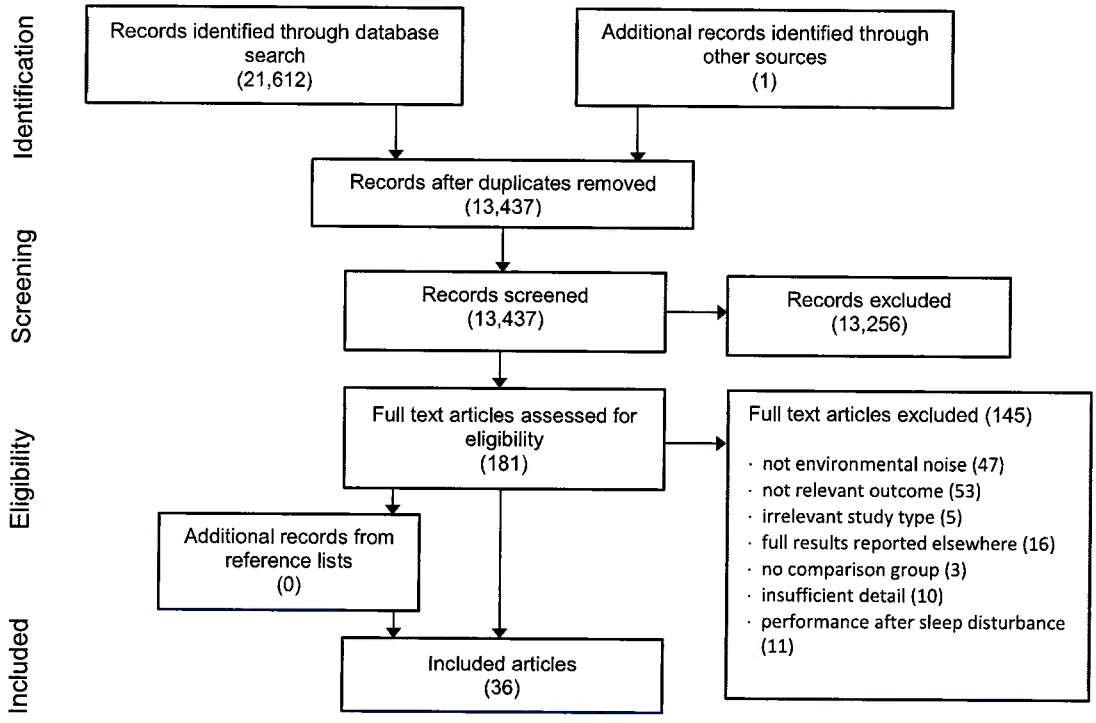


Figure 5-1: PRISMA flow chart. Number of articles identified and reviewed during the systematic review (Moher et al. 2009)

5.2.2 Overview of included studies

Study types and settings

Of the 36 articles identified, 29 were from distinct studies (some articles reported on the same data); a total of 14 observational and 15 experimental studies were included in the review. Most of the observational studies (11) were solely cross-sectional (NHMRC level IV), two included

both cross-sectional and prospective cohort (NHMRC level II) components, and one was a controlled before and after study (NHMRC level III-3).

All of the experimental studies were either non-randomised experimental studies (8) (NHMRC level III-2) or pseudo-randomised studies (7) (NHMRC level III-1). Most were based in sound and temperature controlled laboratories (12), while three were conducted in classrooms.

5.2.3 Noise exposure and how it was measured

Most of the observational studies explored aircraft noise (8 studies), followed by road traffic noise (4) and general community noise (3). Most experimental studies simulated road traffic noise (12 studies), with a small number simulating aircraft noise (3), and rail noise (1).

For observational studies, noise exposure was measured by direct measurement with sound level meters in various locations (5 studies), or estimated using models (8). One study did not clearly specify the measurement method. Experimental studies delivered noise levels in a controlled way to participants.

The most common noise measures used were A-weighted equivalent sound levels (L_{Aeq}) for various periods. Maximum sound pressure levels (L_{Amax}) were also commonly used.

All of the observational studies involved children from seven to 16 years old. Experimental studies involved university students and young adults (7 studies), primary and secondary school students (5), and only one involved adults aged from 35 to 65 years.

5.2.4 Types of outcomes reported

Most studies explored multiple outcomes. The most common outcomes explored in observational studies were reading comprehension (8 studies), memory (7) and attention (6). The most common outcomes explored in experimental studies were memory (8 studies), attention (5) and mathematical tasks (4).

Most studies used standardised or well-known tests to assess outcomes.

5.2.5 Quality ratings

GRADE is a structured process for rating quality of evidence in systematic reviews. Quality ratings according to the GRADE criteria are shown in Table 5-1. This indicates that on aggregate, the quality of the evidence was rated as low.

All included studies are listed in section 8.5. GRADE criteria are detailed in appendix A.

Table 5-1: GRADE evidence profile for environmental noise and cognition (29 studies)

No of studies (design)	Reasons for rating quality down	Reasons for rating quality up	Key findings	Quality score
Reading (skills and comprehension)				
Six (cross-sectional)	Some risk of bias	None	Aircraft noise at school has a detrimental effect on children's reading comprehension	⊕⊕○○ Low
Three (prospective cohort)	Some inconsistency	None	Detrimental effects of aircraft noise on children's reading may not persist over time, especially if noise exposure is changed	⊕⊕⊕○ Moderate

No of studies (design)	Reasons for rating quality down	Reasons for rating quality up	Key findings	Quality score
Two (experimental)	Some risk of bias Some indirectness / applicability (see GRADE guidelines)	None	Road traffic noise may affect reading speed in children but no effect was found on reading comprehension in children	⊕⊕⊕○ Moderate
Memory (short and long term)				
Six (cross-sectional)	Serious risk of bias Some inconsistency	None	Aircraft noise may affect long term memory in children. No effect of road or aircraft noise on short term memory	⊕⊕○○ Low
One (prospective cohort)	Only one small study	None	Chronic exposure may have detrimental effect on long term memory in children which is not immediately resolved by removing noise	⊕⊕○○ Low
Six (experimental)	Serious risk of bias Some indirectness	None	No effect of acute road or aircraft noise on short term memory	⊕⊕⊕○ Moderate
Attention				
Four (cross-sectional)	Some risk of bias Some inconsistency	None	Mixed results	⊕⊕○○ Low
One (prospective cohort)	Only one small study	None	No significant findings	⊕⊕○○ Low
Four (experimental)	Some risk of bias Some inconsistency Some indirectness	None	No effect of noise	⊕⊕⊕○ Moderate
Academic achievement (student, school and borough level measures)				
Five (cross-sectional)	Serious risk of bias Serious inconsistency	None	Noise at school may affect achievement (one high quality study)	⊕⊕○○ Low
Mathematics tasks (arithmetic, mathematical reasoning)				
Four (experimental)	Serious risk of bias Some indirectness	None	No effect of road traffic noise	⊕⊕⊕○ Moderate

5.3 Summary of findings from the systematic review

5.3.1 What is the evidence of the effect of environmental noise on cognition?

This systematic review identified 29 primary studies published between 1994 and 2014 examining the relationship between environmental noise exposure and cognition. Fourteen studies were observational and 15 were experimental. The main findings were mixed and are summarised below.

These studies generally measured the noise exposure, or exposed the participants, within the learning environment.

5.3.2 Observational studies

The observational studies examined the relationships between environmental noise exposure and a range of cognitive outcomes. Eight studies examined aircraft noise and four road traffic noise. A further three assessed general community noise, which included a combination of noise sources but did not allow for the sources to be distinguished from one another. Most studies were conducted on samples of children. Evidence of a relationship between environmental noise exposure and cognition was mixed across these studies.

Aircraft noise

Six of the eight studies indicated a significant relationship between aircraft noise exposure and cognitive outcomes. For example, they reported that exposure to aircraft noise was cross-sectionally associated with poorer reading comprehension (Evans et al., 1995, Evans et al., 1997; Seabi et al., 2010; Seabi et al., 2012, RANCH study and Haines et al., 2001a, b). The RANCH study and Haines (2001a, b) study found that the relationship did not maintain significance when explored through a prospective cohort study. Mixed results were found for memory and attention with four studies finding a significant relationship (Evans et al., 1995; Haines et al., 2001; Seabie et al., 2010 and the RANCH Study). The remaining two indicated aircraft noise exposure was not associated with reading comprehension, memory, attention and academic achievement (Haines et al., 2001c, 2002).

Road traffic noise

Two of the four studies provided some support for an association between road traffic noise and cognition. Belojević et al. (2012) found that higher road traffic noise was associated with poorer executive functioning in boys but not girls. The RANCH study indicated that road traffic noise was associated with impaired recognition memory, but not reading comprehension (Clark et al., 2006; Clark et al., 2012; Stansfeld et al., 2005; Stansfeld et al., 2010). Two studies conducted by Xie et al. (2010, 2011) indicated that road traffic noise was not associated with measures of academic achievement.

Generic environmental noise

Two of the three studies indicated that generic environmental noise (total noise levels measured outside schools or homes) was associated with poorer cognitive outcomes. Lercher et al. (2003) found that increased environmental noise was associated with impaired memory, while Pujol et al. (2014) found increased environmental noise was associated with poorer academic achievement. Another study conducted by Shield et al. (2008) found mixed support for a relationship between environmental noise (excluding aircraft noise) and cognitive outcomes. They found that higher levels of noise were associated with poorer academic achievement in some, but not all, schools.

Study limitations

The quality of the observational studies was generally low, reflecting a combination of factors including study design and a high risk of bias. For example, most of the studies were cross-sectional, with only two studies examining the prospective associations between environmental noise and cognition. This is a major limitation because cross-sectional studies are not able to provide insight into the direction of causation between noise and cognition. The RANCH study and Haines et al. (2001a, b) also reported cross-sectional associations between noise exposure and poorer cognition. However, these associations were not supported in the prospective analysis. This raises further concerns on the validity of the cross-sectional findings.

Most of the studies (eight out of 13) had a high risk of bias, mainly reflecting the lack of control for relevant confounding variables. This is an important consideration because significant results reported by these studies could reflect residual confounding rather than a true relationship between noise and cognition. Three of the nine studies had a moderate risk of bias, reflecting the inclusion of some confounders but omission of some key confounders such as socioeconomic status. The adjustment of confounders differed substantially between studies, particularly for measures of socioeconomic status.

Two studies, the RANCH and the Pujol et al. (2014) studies, had a low risk of bias. Several articles reported on the RANCH study, which demonstrated significant relationships between exposure to aircraft noise and poorer cognition across measures of reading comprehension, memory and attention. The RANCH study also indicated that road traffic noise was associated with some impairments in memory. Pujol et al. (2014) examined a sample of 586 children and found that general environmental noise was associated with impairments in standardised measures of academic achievement.

The observational studies examined several measures of cognition. For example, articles using data from the RANCH study used several standardised measures to assess reading comprehension and different components of memory, such as episodic and prospective memory. Studies also used generic indicators of overall executive functioning (Belojević et al., 2012) or standardised school performance scores (Haines et al., 2002; Shield et al., 2008; Pujol et al., 2014; Xie et al., 2010, 2011). Many other studies assessed domains of cognitive performance including reading, memory, attention, speech perception and intelligence (Haines et al., 2001a, c).

The variations in outcome measures may partly explain the inconsistent findings and limits the conclusions that can be drawn. Further, because most studies examine only a select range of cognitive outcomes, they do not provide a comprehensive insight into the effects of environmental noise on cognition.

The type of noise exposure indicator used is relatively consistent across the studies (usually L_{Aeq} or L_{Amax}). However, how the noise exposed was estimated – such as direct measurement or contour maps – and the site at which it was measured – building façade or participant's ear – varied considerably. This complicates a synthesis of the evidence.

5.3.3 Experimental evidence

Fifteen experimental studies examining the effects of environmental noise on cognitive outcomes were identified in this review. Twelve studies examined road traffic noise, three aircraft noise, and one rail noise, although some examined more than one noise source. The findings of these studies are summarised below.

Road traffic noise

Six of the 12 studies indicated that increased road traffic noise was associated with poorer cognitive performance. One study (Belojević et al., 2001) found that noise was not associated with cognitive performance in the total sample, although a significant effect was observed in introverts, but not extroverts. Three studies indicated that noise was not significantly associated with cognitive outcomes. Finally, two studies reported that increased noise led to improvements in cognitive performance. Alimohammadi et al. (2013) found that exposure to two hours of road traffic noise (71dB(A)) led to improved attention and concentration. However, these findings could feasibly be attributed to practice effects. White et al. (2012) reported that exposure to noise (road traffic and aircraft noise) led to faster reaction times, but this is not necessarily indicative of improved performance as accuracy was not affected by noise.

Aircraft noise

Two studies indicated that aircraft noise was not significantly associated with cognitive performance. As noted above, White et al. (2012) found that road traffic and aircraft noise were significantly associated with faster reaction times, but not differences in performance accuracy.

Rail noise

Klatte et al. (2007) found that rail noise did not lead to any differences in memory, listening comprehension, written language acquisition or visual recall.

Study limitations

The quality of the experimental evidence was moderate, with eight studies found to have a low risk of bias. But several other issues relating to the experimental evidence warranted consideration. One concerned the large variation of cognitive outcomes assessed between studies. The range of cognitive outcomes included attention, memory (short-term, long-term, prospective, cued recall), reading comprehension, speech perception, intelligence and academic performance. When similar outcomes were assessed, different approaches were used. For example, several studies examining the effects of environmental noise on reading comprehension used different measures such as the Suffolk Reading Scale (Haines et al., 2001a) and the Woodcock Reading Mastery Test (Evans et al., 1997). The variation in types of cognitive outcomes, and the measures used to assess them, limits comparisons between studies.

The nature of the experimental manipulation also differed considerably between studies. These related to the duration, mean levels and peak levels of noise exposure. There were also substantial variations in noise levels in the control or 'quiet' conditions used as a reference in these studies. These variations further limit comparisons that can be made between studies.

It is also important to note that these experimental studies assess the acute effects of noise on cognition and may lack external validity. That is, while the risk of bias was low in many studies, the results of these studies do not provide an indication of the effects of chronic noise exposure on longer term cognitive outcomes.

5.3.4 Is there a dose–response relationship between environmental noise and cognition?

None of the studies identified formally examined dose–response relationships between environmental noise and cognitive outcomes. However, some studies did report significant linear associations between noise exposure and cognition, suggesting that the effects on cognition are more pronounced at increased noise levels (Clark et al., 2006; Matheson et al., 2010).

The studies in this review did not provide a clear indication of dose–response relationships or threshold effects. An important consideration is that there may be distinct threshold effects for

different cognitive outcomes, such as memory versus attention. Further, many of these studies examined the acute effects of noise on cognition and provide only a limited insight into the effects of chronic noise exposure. Chronic exposure could have a different relationship with cognitive outcomes.

5.3.5 Is there any evidence that certain populations are vulnerable to the effects of noise on cognition?

Most of the studies were conducted in children, with only a few on adults. For the studies examining children, there was very limited evidence as to whether certain populations were more vulnerable to the effects of environmental noise on cognition. Belojević et al. (2012) found a significant detrimental effect of road traffic noise exposure at home on teacher-rated executive functioning in boys but not girls. However, few other studies in children examined sub-group effects.

Similarly, there was insufficient evidence as to whether any adult sub-populations were more vulnerable to the effects of environmental noise on cognition.

5.3.6 Does the association between environmental noise and cognition vary by noise source?

There was limited evidence as to whether the associations between environmental noise and cognition varied by noise sources. This is primarily because very few studies examined the effects of multiple sources of noise. Because studies used different methods, it was not possible to directly compare results.

Clark et al. (2006) is an example of one study that compared the effects of different noise sources. They found that aircraft noise, but not road traffic noise, was significantly associated with impaired reading comprehension. Clark et al. (2006) suggested that this may occur because aircraft noise is more intense and less predictable than road traffic noise. The transient nature of aircraft flyovers, which have short-term high noise levels, may disrupt children's concentration and distract them from learning tasks. The constant nature of road traffic noise may allow children to habituate and not be distracted.

5.3.7 Is there any evidence that annoyance is a mediator linking environmental noise exposure to cognition?

Clark et al. (2006) examined whether noise annoyance was a mediator linking noise with cognition. Their results indicated that annoyance was not a significant mediator. None of the other studies in this review formally examined the role of annoyance as a mediator of these relationships. However, many studies discussed annoyance as a potential mediator.

5.4 Conclusion

The systematic review identified 29 primary studies (14 observational and 15 experimental) from 35 papers published between January 1994 and March 2014 examining the associations between environmental noise and cognitive outcomes. There is some evidence that increased levels of environmental noise are associated with poorer cognitive performance as reflected by a range of measures assessing reading comprehension, memory and attention. However, many of the findings between studies were mixed, and the nature of the relationship between environmental noise and cognition requires further investigation.

In general, the quality of the observational evidence included in this review was low, and experimental studies were considered to have a lower risk of bias. Regardless of risk of bias, the results were generally inconclusive. From the systematic review, it is therefore not possible to

draw any meaningful conclusions on threshold effects, sub-group differences, or differential effects between noise sources. There is also insufficient evidence to draw any conclusions on the role of annoyance as a mediator. As a result, an NHMRC rating statement of D is applied to the overall body of evidence: the body of evidence is weak and findings cannot be trusted.

It is plausible that a relationship exists between environmental noise and cognitive performance. For example, environmental noise could be a source of distraction and thus interfere with task performance. Environmental noise may also induce hyper-arousal and lead to deficits in performance. It is also plausible that environmental noise has an indirect effect on cognition through disturbed sleep. Although these mechanisms are often discussed, evidence of a strong association is still lacking.

6 Discussion

With future urban population growth, a significant and increasing number of people in Australia are likely to be adversely affected by exposure to environmental noise. The number exposed to potentially harmful levels of environmental noise is yet to be comprehensively quantified.

Chapters 3, 4 and 5 systematically identify and appraise the evidence on the effect of exposure to environmental noise on sleep, cardiovascular and cognitive outcomes. The systematic reviews also considered the evidence for dose–response relationships, vulnerable groups and possible thresholds for risk.

The expert advisory group considered an analysis of the highest quality studies – studies with a risk of bias rating of one or two and an NHMRC higher quality study design – was important for further interpretative guidance.

This guidance can assist regulatory authorities, public health professionals and others by:

- providing insight into the likely causal probability
- identifying if there are broad threshold boundaries for health effects
- indicating the magnitude or importance of the effects described.

The following sections provide an additional synthesis of the available evidence from higher quality studies for sleep, cardiovascular and cognitive outcomes, along with limitations in the current literature.

6.1 Discussion on higher level studies with sleep related outcomes

Outcomes and their importance

Sleep disturbance can be quantified objectively by the number and duration of nocturnal awakenings, the number of sleep stage changes and modifications in their amounts. Subjectively, disturbance can also be measured through social surveys where individuals are asked to self-evaluate their sleep quality. Physiologically, sleep can be monitored using a sleep polygraph that measures total sleep time, sleep efficiency, total time in various sleep stages as well as arousals and awakenings. Motility (body movements) can be detected using accelerometers or actimetry and are also a useful indicator of sleep disturbance. A problem for interpretation in the systematic review was the proliferation of outcome measures. In general electroencephalogram awakenings are an acceptable proxy measure of sleep disturbance. However, small increases in awakenings have uncertain effects on sleep quality and uncertain long-term health consequences.

The systematic review examined a total of 79 studies, 43 of which were observational and 36 were experimental. The evidence base, while extensive, was not rated highly in terms of overall quality. An NHMRC rating statement of C was given. The low quality rating reflected issues around study design (most were cross-sectional) and a high risk of bias within studies (primarily due to measurement of sleep and control of confounders). These issues are detailed in chapter 3.

Higher quality studies

Higher quality studies included field studies with ratings of NHMRC III-2 and risk of bias one or two or NHMRC II and risk of bias one or two (Basner et al., 2006; Horne et al., 1994; Örhström et al., 2006; and Passchier-Vermeer, 2002). They also included experimental studies (all III-1 or III-2) with a risk of bias score one, (Basner and Samel, 2005; Basner et al., 2011; Griefahan et al., 2006a; Saremi et al., 2008). See appendix A, Table A-2 for the risk rating system. These are discussed below.

The field studies by Basner et al. (2006) and Passchier-Vermeer (2002) measured the noise a participant was exposed to indoors in their home and found a significant association between noise and an impact on a sleep parameter. Outcomes included reduced rapid eye movement (REM) sleep duration, increased sleep awakenings and increased motility as measured by actimetry.

Basner et al. (2006) examined awakenings and sleep stage transitions in response to aircraft noise events in a field study of 64 subjects. Sleep outcomes were measured using polysomnography, and sound pressure levels (SPL)($L_{As,max}$) were recorded inside the bedroom at the participant's ear as well as outside at the façade. Awakening probability increased with maximum SPL of an aircraft noise event. A threshold value of 33 dB(A) was found in the study, although it was noted that the effect was small, with only 0.2 per cent probability of awakening at an aircraft noise event maximum SPL of 34 dB(A) ear. The study showed a dose–response relationship with probability of awakenings increasing as maximum SPL increased. A 10 per cent rise in awakening probability corresponded to 73.2 dB(A) ear.

The study by Passchier-Vermeer (2002) measured aircraft noise both indoors and outdoors at the participant's residence and found indoor noise measurements – but not outdoor – were significantly associated with increased motility. Studies that more precisely measured the participants' noise exposure more clearly supported the influence of environmental noise on sleep.

Horne et al. (1994) and Örhström et al. (2006) did not use indoor noise monitoring, but for neighbourhood noise levels or modelled levels for the façade of the house they found less clear relationships. Horne et al. (1994) found that most aircraft noise events were not associated with an awakening, as measured by actimetry, and that other factors such as the presence of young children and concurrent illness, were more important. The study by Örhström et al. (2006) found mixed results, with some sleep parameters improved in high noise areas, although they were unable to adequately control for a government noise insulation program available in the highest noise area.

The higher quality experimental studies found similar outcomes (Basner and Samel, 2005; Basner, 2011; Griefahan et al., 2006a; Saremi et al., 2008). All experimental studies used polysomnography and, owing to their experimental design, tended towards better characterised or controlled noise exposure. The results were similarly small in magnitude of effect but all found statistically significant effects of noise on sleep. This included effects on sleep awakenings, sleep onset latency, sleep structure and micro-arousals.

The magnitude of these effects was low and the impact on sleep uncertain. There was insufficient evidence to determine a dose–response curve. There was also insufficient evidence across all studies to identify a specific threshold. However, there was consistency across higher quality studies when the threshold started at 55 dB L_{Amax} façade.

Other guidance recommendations

In recent years, WHO Europe has published two reports based on extensive reviews of the literature: the WHO Night Noise Guidelines for Europe (2009) and the Burden of Disease from Environmental Noise (2011). The night noise guidelines report identified threshold levels for a series of effects (biological, sleep quality, well-being and medical conditions), for which sufficient evidence was available. It identified children, elderly people, pregnant women and shift workers as at-risk groups. This report concluded with a proposed lowest observable adverse effect level (LOAEL) night noise guideline level of 40 dB $L_{night,outside}$ (WHO, 2009). This is not consistent with the threshold levels identified in the higher level studies described above.

The burden of disease report relied on several assumptions to arrive at estimates for exposure-response relationships. These were used to estimate the disease burden from environmental noise, measured in Disability Adjusted Life Years (DALYs). Such estimates of dose–response relationships and thresholds need to be interpreted with caution.

6.2 Discussion on higher level studies with cardiovascular outcomes

Outcomes and their importance

Cardiovascular outcomes reported in the studies in the systematic review are indisputably important health effects. Outcomes reported are hypertension (56 studies), cardiovascular disease usually comprising myocardial infarction or ischaemic heart disease (14 studies), heart failure and stroke. A variety of studies, equivalent to chamber studies in air pollution research, demonstrated acute effects of noise exposure on heart rate, blood pressure, insulin and catecholamine release.

A total of 65 studies was included in the systematic review. The overall body of evidence was given an NHMRC rating statement of C, where the body of evidence has limitations and care should be taken in interpreting findings. Higher quality non-experimental studies (Babisch et al., 1999; Beelen et al., 2009; Chang, 2009; de Kluizer et al., 2013; Eriksson, 2007 and 2010; Gan et al., 2012; Sørensen et al,. 2012a) included cardiovascular outcomes with a risk of bias rating of one or two and a prospective cohort design (NHMRC Level II evidence for aetiological questions).

Higher quality studies

Three higher quality studies addressed the outcome of hypertension (Eriksson, 2007 and 2010; Chang, 2009). Those by Eriksson used the Stockholm Diabetes Prevention Program Cohort to investigate the effects of modelled aircraft noise on self-reported diagnosis of hypertension. The earlier study found a significant association between increasing noise and escalated rates of self-reported hypertension. The second study by Eriksson (2010), which controlled for more confounders and had a longer follow-up period, found persistent effects only for men. Chang et al. (2009) investigated the effect of environmental noise (measured on a personal device that logged noise levels every five minutes) on blood pressure (measured every 30 to 60 minutes throughout the study period). This study found an association between increasing noise and short-term rises in blood pressure in young adults.

Five higher quality studies, all prospective cohort studies, examined cardiovascular outcomes more generally (including coronary heart disease and cerebrovascular events) as well as coronary heart disease specifically (Babisch et al., 1999; Beelen et al., 2009; de Kluizer et al., 2013; Gan et al., 2012; Sørensen, 2012). Effects seen were small and significant in only the three studies that examined cohorts of more than 50,000 people (Beelen et al., 2009; Gan, 2012; Sørensen, 2012). Sørensen et al. (2012) found a linear dose–response for traffic noise and myocardial infarction throughout the exposure range of the study (42-84 dB). As all these studies assessed exposure to road noise, consideration of air pollution as a potential confounder is important. Only two studies considered both cardiovascular risk factors and air pollution in their analysis, with the smaller cohort (Sørensen, 2012) finding a significant effect of noise, and the larger cohort (Beelen et al., 2009) finding a non-significant trend. A trend towards increased cardiovascular outcomes with noise was observed in all higher quality studies, be it statistically significant or not.

Most of the higher quality studies found an effect of noise on cardiovascular outcomes including hypertension, coronary heart disease and cerebrovascular disease. In general, effect sizes were low. Studies with fewer subjects often found non-significant trends towards an effect, while studies with more subjects found small but more often significant effects. Although the magnitude

of effect was low and the impact of these effects uncertain, it is still possible to reach limited conclusions around adverse effects on cardiovascular health.

Higher level studies suggest a general threshold for cardiovascular disease outcomes, which may be observed as low as 52 dB(A) measured at the façade (or 42 dB(A) at the ear using an assumption of 10 dB loss across the façade) but which are definitely observed as having adverse health effects starting in the range 55–60 dB(A) façade. These outcomes are for chronic exposure to road traffic noise estimated using a standard composite noise metric (usually L_{den}).

Other guidance recommendations

WHO Europe's Burden of Disease from Environmental Noise report (2011) looked at the risk of cardiovascular disease (specifically ischemic heart disease and hypertension) from increased noise levels. It notes that no myocardial risk is detected at noise levels under 60 dB(A). This report relied on several assumptions to arrive at estimates for exposure-response relationships, which in turn were used to estimate the disease burden from environmental noise, measured in Disability Adjusted Life Years (DALYs). Such estimates of thresholds need to be interpreted with caution.

6.3 Discussion on higher level studies with cognitive outcomes

Outcomes and their importance

Cognitive outcomes are not commonly considered a health outcome unless they are persistent and affect the quality of social interaction, life opportunities or activities of daily living. Many of the cognitive outcomes considered by studies covered by the systematic review could be more properly considered educational or learning outcomes. Generally, experimental studies are able to report only short term cognitive deficits arising from noise interference with cognitive tasks. They provide insight into kinds of cognitive functions that noise can interfere with and possible thresholds for this interference. However, they cannot provide direct evidence for the level at which noise may cause persistent cognitive deficit.

The systematic review identified 14 observational and 15 experimental studies. The body of evidence was given an overall NHMRC rating statement of D, where the body of evidence is weak and findings cannot be trusted.

Higher quality studies included observational studies of NHMRC study type II (prospective cohort) and risk of bias rating one or two, or NHMRC study type IV (cross-sectional) and risk of bias rating one, and experimental studies with NHMRC study type (all III-1 or III-2) (Clark, 2006, 2013; Enmarker, 2004; Hygge, 2002; Hygge, 2003a; Klatte, 2007; Ljung, 2009; Pujol, 2014; Sandrock, 2010; Sörqvist, 2010; Stansfield, 2005; Sukowski, 2007; Trimmel, 2012). These are discussed below.

Higher quality studies

A number of these studies (mostly experimental in design) examined the relationship between noise and various aspects of memory. All studies that considered the effect of road or aircraft noise on an aspect of memory found a significant relationship with at least one aspect of memory (Enmarker, 2004; Hygge, 2003a; Sörqvist, 2010; Stansfield, 2005; Hygge, 2002). Klatte (2007), the only study that assessed rail noise, found a non-significant effect of rail noise on short term memory. Enmarker (2004) and Hygge (2002) considered attention in their studies but found noise did not have a significant effect.

The four experimental studies examined a range of noise exposures and outcomes. Three of these found an effect of noise on academic performance (Ljung, 2009; Sukowski, 2007; Trimmel, 2012). The study finding no effect of noise on academic performance (Sandrock, 2010) exposed

participants to higher levels of noise in the control group compared to other studies, which might have been a factor in the non-significant result.

Observational studies that examined the effect of noise on academic performance all considered the influence of aircraft noise alone. The RANCH studies recruited students aged nine to 10 from 98 schools around airports in the Netherlands, Spain and the United Kingdom (Clark, 2006, 2012; Stansfeld, 2005). These considered outcomes related to academic performance such as school-based tests or other academic abilities, including mathematical reasoning, grammatical reasoning and reading comprehension. The RANCH studies found a significant effect on reading comprehension but not attention (Clark, 2006, 2012; Stansfeld, 2005). A study by Pujol (2014) found a significant effect of school noise on language and mathematical performance. A follow-up study by Clark (2013) of primary school children in the London arm of the RANCH study showed only non-significant decreases in reading comprehension persisting after six years.

In general, observational studies reported a large number of cognitive outcomes, did not report consistent direction of effect of cognitive outcomes, and did not report consistent effects across studies. Studies adjusted for a large range of potential confounders. However, we cannot discount a possible residual effect from socioeconomic status or other related confounders.

The high level studies suggest that noise may acutely interfere with some aspects of cognitive performance. Impairment may vary according to type of noise source, type of task and level of difficulty. There was insufficient evidence of what the long-term effects from environmental noise may be, or whether short-term effects persist over the longer term. These mixed findings may be attributable to the quality of the study designs or absence of high quality longitudinal studies but also reflect the inherently complex nature of cognitive processing.

Other guidance recommendations

In its report on the burden of disease from noise assessment, WHO (2011) proposed a hypothetical exposure–response relationship, where it is assumed that no children are affected at levels under 50 dB(A) L_{dn}, and that 100 per cent were affected at levels over 95 dB(A) L_{dn}. However, this report relied on several assumptions to estimate exposure-response relationships that were then used to estimate the disease burden from environmental noise, measured in Disability Adjusted Life Years (DALYs). Such estimates of dose–response relationships and thresholds need to be interpreted with caution.

6.4 Limitations

Limitations imposed by the quality of the body of evidence available for the systematic reviews have been discussed in chapters 3 to 5. Many studies did not consider the duration of exposure to noise, particularly for cardiovascular disease, which could have an impact on findings. Most studies were observational studies with a high risk of bias due to potential confounding, and there are issues with external validity of experimental studies (applicability of experimental findings to real world situations). These and the heterogeneity of measurement of both noise and outcomes restricted any attempt at meta-analysis of results in the systematic reviews.

Causality is difficult to demonstrate without randomised controlled trials or prospective cohort studies, and these studies are difficult or impossible to conduct in the area of environmental noise. Sections 10 and 11 in appendix A detail the overall quality assessment process using the GRADE guidelines (Guyatt et al., 2011), informed by relevant recommendations from the NHMRC (1999).

GRADE is an accepted method of providing a structured process for rating the quality of evidence in systematic reviews. However, it was developed primarily in the context of clinical trials, and there are ongoing debates about its application for public health. This includes

environmental noise health effects, where randomised control trials are often not possible. The limitations in applying GRADE guidelines to public health evidence have been noted previously, including in a study by two members of the GRADE working group (Rehfuess and Akl, 2013). One issue identified was the low quality evidence grading for all observational studies – non-epidemiological evidence, such as experimental studies, is regarded as very low quality. Other issues included uncertainty about how to apply the GRADE criteria to narrative summaries, and potential for policymakers to misinterpret the GRADE terminology to describe the quality of evidence. The authors suggested the GRADE working group consider modifications to the criteria to better suit reviews of public health interventions.

The GRADE criteria used to rate evidence in the systematic reviews cited here have been modified to account for issues with experimental studies (see appendix A, section 7). While the formal GRADE requirement rates all observational studies as 'low quality', the studies we reviewed may have adopted close to the best feasible design for many of the measured noise outcomes.

7 SUMMARY AND RECOMMENDATIONS

This chapter summarises the findings, identifies the gaps in the literature and considers future priorities to protect and promote human health in relation to environmental noise.

7.1 Summary statement on environmental noise and sleep disturbance

There is consistency across higher quality studies to suggest a causal relationship between environmental noise and sleep disturbance above 55 dB(A) ($L_{night,outside}$) at the building façade.

Table 7-1 summarises the findings from the systematic review and areas of concern.

Table 7-1: Summary of current evidence on the effect of noise on sleep disturbance and dose–response, sources, thresholds and individual vulnerability

Concern	Summary of effects on sleep disturbance
Dose–response	It is likely there is a dose–response relationship between noise and physiological effects on sleep which some studies show begins above 32 dB(A) L_{Amax} measured at the ear (about equivalent to 42 dB(A) L_{Amax} at the façade). While physiological effects have been observed at these levels, this does not suggest this is the threshold for adverse health effects.
Variations by source	The systematic review concludes it is plausible that aircraft, rail and road traffic noise have differential effects on sleep quality. However, because available data are limited, it is not possible to draw definitive conclusions on the nature and magnitude of these differences.
Threshold	There is consistency across higher quality studies to suggest sleep disturbance above 55 dB(A) ($L_{night,outside}$) at the façade. Some studies show physiological effects below 55 dB(A) ($L_{night,outside}$) but because of the studies' limitations, the evidence was not sufficient to say when these outcomes constitute an adverse health effect.
Vulnerable populations	Evidence from the systematic review raises the possibility that some effects may be greater in certain populations, but it is not strong or complete enough to draw strong conclusions on vulnerable groups. WHO's night noise guidelines for Europe report identifies children, elderly people, pregnant women and shift workers as potential at-risk groups.
Gaps and research needs	Observational research should ideally be longitudinal in design. Use of standardised sleep measures and accurate noise exposure measures (not proxies), and appropriate control of covariates with potential to confound the findings, would help to compare and pool studies. Studies are needed that allow for further comparison of the effects of different noise sources, as well as formal examination of mechanisms that may link environmental noise and sleep (annoyance).

7.2 Summary statement on environmental noise and cardiovascular disease

The larger prospective cohort studies that more comprehensively controlled for confounders suggested a causal relationship between chronic exposure to environmental noise and cardiovascular outcomes above 60 dB $L_{Aeq,day,16h}$ at the façade. Note that the $L_{Aeq,day,16h}$ metric measures sound from 7 am to 11 pm and is an outdoor value.

Table 7-2 summarises the findings from the systematic review and areas of concern.

Table 7-2: Summary of current evidence on the effects of noise on cardiovascular disease and dose–response, sources, thresholds and individual vulnerability

Concern	Summary regarding effects on cardiovascular health
Dose–response	Variation in research design, study quality, adjustment for confounders, and outcome reporting make construction of dose–response relationships difficult. A small number of studies formally examined whether there was a dose–response relationship between noise exposure and cardiovascular outcomes. These studies were suggestive but not conclusive of a dose–response relationship. Many studies reported that stronger relationships with cardiovascular outcomes were observed as noise levels increased.
Variations by source	The systematic review concludes it is plausible that aircraft, rail and road traffic noise have differential effects on cardiovascular health, but existing evidence is not conclusive.
Threshold	The larger studies that more comprehensively controlled for confounders suggested adverse effect on the cardiovascular system occur above 60 dB $L_{Aeq,day,16h}$ at the façade. Note that the $L_{Aeq,day,16h}$ metric measures sound from 7 am to 11 pm and is an outdoor value. Given the variability in research designs and study quality, summary threshold effects could not be determined from the studies. Some studies offer findings that indicate levels at which adverse outcomes are observed, although these do not indicate clear thresholds.
Vulnerable populations	Evidence from the systematic review suggests the association between aircraft noise exposure and hypertension was stronger in older individuals, in those with high levels of annoyance and in individuals who had lived in noise exposed areas for a longer period. Road traffic noise was found in some studies to be associated with hypertension, coronary heart disease and myocardial infarction in middle aged adults and also in individuals who had lived in noise exposed areas for a longer time. There were significant but inconsistent gender differences in some studies.
Gaps and research needs	There is a need to better identify vulnerable groups and subgroups, and those who have lived in a high noise exposure area for a longer period (>10 years). Future studies should investigate whether factors such as annoyance mediate the association between noise exposure and cardiovascular health. Any further research should use study designs that show causality and use objective outcome measures to reduce bias. Many of the studies that considered cardiovascular outcomes did not comprehensively control for confounding, particularly air pollution.

7.3 Summary statement on environmental noise and cognition

There is some evidence that increased levels of environmental noise are associated with poorer cognitive performance. This is reflected in a range of measures assessing reading comprehension, memory and attention.

Many of the findings between studies were mixed, and the nature of the relationship between environmental noise and cognition requires further investigation.

There is insufficient evidence of a causal effect of environmental noise on persistent cognitive or learning deficits.

Table 7-3 below summarises the findings from the review and areas of concern.

Table 7-3: Summary of evidence on the effects of noise on cognition and dose–response, sources, thresholds and individual vulnerability

Concern	Summary regarding effects on cognition
Dose–response	The systematic review did not identify studies that formally examined dose–response relationships between environmental noise and cognitive outcomes. Some studies did report significant linear associations between noise exposure and cognition, suggesting that the effects on cognition are more pronounced at increased noise levels.
Variations by source	The systematic review noted there is limited evidence as to whether the associations between environmental noise and cognition varied by noise sources. This is primarily because very few studies examined the effects of multiple sources of noise. Because studies used different methods, it was not possible to directly compare results between studies. However, it is possible that aircraft noise is more disruptive to children's concentration.
Threshold	The systematic review did not provide a clear indication of a threshold but it suggested there may be distinct threshold effects for different cognitive outcomes.
Vulnerable populations	Evidence from the systematic review is not sufficient to identify vulnerable groups. Most studies were conducted on children, and it seems reasonable to suggest that children are a vulnerable population with regards to noise and cognition. Subgroup effects among different children groups, such as gender, are inconclusive.
Gaps and research needs	More research is needed to clarify the nature of the relationship between environmental noise and cognition, taking account of specific cognitive outcomes and chronic noise exposure. These should include well-designed prospective studies and experimental studies that involve randomisation and that compare the effects of different noise sources. Observational studies would also be useful to identify vulnerable populations, which could then be further examined in experimental studies. It would be valuable for studies to examine the role of annoyance as a mediator linking environmental noise to cognition.

7.4 Overall summary statement for the effect of environmental noise on health

There is sufficient evidence of a causal relationship between environmental noise and both sleep disturbance and cardiovascular disease, to warrant health based limits for residential uses.

During the night-time, an evidence based limit of 55 dB(A) at the facade using the $L_{eq,night,}$ or similar metric and an eight-hour night-time period is suggested.

During the day-time, an evidence based limit of 60 dB(A) at the facade measured using the $L_{eq,day,}$ or similar metric and a 16-hour day-time period is suggested.

7.5 Recommendations

It is likely that community and public health concern over environmental noise will grow. This is particularly due to increasing urban density along busy transport corridors, growth in urban transportation, significant shifts in inner city land use, growing residential use of rezoned industrial areas, and greater information and evidence.

This report confirms and expands on the findings of the enHealth report on the health effects of environmental noise published in 2004. The current evidence indicates that environmental noise is an ongoing public health problem, and one that deserves more attention than it receives.

Four main recommendations are presented as measures to address the health impacts of environmental noise. They are:

1. recognise that environmental noise is a health risk
2. promote measures to reduce environmental noise and health impacts
3. address environmental noise in planning and development activities
4. foster research to assist policymaking and action.

These recommendations are not considered exhaustive and may be subject to change in light of further evidence.

7.5.1 Recommendation 1: Recognise that environmental noise is a health risk

Policy

Recommended actions	Responsibility	Priority
Consider this review when developing national environmental noise goals	State and territory health agencies	High
State and territory and Australian Government agencies to include noise as an important environmental health issue for strategic and local planning	State and territory health agencies	High
Review adequacy of existing health guidelines in state and territory legislation	enHealth	High

Interventions

Recommended actions	Responsibility	Priority
Promote awareness of the impacts of environmental noise on health	Relevant agencies, stakeholders and non-government organisations	Medium

Information

Recommended actions	Responsibility	Priority
Inform communities and stakeholders of national and international standards and guidelines	State and territory health agencies, other relevant agencies, stakeholders and non-government organisations	Medium

7.5.2 Recommendation 2: Promote measures to reduce environmental noise and associated health impacts

Policy

Recommended actions	Responsibility	Priority
Review consistency of existing legislation across all levels of government	enHealth, state health, environment and planning authorities including the Australian Building and Construction Commission	High

Interventions

Recommended actions	Responsibility	Priority
Review noise arising from transportation, including noise criteria for areas adjacent to transport infrastructure	State health, environment and planning authorities including the Australian Building and Construction Commission	Medium
Promote noise mitigation measures (for example, acoustic barriers or noise insulation in residential buildings) and the use of licensing controls to limit noise impacts	State health, environment, transport and planning authorities including the Australian Building and Construction Commission	Medium

Information

Recommended actions	Responsibility	Priority
Develop a national environmental noise reduction education program, which could be supplemented with additional state-specific campaigns	enHealth, state and territory health agencies	Medium

7.5.3 Recommendation 3: Address environmental noise in planning and development activities

Policy

Recommended actions	Responsibility	Priority
Include environmental noise in the health impact assessment of proposed developments, where warranted	State health, environment and planning authorities including the Australian Building and Construction Commission	High
Determine baseline environmental noise levels to inform planning actions (noise mapping)	State health, environment, transport and planning authorities	High
Review noise control practices and how to further integrate noise control into planning processes, for all levels of government (with attention to future noise research findings)	State health, environment and planning authorities	Medium
Foster national consistency for: • guidelines on how to minimise or prevent environmental noise arising from developments (that is, appropriate attention to layout, design and construction) • limiting noise arising from major sources • methods to set noise limits	State health, environment and planning authorities including the Australian Building and Construction Commission	Medium

Interventions

Recommended actions	Responsibility	Priority
Carry out baseline monitoring of environmental noise levels over time to ascertain existing ambient levels across a broad range of populations and land use areas. This could be used to inform land use planning or burden of disease studies	Environment, transport and health agencies	High
Apply appropriate controls where noise is known to have an effect	Regulatory authorities	High
Develop national and state action plans for both the long and short term to integrate planning and research at all levels of government	enHealth, State health, transport, environment and planning authorities	Medium
Develop guidelines for noise sensitive developments for layout, design and construction	Planning, environment and health agencies	Medium

Information

Recommended actions	Responsibility	Priority
Develop state information strategies to keep communities informed of advances in measures to improve noise	State health, environment and planning authorities including the Australian Building and Construction Commission	Medium

7.5.4 Recommendation 4: Foster research to support policymaking and action

Policy

Recommended actions	Responsibility	Priority
Identify factors giving rise to sensitivity to noise and vulnerability to non-auditory health effects to inform environmental, planning and health policies	State and territory health agencies, enHealth, key researchers	High

Interventions

Recommended actions	Responsibility	Priority
Conduct a rigorous evaluation of national, state and city population exposures to each major noise source	State and territory environment agencies, health agencies, such as National Health and Medical Research Council enHealth, key researchers	High
Support noise mapping projects to determine community noise exposures to each major noise source, which could be used to inform land use planning or burden of disease studies	Health, environment and transport stakeholders	High
Conduct evaluations of noise reduction schemes on community health	State health, environment and planning authorities including the Australian Building and Construction Commission, enHealth, key researchers	Medium
Promote further research on the effects of noise on learning performance in children, sleep disturbance, annoyance and cardiovascular health and mental wellbeing to establish threshold levels	State health, environment and planning authorities including the Australian Building and Construction Commission, enHealth, key researchers	Medium

Information

Recommended actions	Responsibility	Priority
Translate research findings into useful information for community and relevant stakeholders	State health, environment and planning authorities including the Australian Building and Construction Commission, enHealth, key researchers	Medium

Concluding remarks

Although the body of evidence is largely still emerging, there is sufficient evidence to suggest that noise affects health. It is important to consider actions to reduce environmental noise exposure where feasible. This would likely have a positive impact through health benefits.

A number of areas require further investigation and particularly for the Australian context. Environmental noise in rural areas has not been well researched because the low population density makes it difficult to conduct studies with sufficient statistical power to confirm or refute any hypothesis.

Lack of noise mapping and determination of population exposure by noise levels constrains estimates of the burden of disease from noise exposure. Environmental noise therefore needs to be prioritised on the research agenda.

Research that would have a direct impact on policy would be intervention studies examining the effects of change in noise exposure on changes in population health. Health agencies have a critical role to play in developing an appropriate research framework with academic institutions, transport, environment and planning agencies.

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8.5 Chapter 5

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8.6 Chapter 6

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APPENDIX A: REVIEW OBJECTIVES AND METHODOLOGY

1. Evidence reviews

NSW Health convened an expert advisory group to assist in developing this document. It also commissioned the Centre for Health Initiatives at the University of Wollongong to do systematic reviews of the evidence for three health outcomes: sleep disturbance, cardiovascular diseases and cognition.

2. Review objectives

The review identified and appraised international evidence on the influence of exposure to environmental noise on sleep, cardiovascular and cognitive outcomes.

The primary research question was: 'What is the evidence for an effect of environmental noise on sleep, cardiovascular and cognitive outcomes?'

Four sub-questions were:

1. Is there a dose–response relationship between environmental noise and sleep, cardiovascular and cognitive outcomes?
2. Is there any evidence that certain populations, such as children, are particularly vulnerable to the effects of environmental noise on sleep, cardiovascular and cognitive outcomes?
3. Does the association between environmental noise and sleep, cardiovascular or cognitive outcomes vary by noise source, such as rail, road and aircraft?
4. Is there any evidence that annoyance is a mediator linking environmental noise exposure to sleep, cardiovascular and cognitive outcomes?

A protocol was developed with guidance from the expert advisory group for this review. This outlined the scope, research questions and criteria for selecting and appraising studies, templates for extracting data, and methods for synthesising the results.

The review followed established guidelines, such as the NHMRC guidelines (1999) and the Cochrane Collaboration guidelines (Higgins and Green, 2011).

It involved six steps:

1. Refining the research question and scope
2. Conducting an extensive search of the academic literature
3. Searching the websites of international agencies and conducting Google searches to identify grey literature
4. Extracting the relevant data
5. Assessing the quality of the selected studies
6. Systematically synthesising the selected studies.

This review informs chapters 3 to 7 of this document.

3. Literature search

A comprehensive and systematic search identified all relevant studies in peer reviewed and grey literature sources published from January 1994 to March 2014. This updates the previous enHealth review published in 2004, which was not a systematic review.

An initial 'scoping search' in December 2013 provided a brief overview of the evidence base and serve as a basis for scoping decisions. The formal search was done in March to June 2014 (bibliographic database searches) and July 2014 (internet searches). The results of the database searches and citations of relevant reports and articles identified in the grey literature search were uploaded to an EndNote library (EndNote X7, www.endnote.com) for appraisal. Full details of the search process are in the chapters addressing sleep, cardiovascular and cognitive outcomes.

4. Grey literature and hand searching

Primary studies published in the grey literature (not in peer reviewed journals) were identified by searching various online sources. Websites of key organisations (identified by the expert advisory group) and Google advanced search were searched. Full details of the search methods and results of the grey literature search are in the chapters addressing sleep, cardiovascular and cognitive outcomes.

Key journals, where a large proportion of included studies were published, were also hand searched by accessing the journal online and browsing archives for the period January 1994 to March 2014. These included:

- Noise and Health
- Journal of Sound and Vibration
- Journal of the Acoustical Society of America
- Applied Acoustics.

The reference lists of included studies and other relevant reviews were scanned for any additional studies.

5. Study selection and appraisal

Studies were selected for inclusion using a two-stage process conducted by two research team members (with 20 per cent random overlap to ensure consistency). The first stage involved scanning titles and abstracts in EndNote and excluding based on obvious deviations from the inclusion criteria. Full texts were retrieved for all remaining citations. The second stage involved reading the full text to ascertain whether the study fully met the inclusion criteria. The culmination of stage two was a final dataset of included studies.

The criteria used to select studies for review are in Table A-1.

Table A-1: Inclusion criteria for the systematic reviews.

Topic	Details
Participants	The review considered all studies that involve human subjects of any age.
Time periods	The review was limited to articles published between January 1994 and March 2014. This time frame was chosen to include the most relevant and recent studies, including those reviewed for the previous enHealth noise and health guidelines (2004).
Language	English language articles were included.
Noise exposure (source and how it was measured)	Studies were included if they specifically addressed environmental sources of noise. While this primarily means noise emitted from road, rail and air traffic, other sources considered relevant for this review included industrial and capital works, ventilation noise emitted from external sources in neighbouring buildings, and general community noise (not emitted from one's own property). Noise sources not within scope included: Occupational noise experienced by employees in the workplace Domestic sources of noise and their effects (e.g. noise from within neighbouring apartments Infra-sound and wind farms. A number of studies looked at classroom acoustics and cognition. Most of these were excluded because the noise source of interest was either within the classroom or emitted from within the school grounds. Studies were included only if the noise source of interest was external to the school and a sufficient measure of exposure was utilised. Studies were also required to include a reliable measure of exposure. This included a broad array of tools from direct measurement to estimates obtained from models or contour maps. Studies were excluded if only proxy measures of noise exposure were used (e.g. noise annoyance, proximity to a roadway).
Sleep outcomes	Studies were included if they addressed one or more sleep disturbance outcomes. These ranged from self-reported sleep quality to polysomnography. Studies assessing sleep disturbance among shift workers, who may not sleep during night time hours, were also included.
Cardiovascular disease outcomes	The specific focus of this review was on outcomes directly relevant to cardiovascular disease; including hypertension, heart disease, stroke and diabetes. Many studies examined blood pressure on a continuum – participants were not categorised into blood pressure categories. These studies were included as they encompass individuals with high blood pressure. Studies that focused solely on changes in hormone levels (such as catecholamines) or stress responses were excluded. These outcomes are related to cardiovascular health, but they do not provide a direct insight into the effects of environmental noise on cardiovascular disease risk. Rather, these measures are likely to be part of the causal pathway linking environmental noise with cardiovascular disease. In addition, there are numerous studies examining the effects of environmental noise on cardiovascular activity during sleep, such as cardiac arousals. These studies were excluded from the review as they are unlikely to provide an indication of risk.

Topic	Details
Cognition outcomes	Cognition may be defined in a number of ways but the relevant outcomes included in this review were those that were indicators of the cognitive functioning of healthy children, adolescents and adults with normal hearing. These include such functions as memory, comprehension, logical processing, attention and vigilance. Speech perception and the way people hear sounds was the focus of a number of studies, but not deemed relevant for this review as it is more of a mediating factor in the association between noise and cognition, rather than a cognitive outcome in itself. Listening and reading comprehension were considered to be cognitive functions and were included. A number of studies used simulated noise delivered while participants slept in a laboratory setting to study the association between noise-disturbed sleep and cognitive performance the next day. These were deemed to be more focused on the effect of the sleep disturbance on cognition rather than noise exposure itself and were therefore excluded.
Study and publication types	A broad range of study types was included. Studies were excluded if they had: no control or comparison group (e.g. descriptive study); intervention studies, except where relevant cross-sectional data (baseline) was available; and animal studies. Peer reviewed articles, official reports, and conference papers were included. Conference abstracts were included only when sufficient information was available to extract necessary data and appraise for risk of bias. Correspondence, editorials and reviews were excluded.

6. Quality assessment

The overall quality assessment process followed GRADE guidelines (Guyatt et al., 2011), informed by relevant recommendations from NHMRC (1999).

GRADE is a structured process for rating quality of evidence in systematic reviews. This process provides a summary of the evidence – the quality rating for each outcome and the estimate of effect, reflecting the extent we can be confident the estimates of effect are correct.

A range of domains were used to appraise the quality of the evidence. Risk of bias is first assessed at the individual study level. The rest are assessed by looking at the entire body of evidence for that outcome. These domains are:

1. Risk of bias – assessed at individual study level. Used to assess limitations with the study and degree of confidence in the findings
2. Inconsistency of results – inconsistency in participants, methodology and outcomes across the body of evidence. An evaluation of the similarity of point estimates and/or extent of overlap of confidence intervals may be used
3. Indirectness of evidence – the differences between study characteristics (such as participants, exposures and outcomes) and those of interest (such as populations of interest) within the body of evidence. The greater the difference, the more indirect the evidence. May be appropriate to use interchangeably with the terms 'applicability' and 'generalisability'
4. Imprecision – an assessment of 95 per cent confidence intervals (CI) to ascertain whether the estimate of effect for the body of evidence is sufficiently precise. This is more difficult if CIs are not reported and is generally only used in meta-analysis
5. Publication bias – suspected when evidence comes from a number of small studies, most of which have been commercially funded

6. Large magnitude of effect – presence may justify increasing the rating for the quality of the body of evidence
7. Plausible confounding, which would reduce a demonstrated effect
8. Dose–response gradient – presence may justify rating up the quality of the body of evidence.

Quality assessment involved two main stages.

First, the risk of bias within each individual study and each individual outcome within the study was assessed. The NHMRC level of evidence for study type was also recorded.

Second, the overall quality of the body of evidence for each individual outcome was assessed. See 'Evidence quality' below.

7. Risk of bias

Risk of bias is the risk that authors will overestimate or underestimate the true effect of a particular exposure (Higgins et al., 2011). Risk of bias is assessed by looking at features of the design and execution of individual studies that have the potential to affect the validity of findings. Risk of bias is distinguished from the 'methodological quality' of a study. The latter may refer only to the extent to which study authors conducted their research to the highest possible standards and not the extent to which results should be believed. A study may be performed to the highest possible standards and yet still have an important risk of bias (Higgins et al., 2011).

Risk of bias assessment was conducted by two researchers, with inter-coder reliability checked on 20 per cent of the sample to ensure consistency, and taking into account that judgements will involve a certain level of subjectivity. Any discrepancies were reviewed by a third researcher.

Assessment of risk of bias was informed by the GRADE guidelines risk of bias criteria (Guyatt et al., 2011); and the Cochrane Collaboration risk of bias tool (Higgins and Green, 2011). Further information on the GRADE criteria is available in sections 10 and 11 of this appendix.

Quality assessment tools such as GRADE are typically developed for the assessment of randomised controlled trials. Where appropriate, GRADE guidelines were modified to be suitable for assessing the studies in this review. This applied particularly to experimental studies, as GRADE guidelines emphasise allocation concealment and blinding in the risk of bias assessment. These criteria may be less relevant to experimental studies that are not randomised control trials. Therefore, we modified GRADE criteria to include a rating of 'randomisation and counterbalancing of allocation'. Studies using an appropriate method of allocation to experimental conditions (such as randomisation or counterbalancing) are rated as having a low risk of bias.

8. Evidence rating

Once risk of bias ratings were completed for all papers for a given outcome, a rating of the overall body of evidence was done. GRADE offers four levels of evidence quality: high, moderate, low and very low. These levels imply a gradient of confidence in estimates of treatment effect, and thus a gradient in the consequent strength of inference. Randomised trials begin as high quality evidence and observational studies as low quality evidence. Quality may be downgraded as a result of limitations in study design or implementation, imprecision of estimates (wide confidence intervals), variability in results, indirectness of evidence, or publication bias. Quality may be upgraded because of a very large magnitude of effect, a dose–response

gradient, and if all plausible biases would reduce an apparent treatment effect (appendix A, sections 10 and 11).

To be consistent with the NHMRC, we also appraised the evidence according to NHMRC levels of evidence ratings (Table A-7). These ratings were informed by GRADE ratings as well as study design. Details for interpreting each rating are shown in Table A-2.

Table A-2: NHMRC evidence statements

Evidence rating	Description
A	Findings from the body of evidence can be trusted
B	Findings from the body of evidence can be trusted in most situations
C	The body of evidence has limitations and care should be taken in the interpretation of findings
D	The body of evidence is weak and findings cannot be trusted

9. Data synthesis

Narrative synthesis is a textual approach to synthesis to 'tell the story' of the findings. This was chosen as the most appropriate approach to synthesis, given the diverse range of study types and the nature of the research questions.

Formal guidelines for narrative synthesis are not available. However, current guidelines for the conduct of systematic reviews (CRD, 2009) suggest that synthesis should incorporate these elements:

- developing a theory of how the intervention works, why and for whom
- developing a preliminary synthesis of findings of included studies
- exploring relationships within and between studies
- assessing the robustness of the synthesis.

These features are primarily concerned with systematic reviews of intervention studies. Only the last three elements were therefore used to guide the data synthesis stage.

10. GRADE criteria

The GRADE criteria are different for observational and experimental studies (Table A-3). Criteria 1 for experimental trials have been modified to better suit the types of studies in this review (not randomised controlled trials).

Table A-3: GRADE risk of bias criteria

Criteria	Questions
Risk of bias in experimental trials	
1. Lack of allocation concealment (changed in this review to randomisation/ counterbalancing of allocation)	Was there an adequate method of allocation? (randomisation or counterbalancing)
2. Lack of blinding	Were participants, personnel and outcome assessors 'blind' to intervention?

Criteria	Questions
3. Incomplete accounting of patients and outcome events	Was the trial stopped early? Were patients analysed in the groups to which they were randomised?
4. Selective outcome reporting bias	Is there incomplete or absent reporting of some outcomes and not others on the basis of the results?
5. Other limitations	Were there any other limitations that could affect the validity of the findings?
Risk of bias in observational studies	
1. Failure to develop and apply appropriate eligibility criteria (inclusion of control population)	**Cohort** Was the cohort representative of the population of interest? Were participants in different exposure groups recruited from the same population or matched and over the same period? **Case control** Were cases and controls recruited from the same population or matched and over the same period?
2. Flawed measurement of both exposure and outcome	**All** Was the exposure clearly defined and accurately measured? Were the main outcome measures used accurate (valid and reliable)? Did they use subjective or objective measurements? Were the measurement methods similar in different groups? Were the statistical tests used to assess the main outcomes appropriate? **Cohort** Do the analyses adjust for different lengths of follow-up? **Case control** Period between the intervention and outcome the same for cases and controls?
3. Failure to adequately control confounding	**All** Were all relevant prognostic factors measured? What was missed? (genetic, environmental, socio-economic) All relevant confounders addressed in design and/or analysis?
4. Incomplete follow-up	**All** Was follow-up complete enough? Was follow-up long enough? **Cohort** Anything special about people leaving or entering the cohort? **Cross-sectional** NA

Each study (or outcome, where multiple outcomes were assessed in one study) was given a score of 1, 2 or 3 based on the risk of bias found (see Table A-4 for details of scoring). At this stage the scores were not comparable across study types given that a randomised controlled trial may receive a high risk of bias score and a cross-sectional study may receive a low risk of bias score.

Table A-4: Risk of bias summary scores

Bias Score	Definition
1	Low risk of bias for all key criteria
2	Crucial limitations for one criterion or some limitations for multiple criteria sufficient to lower one's confidence in the estimate of effect
3	Crucial limitation for one or more criteria sufficient to substantially lower one's confidence in the estimate of effect

Both GRADE guidelines and the Cochrane Collaboration recommend against the use of scales yielding a score because calculating a score inevitably involves assigning weights to particular domains, which is not always justifiable (Higgins, Altman et al., 2011). However, summarising risk of bias within individual studies is useful when grading the quality of evidence across studies, which occurs at the data synthesis stage.

11. GRADE levels of evidence

Table A-5: Quality assessment criteria (Guyatt, Oxman et al., 2011)

Study design	Quality of evidence	Lower if	Higher if
Randomised trial	High	Risk of bias -1 Serious -2 Very serious	Large effect +1 Large +2 Very large
	Moderate	Inconsistency -1 Serious -2 Very serious	Dose response +1 Evidence of a gradient
Observational study	Low	Indirectness -1 Serious -2 Very serious	All plausible confounding +1 Would reduce a demonstrated effect or
	Very low	Imprecision -1 Serious -2 Very serious	+1 Would suggest a spurious effect when results show no effect
		Publication bias -1 Likely -2 Very likely	

Table A-6: Quality of evidence grades

Grade	Definition
High	We are very confident that the true effect lies close to that of the estimate of the effect
Moderate	We are moderately confident in the effect estimate: the true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different
Low	Our confidence in the effect estimate is limited: the true effect may be substantially different from the estimate of the effect
Very low	We have very little confidence in the effect estimate: the true effect is likely to be substantially different from the estimate of effect

Table A-7: NHMRC evidence hierarchy (NHMRC 2009)

Level	Intervention	Diagnostic accuracy	Prognosis	Aetiology	Screening intervention
I	A systematic review of level II studies	A systematic review of level II studies	A systematic review of level II studies	A systematic review of level II studies	A systematic review of level II studies
II	A randomised controlled trial	A study of test accuracy with: an independent, blinded comparison with a valid reference standard, among consecutive persons with a defined clinical presentation	A prospective cohort study	A prospective cohort study	A randomised controlled trial
III-1	A pseudo randomised controlled trial (alternate allocation or some other method)	A study of test accuracy with: an independent, blinded comparison with a valid reference standard, among non-consecutive persons with a defined clinical presentation	All or none	All or none	A pseudo randomised controlled trial (alternate allocation or some other method)
III-2	A comparative study with concurrent controls: Non-randomised experimental trial Cohort study Case-control study Interrupted time series with a control group	A comparison with reference standard that does not meet the criteria required for Level II and III-1 evidence	Analysis of prognostic factors among persons in a single-arm of a randomised controlled trial	A retrospective cohort study	A comparative study with concurrent controls: Non-randomised experimental trial Cohort study Case-control study

Level	Intervention	Diagnostic accuracy	Prognosis	Aetiology	Screening intervention
III-3	A comparative study without concurrent controls: Historical control study Two or more single-study Interrupted times series without a parallel control group	Diagnostic case-control study	A retrospective cohort study	A case-control study	A comparative study without concurrent controls: Historical control study Two or more single-arm study
IV	Case series with either post-test or pre-test/post-test outcomes	Study of diagnostic yield (no reference standard)	Case series, or cohort study of persons at different stages of disease	A cross-sectional study or case series	Case series

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Title: Transportation Noise Pollution and Cardiovascular Health

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Transportation Noise Pollution and Cardiovascular Health

Short Title: Noise and Cardiovascular Health

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Abstract

Epidemiological studies have found that transportation noise increases the risk for cardiovascular morbidity and mortality, with solid evidence for ischemic heart disease, heart failure, and stroke. According to the World Health Organization, at least 1.6 million healthy life years are lost annually from traffic-related noise in Western Europe. Traffic noise at night causes fragmentation and shortening of sleep, elevation of stress hormone levels, and increased oxidative stress in the vasculature and the brain. These factors can promote vascular (endothelial) dysfunction, inflammation, and arterial hypertension, thus elevating cardiovascular risk. The present review focusses on the indirect, non-auditory cardiovascular health effects of noise. We provide an updated overview of epidemiological research on the effects of transportation noise on cardiovascular risk factors and disease, mechanistic insights based on the latest clinical and experimental studies and propose new risk markers to address noise-induced cardiovascular effects in the general population. Potential effects of noise on alterations of gene networks, epigenetic pathways, circadian rhythm, signal transduction along the neuronal-cardiovascular-axis, oxidative stress, inflammation, and metabolism will be elaborately explained. Current and future noise mitigation strategies will be described. Lastly, we will conduct an overall evaluation of the status of the current evidence of noise as a significant cardiovascular risk factor.

Introduction

Extensive research has been conducted on adverse health impacts of air pollution and cardiovascular disease (CVD), and it is well-established that air pollution encompasses conditions like acute myocardial infarction (MI), heart failure, arrhythmia, hypertension, and stroke (for review see[1]). Recent studies have identified particulate matter with a diameter of <2.5µm (PM2.5) as a leading air pollutant contributing to approximately 8.8 million annual excess deaths.[2] Surprisingly, much less attention has been given to another frequent environmental pollutant: transportation noise. This is somewhat puzzling, given that urban areas experience simultaneous exposure to high air pollution and noise levels.[3,4] Noise is defined as "unwanted and/or harmful sound " and includes transportation, occupational, leisure, residential, and industrial noise (Figure 1).[5]

In their 2020 report, the European Environment Agency (EEA) highlighted that many people remain exposed to high road traffic noise levels.[6] EEA estimated that at least 20% of the population in the European Union (EU) resides in urban areas where traffic noise adversely affects health, though this number is considered strongly underestimated.[6] The population's exposure to environmental noise is projected to increase due to urban expansion and growing demand for mobility.[7] Projections indicate an apparent rise in the number of individuals exposed to road noise exceeding 55 dB(A) in L_{den} (day-evening-night level, Supplement Table 1) by 7.8% and railway noise by 11.8% within urban areas and 16.4% and 8.7%, respectively, outside urban areas by 2030.[7] In contrast, the exposure to aircraft noise will remain unchanged inside and outside metropolitan area.[7]

In this review, our primary focus is to explore the indirect, non-auditory impacts of transportation noise on cardiovascular health. We summarize epidemiological and clinical findings and mechanistic and experimental data. Additionally, we shed light on emerging indicators to better understand the cardiovascular consequences of noise in the general population. Mechanistic data related to the adverse health effects of noise encompass alterations in gene networks, epigenetic pathways, circadian rhythms, signal transmission within the neuronal-cardiovascular connection, oxidative stress, inflammation, and metabolic processes. Finally, we provide an overview of the most promising strategies for mitigating noise-related health issues and assess the existing body of evidence regarding noise as a risk factor for cardiovascular health.

Noise and the Global Burden of Disease

The global health burden arising from noise is substantial. Road traffic noise above 55 dB(A) L_{den} affects 113 million Europeans, mainly in urban areas.[8] Furthermore, 22 million are exposed to railway noise and 4 million to aircraft noise above 55 dB(A). According to the World Health Organization (WHO), adverse health impacts are likely at this noise level.[9] Noise exposure at night is even more detrimental, and the WHO recommends that nocturnal noise from road traffic do not exceed 45 dB(A) (L_{night}).[9] Annually in EU, transportation noise is estimated to result in 12,000 premature deaths, 48,000 new cases of ischemic heart disease (IHD), 6.5 million people experiencing chronic sleep disturbances, and 22 million individuals enduring significant annoyance.[7] Nevertheless, these statistics fail to capture the full extent of the health burden, as adverse effects are now observed at lower noise exposure levels than previously recognized, affecting a broader range of health outcomes.

How Noise Causes Cardiovascular Disease: The Noise Reaction Scheme

Wolfgang Babisch introduced the noise reaction model in which an indirect pathway plays a pivotal role in the development and progression of CVD.[10] A key element of this model is the cognitive perception of noise, which triggers cortical activation and release of stress hormones (Figure 1). Over time, this can lead to the emergence of cardiovascular risk factors, such as diabetes, high cholesterol, and high blood pressure, ultimately manifesting as CVD, including conditions like acute and chronic coronary syndrome, heart failure, persistent hypertension, arrhythmias, and stroke (for review see[11]). Noise also disrupts sleep, daily activities, and communication, causing annoyance and reduced sleep quality and duration, potentially linked with increased risk of CVD[11] (Figure 1). The noise-induced activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system triggers the release of stress hormones like cortisol and catecholamines. This, in turn, induces inflammation, leading to increased levels of IL-6, IL-1β, and proinflammatory monocytes, along with oxidative stress. Nighttime exposure to aircraft noise has even been linked to stress-induced cardiomyopathy, also known as Takotsubo Syndrome.[12] Stress responses can increase blood pressure, potentially impairing vascular function. This affects endothelial nitric oxide synthase (eNOS), increases oxidative stress, and reduces vascular nitric oxide bioavailability, ultimately causing

endothelial dysfunction and rendering blood vessels more sensitive to stress-induced vasoconstriction (Figure 1).

Importantly, a recent study showed that the amygdala, a part of the limbic system primarily involved in stress perception and emotional regulation, acts as a "cerebral" link between noise stimuli, vascular inflammation, and adverse cardiovascular events.[13] Based on clinical imaging to measure amygdala metabolic activity and arterial inflammation in 498 adults without preexisting CVD or active cancer, the study found that road and aircraft noise exposure was associated with increased amygdala activity and vascular inflammation. Furthermore, a mediation analysis indicated that higher noise exposure was associated with significant adverse cardiovascular events (MACE) through a sequence involving heightened amygdala activity and arterial inflammation. This association remained robust, with a hazard ratio (HR) of 1.34 (95% confidence interval [CI]: 1.15–1.57) per 5 dB(A) increase in noise levels, even after multivariable adjustments.[13] Accordingly, a subsequent report demonstrated that among individuals chronically exposed to socioeconomic or environmental stressors, a higher degree of resilience was associated with a >50% reduction in MACE risk, potentially via reduced arterial inflammation, suggesting that enhancing resilience may decrease the CVD burden in response to these stressors.[14]

Epidemiological Evidence

Transportation Noise and Cardiovascular Disease and Death

Over the past years, there has been a growing body of evidence on the effects of environmental noise on cardiovascular health. The meta-analyses commissioned by the WHO that were published as part of the Environmental Noise Guidelines for the European Region from the World Health Organization (WHO ENG)[15] included studies published until 2015. Subsequent studies have been evaluated in a 2023 Umbrella+ review, including meta-analyses to obtain up-to-date exposure-response functions for CVD and mortality.[16] An Umbrella+ review summarizes results from the newest high-quality systematic reviews combined with original studies published after the corresponding review. Only original studies that applied reliable exposure assessment methods and accounted for the most relevant confounding factors were considered in the Umbrella+ review. For mortality and CVD, only cohort studies were included. In contrast, case-control and cross-sectional studies were included for

hypertension if they were large, population-based, and used established methods for outcome assessment.[16] Below is a summary of the Umbrella+ CVD results (Figure 2).

Cardiovascular mortality

The systematic literature search identified 61 potentially eligible cardiovascular and IHD mortality papers. Thereof, twelve prospective cohort studies on road, railway, and/or aircraft noise were eligible for meta-analysis (Supplementary Table 2). Supplement Figure 1 shows the results of the meta-analysis. Based on nine studies, the pooled effect estimate per 10 dB(A) of road traffic noise was 1.045 (95% CI: 1.017–1.073).[16] For railway and aircraft noise, only two studies were available for each exposure, suggesting either no or minimal effect of these two exposures on cardiovascular mortality (Supplement Figure 1).

Ischemic heart disease

A systematic review published as part of the WHO ENG,[15] assessed the association between transportation noise and incidence of IHD and found a relative risk (RR) of 1.08 (95% CI: 1.01–1.15) per 10 dB(A) increase in L_{den} based on three cohort and four case-control studies.[17] The working group rated the evidence for an association to be high. Only ecological or cross-sectional studies were identified for railway and aircraft noise, which led to a very low certainty of evidence.

Since then, many studies have been published investigating noise and IHD incidence. The most recent studies includes a pooled analysis of Danish and Swedish cohorts,[18] and a nationwide study with more than 2.5 million participants.[19] Both studies found road traffic noise to increase IHD incidence with HRs of 1.03 (95% CI: 1.00–1.05) and 1.05 (95% CI: 1.04–1.06), respectively. For railway noise, however, the results are inconsistent.[18 19]

We found that in a meta-analysis combining the WHO ENG review[17] with three new cohort studies and the pooled Scandinavian cohort (Supplement Table 3), a 10 dB(A) increase in road traffic noise was associated an RR of 1.041 (95% CI: 1.023–1.059) for IHD incidence (Figure 2).[16] Corresponding RRs (95% CI) for aircraft and railway noise were 1.009 (0.992–1.026) and 0.996 (0.933–1.062), respectively.

Stroke

In the WHO ENG, only one cohort study on road traffic noise and incident stroke was available, finding an HR of 1.14 (95% CI: 1.03–1.25).[15] Certainty of evidence was thus assessed by the WHO working group to be moderate. For railway and aircraft noise, no cohort studies were identified. Since then, nine studies on stroke were published, mostly indicating positive associations between road traffic noise and risks close to unity for rail and aircraft noise. Among others, a pooled analysis including multiple cohorts from Denmark and Sweden resulted in an increased risk with a HR of 1.06 (95% CI: 1.03–1.08) per 10 dB(A) higher road traffic noise.[20]

In the Umbrella+ review we found that based on six cohort studies (Supplement Table 3), road traffic noise increased in the risk of incident stroke, with an RR per 10 dB(A) of 1.046 (95% CI: 1.013–1.081).[16] In contrast, aircraft and railway noise, both based on two studies, was not associated with stroke, with pooled RR (95% CI) of 0.995 (0.875–1.131) and 0.969 (0.955–0.984), respectively.

Heart failure

Neither the WHO ENG[15] nor any recent systematic review addressed heart failure in the context of noise exposure. However, six longitudinal studies addressed this association in recent years, all showing positive associations with an RR between 1.01 and 1.09 per 10 dB(A) increase in road traffic noise (Supplement Table 3). In an updated meta-analysis, we found road traffic noise to increase risk of heart failure, with a RR of 1.044 (95% CI: 1.017–1.071) per 10 dB(A) increase (Figure 2).[16] Two studies assessing railway noise indicated no or a positive association. For aircraft noise, a German study[21] showed a decrease in risk, whereas a Danish nationwide study presented a positive association with a RR of 1.06.[19] The pooled RR for railway and aircraft noise, derived from two studies each, were 1.011 (95% CI: 0.998–1.035) and 1.017 (95% CI: 0.934–1.107) per 10 dB(A) L_{den}, respectively.

Arrhythmia

Although a few reviews have addressed the association between noise and arrhythmia, no meta-analyses have been conducted. Further, only a few cohort studies exist. These studies include a Danish nationwide cohort study[22] with over 3.5 million participants of whom 269,756 developed atrial fibrillation. This study reported weak positive associations with road, railway, and aircraft noise, with risk increases of 1–2% per 10 dB(A).

In an updated meta-analysis for road traffic noise based on three cohort studies, we found an RR of 1.006 (95% CI: 1.001–1.011) per 10 dB(A), whereas the pooled RR for aircraft noise, which was based on two studies, was 1.207 (95% CI: 0.699–2.084) per 10 dB(A).[16] Only one study provided an estimate for railway noise, which was 1.017 (95% CI: 1.008–1.027) per 10 dB(A).

Conclusions on cardiovascular incidence studies

Based on the Umbrella+ review, we conclude that solid evidence exists for an association between road traffic noise and IHD, stroke, and heart failure. For all cardiovascular diagnoses combined, the risk increased by 3.2% (95% CI: 1.1–5.2%) per 10 dB(A) higher road traffic noise (L_{den}, Figure 2). Associations tended to be less pronounced for railway and aircraft noise. It is conceivable that noise from railways and aircraft is often masked by the substantially more prevalent road traffic noise, and this may explain why the exposure-response association for these two sources may not be as accurately estimated as for road traffic.

Lower effect threshold of noise

The lower effect threshold of noise is defined as the level below which no health effects of noise are expected.This threshold has not been determined with certainty. It is currently uncertain at which level this lower effect threshold is. Also, the threshold likely varies between sources of noise. Different noise recommendation limits exist across the world. E.g., the EU currently incorporates a noise mapping threshold of 55 dB(A) L_{den} as part of the European Noise Directive. In contrast, the WHO, in their 2018 guidelines, recommended a 53 dB(A) threshold for road traffic noise (L_{den}).[9]

Recently, several studies based on large cohorts have investigated the shape of the exposure-response function for transportation noise in relation to various outcomes with large statistical power across the entire exposure span, starting from around 35–40 dB(A) (app. background level of noise) until 80–85 dB(A).[19,20,23-25] Many of these studies suggest that the effects of noise on morbidity and mortality start already from 35–40 dB(A), e.g., for stroke,[20,23] diabetes,[26] and cardiovascular mortality.[25,27] For IHD, studies have suggested a threshold between 50 and 55 dB(A).[19,24]

As illustrated in Table 1, correct assignment of the effect threshold level is highly important when performing a health impact assessment, an essential tool for

decision-makers prioritizing noise prevention. This table shows results from health impact assessments concerning CVD mortality and incident IHD for Denmark and Switzerland using four potential lower effect thresholds scenarios for road traffic noise. These calculations are possible because researchers in Denmark and Switzerland have performed estimations of road traffic noise for the entire population throughout the exposure range.[28,29] We observed that the calculated numbers of IHD and CVD deaths due to road traffic noise were three to four times higher when using 45 dB(A) as a lower effect threshold than 55 dB(A), highlighting the importance of identifying the correct effect threshold for noise.

Effects of Noise on Cardiovascular Risk Factors

Arterial hypertension

A 2021 review assessed hypertension in relation to transportation noise and derived pooled estimates stratified by noise source and study type.[30] While the meta-analysis for road traffic noise showed no association for cohort and case-control studies, an increased odds of 9% per 10 dB(A) was seen in cross-sectional studies. No associations were observed for aircraft and railway noise. Since then, three studies have been published on aircraft noise and hypertension, of which two studies were based on populations from the USA, showing that comparing individuals exposed to above 45 dB(A) versus below 45 dB(A) resulted in increased risk of 0%[31] and 3%.[32] A French aircraft study found a significant risk increase of 36% per 10 dB(A).

In a meta-analysis pooling the risk estimates from cohort and cross-sectional studies, we found an RR of 1.04 (95% CI: 0.970–1.126) per 10 dB(A) road traffic noise and 1.031 (95% CI: 1.008–1.053) per 10 dB(A) aircraft noise.[16 30] No studies were identified for railway noise. This suggests that transportation noise may increase the risk of hypertension, though more longitudinal studies are needed to investigate this further.

Diabetes and obesity

Type 2 diabetes and obesity are frequent comorbidities in CVD patients,[33,34] and adverse effects of noise on these two conditions may, therefore, contribute to the link between noise and CVD. Several recent cohort studies have consistently linked transportation noise, especially from road traffic, with a higher risk of diabetes.[26,35-41] A meta-analysis from 2023 found that a 10 dB(A) higher road traffic noise was

associated with an RR of 1.06 (95% CI: 1.03–1.09).[42] Interestingly, two studies have investigated effects of road traffic noise at both the most exposed façade (standard noise measure) and the least exposed façade, considered to be a proxy for bedroom noise exposure, as people often chose to sleep in a room facing away from a busy street.[26,43] These two studies found more potent effects of noise at the least exposed façade than the most exposed façade, suggesting that for diabetes, the effects of noise on sleep are an essential mechanism. Furthermore, noise-induced endothelial dysfunction is suggested to contribute to the adverse metabolic effects noise.[44] In line, a recent study demonstrated that endothelial dysfunction can predict the onset and progression of type 2 diabetes.[45]

A handful of prospective cohort studies have investigated the effects of exposure to transportation noise on the risk of developing adiposity.[46-51] The measures of adiposity varied across the studies, with four studies investigating effects on weight gain/BMI,[46,47,49-51] two studies on waist circumference changes,[46,47] and two studies on the risk of obesity/overweight.[47,48] While evidence is still emerging, the currently available research suggests an association between exposure to road traffic noise and an increased risk of adiposity. Interestingly, this was shown in both child cohorts,[50,51] a pregnancy cohort,[49] and adult and elderly populations,[46-48] suggesting that exposure to road traffic noise may affect the risk of adiposity throughout life.

Mental health

Major depression and other psychological factors are acknowledged risk factors for CVD,[52] and the effects of transportation noise on mental health could, therefore, be an essential mediator in explaining the effects of transportation noise on CVD risk. A 2020 meta-analysis found that road traffic and aircraft noise were associated with a higher risk of depression, with RRs (95% CI) per 10 dB(A) of 1.03 (0.99–1.06) and 1.12 (1.02–1.23), respectively.[53] Since this meta-analysis, new longitudinal studies have supported that road traffic noise may increase the risk of depression and/or poorer mental health-being[54,55] and suicide.[56] One study furthermore suggested that the effects of road traffic noise on depression seemed to be partly mediated by annoyance towards noise,[54] supported by a prospective study showing that noise annoyance at baseline was associated with risk of depression and anxiety symptoms

five years later.[57] The observed linkage between transportation noise and depression needs confirmation in more high-quality prospective studies.

Unhealthy behaviour

A few studies have investigated whether road traffic noise is associated with unhealthy lifestyle habits,[58-60] which seems plausible as noise is believed to exert its harmful effects through two main pathways associated with lifestyle changes, namely stress and sleep disturbance. Effects of noise on leisure-time physical activity have been studied in two studies, investigating effects of estimated residential road traffic noise and self-reported transportation noise annoyance.[58,59] Both studies suggested that road traffic noise might have a negative impact on physical activity.[59] One study investigated the effects of road traffic noise on smoking and alcohol habits.[60] Although the study showed that road traffic noise was positively associated with smoking and alcohol intake in cross-sectional analyses, longitudinal analyses showed no associations with lifestyle changes. New, well-designed cohort studies are highly necessary to investigate whether the effect of noise on lifestyle is an important mediator on the pathway between noise and CVD.

Sleep disturbance

Sleep societies recommend that adults obtain 7-9 hours of high-quality sleep to promote well-being and health.[61] Conversely, sleep that is disturbed or too short has been associated with increased mortality and several adverse health outcomes, first and foremost CVD.[62] In 2022, the American Heart Association added sleep to their list of lifestyle factors with critical importance for cardiovascular health. Noise disrupts sleep by causing intermittent awakenings or brief arousals, reducing continuity.[63] Consequently, sleep will be shorter and lighter with less time spent in deep and REM sleep, which is crucial for sleep recovery. This is evidenced by increased daytime sleepiness and decreased performance after noisy nights.[64] Even when sleep stages are unaffected, noise can increase the number and intensity of autonomic arousals, negatively affecting metabolic and cardiovascular function.[63,65] In addition to the sound level, the sleep-disturbing properties depend on other noise characteristics like rise time and frequency content, but also the meaning of the sound to the recipient.[63]

Epidemiological studies suggest that nighttime noise exposure has a more significant impact on long-term health outcomes than daytime noise exposure, likely

also because people are more consistently at home during the night.[66] Studies have shown that even a single night of exposure to rail or aircraft noise can impair flow-mediated dilation of the brachial artery, a finding replicated in individuals with or at high risk for coronary artery disease.[67,68] Noise also prevents blood pressure from dipping during the night,[69] with plausible long-term effects on CVD risk. Analysis of blood proteins showed that noise exposure induces changes indicative of a pro-thrombotic and pro-inflammatory state, providing a biological basis for the increased risks of CVD and other diseases like neurodegenerative disease, obesity, diabetes, and breast and colon cancer.[68] In one animal study, adverse effects on blood vessels and composition were primarily observed if the noise exposure was intermittent during the sleep phase, again highlighting the importance of undisturbed sleep for health.[70] A recent retrospective case-crossover study at Zurich airport revealed an association between aircraft noise exposure levels in the two hours before an event and cardiovascular mortality.[71] Thus, nocturnal noise exposure may contribute to physiological changes that elevate CVD risk and trigger fatal events through physiological arousal.

Noise annoyance

Noise annoyance is a psychological response to unwanted or disturbing sounds, encompassing cognitive, emotional, and behavioral reactions. Globally, road traffic noise is the primary source of noise annoyance, while neighborhood noise is another substantial but understudied contributor. ISO standards provide a framework for assessing noise annoyance through population surveys, utilizing the "percentage highly annoyed" (%HA) metric for clear communication.[72,73]

Acoustic characteristics of noise exposure, such as intensity, frequency, complexity, and duration, do not solely determine noise annoyance reactions. They are significantly influenced by personal, social, and situational factors, encompassing age, sex, health status, noise sensitivity, attitude towards noise, socioeconomic status, public perception, perceived stress, and coping abilities.[74] In the WHO ENG, exposure-response relationships for road, railway, and aircraft noise estimated in meta-analyses, ranked aircraft noise as the most annoying.[75] Recent trends showed increased annoyance for aircraft and railway noise while remaining relatively stable for road traffic noise, necessitating further investigation into these variations.[76,77]

Noise annoyance can be an early indicator of more severe health risks due to its rapid onset compared to physical illnesses. Historically, annoyance has been a critical indicator for shaping noise policies, with noise protection limit values partially based on exposure-response functions for annoyance. WHO recommends noise level reductions to protect against adverse health effects, primarily informed by noise annoyance surveys. However, evolving epidemiological studies are focusing on cardiometabolic health outcomes.[78]

Mechanistic Noise Studies in Humans and Animals

Cardiovascular effects of noise

The concept that non-auditory effects of noise contribute substantially to health consequences was suggested in a 1970 monography.[79] The monography described results showing that acute noise exposure had cardiovascular effects, e.g., a study establishing that noise exposure led to constricting peripheral blood vessels in individuals engaged in physical exercise.[80] Also, a study showed that exposure to noise or music elicited varying hemodynamic responses, including cardiac output and minute flow, and the authors concluded that the intensity of the sound, rather than its aversive (noise) or pleasurable (music) qualities, governed the somatic responses.[81]

Among 1,005 German industrial workers, it was observed in 1968 that workers in very noisy industries exhibited more pronounced peripheral circulation problems, heart issues, and equilibrium disturbances than workers in less noisy industries.[82] In 1993, the Speedwell study reported significant associations between road traffic noise and risk factors for IHD. These included increases in total triglycerides, platelet count, plasma viscosity, glucose levels, and systolic and diastolic blood pressure.[83] Moreover, higher noise levels were significantly linked to elevated systolic and diastolic blood pressure and heart rate.[84] Later, a study found that nighttime transportation noise led to more pronounced increases in blood pressure compared to daytime exposure.[85] It was suggested that repeated nighttime autonomic arousals may disrupt the natural nocturnal decline in blood pressure.[69] Also, in workers exposed to occupational noise exceeding 80 dB(A) LAeq, a significant increase in systolic and diastolic blood pressure was observed along with elevated levels of glutathione peroxidase and DNA damage, compared to office workers exposed to between 40-50 dB(A) LAeq.[86]

Translational Studies of Transportation Noise on Endothelial Function in Humans

Through a series of field investigations (studies performed in the home of the subject), the adverse effects of aircraft and railway noise on vascular (endothelial) function, sleep quality, stress hormone release, and inflammation markers, both in healthy individuals and CVD patients were established.[67,87] Noise recorded in a bedroom near Düsseldorf airport was played back (30 or 60 times per night; noise30 and noise60) on a standard portable audio system with a fixed speaker position relative to the head of the subject.

These studies revealed that in healthy subjects, nighttime exposure to aircraft noise with an equivalent sound level (Leq) of 46.3 dB(A) and a peak level of 60 dB(A) for one night caused decreased sleep quality, elevated levels of adrenaline, endothelial dysfunction (impaired flow-mediated dilation, a subclinical marker for atherosclerosis), and a reduction in pulse transit time, indicating sympathetic activation (Figure 3).[87] Notably, the acute administration of vitamin C of 2g improved endothelial dysfunction 2h after administration, indicating the involvement of reactive oxygen species (ROS) in causing vascular dysfunction. Interestingly, a priming effect of aircraft noise on endothelial function was observed, i.e., previous exposure to Noise30 caused Noise60 to have a significantly larger adverse effect on endothelial function. These data demonstrate that aircraft noise can affect endothelial function and suggest that rather than habituation, prior noise exposure seems to amplify the negative effect of noise on endothelial function. Further support for the oxidative stress concept was established by the demonstration of a significant increase in oxidative stress markers 3-nitrotyrosine [3-NT] and 8-isoprostane serum levels in response to aircraft noise exposure (Figure 3).[70]

The adverse effects of aircraft noise on endothelial function were more pronounced in patients with established coronary artery disease, suggesting that an already compromised endothelium is more susceptible to further deterioration.[67] Similar investigations were conducted in healthy subjects exposed to either 30 (Noise30) or 60 train events (Noise60) during the nighttime, with LAeq levels ranging from 33 to 54 dB(A) for one night (Figure 3).[88] This exposure decreased sleep quality and impaired the brachial artery's flow-mediated dilation (FMD). Once again, acute challenges with vitamin C significantly ameliorated railway noise-induced endothelial

dysfunction. In-depth proteomic analysis identified significant impacts on plasma proteins involved in redox, pro-thrombotic, pro-inflammatory, and fibrotic pathways compared to controls.[88]

We have furthermore explored the influence of nighttime noise event loudness and frequency on endothelial function by exposing patients with established coronary artery disease to two nighttime aircraft noise scenarios, with comparable mean sound pressure levels: one with loud and infrequent noise events and one with less loud but more frequent noise events (Leq values of 37 dB(A) for control and 45 dB(A) for noise exposure for one night).[89] Both scenarios resulted in similar worsening of endothelial function (FMD). For the first time, we also observed a diastolic heart dysfunction (an increase in the E/E' ratio) as indicated by serial echocardiography. An exploratory protein analysis through proximity extension assay revealed significant decreases in three biomarkers (follistatin, glyoxalase I, and ACE-2) associated with regulating heart function, oxidative stress, inflammation, and fibrosis.[89]

Noise Causes Endothelial Dysfunction, Epigenetic Changes, and an Adverse Impact on the Immune System

Cross-sectional cohort studies have found that exposure to transportation noise can impact the immune system. Two studies observed noise to increase levels of IL-12 and high-sensitivity C-reactive protein (hsCRP) while decreasing natural killer cell populations and activity,[90,91] although these results are not consistently uniform across all studies.[65] Furthermore, alterations in the immune system have been linked to elevated circulating cortisol levels and heightened noise sensitivity.[90,91] Interestingly, a study based on the Swiss SAPALDIA cohort showed that long-term exposure to transportation noise and air pollution led to distinct and shared DNA methylation patterns, with enrichments in pathways related to inflammation (e.g. CRP), cellular development, and immune responses.[92] Based on the same cohort, chronic exposure to nocturnal intermittent train or road traffic noise was suggested to increase arterial stiffness (reflecting endothelial dysfunction), as determined by pulse wave velocity.[93] This finding is supported by a German cohort study, showing that long-term exposure to night-time road traffic noise was associated with subclinical atherosclerosis, especially in participants with early arterial calcification.[94,95] Thus, in summary, these findings offer pathophysiological and molecular evidence from

human studies, shedding light on observed effects of transportation noise on incident CVD. Notably, the results from these human studies, including stress pathways, inflammation, oxidative stress, parameters of arterial stiffness, and endothelial/cardiac dysfunction, align with mechanistic data derived from animal studies (see subsequent sections).

Development of Animal Models to Study the Molecular Mechanisms of the Cardiovascular and Cerebral Side Effects of Transportation Noise

Noise causes vascular dysfunction, oxidative stress, inflammation, and dysregulation of gene networks

A novel animal model was established to study the molecular mechanisms underlying noise-induced cardiovascular and cerebral adverse effects. . This involved exposing mice to continuous aircraft noise (with a constant Leq of 72 dB(A) and peak levels of 85 dB(A) for 24 hours a day, for 1, 2, and 4d).[96] This exposure significantly increased stress hormones, blood pressure, endothelial dysfunction, and oxidative stress in both vascular and cerebral systems, primarily from phagocytic nicotinamide adenine dinucleotide phosphate (NADPH) oxidase (NOX-2), and secondary to inflammation due to infiltrating immune cells, which was associated with diminished endothelial nitric oxide bioavailability, increased vascular superoxide, enhanced endothelin-1 expression, and sensitivity to vasoconstrictors.[70,96] Importantly, endothelial dysfunction was associated with an up- rather than downregulation of the eNOS. Further studies revealed that eNOS was uncoupled due to enhanced S-glutathionylation of eNOS, an established mechanism of eNOS uncoupling in the aorta.[97] When mice were exposed to white noise under similar conditions (comparable exposure time and mean sound pressure level), they did not exhibit these cardiovascular side effects. This suggests that it is not merely the sound pressure level itself, but rather the characteristics of the noise, such as frequency or noise pattern, that play a determining role in exerting cardiovascular damage.[96]

A dysregulation of gene networks in response to noise, identifying potential marker genes associated with noise within the vasculature was also observed. This was accompanied by impaired endothelial and vascular signaling. Among the four most up-regulated genes compared to controls were Zbtb44, Setad4, Ypel2, and Ihh. Conversely, the expression of Sacs, Nbeal1, PTPN4, and NR4A3 transcripts in aortic

tissue was significantly reduced by noise. These genes are linked to TGF-beta signaling, autophagy, growth, matrix metalloprotease regulation, and fibrosis.[96]

Cellular pathway analysis unveiled alterations in NF-κB and adrenergic signaling, focal adhesion, cell cycle control, apoptosis, and kinase-mediated growth and proliferation signaling, with Foxo transcription factors playing a central role. Noise also adversely influenced the circadian rhythm, insulin, and calcineurin signaling pathways.[70,96]

The central role of nighttime noise, inflammatory cells, and changes in the circadian rhythm

As opposed to the awake phase, exposure to aircraft noise during sleep had a substantially more detrimental impact on the cardiovascular system, leading to endothelial dysfunction, increased blood pressure responses, higher levels of neurohormones, the vasoconstrictor endothelin-1, and oxidative stress in the plasma, the vasculature, and the brain (Figure 4).[70] Additionally, it resulted in dysregulation of central and peripheral Foxo3/circadian clock signaling, shown using RNA sequencing. A crucial finding was that aircraft noise-induced vascular and cerebral damage was strongly mitigated by knockout of the Nox2 gene, underscoring the vital role of inflammatory cells in mediating noise-induced cardiovascular and cerebral side effects.[70] Surprisingly, within 4d of continuous noise exposure, noise also triggered a significant downregulation and uncoupling of neuronal nitric oxide synthase (nNOS), creating a neuroinflammatory phenotype, as indicated by markers of inflammation and astrocyte activation.[70] This enhanced formation of cerebral ROS could partially explain the observed impairments in cognitive development, especially in learning and memory, in children exposed to aircraft noise.[98]

Particularly interesting is that the molecular mechanisms underlying vascular dysfunction in response to continuous and intermittent (nighttime) aircraft noise closely resemble the mechanisms through which traditional cardiovascular risk factors, such as diabetes,[99] arterial hypertension,[100] and smoking,[101] induce endothelial and vascular dysfunction. This suggests that noise-induced stress and pre-existing cardiovascular risk factors may accelerate vascular and cerebral atherosclerosis and neurodegenerative diseases due to shared molecular pathomechanisms.

In line with this, we noted that the adverse cardiovascular effects of aircraft noise were exacerbated in mice with pre-existing arterial hypertension (angiotensin-II infused animals, exposed to a continuous Leq of 72 dB(A) and peak levels of 85 dB(A) for 24 hours a day for seven days).[102] This phenomenon was primarily driven by increased blood pressure, vascular inflammation, and oxidative stress. Noise further potentiated neuroinflammation and cerebral oxidative stress in hypertensive animals.[102] In addition, noise with a mean sound pressure level of 72 dB(A) for 4d induced oxidative DNA damage and enhanced NOX-2 expression in C57BL/6 mice.[103] In Ogg1-/- mice (DNA-repair deficient 8-oxoguanine glycosylase knockout mice), we observed additive effects of noise on the degree of oxidative burst in blood leukocytes and other oxidative stress and inflammation markers.

To answer the question of whether microvascular endothelial/vascular dysfunction occurs in response to noise and whether there is a connection to inflammation, control mice and mice with genetic deletion of the phagocytic NADPH oxidase catalytic subunit (gp91phox or NOX-2) were exposed to aircraft noise for 4d. In vivo fluorescence microscopy established a higher number of leukocytes adhering to the vasculature in noise-exposed wild-type mice. Microvascular diameter, red blood cell velocity, and segmental blood flow were decreased by noise exposure, indicating microvascular constriction. All adverse effects on functional parameters were normalized or improved in noise-exposed gp91phox$^{-/y}$ mice. Noise exposure also induced substantial endothelial dysfunction in cerebral microvessels, associated with higher oxidative stress burden and inflammation, demonstrating a link between a pro-inflammatory phenotype of plasma, activation of circulating leukocytes, and microvascular dysfunction. Again, the phagocytic NADPH oxidase was identified as a central player in the underlying pathophysiological mechanisms.[104]

No tolerance to cardiovascular side effects of noise

To test whether chronic exposure to aircraft noise results in noise habituation, we exposed animals to noise for up to 28d, revealing a persistent endothelial dysfunction and elevated blood pressure.[105] Additionally, there was a time-dependent increase in formation of ROS, as observed through dihydroethidium (DHE) staining and HPLC-based superoxide measurements in the aorta, heart, and brain. The oxidative burst in whole blood peaked after 4-7d of noise exposure. Increased superoxide in the brain coincided with downregulation of neuronal nitric oxide synthase (Nos3) and the

transcription factor Foxo3 genes. Conversely, Vcam1 mRNA, a marker of inflammation, was upregulated in all noise exposure groups. Endothelial dysfunction and inflammation persisted throughout 28d of aircraft noise exposure. The formation of ROS increased gradually with ongoing exposure, indicating that mice did not habituate to chronic noise stress at moderate levels.[105]

Noise preconditioning and myocardial infarction

A significant clinical question concerns whether the side effects of noise are aggravated in vulnerable patients, e.g., patients with acute coronary syndromes. We addressed this by exposing mice to an average sound pressure level of 72 dB and a peak level of 85 dB for up to 4d, activating pro-inflammatory aortic gene expression related to myeloid cell adhesion and diapedesis pathways.[106] Noise exposure promoted adhesion and infiltration of inflammatory myeloid cells in vascular and cardiac tissues, accompanied by increased percentage of leukocytes exhibiting a pro-inflammatory phenotype, characterized by ROS and upregulation of NADPH oxidase type 2 (Nox2) and phosphorylation of nuclear factor 'kappa light chain enhancer' of activated B-cells (phospho-NFκB) in peripheral blood.[106] This pro-inflammatory phenotypic switch of circulating immune cells and cardiac tissue suggests a preconditioning of the heart for future ischemic heart damage. Subsequently, ligation of the left anterior descending artery was performed to induce MI. This resulted in a decline in cardiac function, substantial infiltration of $CD11b^{+}$ myeloid cells and $Ly6C^{high}$ monocytes into the cardiac tissue, and the induction of IL-6, IL-1β, CCL-2, and Nox2. These effects were intensified when noise exposure had occurred before MI (Figure 5). There was also an increase in mitochondrial $O_2^{\cdot-}$ production due to a reduction in the oxygen consumption rate (OCR). The MI induced more pronounced endothelial dysfunction and increased vascular ROS levels in animals preconditioned with noise.[106] These observations are in accordance with the previous finding that noise-mediated vascular damage was efficiently prevented when pro-inflammatory subsets of myelomonocytic cells were ablated using a genetic mouse model of specific depletion of LysM-positive cells based on LysM-specific overexpression of a diphtheria toxin receptor.[107]

In a translational approach, we prospectively investigated participants in the Gutenberg Health Study Cohort. Among cases with an incident MI during follow-up, we observed that individuals with a history of noise exposure and annoyance

exhibited elevated C-reactive protein levels at baseline and a more substantial decline in left ventricular ejection fraction (LVEF) after the MI.[106] Accordingly, in a prospective study, persons with verified acute coronary syndromes were found particularly vulnerable to effects from aircraft noise.[108] The HR for recurrence of cardiovascular events was 1.24 (95% CI: 0.97–1.58) per 10 dB increase in L_{den} aircraft noise. A combined analysis of recurrence (defined as MI, stroke, bypass surgery, or percutaneous coronary intervention with stent implantation) and all-cause mortality yielded an HR of 1.31 (95% CI: 1.03–1.66). Similar HRs were found for L_{day} and L_{night} aircraft noise exposure. HRs for road traffic and railway noise were above unity but insignificant. In summary, studies on humans and animals indicate that CVD patients are highly vulnerable to noise.

How long does the cardiovascular system need to recover after noise stress?

Following a continuous exposure period lasting 4d, the cessation of noise for either 1, 2, or 4d proved sufficient to completely normalize noise-induced endothelial dysfunction in the aorta (in mice).[109] This improvement was assessed through the measurement of acetylcholine-dependent relaxation. Furthermore, vascular oxidative stress and increased blood pressure exhibited partial correction, and markers of inflammation, including VCAM-1, IL-6, and leukocyte oxidative burst, returned to normal levels within 4d of noise cessation. In contrast, the endothelial dysfunction, oxidative stress, and inflammation observed in the cerebral microvessels of noise-exposed mice showed no improvement. These results emphasize that the recovery process from noise-induced damage is more intricate than anticipated. While large conductance vessel function could be completely restored, persistent endothelial dysfunction in the microcirculation was evident. These findings suggest that, in general, more extended periods of noise cessation are required to reverse noise-induced vascular dysfunction, including the resistance vessels.[109]

Noise-induced side effects can be modified via non-pharmacological and pharmacological activation of the α1AMPK

Nondrug approaches, including maintaining a routine of physical activity, adopting a well-balanced, healthy diet, and managing weight, have proven effective in preventing and treating CVDs and diabetes.[110] Also, regular exercise is considered a mean to mitigate the impact of air pollution-induced CVD and mortality.[111]

We recently explored the potential protective effects of α1AMPK activation through exercise, intermittent fasting and pharmacological activation by AICAR (5-aminoimidazole-4-carboxamide riboside) in a murine model of vascular dysfunction-induced aircraft noise.[112] Mice were subjected to aircraft noise exposure, significantly impairing endothelial function in the aorta, mesenteric arteries, and retinal arterioles. This dysfunction was accompanied by an elevation in vascular oxidative stress and the formation of asymmetric dimethylarginine. Importantly, activation of α1AMPK using all three approaches effectively prevented the onset of endothelial dysfunction and vascular oxidative stress, a conclusion supported by RNA sequencing data (Figure 6). Notably, the absence of endothelium-specific α1AMPK worsened noise-induced vascular damage and nullified the positive effects of exercise or intermittent fasting.[112] These outcomes substantiate that activating endothelium-specific α1AMPK through pharmaceutical stimulation, exercise, and intermittent fasting effectively mitigates noise-induced cardiovascular damage.

The Interplay between Noise Pollution and the Neuroendocrine Axis

When noise exposure occurs during sleep, sleep fragmentation and abbreviated sleep periods may lead to significant life stress. This situation is known to initiate cerebral oxidative stress, primarily driven by heightened angiotensin-II signaling and activation of NOX-2; both have the potential to incite inflammation in the brain's microvasculature.[113] In support, noise-exposed animals exhibit elevated circulating levels of the neurohormone angiotensin-II (Figure 7).[96,114]

In animals, activation of the sympathetic nervous system due to oxidative stress induced by NADPH oxidase is the connecting link between RAAS-mediated NOX-2 activation and subsequent release of catecholamines.[115,116] Conversely, catecholamines can incite oxidative stress in rats by modulating monoamine oxidase activity or activating astrocytes, microglia, and NOX-2.[117] Consistent with the concept of an RAAS-ROS-SNS axis, administering NADPH oxidase inhibitors reduces blood pressure and levels of angiotensin-II and noradrenaline in hypertensive mice.[118] Furthermore, oxidative stress in the heart and vasculature is mitigated through the blockade of the AT1 receptor and inhibition of the angiotensin-converting enzyme.[119,120]

Aircraft noise triggers an increase in the expression of endothelin-1 in the aorta of mice secondary to increased oxidative stress.[121] Endothelin-1 is a potent

vasoconstrictor and activates NOX-2, which is partially dependent on the RAAS.[70,96,122]

These findings put forth a comprehensive molecular and pathophysiological framework that could account for the endothelial dysfunction and hypertension observed in animal models of noise exposure. This framework attributes a central role to NOX-2-induced oxidative stress and inflammation and the disruption of circadian rhythm caused by sleep fragmentation and deprivation. Most notably, these animal-based data strongly support the pivotal role of stress-response pathways in inducing adverse cardiovascular and cerebral effects in humans exposed to noise. They also provide detailed molecular mechanisms that outline the sequence of events within the brain and along the stress-response axis (Figure 7).

Co-Exposure to Noise and Air Pollution

Comparative analyses of the disease burden reveal that air pollution is the foremost environmental contributor to disability-adjusted life years lost (DALYs), whereas environmental noise ranks second.[123] Air and noise pollution often share common sources, including aircrafts, trains, and road vehicles, resulting in simultaneous exposure to noise and air pollution. Research suggests that the EU faces substantial noise and air pollution costs, encompassing excess deaths and diseases, reaching nearly 1 trillion EUR. In comparison, the cost related to alcohol consumption in the EU is estimated at 50-120 billion EUR, and smoking at 544 billion EUR.[124] Several studies on transportation noise have incorporated mutual adjustment for air pollution. A review from 2023 concluded that air pollution did not appear to confound the association between noise and cardiovascular health, strongly indicating that transportation noise and air pollution independently increase the risk of CVD.[125] The review also concluded that more studies on potential interactions between these two exposures were needed.

To investigate this further, we employed an exposure system with an aerosol generator and loudspeakers, subjecting mice to acute exposure for 3d to ambient particulate matter and/or aircraft noise. Both stressors led separately to a significant degree of endothelial dysfunction in arterial conductance and cerebral resistance vessels, increased blood pressure, oxidative stress, and inflammation.[126] An additional impairment of endothelial function was observed in isolated aortic rings and was even more pronounced in cerebral and retinal arterioles. The increase in

oxidative stress and inflammation markers, coupled with RNA sequencing data, pointed to noise primarily affecting the brain and PM affecting the lungs. The combined impact of both stressors exhibited additive adverse effects on the cardiovascular system, likely driven by PM-induced systemic inflammation and noise-induced stress hormone signaling.[126] These studies also revealed an additive upregulation of ACE-2 in the lungs, potentially explaining the increased vulnerability to COVID-19 infection in populations residing in highly air and noise-polluted areas.[127]

These data underscore the need for further mechanistic studies to elucidate the propagation of primary target tissue damage from the lung and brain to remote organs, such as the aorta and heart, resulting from combined noise and PM exposure.[126]

Noise Mitigation Maneuvers

Local authorities can employ various strategies to mitigate the noise from roads, railways, and aircraft. For road traffic, it is important to note that at speeds exceeding 30-35 km/h for cars and 55-65 km/h for heavy vehicles, emitted noise arises primarily from the contact between tires and the road surface. Consequently, the transition from combustion engine cars to electric vehicles will only result in minor reductions in road traffic noise, approximately 1 dB(A). Several established strategies can be employed to reduce road traffic noise. These include noise barriers erected along busy roads in densely populated areas, which will significantly reduce noise levels (up to 10 dB(A)), and noise-reducing asphalt, which can lead to noise reductions ranging from 3 to 6 dB(A). Speed limit adjustments lead to approximately 1 dB(A) reduction per 10 km/h reduction in the speed limit. Developing and promoting low-noise tires can potentially reduce noise levels at a national scale by approximately 2-3 dB(A). Since these individual abatement strategies often result in relatively small noise reductions, combining these approaches may be needed in densely populated areas.

For aircraft noise, strategies to reduce population exposure include the implementation of optimized air traffic routes via GPS guiding to minimize overlap with densely populated areas. Implementing night flight bans, during which take-offs and landings are not allowed, can significantly reduce nighttime aircraft noise. A

continuous descend approach using steeper descents with lower and less variable throttle settings can help to minimize noise during aircraft approaches and landings.

In the context of railway noise, preferred strategies for reducing noise include rail grinding, meaning regular maintenance and grinding of railway tracks to reduce wear and noise. Using brake upgrades means replacing cast-iron block brakes with composite materials that generate less noise during train operations. Nighttime operation bans on railway operations near residential zones are a powerful tool to reduce nighttime noise disturbances.

Conclusions, Political, and Societal Consequences

The comprehensive compilation of preclinical, clinical, and epidemiological evidence strongly reinforces the notion that transportation noise serves as a significant environmental factor contributing to the development of various cardiovascular and cerebrovascular conditions, including chronic coronary artery disease, acute coronary syndrome, arterial hypertension, stroke, and heart failure.

The findings from recent translational studies involving animals and humans support the idea that noise is linked to disruptions in redox balance and vascular function and disturbances in autonomic and metabolic processes. These noise-related effects not only exacerbate the adverse health consequences of traditional cardiovascular risk factors, such as arterial hypertension and diabetes, but also accelerate atherosclerotic processes and increase overall risk of CVD. Nevertheless, it is essential to acknowledge that while preclinical animal studies provide valuable insights, they may only sometimes be entirely applicable to human noise-related health effects due to species-specific differences in hearing range and noise perception. As a result, these findings should be interpreted cautiously.

Noise and air pollution are correlated as they are emitted from common sources, most importantly aircrafts, trains, and road vehicles. The estimated costs of co-exposure to noise and air pollution greatly surpass those associated with alcohol and smoking.[124] Consequently, numerous research gaps warrant attention, including the assessment of the magnitude and time course of responses to co-exposure to noise and air pollution, the exploration of the synergistic effects of both exposures on surrogate measures like blood pressure and diabetes, and the determination of the duration of effects and their potential for reversal. There is also a need for investigating the impact of cardiovascular therapies, such as statins, ACE inhibitors,

AT-1 receptor blockers, and AMP-kinase activators, on noise- and air pollution-induced health effects and their implications for future cardiovascular risk by studying the influence of noise on circadian rhythms and understanding the combined effects of noise in conjunction with lifestyle factors such as diet, stress, and exercise.

Regarding political consequences, the cardiovascular community is responsible for raising awareness of the impact of environmental pollutants. This goes beyond promoting healthy lifestyles and diets; it also involves taking steps to minimize the effects of noise pollution on cardiovascular health. However, recommendations to reduce noise pollution were conspicuously absent from the ESC[110] and the ACC/AHA guidelines for prevention.[128]

Considering the impact of noise on the cardiovascular system, does it still make sense to ask our patients only about traditional cardiovascular risk factors to assess the overall cardiovascular risk? We do not believe so. Rather we must assess the so-called exposome or individual encounters throughout life and how these exposures impact biology and health.[129] It encompasses external and internal factors, including chemical, physical, biological, and social factors that may influence human health. The exposome will help to predict the risk for future CVD more precisely.

Conflict of Interest

None.

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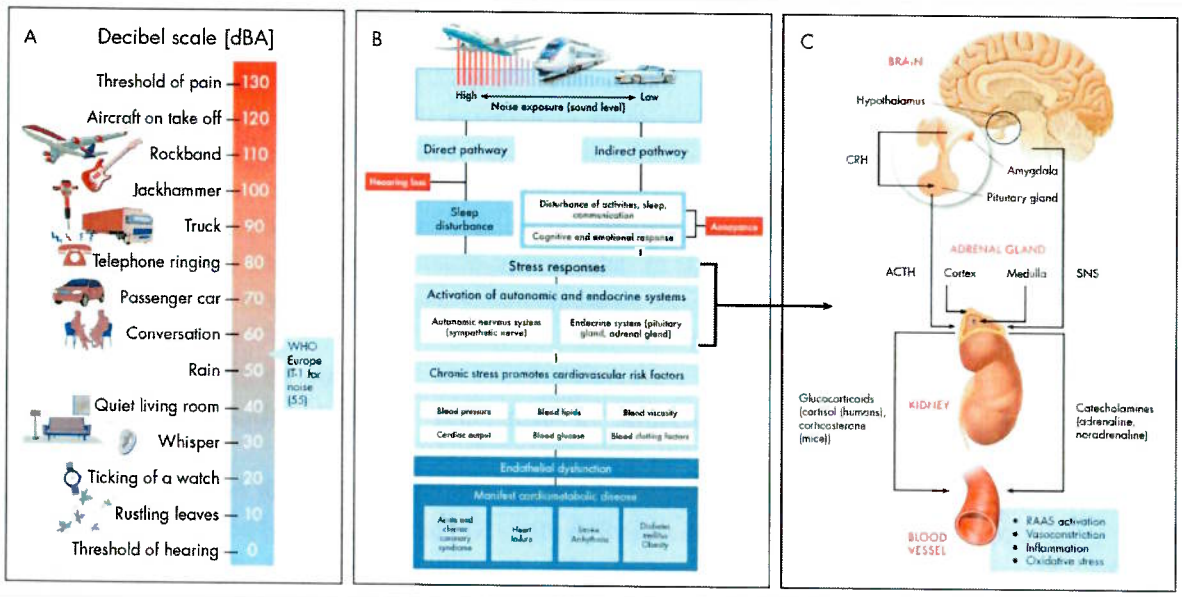
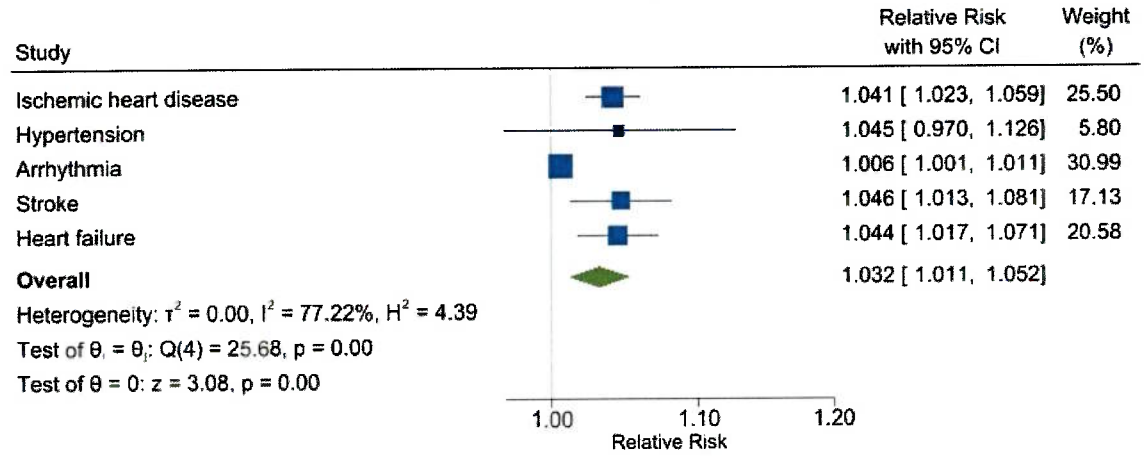
Figure 1



A: Sound pressure levels (SPLs) of different noise sources.[130]
B: Noise reaction model for noise exposure's direct (auditory) and indirect (non-auditory) effects.[131]
C: Neuronal activation (arousals) induced by noise triggers signaling via the hypothalamic–pituitary–adrenal axis and sympathetic nervous system (SNS). This lead to release of corticotropin-releasing hormone from the hypothalamus into the pituitary gland, which stimulates the release of adrenocorticotropic hormone (ACTH) into the blood. ACTH induces the production of glucocorticoids by the adrenal cortex, and the activation of the SNS stimulates the production of catecholamines by the adrenal medulla. The release of glucocorticoids and catecholamines, in turn, leads to the activation of other neurohormones and pathways, such as the renin–angiotensin–aldosterone (RAAS) system, and increased inflammation and oxidative stress. Panel A reprinted from [130] with permission. Copyright © 2018 The Authors. Panel B and C adapted from [11] with permission. Copyright © 2021, Springer Nature Limited.

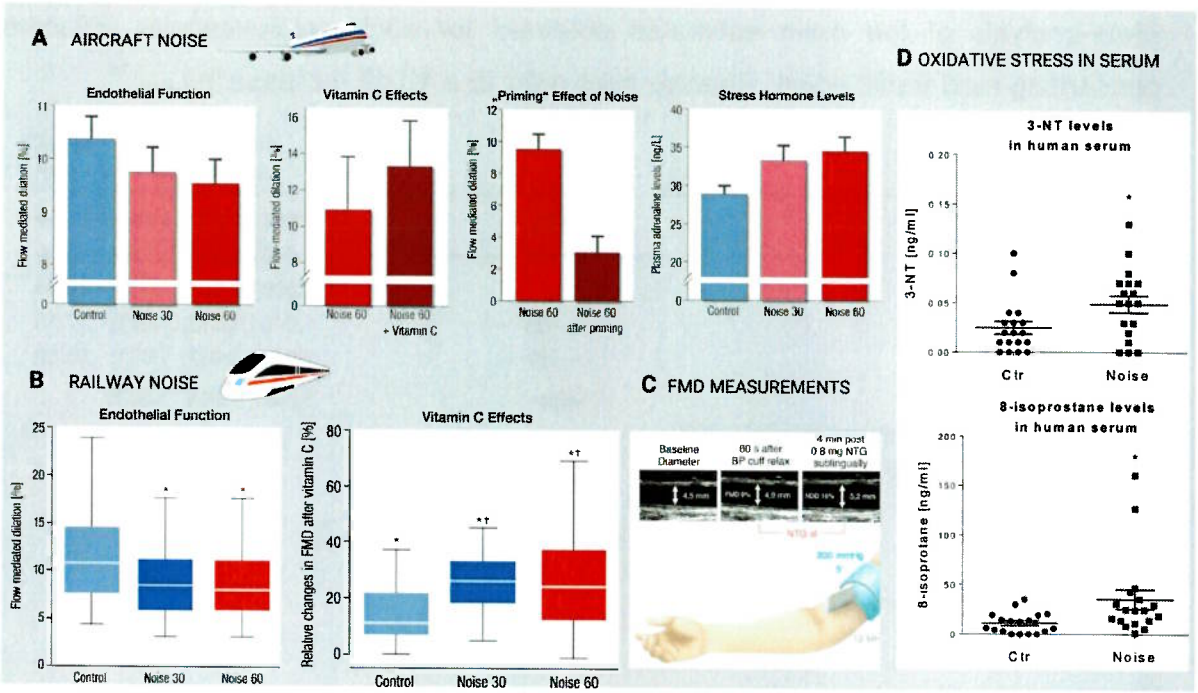
Figure 2

Meta-analysis of the main estimates obtained for each cardiovascular outcome concerning road traffic noise. Relative risks refer to a 10 dB increase in L_{den}.[16]



Random-effects REML model

Figure 3



Key effects of noise observed in human field studies. A: effects of 30 and 60 aircraft noise events on flow-mediated dilation (FMD) of the brachial artery (noise 30 and 60) of 70 healthy subjects. Vitamin C effects were assessed in a subgroup of the cohort. A priming effect of aircraft noise on endothelial function was observed, i.e., previous exposure to Noise30 caused Noise60 to have a significantly stronger reduction of flow-mediated dilation [87]. Serum adrenaline levels also increased significantly. B. Effects of 30 and 60 railway noise events on flow-mediated brachial artery dilation in 70 healthy subjects. Vitamin C effects were assessed in a subgroup. C. Methodology of FMD. D. Effects of aircraft noise on oxidative stress markers (3-nitrotyrosine [3-NT] and 8-isoprostane) in serum that were measured in the samples of the aircraft noise study and published in [70]. Adapted from [70] with permission. Copyright ©2018, Oxford University Press.

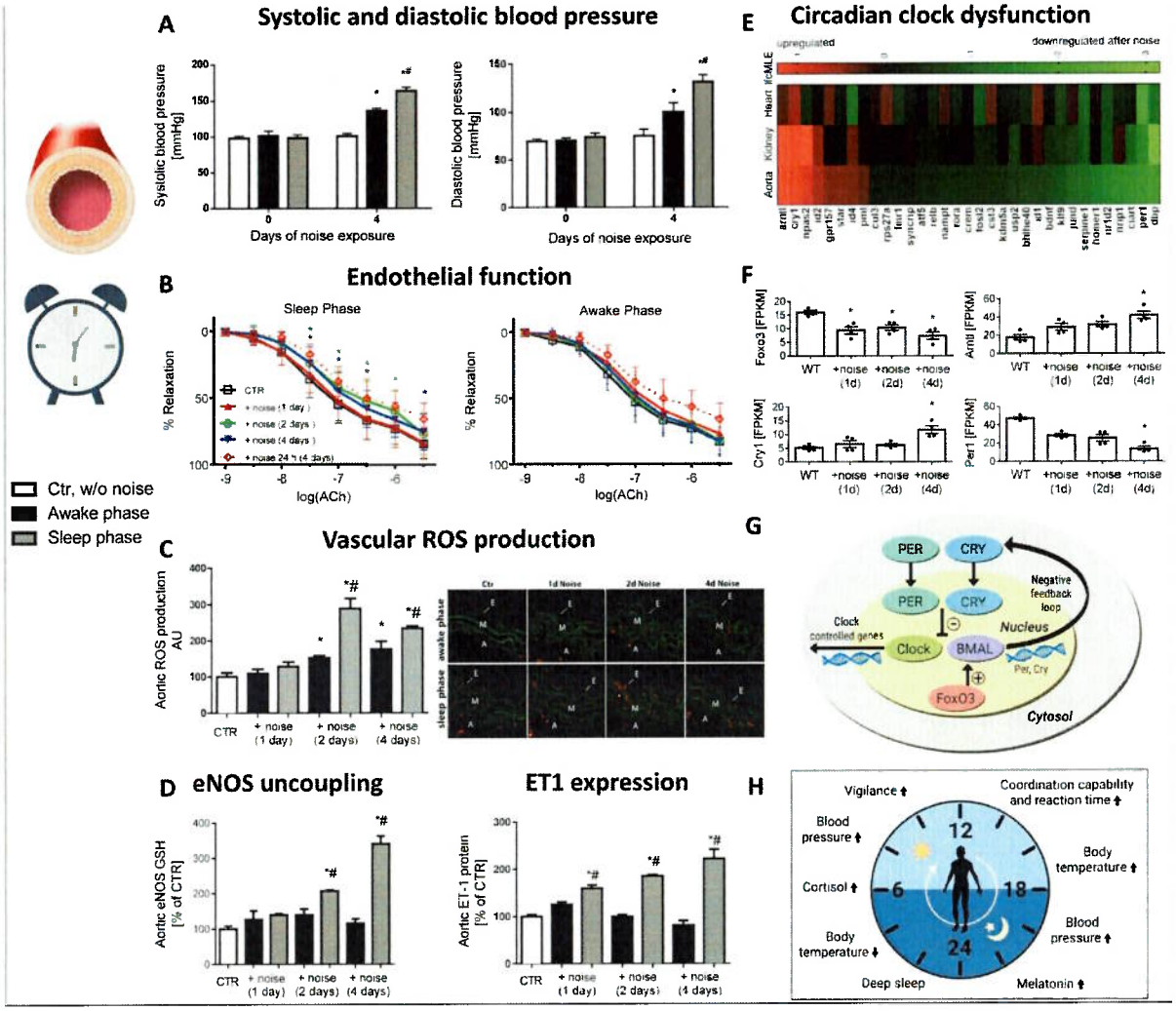
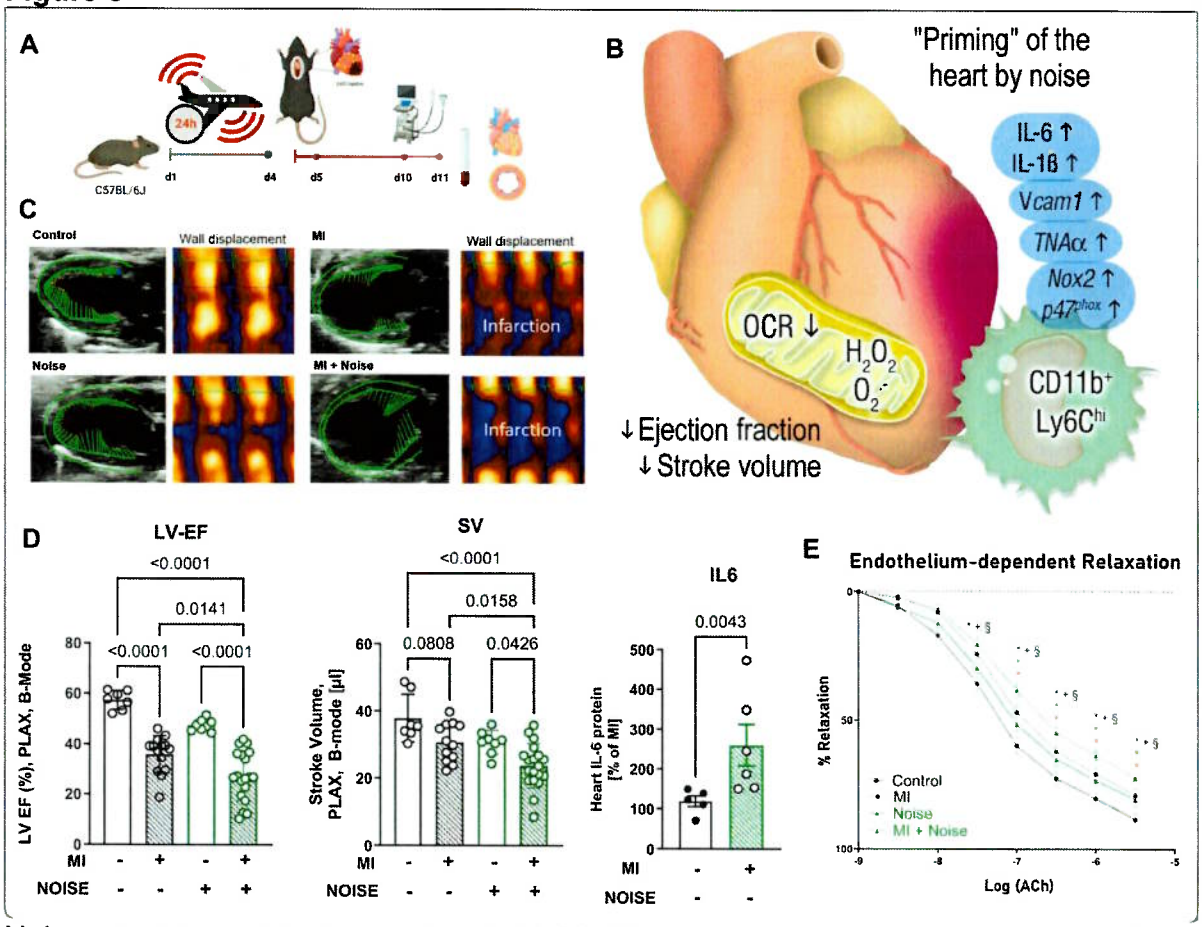


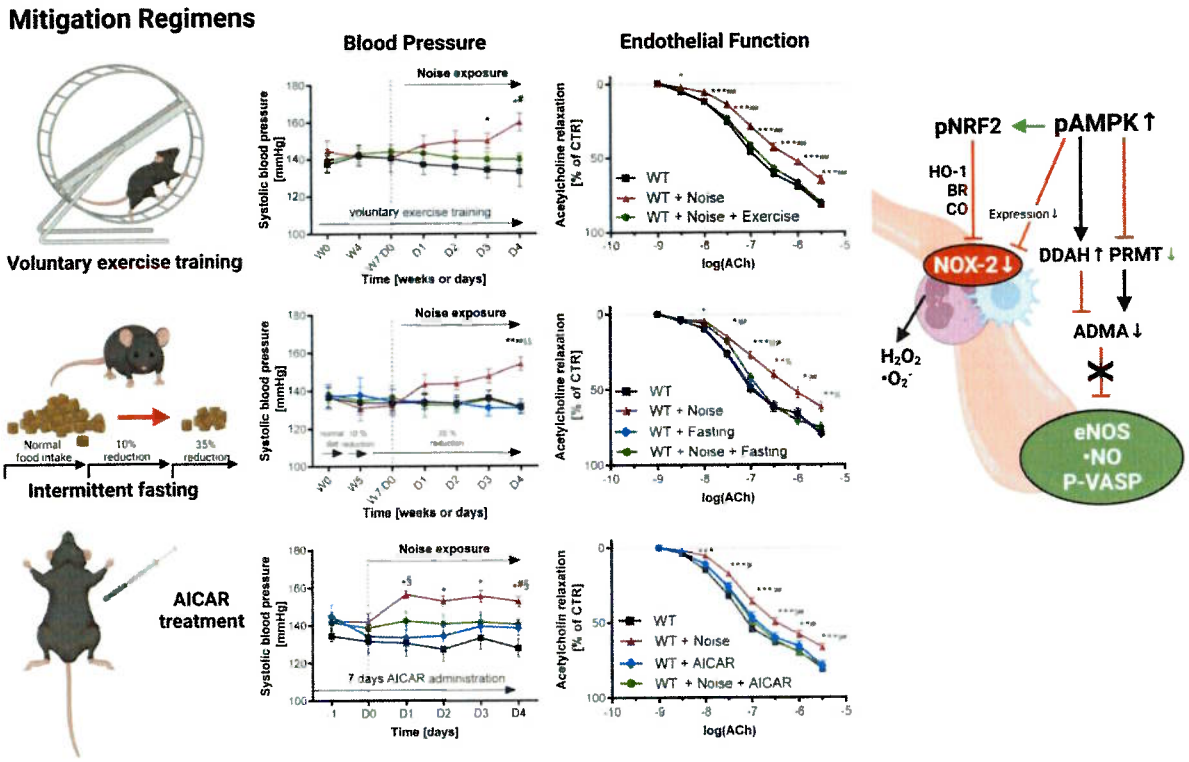
Figure 4 Effects of sleep and awake phase aircraft noise [mean sound pressure level 72 dB(A) for 12 h per day for 1, 2, and 4 days] on the murine vasculature. Sleep phase noise showed a more pronounced increase in systolic and diastolic blood pressure (A) and caused significant endothelial dysfunction (diminished response to acetylcholine [ACh]) (B). For comparison, the impaired endothelium-dependent relaxation in response to 24 h noise exposure is shown in dotted line. Sleep phase noise induced more vascular oxidative stress (red fluorescence staining by oxidized hydroethidium in aortic cryo sections) (C), eNOS uncoupling by immunostaining against S-glutathionylated (= uncoupled) enzyme (eNOS-GSH) and endothelin-1 (ET-1) protein expression by immunohistochemistry (D). Sleep phase noise also caused substantial dysregulation of expression of circadian clock genes in the aorta and kidney as revealed by Illumina RNA sequencing (E). Aortic gene expression of the transcription factor Foxo3 (regulates BMAL1) and period-1 (Per1) were down-regulated. In contrast, brain and muscle aryl hydrocarbon receptor nuclear translocator (Arnt)-like (Bmal) 1 and cryptochrome-1 (Cry1) were up-regulated (F). The clock core components consist of the positive regulators CLOCK and BMAL that directly control circadian gene expression and the negative regulators PER and CRY (G). The circadian clock regulates several essential biological functions (H). Adapted from [70] (A-F) and [68] (G-H) with permission. Copyright ©2018, Oxford University Press and Copyright © 2020 by Annual Reviews.

Figure 5



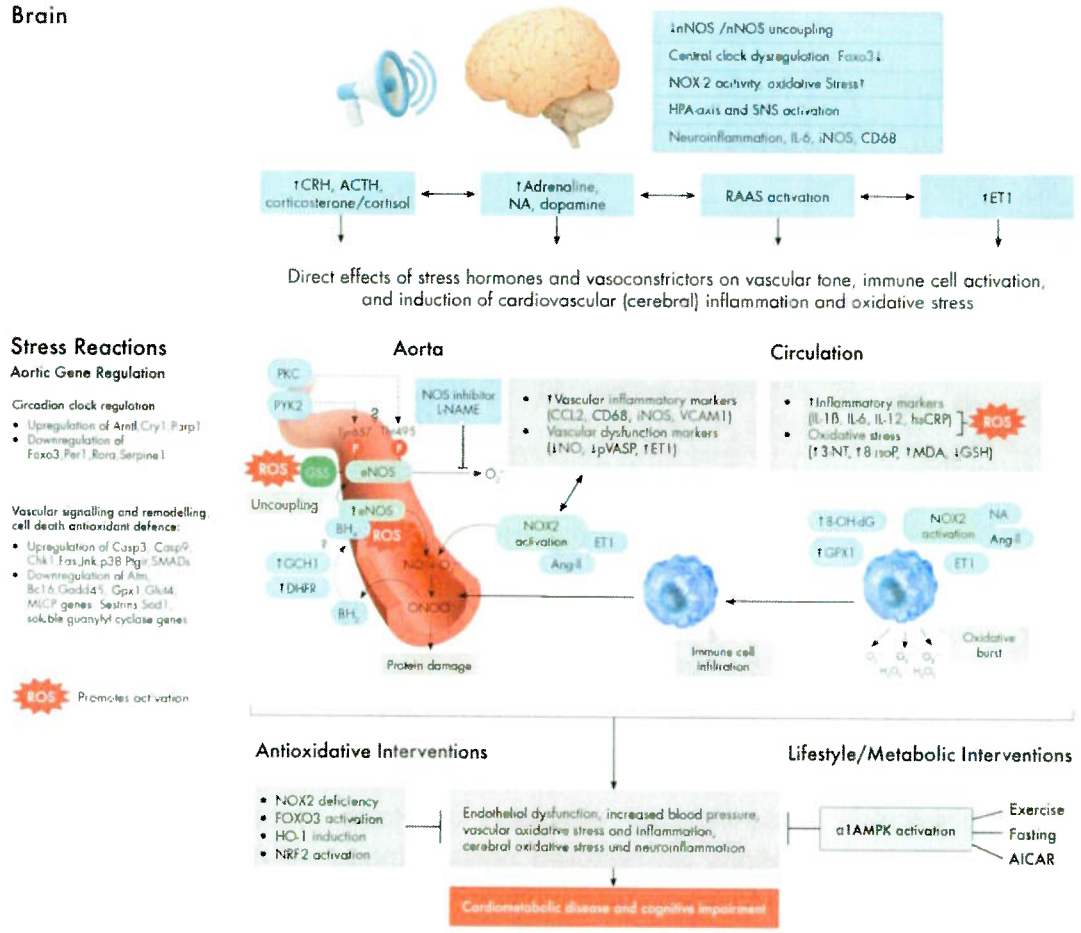
Noise exposure worsens cardiac and endothelial function after acute myocardial infarction (MI). (A) Experimental study scheme with noise exposure before experimental MI by LAD ligation, followed by delayed echocardiography. (B) Noise exposure induces an inflammatory and pro-oxidative phenotype of the heart promoting exacerbation of impaired cardiac function and decreased oxygen consumption rate (OCR) leading to increased mitochondrial production of reactive of reactive oxygen species after MI (C). Representative B-mode images in parasternal long axis (PLAX) and heat map of wall displacement. (D) High-frequency small-animal echocardiography 6d after permanent LAD-ligation or sham operation with or without noise exposure revealed additively decreased left-ventricular ejection fraction (LV-EF in %) and stroke volume (SV in µl). Increased cardiac IL-6 levels support the noise-mediated "priming" of the heart. (E) Noise-induced additive endothelial dysfunction (impaired response to acetylcholine [ACh]) of the aorta after MI. Adapted from [106] with permission. Copyright © 2023 European Society of Cardiology.

Figure 6



Exercise, caloric restriction, and AICAR (5-aminoimidazole-4-carboxamide riboside) treatment prevented noise-induced elevation of blood pressure and endothelial dysfunction. The interventions all restored noise exposure-induced blood pressure elevation to the level of unexposed control. Also, noise-triggered endothelial dysfunction (impaired response to acetylcholine [ACh]) in the aorta was prevented by the three interventions. The proposed protective mechanism of the three mitigation regimens is the activation of α1AMPK leading downstream to an increase of the dimethylarginine dimethylaminohydrolase (DDAH), the degrading enzyme of asymmetric dimethylarginine (ADMA), and a decrease of protein arginine methyltransferase 1 (PRMT1), an enzymatic source of ADMA. This will lead to lower levels of ADMA, the most potent endogenous eNOS inhibitor, and thereby to more efficient nitric oxide signaling, all of which prevents noise-induced endothelial dysfunction. Activation of α1AMPK also prevents noise-triggered NOX-2 expression / activation and oxidative stress, in part by cross-activation of NRF2 via specific phosphorylation and subsequent heme oxygenase-1 (HO-1) induction followed by carbon monoxide (CO) and bilirubin (BR) production. Adapted from [112] with permission. Copyright © 2023 European Society of Cardiology.

Figure 7



Proposed mechanistic pathway between transportation noise and cerebral and cardiovascular disease.

Abbreviations:

HPA, hypothalamic-pituitary–adrenal; SNS, sympathetic nervous system; RAAS, renin–angiotensin–aldosterone system; Ang-II, angiotensin-II; ET1, endothelin-1; NOX2, nicotine adenine dinucleotide phosphate oxidase isoform 2 (phagocytic NADPH oxidase); nNOS, neuronal nitric oxide synthase. FOXO3, forkhead box protein O; iNOS, inducible nitric oxide synthase; eNOS, endothelial nitric oxide synthase; $O_2^{\bullet-}$, superoxide; $ONOO^-$, peroxynitrite; BH_4, tetrahydrobiopterin; GSS-, S-glutathionylation; GSH, glutathione; 8-OH-dG, 8-oxo-2'-deoxyguanine; GPX1, glutathione peroxidase 1; AICAR, 5-aminoimidazole-4-carboxamide riboside; 3-NT, 3-nitrotyrosine; 8-isoP: 8-isoprostane; IL, interleukin; CD68, cluster of differentiation 68 (macrosialin); ACTH: adrenocorticotropic hormone; BH_2, dihydrobiopterin; CCL2, CC-chemokine ligand 2; CRH, corticotropin-releasing hormone; DHFR, dihydrofolate reductase; GCH1, GTP cyclohydrolase 1; HO-1, heme oxygenase-1; NRF2, nuclear factor erythroid 2-related factor 2; α1AMPK, alpha1 subunit of adenosine monophosphate-activated protein kinase; GFAP, glial fibrillary acid protein; H_2O_2, hydrogen peroxide; hsCRP, high-sensitivity C-reactive protein; MDA,

malondialdehyde; NA, noradrenaline; pVASP, phosphorylated vasodilator-stimulated phosphoprotein; L-NAME, N^G-nitro-L-arginine methylester; PKC, protein kinase C; PYK2, protein-tyrosine kinase 2; VCAM1 vascular cell adhesion molecule; ROS, reactive oxygen species. Adapted from [11] with permission. Copyright © 2021, Springer Nature Limited.

Table 1

Estimated number of persons who died and/or had an incident ischemic heart disease (IHD) due to exposure to road traffic noise in Denmark and Switzerland. The lower effect threshold of road traffic noise, corresponding the level below which no health effects of noise is expected, is presently unknown, and therefore numbers are estimated for four "lower harmful level" scenarios. For both Denmark and Switzerland, numbers are estimated using road traffic noise calculated for nationwide epidemiological projects estimating noise from 35 dB and up.[132,133]

Lower effect threshold (Lden)	Denmark (5.75 million)		Switzerland (8.48 million)	
	Cardiovascular mortality[1]	IHD incidence[2]	Cardiovascular mortality[1]	IHD incidence[2]
55 dB	206	256	166	291
53 dB	270	336	234	412
50 dB	385	478	371	651
45 dB	616	764	688	1207

[1] Population Attributable Fraction (PAF) estimated using a relative risk of 1.045 for CVD mortality and proportion of people in different noise categories. Number of persons with CVD mortality calculated as PAF*CVD mortality in Denmark (N=12,455, 2017) and Switzerland (N=19,645, 2021), respectively.

[2] Population Attributable Fraction (PAF) estimated using a relative risk of 1.041 for ischemic heart disease (IHD) and proportion of people in different noise categories. Number of persons with incident IHD calculated as PAF*IHD in Denmark (N=16,984, 2017) and Switzerland (N=37,878, 2021), respectively.

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PERSPECTIVE OPEN

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Noise causes cardiovascular disease: it's time to act

Thomas Münzel[1,2,3]✉, Andreas Daiber[1,2,3], Nicole Engelmann[4,5], Martin Röösli[4,5], Marin Kuntic[1,2] and Jamie L. Banks[6]

BACKGROUND: Chronic transportation noise is an environmental stressor affecting a substantial portion of the population. The World Health Organization (WHO) and various studies have established associations between transportation noise and cardiovascular disease (CVD), such as myocardial infarction, stroke, heart failure, and arrhythmia. The WHO Environmental Noise Guidelines and recent reviews confirm a heightened risk of cardiovascular incidents with increasing transportation noise levels.
OBJECTIVE: We present a narrative review of the evidence from epidemiologic studies and translation studies on the adverse cardiovascular effects of transportation noise.
METHODS: We describe the results of a recent Umbrella+ review that combines the evidence used in the 2018 WHO Environmental Noise Guidelines with more recent (post-2015) high-quality systematic reviews of original studies. High-quality systematic reviews were included based on the quality of literature search, risk of bias assessment, and meta-analysis methodology using AMSTAR 2.
RESULTS: Epidemiologic studies show that exposure to high levels of road traffic noise for several years lead to numerous adverse health outcomes, including premature deaths, ischemic heart disease (IHD), chronic sleep disturbances, and increased annoyance. Mechanistically, noise exposure triggers oxidative stress, inflammation, endothelial dysfunction, and circadian rhythm disruptions. These processes involve the activation of NADPH oxidase, mitochondrial dysfunction, and nitric oxide synthase uncoupling, leading to vascular and cardiac damage. Studies indicate that chronic noise exposure does not result in habituation, and susceptible individuals, such as those with pre-existing CVD, are particularly vulnerable.

Keywords: Epidemiology; Exposure assessment; Health studies; Human well-being; Meta-analysis; Noise pollution

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INTRODUCTION

Extensive research has established the adverse health impacts of environmental exposures contributing to the exposome, specifically air pollution, on cardiovascular disease (CVD), including conditions such as myocardial infarction (MI), heart failure, arrhythmia, hypertension, and stroke [1]. Recent studies have highlighted particulate matter with a diameter of ≤2.5 µm (PM2.5) as a major air pollutant, contributing to ~7.9 million annual deaths [2]. Various studies have observed that proximity to major roads increased cardiovascular health problems such as ischemic heart disease (IHD) or hypertension [3, 4]. These studies cannot clarify whether the observed adverse effects are from air pollution or from noise.

Surprisingly, much less attention has been given to transportation noise despite urban and suburban areas experiencing high levels of both air pollution and noise. Noise, defined as "unwanted and/or harmful sound," comes from transportation, occupational, leisure, residential, and industrial sources. With the present brief review, we want to focus on cardiovascular and metabolic health effects of transportation noise.

TRAFFIC NOISE EXPOSURE AND THE BURDEN OF DISEASE

In 2020, the European Environment Agency (EEA) reported that many people remain exposed to high road traffic noise levels, estimating that at least 20% of the EU population lives in areas where transportation noise exceeds 55 dB L_{den} (reviewed in refs. [5, 6]). The World Health Organization (WHO) indicates adverse health impacts are likely at these noise levels, particularly at night when noise should not exceed 45 dB(A) (Table 1). The U.S. Department of Transportation estimated that in 2018 7.3% of the U.S. population was exposed to road traffic noise levels above 50 dB $L_{aeq,24}$ (corresponding to a L_{den} of ≈53 dB), a number that the authors of the present review and even the U.S. Department of Transportation consider an underestimation of the real exposure levels of the U.S. population. In the EU, environmental noise, mainly from road transportation, is estimated to cause 12,000 premature deaths, 48,000 new cases of IHD, 6.5 million people experiencing chronic sleep disturbances, and 22 million individuals enduring significant annoyance annually. In 2020, it was estimated that 7.8 million, 5.2 million, and 7.9 million people in the U.S. were highly annoyed by aircraft, road, and rail traffic,

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Table 1. WHO recommended limits for noise exposure levels and national legal thresholds for average noise exposure (https://www.euro.who.int/__data/assets/pdf_file/0009/383922/noise-guidelines-exec-sum-eng.pdf)[a].

Noise source	L_{den}	L_{night}	Quality of evidence	EU/US threshold
Road noise	<53 dB	<45 dB	strong	No legally binding limits for ambient noise. Legal limits for $L_{REL,8h}$ of 85–90 dB (US) and L_{EX} of 80–85 dB (EU) for occupational noise sources; peak (impulse) noise limits 135–140 dB.
Railway noise	<54 dB	<44 dB	strong	
Aircraft noise	<45 dB	<40 dB	strong	
Wind turbines	<45 dB	–	limited	
Leisure ambient noise	<70 dB (L_{Aeq},24 h)	–	limited	

[a]dB, decibel; L_{den}, average sound pressure level over 24 h adjusted for day-evening-night with a penalty of 5 dB for the evening time (7–11 pm or 6–10 pm) and a penalty of 10 dB for the night time (11pm–7am or 10pm–6am); L_{night}, average sound pressure level for night time (11pm–7am or 10pm–6am); L_{Aeq}, average sound pressure level over 24 h (A-weighted means adjusted for the human acoustic range). The recommended limits are related to the most seriously exposed face of the building. Strong quality of evidence requires fast action of policy makers, whereas limited quality of evidence requires substantial discussions among the decision makers, also considering the opinion of scientists, clinicians and health care system representatives. $L_{REL,8h}$, recommended exposure level over 8 h at workplace by the US CDC-associated National Institute for Occupational Safety and Health (NIOSH). L_{EX}, recommended exposure level over 24 h or 7 d at workplace by the European agency for occupational safety and health (EU-OSHA). Reused with permission [46].

respectively [7]. Urban expansion and increasing mobility demand are expected to raise the number of individuals exposed to road and railway noise by 2030, while aircraft noise exposure remains unchanged.

TRANSPORTATION NOISE AND CARDIOVASCULAR DISEASE AND DEATH

Recent evidence highlights the impact of environmental noise on cardiovascular health [5, 6]. The WHO Environmental Noise Guidelines for the European Region (WHO ENG) included studies up to 2015 [8]. A recent Umbrella+ review identified subsequent studies, combining the newest high-quality systematic reviews with original studies post-2015 [9]. High-quality systematic reviews were included based on the quality of literature search, risk of bias assessment, and meta-analysis methodology using AMSTAR 2 [10]. Eligible original studies were required to use reliable noise exposure assessment methods and account for relevant confounders. For mortality and incident CVDs, only cohort studies were included, whereas prevalent hypertension studies also considered case-control and cross-sectional studies if they were population-based, large, and methodologically sound [9].

Cardiovascular mortality

The Umbrella+ review identified 61 cardiovascular (ICD-10: I00-I99) and IHD (I20-I25) mortality papers, out of which 12 prospective cohort studies on road, railway, and/or aircraft noise were eligible for meta-analysis. The pooled effect estimate for cardiovascular mortality per 10 dB(A) of road traffic noise was 1.05 (95% CI: 1.02–1.07) [5] based on nine studies (1.05 (95% CI: 1.03–1.08) for ischemic heart disease mortality). Only two studies each were available for railway and aircraft noise, both finding minimal effects on cardiovascular mortality. Figure 1 shows the meta-analysis results. A Swiss case-crossover study found that short-term exposure to aircraft noise was associated with CVD mortality, particularly exposure to nighttime aircraft noise of 40–50 dB(A) and >50 dB(A) within two hours prior to a CVD death [11].

Ischemic heart disease (IHD)

The WHO ENG review found a relative risk (RR) of 1.08 (95% CI: 1.01–1.15) per 10 dB increase in L_{den} for IHD incidence due to road traffic noise [8, 12]. Recent studies, including a pooled Danish and Swedish cohort analysis [13] and a nationwide study from Denmark with over 2.5 million participants [14], reported hazard ratios (HRs) of 1.03 (95% CI: 1.00–1.05) and 1.05 (95% CI: 1.04–1.06) for road traffic noise. A meta-analysis combining WHO ENG [8, 12] with new studies found an RR of 1.04 (95% CI: 1.02–1.06) per 10 dB(A) increase in road traffic noise for IHD incidence [9].

Stroke

The WHO ENG included one cohort study on road traffic noise and incident stroke, finding an HR of 1.14 (95% CI: 1.03–1.25) [8]. Nine subsequent studies mostly indicated positive associations with road traffic noise, with risks near unity for rail and aircraft noise. A pooled Danish and Swedish cohort analysis found an HR of 1.06 (95% CI: 1.03–1.08) per 10 dB(A) increase in road traffic noise [15]. The Umbrella+ review reported an RR of 1.05 (95% CI: 1.01–1.08) per 10 dB(A) increase in road traffic noise for incident stroke [9].

Heart failure

The WHO ENG [8] did not address the effects of noise on heart failure. A 2023 meta-analysis found road traffic noise was associated with a 5% higher risk of heart failure per 10 dB(A) [16]. An updated meta-analysis found an RR of 1.04 (95% CI: 1.02–1.07) per 10 dB(A) increase in road traffic noise for heart failure [9].

Arrhythmia and/or atrial fibrillation

Few reviews and cohort studies exist on noise and arrhythmia and/or atrial fibrillation. A Danish nationwide cohort study with over 3.5 million participants reported weak associations between atrial fibrillation and road, railway, and aircraft noise [17]. An updated meta-analysis found an RR of 1.01 (95% CI: 1.00–1.01) per 10 dB(A) increase in road traffic noise [9]. After the publication of the Umbrella review, a pooled analysis of eleven prospective Nordic cohorts found a RR of 1.02 (95% CI: 1.00–1.04) per 10-dB of 5-year mean time-weighted exposure, which changed to 1.03 (1.01–1.06) when implementing a 53-dB cut-off [18]. It should be mentioned that we did not identify literature on associations of transportation noise with sudden death, out-of-hospital cardiac arrest, or ventricular arrhythmias.

Conclusions

The Umbrella+ review confirms associations between road traffic noise and various CVD diagnosis groups. Combining pooled effect estimates of IHD, stroke, hypertension, arrhythmia and heart failure results in a global CV risk increase of 3.2% (95% CI: 1.1–5.2%) per 10 dB higher road traffic noise (L_{den}) [9]. Evidence is less pronounced for railway and aircraft noise, as road traffic noise is more prevalent, many people exposed to moderate levels of railway and aircraft noise may not hear it because the road traffic noise is substantially higher. As a consequence, road traffic noise is

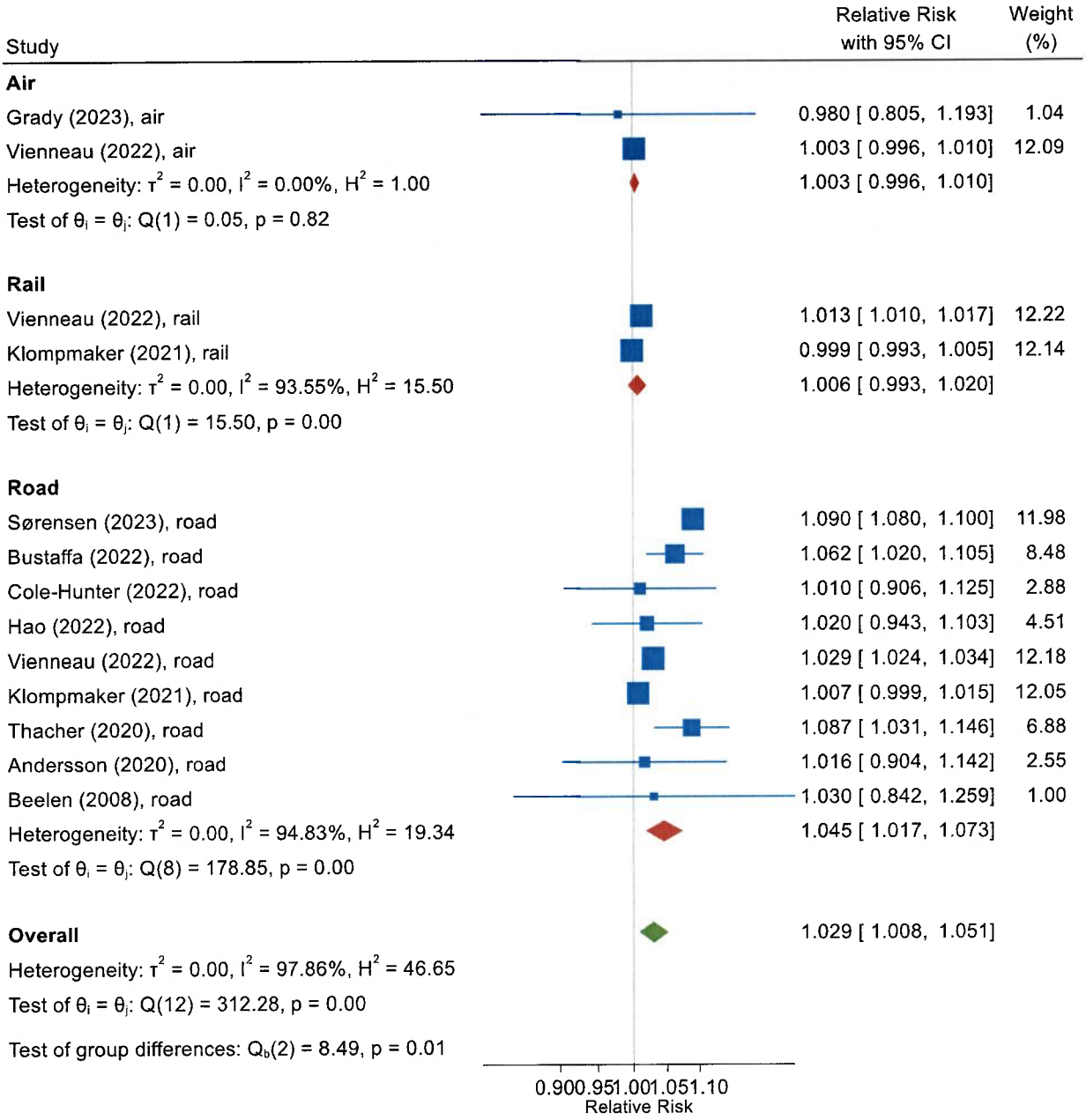

Fig. 1 Meta-analysis of cohort studies on cardiovascular mortality in relation to transportation noise, stratified by source. Relative risks refer to a 10 dB increase in L_{den}. Adapted with permission from [9].

potentially masking the effects of railway and aircraft noise in source-specific analysis. Intervention studies are needed to demonstrate risk reduction after noise mitigation. It would be also important to design studies to include measures of traffic related pollutants (NO_2 and PM2.5) and proximity of residence to roadway as complements to noise.

LOWER EFFECT THRESHOLD OF NOISE

The lower effect threshold of noise, below which no health effects are expected, is undetermined and likely varies by noise source and the different characteristics of noise (e.g., tonality, frequency). Different noise recommendations exist worldwide, with the EU using a 55 dB L_{den} threshold [8, 12] and the WHO recommending 53 dB(A) for road traffic noise [19]. Recent large cohort studies suggest effects starting around 45 dB L_{den} for various cardiovascular diagnoses and diabetes [9].

TRANSPORTATION NOISE AND DIABETES AND OBESITY

Recent cohort studies have linked transportation noise, especially from road traffic, with a higher risk of diabetes (reviewed in ref. [5]). A meta-analysis found that a 10 dB(A) increase in road traffic noise was associated with a relative risk (RR) for diabetes of 1.06 (95% CI: 1.03–1.09). Studies have suggested that noise may affect sleep quality and contribute to metabolic changes leading to diabetes.

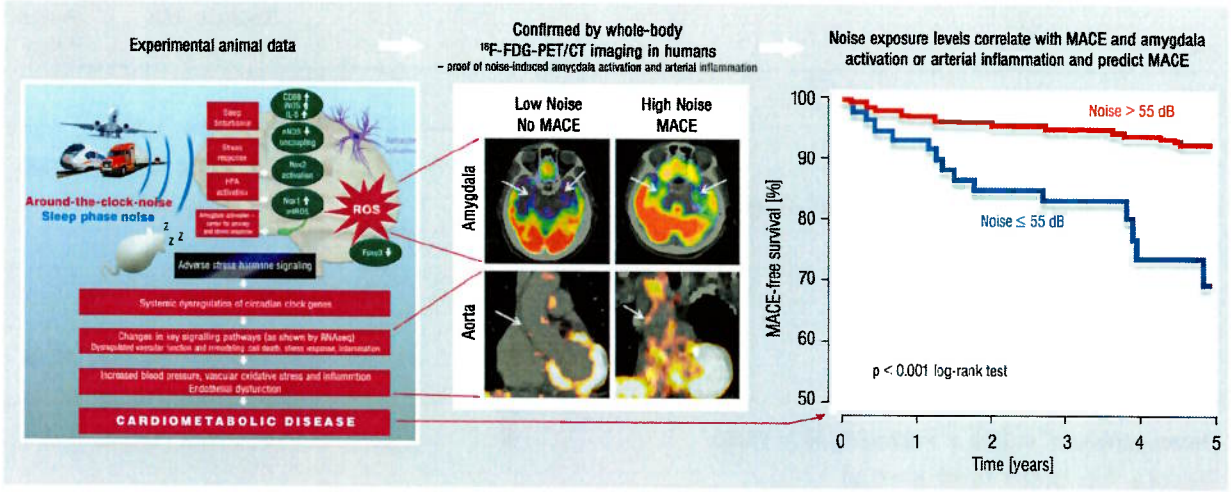

Fig. 2 Key data on health effects of noise through the brain-heart/vessel-axis. *Left panel:* Main results of animal studies regarding brain-heart/vessel interaction. *Middle and right panel:* Proof-of-concept translational study in humans demonstrating the association between transport (road and aircraft) noise-induced cerebral (amygdala relative to cortical) metabolic activity and arterial inflammation increasing major adverse cardiovascular events (MACE) [29, 30]. Reused with permission from ref. [30].

Research also indicates that noise exposure may contribute to obesity (reviewed in ref. [5]). Several studies found associations between road traffic noise and increased measures of adiposity, suggesting that noise can affect weight gain throughout life.

NOISE AND EPIGENETIC CHANGES: ADVERSE IMPACTS ON THE IMMUNE SYSTEM AND VASCULAR FUNCTION

Cross-sectional cohort studies have found that exposure to transportation noise can have an impact on the immune system. Two studies observed that noise increases levels of IL-12 and high-sensitivity CRP (C-reactive protein) while decreasing natural killer cell populations and activity, although the extent of noise effects on the immune system is not consistently uniform across all studies [20–22]. Furthermore, alterations in the immune system have been linked to elevated circulating cortisol levels and heightened noise sensitivity [21, 22]. Higher cortisol levels may also be related to nocturnal noise exposure and impaired circadian rhythm [23, 24].

Interestingly, a study based on the Swiss SAPALDIA cohort showed that long-term exposure to transportation noise and air pollution led to distinct and shared DNA methylation patterns, with enrichments in pathways related to inflammation (e.g., CRP), cellular development, and immune responses [25]. Findings in the same cohort suggested that chronic exposure to nocturnal intermittent train or road traffic noise increases arterial stiffness (reflecting endothelial dysfunction), as determined by pulse wave velocity [26]. This finding is supported by a German cohort study, showing that long-term exposure to nighttime road traffic noise is associated with subclinical atherosclerosis, especially in participants with early arterial calcification [27, 28].

In summary, these findings offer pathophysiological and molecular evidence from human studies, highlighting the effects of transportation noise on incident CVD. Notably, the results from these human studies, including stress pathways, inflammation, oxidative stress, arterial stiffness, and endothelial/cardiac dysfunction, align with mechanistic data from animal studies (reviewed in refs. [5, 6]).

NOISE ANNOYANCE

Noise annoyance, a psychological response to unwanted sounds, can be an early indicator of more severe health risks. It involves cognitive, emotional, and behavioral reactions influenced by personal, social, and situational factors. Recent studies have shown that transportation noise, particularly from roads and aircraft, is associated with increased annoyance levels, contributing to stress and negatively impacting cardiovascular health (reviewed in refs. [5, 6]). Annoyance from noise can lead to increased stress hormone levels and inflammation, further contributing to cardiovascular risk.

NOISE AND AMYGDALAR ACTIVATION

The link between noise exposure and major adverse cardiovascular events (MACE) was observed in a 2020 study where stress-associated neural activity was associated with arterial inflammation in 498 healthy subjects without active cancer or CVD [29]. The neural activity was determined as the ratio of amygdala to regulatory cortical metabolic activity envisaged by the ^{18}F-fluorodeoxyglucose positron emission tomography-computed tomography (PET–CT) imaging. At the same time, aortic inflammation was also determined using PET-CT to observe ^{18}F-fluorodeoxyglucose uptake. The results indicated that the increased noise exposure level at the individual's home address was linked to elevated amygdala activity, arterial inflammation, and higher risk of MACE, independently of confounders such as air pollution, socioeconomic status, and other established CVD risk factors. The study's authors conclude that the association between higher noise exposure and MACE occurred via elevated amygdala activity and arterial inflammation (Fig. 2) [29, 30]. Interestingly, a similar pathway was previously observed to be responsible for the association between perceived stress and socioeconomic disparities (e.g., lower education or income) and CVD [31].

MECHANISTIC INSIGHTS: TRANSLATIONAL STUDIES IN HUMANS AND ANIMALS

Oxidative stress and endothelial dysfunction

Translational studies in humans with and without CVD have demonstrated that exposure to transportation noise for one night (47 dB(A) L_{eq}) leads to a significant increase in oxidative stress markers such as 3-nitrotyrosine and 8-isoprostane in serum and a significant degree of endothelial dysfunction as indicated by a reduction of flow-mediated dilation (FMD) (Fig. 3) (reviewed in refs. [5, 6]). Importantly, the deterioration of FMD was stronger in

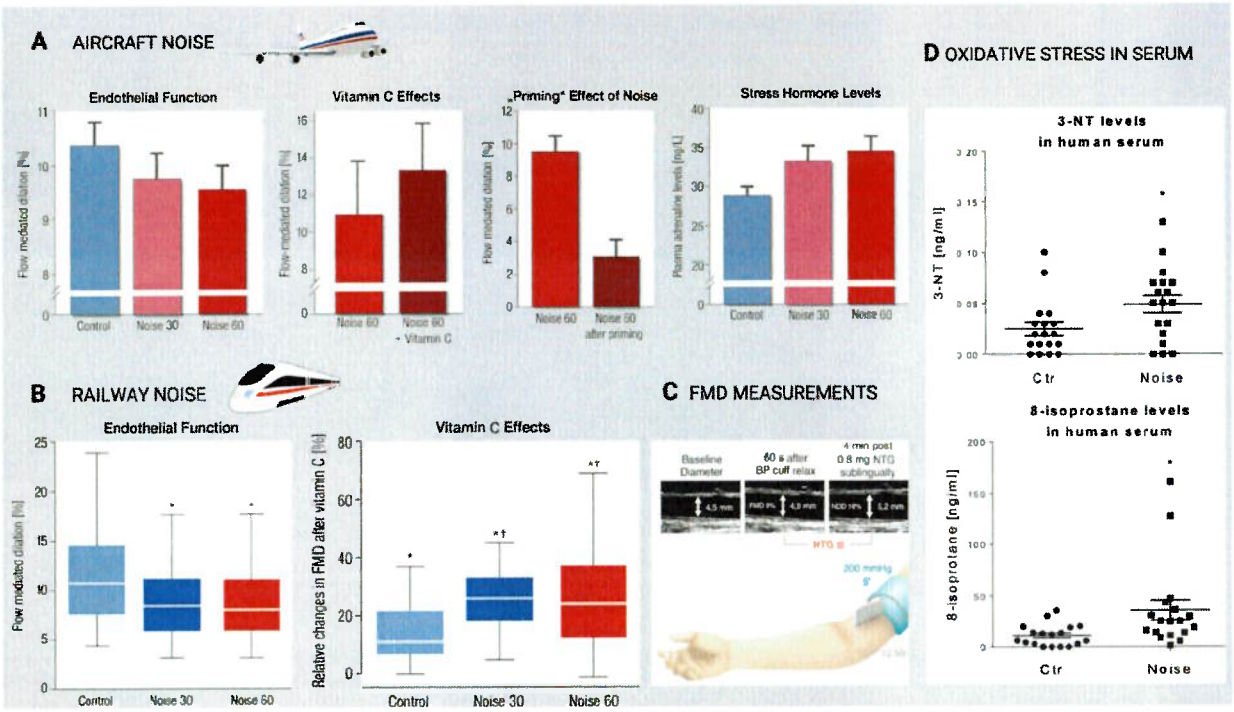

Fig. 3 Key effects of noise observed in human field studies. A Effects of 30 and 60 aircraft noise events on the brachial artery (Noise30 and Noise60) of 70 healthy subjects. Vitamin C effects were assessed in a subgroup of the cohort. A priming effect of aircraft noise on endothelial function was observed, i.e., previous exposure to Noise30 caused Noise60 to have a significantly stronger reduction of flow-mediated dilation [52]. Serum adrenaline levels also increased significantly. **B** Effects of 30 and 60 railway noise events on flow-mediated brachial artery dilation in 70 healthy subjects. Vitamin C effects were assessed in a subgroup. **C** Methodology of FMD. **D** Effects of aircraft noise on oxidative stress markers (3-nitrotyrosine [3-NT] and 8-isoprostane) in serum that were measured in the samples of the aircraft noise study and published in ref. [32]. Adapted from [32] with permission. Copyright ©2018, Oxford University Press.

subjects with already established CVD. The acute administration of the antioxidant vitamin C has been shown to improve endothelial function, indicating the roll of oxidative stress in noise-induced vascular damage.

Preclinical studies revealed that oxidative stress in noise-exposed mice (72 dB(A) L_{eq}, around-the-clock for 4 days) is primarily driven by the activation of NADPH oxidase (NOX-2), a key enzyme in inflammatory cells like leukocytes and macrophages that produces reactive oxygen species (ROS) but is also driven by a dysfunctional, uncoupled endothelial nitric oxide synthase [32]. Noise exposure also activates inflammatory pathways. It triggers the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system, leading to the release of stress hormones like cortisol and catecholamines (Fig. 4) (reviewed in refs. [5, 6]). These hormones induce a pro-inflammatory state characterized by elevated levels of interleukins (IL-6, IL-1β) and proinflammatory monocytes. This inflammation can lead to vascular changes that contribute to the progression of atherosclerosis and other cardiovascular conditions.

Recently we also demonstrated that noise exposure can dysregulate gene networks within the vasculature. This includes the upregulation of genes involved in TGF-beta signaling, autophagy, and growth regulation and downregulation of genes associated with cell cycle control and apoptosis [33]. These changes in gene expression further impair endothelial and vascular signaling, contributing to cardiovascular dysfunction.

An additional finding from our preclinical studies is that nighttime noise exposure has a more detrimental effect than daytime noise. We demonstrated that nighttime noise, as opposed to daytime noise, led to significantly higher blood pressure, a greater increase in neurohormonal release, elevated oxidative stress in vascular tissue, increased endothelin-1 expression within the vasculature and interestingly no endothelial dysfunction at all. These factors may explain, at least in part, why nighttime noise contributes to greater vascular stiffness and higher blood pressure compared to daytime noise (for review, see [34]). Moreover, we observed circadian clock dysregulation, primarily involving the downregulation of FOXO3, a transcription factor serving as a central signaling hub. We tested the effect of bepridil, a FOXO3 activator, calcium antagonist, anti-anginal, and class IV anti-arrhythmic drug. Bepridil prevented noise-induced endothelial dysfunction, increased FOXO3 mRNA expression, and reduced vascular and cerebral oxidative stress [32]. Based on these findings, we hypothesized that the adverse effects of nighttime noise are partly due to circadian rhythm disruption, as noise during sleep causes sleep fragmentation and reduced sleep quality, thereby amplifying stress responses leading to more pronounced oxidative stress and endothelial dysfunction [32–34]

This disruption affects central and peripheral circadian clocks, contributing to metabolic and cardiovascular dysfunction. Nighttime noise exposure causes also a significant downregulation and uncoupling of neuronal nitric oxide synthase (nNOS), leading to a neuroinflammatory phenotype [32] that probably affects cognitive functions and increases cardiovascular risk.

Noise exposure affects the neuroendocrine system by elevating levels of angiotensin II and endothelin-1, hormones that regulate blood pressure and fluid balance (Fig. 4). This elevation increases oxidative stress and inflammation in the brain's microvasculature and conductance vessels, contributing to hypertension and other cardiovascular issues (reviewed in refs. [5, 6]). The sympathetic nervous system activation due to oxidative stress further releases catecholamines, which can exacerbate cardiovascular damage. The following sections briefly explain how stress hormone-mediated receptor signaling can activate sources of ROS

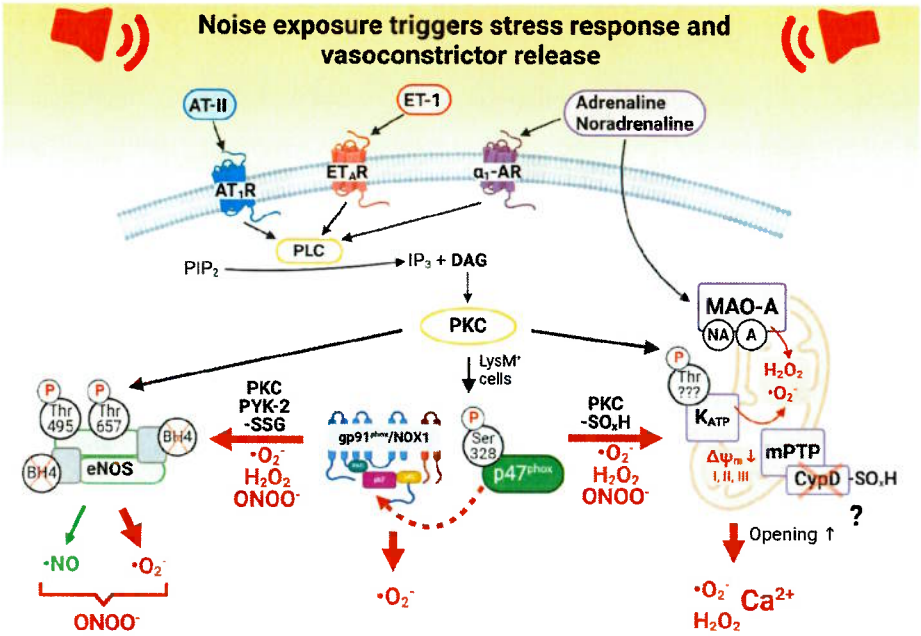

Fig. 4 Oxidative stress pathways activated by noise. Noise causes stress hormone release (catecholamines and cortisol) and downstream endocrinal activation of vasoconstrictors activating common disease pathways, such as oxidative stress. Angiotensin II (AT-II) and endothelin-1 (ET-1) lead to the formation of diacylglycerol (DAG) from phosphatidylinositol 4,5-bisphosphate (PIP_2), a potent activator of protein kinase C (PKC), via their receptors and the activation of phospholipase C (PLC). (1) PKC via phosphorylation of $p47^{phox}$ at serine 328 causes activation of the phagocytic NADPH oxidase (NOX-2) and potentially NOX-1. The expression of NOX-2 is upregulated by noise-triggered immune cell infiltration (lysozyme M-positive ($LysM^+$) cells) and systemic inflammatory conditions. NOX-2 (and NOX-1, especially in the brain) produces superoxide ($O_2^{\bullet-}$) and via dismutation also hydrogen peroxide (H_2O_2). NOX-4 was not changed by noise and NOX-5 (relevant for humans) was not studied so far. (2) Dysfunction of endothelial nitric oxide synthase (eNOS) is mediated by noise-dependent activation of PKC and phosphorylation of threonine 495. Alternatively, NOX-2-dependent ROS formation may activate PKC [53] and protein tyrosine kinase 2 (PYK-2) [54, 55], causing adverse phosphorylation at tyrosine 657 and threonine 495. Uncoupling of eNOS may be induced by noise-driven oxidative depletion of tetrahydrobiopterin (BH_4) and S-glutathionylation (-SSG) of eNOS by ROS originating from NOX-2 [56]. Semi-uncoupled eNOS may represent a potent source of peroxynitrite. (3) Noise also leads to mitochondrial ROS formation, generating both $O_2^{\bullet-}$ and H_2O_2. Noradrenaline (NA) and adrenaline (A) originating from sympathetic activation are substrates of monoamine oxidases (MAO) that produce H_2O_2. NA and A can also activate PKC through adrenergic receptor (α_1-AR). PKC seems to activate the mitochondrial K_{ATP} channel by phosphorylation of a threonine residue with subsequent depolarization of the mitochondrial membrane ($\Delta\Psi_m\downarrow$) and $O_2^{\bullet-}$ formation from respiratory complexes I, II and III. Mitochondrial H_2O_2 / $O_2^{\bullet-}$ and calcium are released to the cytosol upon the mitochondrial permeability transition pore (mPTP) opening (e.g., by thiol oxidation of the regulatory subunit cyclophilin D (CypD) [57]). K_{ATP} channel activation and mPTP opening can also be stimulated by redox-crosstalk with H_2O_2 (probably also $O_2^{\bullet-}$ via peroxynitrite) derived from NOX-2 [58]. So far, there is no evidence for the role of xanthine oxidase in noise's non-auditory (indirect) effects. This scheme was adapted from [35] with permission and created using biorender.com.

(superoxide, $O_2^{\bullet-}$ and hydrogen peroxide, H_2O_2) and lead to oxidative stress conditions (reviewed in ref. [35]).

NADPH oxidases

NOX-2 (gp91phox), the phagocyte isoform of NADPH oxidases, is a key enzyme in host defense. Whereas other NADPH oxidase isoforms (e.g., NOX-1, NOX-4, NOX-3, and DUOX-2) play a role in noise-induced hearing loss, the role of NADPH oxidases in the non-auditory (indirect) pathology is less explored. Upon noise exposure, NOX-2 protein and NOX-2 mRNA levels are consistently upregulated in the murine aorta and heart [32, 33]. Also, a more pronounced activation state of NOX-2 was reported for noise-exposed mice, which was driven by angiotensin-II or endothelin-1 dependent diacylglycerol-mediated protein kinase C (PKC) activation with subsequent Ser328 phosphorylation of p47phox, the cytosolic regulator, and activation of NOX-2 (Fig. 4). Evidence of oxidative stress is readily detectable in the aorta, heart, and brains of mice exposed to noise [32, 33] and the serum of noise-exposed healthy subjects. Importantly, mice with a genetic deletion of NOX-2 gene or ablation of inflammatory monocytes ($LysM^+$ cells) are protected from this oxidative stress and the subsequent endothelial dysfunction [32, 36]. Further support for a central role of NOX-2 in noise-mediated pathophysiology comes from studies showing an additive upregulation of NOX-2 protein in noise-exposed hypertensive and MI mice (reviewed in ref. [5]).

Mitochondria

Mitochondria are well-known producers of ROS and are known to contribute to oxidative damage in IHD and hypertension (reviewed in ref. [35]). Different noise sources and patterns were reported to cause mitochondrial damage in the form of cardiac fibrosis, enlarged cardiac mitochondria, swelling, matrix dilution, cristolysis, DNA damage and reduced connexin 43 contents (reviewed in ref. [5]). These observations can be linked to high noradrenaline levels, monoamine oxidase (MAO) activity, disturbed mitophagy, potentially negatively impacting permeability transition (e.g., mPTP), and calcium handling. Catecholamines (or serotonin) serve as MAO substrates enabling significant ROS formation. Accordingly, an additive increase in mitochondrial superoxide levels was seen in the hearts of noise-exposed mice with MI in conjunction with impaired mitochondrial respiration and oxygen handling [37]. Pathways that could be involved in noise-dependent mitochondrial ROS formation are shown in Fig. 4.

Uncoupled nitric oxide synthases

Due to the excessive superoxide formation in noise-exposed animals, endothelial NOS (eNOS) in the aorta (and neuronal - nNOS in the brain) uncouples, which means that it transforms into a source of $O_2^{\bullet-}$ and H_2O_2 instead of proper synthesis of nitric oxide ($^{\bullet}NO$). NOS uncoupling was previously demonstrated in tissues of noise-exposed mice by dihydroethidium staining in the presence of the eNOS inhibitor N^G-nitro-L-arginine methyl ester (L-NAME) [32, 33]. eNOS is redox-sensitive because of its reliance on a readily oxidizable cofactor, tetrahydrobiopterin (BH_4). Without BH_4, eNOS cannot produce $^{\bullet}NO$, but instead produces $O_2^{\bullet-}$. The concomitant formation of $^{\bullet}NO$ and $O_2^{\bullet-}$ by uncoupled eNOS generates peroxynitrite, which in term reacts with proteins to result in their tyrosine nitration as observed in noise-exposed mice and humans [32, 33]. eNOS uncoupling diminishes $^{\bullet}NO$ bioavailability in the aortas of noise-exposed mice as determined by the direct quantification of $^{\bullet}NO$ using electron spin resonance spectroscopy. Adverse dysregulated phosphorylation by excessive ROS could further aggravate eNOS dysfunction. Another redox-dependent uncoupling mechanism is eNOS S-glutathionylation, which was also increased in the aorta and heart of noise-exposed mice [32, 33]. The latter effect was not observed in NOX-2–deficient mice and was aggravated in noise-exposed hypertensive mouse hearts. The noise-triggered adverse regulation of eNOS that switches the enzyme to a peroxynitrite and superoxide source is shown in Fig. 4.

LACK OF TOLERANCE DEVELOPMENT TO CARDIOVASCULAR HEALTH EFFECTS OF NOISE

Chronic exposure of mice to aircraft noise for 4 weeks does not result in habituation concerning the cardiovascular side effects. Persistent endothelial dysfunction and elevated blood pressure were observed in studies exposing animals to noise for up to 28 days [38]. The formation of ROS increased over time, particularly in the aorta, heart, and brain. This oxidative stress was marked by a peak oxidative burst in whole blood after 4–7 days. Additionally, increased superoxide in the brain was associated with the downregulation of neuronal nitric oxide synthase (NOS3) and FOXO3 genes. Inflammatory markers like VCAM-1 mRNA were consistently upregulated, indicating that mice did not acclimate to chronic noise stress, and endothelial dysfunction and inflammation persisted throughout the exposure period.

NOISE PRECONDITIONING AND MYOCARDIAL INFARCTION

The impact of noise on susceptible patients, such as those with acute coronary syndromes [39], was also studied on a mechanistic basis by exposing mice to 72 dB(A) noise levels with peaks at 85 dB(A) for up to 4 days [37]. This exposure activated pro-inflammatory gene expression related to myeloid cell adhesion and diapedesis pathways. Noise exposure led to increased adhesion and infiltration of inflammatory myeloid cells in vascular and cardiac tissues, and a higher percentage of leukocytes showed a pro-inflammatory phenotype characterized by ROS and upregulation of NOX-2 and NF-κB phosphorylation. This resulted in "priming" of the heart for ischemic damage. Subsequent MI caused more pronounced endothelial dysfunction and elevated vascular ROS levels in noise-preconditioned animals (reviewed in ref. [5]).

Translational studies in the Gutenberg Health Study Cohort found that individuals with prior noise exposure and annoyance had elevated baseline CRP levels and a more significant decline in left ventricular ejection fraction after an MI [37]. People with acute coronary syndromes were particularly susceptible to aircraft noise, with a hazard ratio (HR) of 1.24 (95%-CI: 0.97–1.58) per 10 dB increase in L_{den} aircraft noise [39]. Combined analysis showed an HR of 1.31 (95%-CI: 1.03–1.66) for recurrence of cardiovascular events and all-cause mortality, indicating high susceptibility of CVD patients to noise.

RECOVERY TIME FOR CARDIOVASCULAR SYSTEM AFTER NOISE STRESS

Recovery from noise-induced endothelial dysfunction in the aorta was possible within 1–4 days of noise cessation in mice [40]. Acetylcholine-dependent relaxation measurements confirmed this recovery. Partial correction of vascular oxidative stress and blood pressure and normalization of inflammatory markers such as VCAM-1 and IL-6 were observed. However, endothelial dysfunction and inflammation in cerebral microvessels did not improve, suggesting that microcirculation requires longer recovery to reverse noise-induced vascular dysfunction (reviewed in ref. [5]).

MODIFYING NOISE-INDUCED HEALTH EFFECTS THROUGH α1AMPK ACTIVATION

Non-pharmacological approaches like physical activity, a balanced diet, and weight management are effective in preventing and treating CVD and diabetes [41]. It is known that exercise can mitigate the impact of air pollution-induced CVD and mortality. Activation of α1AMPK through exercise, intermittent fasting, and pharmacological methods (e.g., AICAR) was explored in mice exposed to aircraft noise [42]. Noise exposure-impaired endothelial function in the aorta, mesenteric arteries, and retinal arterioles was accompanied by increased vascular oxidative stress and asymmetric dimethylarginine formation. α1AMPK activation effectively prevented endothelial dysfunction and oxidative stress, supported by RNA sequencing data. Absence of endothelium-specific α1AMPK worsened noise-induced vascular damage, nullifying the protective effects of exercise or fasting, highlighting the importance of α1AMPK activation in mitigating noise-induced cardiovascular damage (reviewed in ref. [5]).

PREVENTION AND MITIGATION STRATEGIES TO REDUCE TRANSPORTATION NOISE

Transportation noise is a public health problem affecting large swaths of the global population. The onus is on policymakers and other decision makers to take action to protect the public from the harms of environmental noise. Particular attention should be paid to populations exposed to the highest levels of noise which, at least in the United States, tend to affect low income communities disproportionately [43, 44], as well as other vulnerable populations, such as those with pre-existing CVD. Professional societies, e.g., American Heart Association, American College of Cardiology, European Society of Cardiology, should incorporate environmental noise into CVD prevention guidelines and educational materials. Policymakers should incorporate environmental noise criteria into program and policy screening tools and enact measures to mitigate existing sources of harmful environmental noise. The US EPA has provided a cumulative risk assessment framework that could accommodate noise (https://www.epa.gov/risk/framework-cumulative-riskassessment). By applying the cumulative risk framework provided by the US EPA (Environmental Protection Agency) [45], a more holistic approach to policy development and mitigation is potentially achieved.

The mitigation measures proposed below were reviewed extensively in refs. [5, 46]. Local authorities can employ several strategies to mitigate road, railways, and aircraft noise as outlined in a policy brief of the European Commission [47]. Priority measures are at the source. Special noise-reducing asphalt can decrease noise by 3 to 6 dB(A). Reducing speed limits can decrease noise by ~1 dB(A) per 10 km/h reduction. Low speed (20 miles per hour) in combination with electrification of cars,

reduces noise in urban areas to a large extent. At higher speed, electrification is less effective as the sound generated by the interaction of tires and pavement is the dominating noise source. Promoting the use of low-noise tires can potentially reduce noise by 2–3 dB(A). Investing in urban infrastructure such as bike lanes, ride-sharing programs, and public transportation can also help to reduce urban noise levels as well as air pollution levels. As an ultimate measure, sound proof windows reduce indoor noise substantially and for road and railway noise, erecting barriers along busy lines in densely populated areas can reduce noise levels by up to 10 dB(A).

To address aircraft noise, implementing GPS-guided routes can help avoid densely populated areas, thus reducing noise impact. Prohibiting take-offs and landings during nighttime hours can significantly reduce sleep disturbances. Continuous descent approaches with steeper descents, and lower throttle settings can minimize noise during landings. Furthermore, promoting the development and use of quieter aircraft technology can have a long-term impact on reducing noise pollution from aviation.

For railway noise, regular maintenance and grinding of tracks can help reduce noise generated by train operations. Replacing traditional cast-iron block brakes with composite materials can lower noise levels during braking. Prohibiting railway operations near residential areas during nighttime can help reduce disturbances. Investing in vibration-damping track systems and sound barriers along railway lines can also mitigate noise pollution from trains.

By combining these strategies, authorities can substantially reduce noise, particularly in densely populated areas. A comprehensive approach that includes technological advancements, infrastructure improvements, and policy changes will most effectively address transportation noise pollution and help to improve public health.

INTERACTION BETWEEN TRANSPORTATION NOISE AND AIR POLLUTION

Noise and air pollution often co-occur, having a common source in fossil fuel-powered vehicles, equipment, and machinery. Their combined exposure can adversely affect cardiovascular health, leading to cumulative risk increases in humans as reported for diabetes [48], stroke [49], and MI [50]. Animal studies using combined exposure systems revealed that both stressors independently cause endothelial dysfunction and oxidative stress, and their combination leads to more severe cardiovascular damage [51]. This interaction underscores the importance of considering multiple environmental factors in assessing cardiovascular risk.

CONCLUSIONS

Transportation noise significantly impacts cardiovascular health through mechanisms involving oxidative stress, inflammation, endothelial dysfunction, gene dysregulation, circadian rhythm disruption, metabolic changes, and psychological stress. Translational studies in humans and experimental animals have provided valuable insights into these processes, emphasizing the need for comprehensive strategies to mitigate noise pollution and its health impacts. Based on the existing evidence concerning noise, it's time now for immediate policy actions to decrease exposure to noise. While this is an action that is supported by the evidence, translation of science into policy and then the implementation of such policies through regulation, rules or guidance takes a long time. Thus we have to consider tactical approaches that can be implemented in the short-term together with health professionals, who can advise their patients to reduce exposure. Thus, we are recommending both a longer-term strategic approach as a solution and a shorter-term tactical approach.

Noise should be acknowledged as a significant cardiovascular risk factor along with other environmental hazards such as ambient air pollution and exposure to chemicals, e.g., in the CVD prevention guidelines of professional societies. Public officials and decision makers should act to reduce public exposure to harmful levels of noise and adhere to the national limits as well as WHO recommendations (Table 1). Further studies are needed to explore the interactions between noise and other environmental stressors and effective public protection interventions.

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AUTHOR CONTRIBUTIONS

TM and JLB contributed to the conception of the article. TM conducted the literature review and prepared the draft manuscript. AD, TM, MR, and MK contributed to the research content. JLB provided technical support and policy insight. All authors provided writing support and interpretation of results, and reviewed the manuscript.

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The authors declare no competing interests.

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ADDITIONAL INFORMATION

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The effects of aircraft noise on sleep quality, sleepiness and annoyance in individuals sleeping during daytime vs. nighttime

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EXTENDED ABSTRACT

INTRODUCTION

Nighttime work is a vital factor for the functioning of modern societies [1]. Around 15 % of the population in Germany work night shifts, requiring them to sleep during the day [2]. Night workers often work between 23:00 and 07:00 h [3] and consequently have to sleep at daytime. Due to the circadian misalignment associated with shift work sleep is more fragile [4].

As ample evidence shows, environmental noise, including aircraft noise, can disturb sleep by increasing sleep fragmentation and reducing sleep depth [5]. Moreover, with increasing noise exposure, self-reported sleep quality decreases [5] and annoyance increases [6]. However, evidence exists mostly for the healthy population, whereas less is known about noise effects in vulnerable groups such as shift workers, the elderly and individuals with an illness who may be more susceptible to the adverse effects of noise [7].

Up to now, the effect of noise on daytime sleep has scarcely been investigated. Research on the specific effect of aircraft noise on daytime sleep is lacking completely. The present laboratory study examined the effect of aircraft noise exposure during both nighttime sleep and daytime sleep, with the latter serving as model for intrinsically more fragile sleep. To this end we quantified objective and subjective sleep quality, sleepiness as well as short-term annoyance.

MATERIALS AND METHODS

In the present study, 33 participants (aged 18-40 years; 18 females) slept in the laboratory during two visits that included two consecutive sleep episodes, each. We assigned participants randomly to either a daytime sleep group ("day sleepers") or a nighttime sleep group ("night sleepers"). The two laboratory visits, during one of which participants were exposed to aircraft

noise during both sleep episodes, were separated by a recovery break of at least seven days. During the sleep episodes with aircraft noise exposure, a noise scenario composed by 81 aircraft fly-over sounds from eight different aircraft types was played back in the bedrooms. The resulting energy-equivalent sound pressure level across the time in bed was L_{eq} = 46.8 dB(A) representing a common indoor aircraft noise exposure near Cologne/Bonn Airport, which is a German hub with a 24/7 operation scheme. For the purpose of a typical aircraft noise scenario, we recorded aircraft sounds in an apartment with tilted windows located six km away from the airport and directly under the flight path.

We measured sleep via polysomnography and derived the following sleep parameters: Sleep efficiency, number of awakenings, time spent in sleep stages 1, 2 and slow wave sleep as well as REM sleep. Sleep quality was also assessed via self-reports using a six-item questionnaire.

After getting up, participants rated their acute sleepiness using the Karolinska Sleepiness Scale [8] and their annoyance due to aircraft noise using an adapted version of the 5-point verbal ICBEN scale [9]. Sleepiness assessments were repeated after 10 to 11 hours awake as well as shortly before the next sleep episode.

The effect of aircraft noise exposure on the selected criterion variables were analyzed separately in the daytime sleep and the nighttime sleep group using linear mixed models. We included noise exposure, sleep episode, and an interaction between noise exposure and sleep episode as predictors plus a random intercept for the participants.

RESULTS

Results showed significant effects of aircraft noise exposure on subjective sleep quality and annoyance reported after getting up in both day and night sleepers. Sleepiness averaged across assessment times (after getting up, after 10/11 hours awake, prior to the next sleep episode) was significantly increased after sleep under aircraft noise exposure in day sleepers, but not in night sleepers.

An effect of aircraft noise exposure on sleep efficiency was found neither for night sleepers nor day sleepers. Time spent in sleep stages 1 and 2 was higher under noise exposure in day sleepers, but not in night sleepers. Likewise, the number of awakenings was increased during noise exposure in day sleepers, but not in night sleepers. Noise exposure reduced the time spent in REM sleep and slow wave sleep on a trend level ($p < 0.1$) during noise exposure in day sleepers, but not in night sleepers.

Besides, in day sleepers, an interaction between the noise exposure and the sleep episode was found such that slow wave sleep was reduced in the first but not in the subsequent sleep episode with noise exposure. On a trend level, an interaction between noise exposure and the sleep episode was found for the number of awakenings in day sleepers. Whilst the number of awakenings was higher in the first sleep episode with noise exposure, it was not in the second one. Similarly, we found an interaction for noise annoyance and subjective sleep quality in day sleepers. Annoyance was increased and self-rated sleep quality was decreased in the first noise-exposed sleep episode whilst noise exposure showed no effect in the subsequent sleep episode.

DISCUSSION AND CONCLUSION

The present findings indicate a stronger effect of aircraft noise exposure on participants sleeping during daytime than on participants sleeping during nighttime. The data suggest that

intrinsically more fragile sleep of night workers may be more vulnerable to the effects of transportation noise.

The significant interactions between noise exposure and the sleep episode may indicate that a compensation takes place between the first and the second noise-exposed sleep episode in day sleepers. The loss in slow wave sleep and the higher number of awakenings seem to have activated homeostatic compensatory mechanisms that prevented effects of noise exposure to become manifest during and immediately after the second sleep episode. Lower annoyance scores and higher subjective sleep quality given after the second sleep episode may reflect the assumed compensatory response.

Since the current study investigated the effects of aircraft noise exposure on people who are required to sleep during daytime for the first time, a discussion of the results in the light of previous findings is hardly feasible. However, the current findings warrant further examination of the effect of transportation noise on sleep in night workers, preferably across a longer time span.

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CLINICAL RESEARCH

Effect of nighttime aircraft noise exposure on endothelial function and stress hormone release in healthy adults

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Aims Aircraft noise disturbs sleep, and long-term exposure has been shown to be associated with increases in the prevalence of hypertension and an overall increased risk for myocardial infarction. The exact mechanisms responsible for these cardiovascular effects remain unclear.

Methods and results We performed a blinded field study in 75 healthy volunteers (mean age 26 years), who were exposed at home, in random order, to one control pattern (no noise) and two different noise scenarios [30 or 60 aircraft noise events per night with an average maximum sound pressure level (SPL) of 60 dB(A)] for one night each. We performed polygraphy during each study night. Noise caused a worsening in sleep quality ($P < 0.0001$). Noise60, corresponding to equivalent continuous SPLs of 46.3 dB (Leq) and representing environmental noise levels associated with increased cardiovascular events, caused a blunting in FMD ($P = 0.016$). As well, although a direct comparison among the FMD values in the noise groups (control: 10.4 ± 3.8%; Noise30: 9.7 ± 4.1%; Noise60: 9.5 ± 4.3%, $P = 0.052$) did not reach significance, a monotone dose-dependent effect of noise level on FMD was shown ($P = 0.020$). Finally, there was a priming effect of noise, i.e. the blunting in FMD was particularly evident when subjects were exposed first to 30 and then to 60 noise events ($P = 0.006$). Noise-induced endothelial dysfunction (ED) was reversed by the administration of Vitamin C ($P = 0.0171$). Morning adrenaline concentration increased from 28.3 ± 10.9 to 33.2 ± 16.6 and 34.1 ± 19.3 ng/L ($P = 0.0099$). Pulse transit time, reflecting arterial stiffness, was also shorter after exposure to noise ($P = 0.003$).

Conclusion In healthy adults, acute nighttime aircraft noise exposure dose-dependently impairs endothelial function and stimulates adrenaline release. Noise-induced ED may be in part due to increased production in reactive oxygen species and may thus be one mechanism contributing to the observed association of chronic noise exposure with cardiovascular disease.

Keywords Endothelial function • Aircraft noise • Cardiovascular risk

Introduction

The WHO estimates that in high-income Western European countries (population ~340 million) at least 1 million healthy life years are lost every year due to environmental noise.[1] The negative health outcomes of noise include annoyance,[2] sleep disturbance,[3] cardiovascular disease,[4,5] and impairment of cognitive performance in children.[6]

Aircraft noise has been shown to be more annoying than road- and railway noise at the same equivalent noise level.[7] Epidemiologic studies have demonstrated associations between long-term

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exposure to aircraft noise and an increased incidence of arterial hypertension and therefore cardiovascular disease.[7,8] The mechanisms underlying these adverse cardiovascular effects of aircraft noise are not fully understood. Nocturnal noise exposure seems to be more relevant for the genesis of cardiovascular disease than daytime noise exposure,[9] probably due to repeated autonomic arousals that have been shown to habituate to a lesser degree to noise than, e.g. cortical arousals.[10] In general, the risk increases with exposure duration, and is higher in those who decide to sleep with open windows.[11,12]

Undisturbed sleep of sufficient length is obligatory for the maintenance of daytime performance and health.[13] The human organism recognizes, evaluates, and reacts to environmental sounds even while asleep.[14] These reactions are part of an integral activation process of the organism that expresses itself, e.g. as changes in sleep structure or increases in blood pressure and heart rate.[10,15] Environmental noise may decrease the restorative power of sleep by means of repeatedly occurring activations (so-called *sleep fragmentation*) that are associated with more awakenings/arousals, less deep sleep and rapid eye movement sleep, and early awakenings in the morning. Although healthy subjects have been shown to habituate to aircraft noise exposure to a certain degree,[10] the habituation is not complete, and noise-induced awakenings and, especially, activations of the autonomic nervous system can still be observed in subjects that have been exposed to aircraft noise for several years.[16] Sleep disturbance and especially sleep restriction in turn have been shown to cause hormonal and metabolic changes,[17–19] which could predispose to a future development of cardiovascular disease.

Circadian changes related to altered sleep may also adversely affect the immune system[20,21] and may increase the responsiveness of the heart to hypertrophic stimuli.[22] Although plausible, the link between polysomnographic evidence of sleep disturbance during aircraft noise exposure and cardiovascular outcomes is not well established. It is largely unknown which changes or indices predict long-term risk.[23]

Furthermore, polysomnography (i.e. the simultaneous measurement of the electroencephalogram, electrooculogram, and electromyogram) is a complex and cumbersome method, which is not very well suited for larger studies in the general population.[24] Therefore, other methods, like actigraphy (a non-invasive technique to monitor human rest/activity cycles) and behaviourally confirmed awakenings, have been used in this context.

In the case of aircraft noise, hypertension may be a consequence of the noise-induced release of stress hormones such as epi- and norepinephrine and/or the development of vascular (endothelial) dysfunction. Endothelial dysfunction (ED) is considered an early step in the development of atherosclerotic changes of the vasculature (for review see[25]) and can be assessed non-invasively. Recent studies indicate that in patients with coronary artery disease and hypertension, ED assessment in the forearm may have prognostic implications.[25]

Based on these considerations, the primary aim of the present study was to test whether nocturnal exposure to aircraft noise may induce ED. The morning plasma level of adrenaline was a secondary endpoint. In a subgroup of noise 60 subjects, we also tested whether acute vitamin C challenges may improve ED.

Methods

The study was approved by the ethics committee of University Medical Center Mainz. All participants were volunteers and signed informed consent. Anti-aircraft noise activists were excluded from the study as were persons with high nighttime traffic noise exposure at home as determined by noise maps available from municipal online resources ($L_{A,eq,22\text{-}6h} > 40$ dB for aircraft noise and $L_{A,eq,22\text{-}6h} > 45$ dB for road and rail traffic noise).

Study population

The study enrolled 75 healthy non-smokers between 20 and 60 years of age. Before the study, audiometry was performed in all participants. Persons with an age-adjusted hearing loss of 20 dB or more on one or both ears were excluded from the study. Subjects with sleep disorders [score >10 on the Pittsburgh Sleep Quality Index (PSQI)][26] or psychiatric disorders (assessed by M.I.NI. Screen interview) were also ineligible. Study participants were instructed to refrain from consumption of coffee, tea, alcohol, sleep altering medications, and nicotine on the day prior to the study night. Otherwise, they were told to continue their usual diet and daily routines. Hormonal contraception was allowed but care was taken to synchronize study nights with the hormonal status. Other hormonal therapies were excluded.

Study procedures

After inclusion, participants returned to the laboratory for three visits. During the night preceding each visit, subjects were exposed in a randomized order to one of three noise patterns. One night served as the control night, and subjects were exposed to normal background noise. During the other two nights, subjects were exposed to recording reproducing different numbers of flights: Noise30 with playback of 30 aircraft noise events, and Noise60 with playback of 60 aircraft noise events. Study visits were prescheduled with at least three non-study nights between two study nights and on the same weekday if possible. In premenopausal women, the visits were scheduled to occur in the same phase of the hormonal cycle. Supplemental vitamins, alcohol, and caffeine containing beverages were prohibited on the evening and night before the study.

Participants were randomly given one of six different sequences of noise and control nights according to the randomization plan (C-30-60, C-60-30, 30-C-60, 30-60-C, 60-C-30, 60-30-C). At study onset, subjects and investigators were both blinded to the noise pattern sequence. Participants slept in their usual home environment and were asked to maintain their usual sleep–wake rhythm. They wore portable polygraphic screening devices (SOMNOwatch™ plus, SOMNOmedics, Randersacker, Germany) during the night with continuous recording of ECG, SpO_2, actimetry, light, and derived parameters as described in previous studies.[27–29]

In the noise exposure nights, the same aircraft noise event was played back repeatedly. It was originally recorded in the bedroom of a resident living in the vicinity of Düsseldorf airport (window tilted open), and was already used in previous studies on the effects of aircraft noise on sleep.[30,31] Noise patterns were recorded as MP3 files and played back on a standard portable audio system with a fixed speaker position relative to the head of the subject. The playback volume was levelled at each measurement site to guarantee similar SPLs at all study sites. During the night, the SPL was continuously recorded in the bedroom with class-2 sound level meters (Datalogger DL-160S, Voltcraft, Germany; Model 407764A Datalogger, Extech Instruments, USA) to assure subject compliance. They were placed on the nightstand close to the participants. All sound files were coded with a study number and were of equal length and file size, making inadvertent unblinding less likely. All noise patterns started with a constant tone of 30 s duration to allow

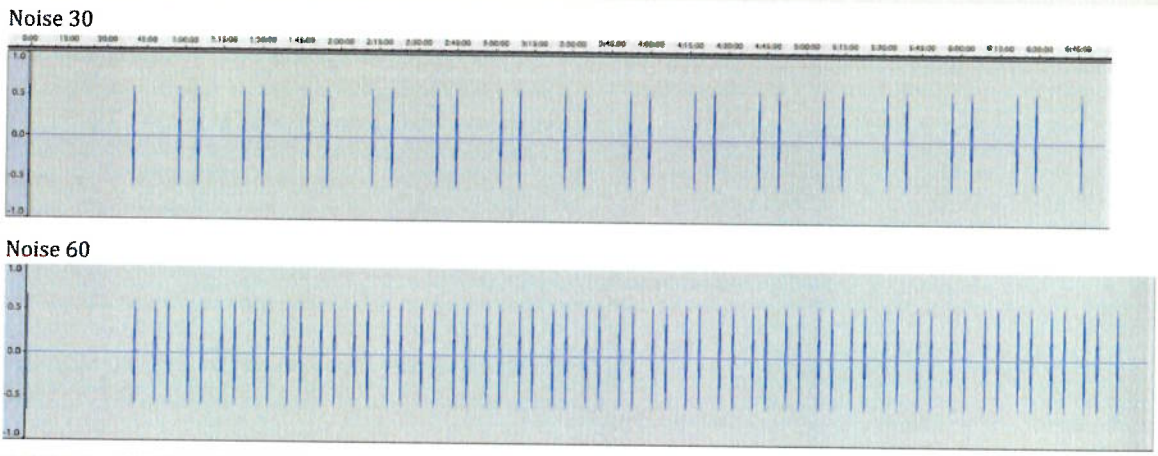

Figure 1 Schematic representation of the noise events.

testing of equipment function. The first aircraft noise event was played back after 39.5 min to facilitate sleep onset. The last aircraft noise event was played back after 415 min. Each noise event lasted roughly 45 s. Noise events followed a short–long–short pattern with time between events roughly 6:40 min and 16:40 min for Noise30 and 4:05 min and 6:40 min for Noise60 (*Figure 1*).

After the study night, participants returned to the study centre in a fasting state for further testing. Flow-mediated dilatation of the brachial artery was measured at the same time in the early morning and before 10 a.m. by a technician using standardized techniques described previously.[25,32,33] Briefly, brachial artery diameter is measured with a linear ultrasound probe at rest and after a 5 min occlusion period with a pressure cuff. Changes in diameter are given in percent and reflect the endothelial release of vasodilatory substances such as nitric oxide (NO). To address the role of reactive oxygen species in causing ED, FMD was also measured in a subset of five subjects exposed to Noise60 before and after administration of vitamin C (2 g, p.o.) as previously described.[34] After FMD measurement, blood samples were drawn and questionnaires were filled out. Blood samples were transported directly to a clinical laboratory for evaluation. Part of the blood was centrifuged, aliquoted, and frozen at below −62 °C for later testing. Global noise sensitivity was measured using the Dortmund Noise Sensitivity Questionnaire.[35] The Horne-Ostberg Morningness-Eveningness Questionnaire (MEQ)[36] was used to assess individual chronotype. Pulse transit time (PTT, time between the R wave in the ECG and peak oxygen saturation measured at the tip of the first finger of the right hand) and heart rate accelerations (number of accelerations >20 bpm and >2 s per h) were calculated. Interleukin-6 and cortisol were measured in serum with chemiluminescence immunoassay. Adrenaline was measured from NH4-heparine anticoagulated blood drawn 30 min after puncture and cooled during transport to the lab.

Statistical analysis

The primary endpoint of the study was the change in %FMD induced by the different levels of noise. Secondary outcomes included the changes in all variables measured (neurohormones, PTT, inflammatory markers, etc), the existence of a relationship between dose of noise and blunting of FMD (dose–effect relationship), and whether Noise30 or Noise60 had a priming effect on the blunting in FMD induced by, respectively, Noise60 or Noise30. A separate study was conducted to test the effect of Vitamin C on FMD in subjects exposed to Noise60. Data are presented as mean ± standard deviation. The Kolmogorov–Smirnov test was used to assess whether the data were normally distributed. To address the primary endpoint, we first compared the effect of Noise60, which reproduces the increase in night noise previously shown to be associated with an increased incidence of cardiovascular events and prevalence of hypertension,[9] with the control visit. Further, a multi-factor ANOVA [taking into account noise exposure, night of exposure, and subject id (for subject-related differences)] was performed. A test for a monotone effect of the exposure (dose of nighttime aircraft noise: 0, 30, or 60) was performed by using exposure as a pseudo-continuous factor in the ANOVA. Further, a (*post hoc*) multi-factor ANOVA was performed with two additional factors: one for the comparison of FMD values after Noise60 in all subjects allocated to control–Noise30–Noise60 or Noise30–Noise60–control to FMD values of all other patients, and the other for the same comparison after Noise30 in all subjects exposed to Noise60 directly preceding Noise30. *P*-values <0.05 were considered significant. All tests were two-sided. *P*-values for secondary outcome variables are shown without adjustment for multiple testing. Based on the paper by Ghiadoni *et al.*,[37] a difference between means of 2% could be expected (with SD of about 3%). With a sample size of 75 and a standard deviation of FMD differences between Noise60 and control of 3%, one may expect to detect a FMD difference of 0.98% with a power of 80% at the alpha-level 0.05.

Results

Study population and setting

A total of 88 subjects were enrolled. Thirteen of them were excluded from the final analysis. Reasons for dropouts (3 study subjects before and 10 after the first study night) included the diagnosis of hyperthyroidism, relocation to noise-affected areas, protocol violations, and inadequate data recording quality. The study subjects included in the final analysis were on average 26 years (range 20–54 years) old, 61% were females. FMD data could not be analysed for one visit in two subjects. The study population did not have relevant sleep disorders as assessed with the PSQI, and had a moderate

Table 1 Baseline characteristics of the study population

Age	(min–max)	25.7 (20–54)
Gender	% female	61.3
Height	cm	174.6 ± 10.2
Weight	kg	67.7 ± 11.9
BMI	kg/m^2	22.1 ± 2.4
Baseline noise sensitivity, chronotype, sleep quality index		
NoiSeQ	0–3	1.22 ± 0.38
Horne–Östberg	14–86	49.41 ± 9.79
PSQI	0–21	3.73 ± 1.72
Laboratory values		
Total cholesterol	mg/dL	182.9 ± 32.9
LDL	mg/dL	104.7 ± 25.6
HDL	mg/dL	60.7 ± 15.3
Triglycerides	mg/dL	87.2 ± 41.9
C-reactive protein	mg/L	1.3 ± 1.5
Creatinin	mg/dL	1.0 ± 0.5
HbA1C	%	5.3 ± 0.5

Data are presented as mean ± SD.
NoiSeQ, Dortmund Noise Sensitivity Questionnaire with three greatest noise sensitivity; Horne-Östberg, Morningness-Eveningness Questionnaire; PSQI, Pittsburgh Sleep Quality Index.

trend towards evening chronotype (characteristics shown in *Table 1*). None reported significant diseases.

The average maximum SPL of aircraft noise events recorded in participants' bedrooms is presented in *Table 2*. Overall nighttime SPLs had average peak levels of 49.6 dB(A) (control), 59.9 dB(A) (Noise30), and 60.9 dB(A) (Noise60) (both $P < 0.0001$ compared with control). Corresponding equivalent continuous SPLs Leq(3) were 35.4 dB(A), 43.1 dB(A), and 46.3 dB(A), respectively. The mean time between awakening and start of image acquisition for FMD did not differ across visits ($P > 0.5$).

Control and noise exposure nights did not differ significantly with regard to outside and body temperatures, total time in bed or subjective well being prior to the study night (data not shown). All data were normally distributed.

Haemodynamic changes in response to night noise

As a secondary predefined endpoint, we also found a dose-dependent decrease in minimum PTT (*Table 2*) after the noise nights, which was mirrored by the changes in systolic blood pressure ($P = 0.11$ for the changes among visits, *Table 2*). Automated heart rate analysis detected no significant change in mean and maximum heart rate. Heart rate acceleration index as detected by the polygraphic device did not differ between noise exposure and control nights.

With increasing number of noise events, study subjects reported deteriorating sleep quality in the morning after the respective study night ($P = 0.001$).

Effects of nocturnal noise on endothelial function

The comparison of the FMD values measured after the control visit and the Noise60 visit demonstrated a blunting in endothelial responses after noise ($P = 0.016$). When all three levels of noise were compared, and noise exposure (0, 30, 60) was used as a pseudo-continuous covariate in the AN(C)OVA in order to test for a dose-dependency in the effect of noise on FMD, a linear relationship between FMD values and exposure was found ($P = 0.020$), confirming that the exposure to more severe noise causes more severe ED. Although a standard comparison among the three noise levels within the ANOVA, i.e. without assuming a monotone effect for dose as a pseudo-continuous covariate, did not reach statistical significance (control night: 10.4 ± 3.8%; after 30 noise events: 9.7 ± 4.1%; after 60 noise events: 9.5 ± 4.3%, $P = 0.052$, *Figure 2A*), the introduction of the two additional factors described in the Methods section evidenced a priming effect of Noise30 nights on the blunting in FMD induced by Noise60 ($P = 0.006$), i.e. Noise60 had the largest impact on FMD in the subjects who had already been exposed to Noise30. Finally, there was no effect of the randomization sequence (means after each visit adjusted for the effect of effect of noise: first visit: 9.8%, second visit: 10.0%, third visit: 9.4%, $P = 0.757$).

Noise had no effect on blood flow and reactive hyperaemia (control: 855 ± 357%; Noise30: 900 ± 423%; Noise60: 900 ± 389%, $P = 0.55$). As well, baseline arterial diameter did not significantly influence the effect of noise on FMD.

In order to study the mechanism of the blunting in FMD induced by Noise60, we tested the impact of acute challenges with vitamin C in five control subjects. In these subjects, 2 h after the administration of Vitamin C, FMD was markedly improved (*Figure 2B*, $P = 0.0171$). In contrast, in a separate control group of subjects exposed to Noise60 without Vitamin C, FMD did not change as an effect of time (11.21 ± 5.56%; FMD at 2 h: 11.47 ± 5.80%; $P = 0.842$).

Effects of night noise on neurohormones and markers of inflammation (*Table 2*)

We found a marked increase in plasma adrenaline concentrations between control and Noise30 and 60 exposure nights, respectively (control: 28.3 ± 10.9 ng/L; Noise30: 33.2 ± 16.6; Noise60: 34.1 ± 19.3 ng/L, $P = 0.0099$, *Figure 3*). In contrast, morning plasma levels of cortisol did not increase with noise exposure. Likewise, inflammatory markers IL-6 and C-reactive protein were unaffected by noise exposure.

Discussion

We demonstrate cardiovascular effects of nighttime aircraft noise in young and healthy individuals with low cardiovascular risk. Nighttime aircraft noise increased plasma epinephrine levels, worsened sleep quality, and decreased pulse transit time, a parameter of arterial stiffness, which varies inversely to arterial blood pressure. A dose-dependent decrease in endothelial function after exposure to increasing levels of noise was also observed. Acute Vitamin C challenges improved endothelial function in a separate group of subjects exposed to Noise60. We found no effect of aircraft noise

Table 2 Effects of nighttime noise on the quality of sleep, haemodynamic parameters, cortisol levels, and inflammation parameters

	Control	Noise 30	Noise 60	P (ANOVA)
PeakdB(A)	48.63 ± 3.47	59.89 ± 3.28	60.87 ± 2.46	<0.001
Leq3dB(A)	35.44 ± 8.08	43.12 ± 4.91	46.28 ± 3.89	<0.001
Sleep quality	6.70 ± 1.92	5.20 ± 2.28	4.37 ± 2.23	<0.001
Movement index	3.94 ± 5.40	3.06 ± 2.85	3.23 ± 3.44	0.639
Haemodynamic parameters				
HR mean	58.7 ± 7.6	59.5 ± 7.7	59.7 ± 7.8	0.345
HR max	102.6 ± 13.3	104.3 ± 13.2	106.9 ± 17.5	0.325
BPsys mean (mmHg)	109.8 ± 15.4	114.9 ± 13.9	115.2 ± 12.4	0.120
BP rise Index	2.3 ± 2.3	2.5 ± 2.32	3.8 ± 5.9	0.397
HR_accel Index	25.8 ± 32.4	22.8 ± 23.0	23.9 ± 26.5	0.215
Pulse transit time (ms)	271.8 ± 12.3	270.9 ± 18.7	264.9 ± 15.7	0.003
Laboratory parameters				
Adrenaline (ng/L)	28.3 ± 10.9	33.2 ± 16.6	34.1 ± 19.3	0.010
Cortisol (μg/L)	15.34 ± 5.47	16.43 ± 5.55	15.76 ± 5.78	0.197
Neutrophils (%)	51.0 ± 11.39	49.77 ± 9.48	50.04 ± 7.87	0.353
IL-6 (pg/mL)	2.6 ± 3.45	2.27 ± 1.25	2.57 ± 3.29	0.383
C-reactive protein (mg/L)	2.26 ± 6.30	2.27 ± 4.82	1.55 ± 2.16	0.512

Data are presented as mean ± SD.
Leq3 dB, long-term equivalent continuous sound level; PTT, pulse transit time; BP, blood pressure; HR accel, heart rate acceleration; IL-6, interleukin 6.

exposure on nocturnal motility, heart rate or blood cortisol, neutrophils, IL-6, or C-reactive protein.

Interestingly, a priming effect of aircraft noise on ED was observed, i.e. previous exposure to Noise30 caused Noise60 to have larger effects on endothelial function. These data demonstrate that aircraft noise can affect endothelial function, and that rather than habituation, prior exposure to noise seems to amplify the negative effect of noise on endothelial function. Although the mechanisms of these observations cannot be characterized at a molecular level *in vivo* in humans, it has been previously shown that other forms of mental stress lead to a decrease in endothelial function.[37–40] With regards to the molecular mechanisms, previous studies indicate that noise leads to an up-regulation, rather than a downregulation, of the eNOS.[41] Interestingly, such an increased eNOS activity does not necessarily result in improved endothelial responses. For instance, in animal models of diabetes and/or hypertension, increased expression of an uncoupled (superoxide-producing) eNOS is associated with impaired endothelial function (reviewed in[42]). Since measurements of NO and/or superoxide production in the local vascular microenvironment are impossible to perform in humans, this question cannot be addressed at the present time. The improvement in FMD observed in our study 2 h after application of the antioxidant vitamin C in subjects exposed to Noise60 is compatible with this evidence, and it suggests that exposure to aircraft noise might lead to ED due to increased vascular oxidative stress.[34]

We also demonstrate changes in PTT, a parameter that correlates inversely with changes in blood pressure. Briefly, PTT is measured as the time it takes a pulse wave to travel between two arterial sites. Rises in blood pressure cause vascular tone to increase, leading to increased arterial stiffness and a shorter PTT. As mentioned above, these data are compatible with those of the HYENA project, in which an increase prevalence of hypertension was reported in subjects exposed to nocturnal noise in the range of 50 dB (similar to our Noise60 condition; 46.3 dB).[9] Similarly, acute noise events were associated in this study with increased systolic and diastolic blood pressure by 6.2 and 7.4 mmHg, a phenomenon which, interestingly, was not necessarily associated with awakenings.

With regard to the pathophysiological mechanism behind the changes in blood pressure and vascular function, we also report elevated epinephrine levels after exposure to noise. It has been demonstrated that intermittent release of adrenaline may be implicated in the development of hypertension.[43] Epinephrine is released as a response to different stressors such as noise[44] and increases the release and the effects of norepinephrine.[45] Interestingly, increased epinephrine levels have been found in patients with borderline hypertension,[45,46] suggesting a role in the early history of hypertension.

Importantly, increased plasma catecholamines have also been shown to correlate negatively with endothelial function as measured by FMD.[47] A recent study has linked autonomic sympathetic activation to the development of hypertension in elderly patients independent of the cause of activation of the autonomic nervous system.[48]

Our results are congruent with the growing amount of data linking short sleep duration or sleep disturbances of various kinds to the development of cardiovascular disease. For example, shift work has been shown to cause impaired endothelial function, sympathetic activation, and metabolic changes.[49,50] Extensive evidence exists for the relation between obstructive sleep apnoea, hypertension, ED, and

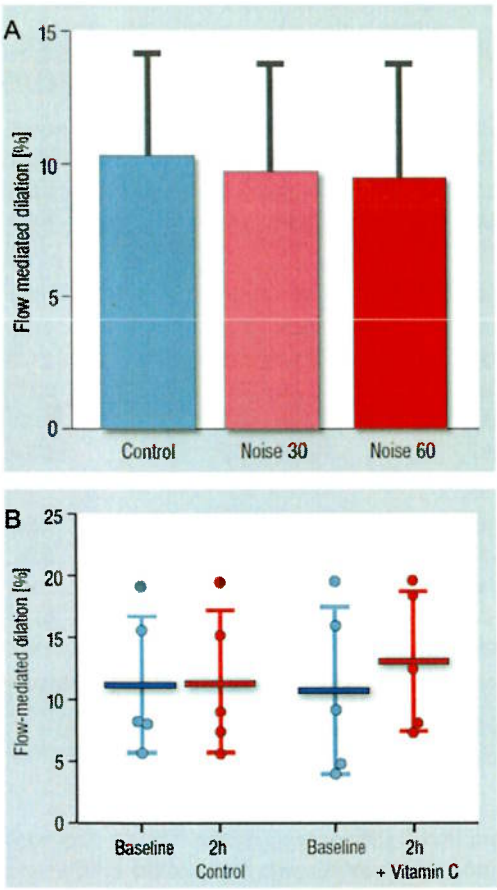

Figure 2 (*A*) Effects of Noise30 and Noise60 on flow-mediated dilation (FMD). Data are mean ± SD; $P = 0.020$ for a test using the level of noise a pseudo-continuous variable, demonstrating a linear relationship between FMD values and noise exposure. (*B*) Effects of Vitamin C (2 g, p.o.) in FMD of the brachial artery. 2 h after Vitamin C administration, the antioxidant improved significantly FMD in five control subjects exposed to Noise60. Date are presented as mean ± SD; $P = 0.0171$ for the effect of Vitamin C on FMD, paired *t*-test.

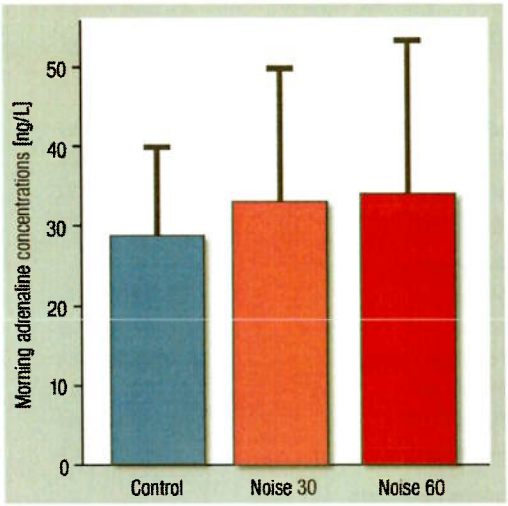

Figure 3 Effects of Noise30 and Noise60 on plasma adrenaline levels. Nighttime noise exposure significantly increases circulating catecholamine levels. Data are mean ± SD. $P < 0.01$, ANOVA.

subsequently cardiovascular disease.[51] Recently, the restless legs syndrome has been identified as another cause for sleep disruption, and it has been shown to increase the risk for myocardial infarction in women.[52] There is ample evidence that nocturnal aircraft noise exposure disturbs and fragments sleep, leads to changes in sleep structure, increases sleepiness during the following day, and leads to impairments of cognitive performance.[10,23,53,54] The results of our study suggest that these changes in sleep structure negatively affect the cardiovascular system, and that these changes, in the case of long-term exposure, may predispose to the development of hypertension and cardiovascular disease.

The study by design eliminated noise adaptation processes, which can often mask effects of environmental influences. Therefore, it is unclear whether the negative cardiovascular effects observed in this study persist after weeks or months with continued noise exposure. However, biologic adaptation is often incomplete and requires physiologic resources therefore also putting strain on the system as a whole. Effects of aircraft noise in population-based studies are likely to be mitigated by partial physiologic adaptation and avoidance of residential areas with high levels of noise exposure by highly sensitive individuals. Other environmental factors like air pollution, which has also been shown to influence endothelial function,[55] may interfere with noise effects in epidemiological studies. Therefore, data from interventional studies may be helpful in judging the effect of nocturnal noise on cardiovascular health and disease.

Limitations of the study

The protocol was designed as a field study with minimal sleep disruption due to environment and equipment, thus creating ecologically valid conditions. We avoided on purpose a pure laboratory environment where ambient conditions, sound levels, and external stimuli can be controlled at the expense of creating artificial rather than familiar conditions. Sleep quality is very sensitive to changes in surroundings and study subjects usually show more pronounced alterations of sleep in the laboratory than in the field.[56] There were no adaptation nights prior to study nights due to logistic constraints and because, since subjects were not required to sleep in non-familiar environments, our study design did not demand such adaptation. Reinforcing this, the analysis did not show a significant first-night effect for our primary outcome,[57] which supports the validity of our study design and results. Study subjects were healthy, young, and with a female majority and are therefore not representative of the whole population. In general, younger adults usually show less sleep problems and disturbance than older persons when exposed to noise, and the fact that noise had an impact also on such a low-risk population rather emphasizes the potential clinical relevance of the present

findings. Finally, endothelium-independent vasodilation was not systematically measured and the data are not presented: nitroglycerin responses were measured initially, but these measures were discontinued due to refusal by many study participants related to the side effects of the drug.

Summary and conclusions

In a group of young and healthy volunteers, we found evidence for significant impairment of endothelial function after only one night of aircraft noise exposure with 60 noise events. Pointing to a significant contribution of oxidative stress in this phenomenon, these adverse changes of the vasculature were markedly improved by acute Vitamin C challenges. Endothelial dysfunction was paralleled by significant increases in circulating adrenaline levels and a substantial, dose-dependent decrease in sleep quality and an increase in systolic blood pressure. These findings indicate that hypertension observed in response to nighttime exposure to noise might be explained by increased sympathetic activation but also by the occurrence of vascular dysfunction. Accumulating data increasingly confirms that sleep disturbance of different causes might represent a novel, important health risk. An undisturbed night's sleep is important for health and well-being and should be protected as far as possible, and reducing nocturnal aircraft noise can therefore be regarded as a preventive measure for cardiovascular disease. Since the present studies demonstrate adverse effects of endothelial function and stress hormones in healthy adults, the implications for patients with known cardiovascular disease will need to be tested in further studies.

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Review

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Environmental Noise and Effects on Sleep: An Update to the WHO Systematic Review and Meta-Analysis

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Background: Nighttime noise carries a significant disease burden. The World Health Organization (WHO) recently published guidelines for the regulation of environmental noise based on a review of evidence published up to the year 2015 on the effects of environmental noise on sleep.

Objectives: This systematic review and meta-analysis will update the WHO evidence review on the effects of environmental noise on sleep disturbance to include more recent studies.

Methods: Investigations of self-reported sleep among residents exposed to environmental traffic noise at home were identified using Scopus, PubMed, Embase, and PsycINFO. Awakenings, falling asleep, and sleep disturbance were the three outcomes included. Extracted data were used to derive exposure–response relationships for the probability of being highly sleep disturbed by nighttime noise [average outdoor A-weighted noise level (L_{night}) 2300–0700 hours] for aircraft, road, and rail traffic noise, individually. The overall quality of evidence was assessed using Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) criteria.

Results: Eleven studies ($n = 109{,}070$ responses) were included in addition to 25 studies ($n = 64{,}090$ responses) from the original WHO analysis. When sleep disturbance questions specifically mentioned noise as the source of disturbance, there was moderate quality of evidence for the probability of being highly sleep disturbed per 10-dB increase in L_{night} for aircraft [odds ratio (OR) = 2.18; 95% confidence interval (CI): 2.01, 2.36], road (OR = 2.52; 95% CI: 2.28, 2.79), and railway (OR = 2.97; 95% CI: 2.57, 3.43) noise. When noise was not mentioned, there was low to very low quality of evidence for being sleep disturbed per 10-dB increase in L_{night} for aircraft (OR = 1.52; 95% CI: 1.20, 1.93), road (OR = 1.14; 95% CI: 1.08, 1.21), and railway (OR = 1.17; 95% CI: 0.91, 1.49) noise. Compared with the original WHO review, the exposure–response relationships closely agreed at low (40 dB L_{night}) levels for all traffic types but indicated greater disturbance by aircraft traffic at high noise levels. Sleep disturbance was not significantly different between European and non-European studies.

Discussion: Available evidence suggests that transportation noise is negatively associated with self-reported sleep. Sleep disturbance in this updated meta-analysis was comparable to the original WHO review at low nighttime noise levels. These low levels correspond to the recent WHO noise limit recommendations for nighttime noise, and so these findings do not suggest these WHO recommendations need revisiting. Deviations from the WHO review in this updated analysis suggest that populations exposed to high levels of aircraft noise may be at greater risk of sleep disturbance than determined previously. https://doi.org/10.1289/EHP10197

Introduction

Sleep is a vital component of human life that serves many critical roles in physical and mental health and well-being.[1] Sufficient quantity and quality of sleep are requirements for optimal daytime alertness and performance, and high quality of life.[2] Experimental studies suggest that restricted sleep duration causes blood vessel dysfunction,[3] induces changes in glucose metabolism[4,5] and appetite regulation,[6] and impairs memory consolidation.[7] Accordingly, epidemiological studies have consistently found that chronic short or interrupted sleep is associated with negative health outcomes, including obesity,[8] diabetes,[9] hypertension,[10] cardiovascular disease,[11] all-cause mortality,[12] and poorer cognitive function.[13] Chronic insufficient or disrupted sleep is therefore of public health relevance, and sleep disturbance is considered a major adverse consequence of exposure to environmental noise.[14]

In Europe, there is a substantial burden of disease from environmental noise, primarily from aircraft, road, and rail traffic.[15,16] In 2011, the World Health Organization (WHO) attributed the majority of this disease burden to noise-induced sleep disturbance, with 903,000 disability-adjusted life years lost annually in Western Europe alone.[14] Environmental noise is also a problem outside of Europe, for example, recent data from the U.S. Bureau of Transportation Statistics estimates that 41.7 million people in the United States are exposed to air and road traffic noise at 24-h average levels ($L_{AEq,24h}$) >50 dB.[17] This noise level, per conversion data from Brink et al.[18] is equivalent to a nighttime (2300–0700 hours) level of 45.3 dB (L_{night}), which is around or above the level associated with adverse effects on sleep.[15] Nighttime noise can fragment sleep structure by inducing awakenings and shifts to lighter, less restorative sleep.[19] Importantly, these effects do not seem to habituate fully, and arousals and awakenings induced by aircraft noise can occur even among chronically exposed individuals.[20–22] Although noise-induced sleep fragmentation and reductions in total sleep time are less severe than in sleep restriction studies, sleep disturbance by chronic noise exposure may lead to the development of disease in the long term. Experimental studies have found adverse effects of nocturnal aircraft noise on parameters of endothelial function, oxidative stress, and inflammation.[23,24] This points to the importance of noise-induced sleep disturbance for cardiovascular disease risk, and, indeed, this is supported with epidemiological data where nighttime noise is more strongly associated with indicators of vascular stiffness and hypertension compared with daytime noise.[25] The ubiquity of exposure to environmental noise in industrialized nations, and the chronic nature of that exposure, therefore poses a significant threat to health.[26]

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In 2018, the WHO published recommendations for protecting human health from exposure to environmental noise.[15] These guidelines included strong recommendations for target nighttime noise levels to mitigate adverse effects of traffic noise on sleep, which were 45 dB L_{night} for road traffic, 44 dB L_{night} for rail traffic, and 40 dB L_{night} for air traffic. These recommendations were based primarily on a systematic review and meta-analysis on the effects of noise on sleep, which included studies published up to the year 2015 only.[19] There has been continued and substantial interest and research in the domain of noise and sleep during the intervening years. We therefore updated the earlier systematic review and meta-analysis to include studies published up to the year 2021. This updated analysis is restricted to field studies on the effects of nocturnal traffic noise on self-reported sleep in adults, and it has the overarching aim of synthesizing updated exposure–response relationships for the probability of being highly sleep disturbed.

Methods

This review and analysis was prepared following Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement guidelines.[27] The completed PRISMA checklist is given in Table S1. The review and analysis protocol was defined *a priori* and registered in PROSPERO (record CRD42021229587) before conducting any preliminary searches, screening of articles, or data extraction. The University of Pennsylvania institutional review board (IRB) determined that the study did not meet the definition of human subjects research given that no identifiable information was being obtained, and therefore review or approval of the study by the IRB was not required.

The analytic approach is described in detail below and was consistent with the previous WHO review,[19] with the following exceptions: *a*) Exposures were limited to traffic noise from aircraft, road, and rail traffic, and *b*) effects on sleep were limited to self-reported questionnaire outcomes. These form the basis of the highly sleep disturbed exposure–response relationships and calculations of the burden of disease by noise and are, therefore, are critical outcomes from a noise policy perspective. Studies on acute noise-induced awakenings using objective measures, such as actigraphy or polysomnography, were not included.

Eligibility Criteria

Studies were restricted to primary investigations in humans exposed to environmental noise from aircraft, road, and rail traffic at home. Studies investigating other sources, such as wind turbine noise or hospital noise, were excluded. Studies were eligible only if sound pressure levels were measured or predicted at the participant's home. Studies with subjective evaluation of the noise levels, distance to the noise source as a surrogate measure of noise level, or noise levels not specific to a participant's home address were excluded. A minimum of two different noise level categories were required so that exposure–response relationships for sleep disturbance could be constructed.

Studies were eligible if they employed prospective, retrospective, cohort, longitudinal, cross-sectional, or case–control study designs. Laboratory studies, intervention studies, or studies in which noise was introduced artificially were excluded due to low generalizability in real-world settings. Studies were restricted to original research published or accepted for publication in the year 2000 or later. Article language was restricted to English, Dutch, French, and German.

This review and analysis focuses on self-reported sleep disturbance by traffic noise. Eligible studies included at least one of the three most common outcomes of self-reported disturbance that were identified in the original WHO review[19]:

- Awakenings from sleep
- The process of falling asleep
- Sleep disturbance

Studies were eligible if they either explicitly mentioned noise as the source of disturbance, for example, "How often is your sleep disturbed by noise from aircraft?", or included more general sleep questions that did not explicitly mention noise, for example, "How often do you have difficulties sleeping?". So that the probability of being highly sleep disturbed could be determined, eligible studies were required to include outcome scales that indicated either the severity or the frequency of symptoms or disturbance on a nonbinary scale. A binary response scale was, however, permitted if the phrasing of the question was such that a binary response would indicate being highly sleep disturbed, for example, "Is your sleep highly disturbed by noise from road noise?". Studies reporting other measures of self-reported sleep not described above (e.g., perceived sleep quality, estimated total sleep time, morning sleepiness), and studies on objective sleep (e.g., polysomnography, actigraphy) or sleep medication use, were excluded.

Study Selection

All studies identified in the WHO evidence review[19] for which data were already available for meta-analysis were included in the updated synthesis. We also identified studies published later than the WHO review from a scoping synthesis by van Kamp et al.[28] Because van Kamp et al.[28] included studies published up to June 2019 only, we further searched four electronic databases (Scopus, PubMed, Embase, PsycINFO), to identify more recent relevant studies published up to 31 December 2021. This search was done with the same search terms and strategy from van Kamp et al.[28] that were relevant for traffic noise and self-reported sleep. The full electronic search strategy is given in Table S2. Any studies of which we were aware but that were not identified during the literature search were also screened for eligibility.

Two reviewers (M.G.S. and M.C.) independently and manually screened the title and abstract of each identified study against the study eligibility criteria. If eligibility could not be determined from the title and abstract alone, the full text was reviewed. Any differences in eligibility judgments were resolved by discussion and consensus, with input from a third reviewer (M.B.) if needed.

Data Extraction and Synthesis

The following variables were extracted by a single investigator from the original records for review by the authorship team: article title, authors, publication year, traffic mode, noise level, noise metric and time base, noise exposure methodology, sleep disturbance question(s) and response scale(s), study design, country, city, effective sample size, number of data points per respondent, and sleep disturbance point estimates. If data could not be extracted directly from the published articles and supplemental materials, we directly contacted all study authors for whom contact details were available to request data. We requested a list of relevant questions on sleep and the response scales used, the total number of respondents in 5-dB bins, and the percentage of respondents reporting being highly sleep disturbed in each 5-dB bin. We requested only these summary data, and no identifiable information on any study respondents was requested or obtained. If the study authors did not reply after they were sent two reminders, the contact was considered a nonresponse and the study was excluded.

The exposure variable of interest for the meta-analysis was average nighttime outdoor A-weighted noise level from a single traffic mode (air, road, and rail) during the night, hereafter termed L_{night}, measured in decibels. A-weighting is a filter network that is used to simulate the nonlinear frequency response of human hearing. The night period was defined as 2300–0700 hours, in line with EU Environmental Noise Directive 2002/49/EC.[29] In studies where noise levels were reported as a different metric, we converted to L_{night} using the conversion formulae from Brink et al.[18] given below. L_{night} was not treated as a continuous variable but, rather, was categorized into 5-dB bins, following the approach used in the WHO review.[19] For open-ended noise level categories, we assigned a noise level that was 2.5 dB above or below the cutoff, for instance, <50 dB and >50 dB would be coded as 47.5 dB and 52.5 dB, respectively. The midpoints of each 5-dB bin were used as the noise exposure levels in the statistical analyses.

The primary outcome of interest was the probability of self-reporting high sleep disturbance for a given noise level. We *a priori* defined three separate domains of questions that were used to determine sleep disturbance. First, "awakenings from sleep," referring to the period between sleep onset and final awakening. These awakenings are defined as events where a participant wakes from sleep, regains consciousness, and recalls the awakening the following morning. Second, the "process of falling asleep," defined as the transition from wakefulness to sleep. Third, "sleep disturbance," defined as the internal or external interference with sleep onset or sleep continuity. Included studies had to address at least one of these domains in the form of at least one self-reported question. For each of these three question types, the coding of whether a respondent was highly sleep disturbed depended on the response scale used. For responses using 5- or 11-point scales referring to the severity of the disturbance, the top two and top three categories were, respectively, defined as highly sleep disturbed, following previous conventions for the International Commission on the Biological Effects of Noise (ICBEN) annoyance scale.[30] For responses that referred to the frequency of symptoms, a frequency of "often" or at least three times per week was considered as highly sleep disturbed because this frequency of difficulty sleeping is a diagnostic criterion of insomnia.[31] One study used a dichotomous filter question, "Do you have any trouble with your sleep?", to determine if a respondent would answer a question on the frequency of difficulty falling asleep.[32] Any responses of "no" to this filter question were coded as not highly sleep disturbed.

Study-Specific Exposure and Response Characterization

One study reported noise exposure as 24-h average levels ($L_{AEq,24h}$).[33] These noise levels were converted to L_{night} using the following conversion equations[18]:

$$\text{Road traffic: } L_{night(23\text{–}07)} = L_{AEq,24h} - 4.7\,\text{dB, and}$$

$$\text{Railway traffic: } L_{night(23\text{–}07)} = L_{AEq,24h} - 0.6\,\text{dB.}$$

One study reported road noise as the day-evening-night level (L_{den}),[34] which was converted to L_{night} as follows[18]:

$$L_{night(23\text{–}07)} = L_{den} - 8.3\,\text{dB.}$$

One study reported noise level as Livello di Valutazione del Aeroportuale (LVA),[35] which is similar to the day-night level (L_{dn}), except that the night period is 7 h (2300–0600 hours) rather than 8 h.[36] Formulae to convert directly from LVA to L_{night} are unavailable; therefore, we made the following assumptions in converting to L_{night}: The 1-h shorter night when using LVA means that the same exposure assessed as L_{dn} will be lower because L_{dn} applies a 10-dB penalty to the night period. We assume −0.7 dB given that that is the difference in L_{dn} metrics with a 1-h difference in the night period (8 vs. 9 h) for aircraft noise.[18] We then incorporated this difference into an appropriate conversion equation to convert from LVA to L_{night}[18]:

$$L_{dn} = \text{LVA} - 0.7\,\text{dB}; L_{night(23\text{–}07)} = L_{dn} - 8.9\,\text{dB, and}$$

$$\therefore L_{night(23\text{–}07)} = \text{LVA} - 0.7\,\text{dB} - 8.9\,\text{dB} = \text{LVA} - 9.6\,\text{dB.}$$

One study used a noise category that was 10-dB-wide (65–75 dB LVA).[35] We subdivided these data into 5-dB-wide bins, assuming (n)/2 respondents in each bin (35 respondents per bin) and the same prevalence of high sleep disturbance in each bin as in the 10-dB-wide category.

Two studies assessed noise exposure as both calculated long-term outdoor noise levels and measured indoor noise levels over 3–6 nights.[20,21] We used the calculated outdoor noise levels as the exposure metric to be consistent with other studies in the meta-analysis.

In one study,[21] sleep in the previous night was assessed repeatedly over several mornings. Because of these repeated measures, we first calculated the probability of being highly disturbed using all five to six responses per respondent. We then used these probabilities to determine the number of individuals that would have reported being highly sleep disturbed if only one response was obtained per person. In this way, each respondent contributed only a single data point to the analysis.

One study calculated exposure to railway traffic as including noise from trains, trams, and subways.[37] The questions regarding "sleep disturbance by tram/subway noise" and "sleep disturbance by train noise" in this study were therefore averaged into a single sleep disturbance variable.

Risk of Bias and Quality of Evidence

The risk of bias at the outcome level within individual studies was assessed using the methodology developed within the WHO review,[19] with the following two amendments to the assessment criteria (Table 1). First, in line with recommendations for cross-sectional studies by the National Institutes of Health,[38] a study was considered at high risk of selection bias if the response rate was <50%, down from the 60% criterion in the WHO review. Second, bias due to the sleep measurement outcome was not assessed because our updated analysis focused on only a single sleep measurement outcome (sleep questionnaires), whereas the WHO review included also heart rate or blood pressure, actigraphy, polysomnography, and other objective physiologic measurements. The risk of bias in each domain was assessed independently by two investigators (M.G.S. and M.C.). All studies were included in the meta-analysis regardless of the bias assessment.

To evaluate heterogeneity between studies, we calculated odds ratios (ORs) for each outcome within each study using binary logistic regression in SPSS (version 26; IBM Corp.). For consistency with the WHO review,[19] the range of L_{night} was not restricted in this analysis. Forest plots for all outcomes across studies were generated using RevMan (version 5.4.1; Cochrane Collaboration) using an inverse-variance (IV) random effects method. Heterogeneity between studies for each outcome was assessed using the I^2 statistic. We interpreted I^2 values using thresholds defined by the Cochrane Collaboration.[39] Publication bias across studies was investigated using funnel plots of the individual study estimates.

The quality of evidence across studies for the effects of exposure to aircraft, road, and rail traffic noise on self-reported sleep outcomes where noise was specified, and self-reported sleep outcomes where noise was not specified, was assessed independently

Table 1. Criteria for assessing risk of bias of individual studies (adapted from the WHO review[19]).

Bias domain	Criteria	Risk of bias
A. Selection bias	Random sampling, areas selected based on noise exposure. ≥50% response rate.[a] inclusion criteria not contingent on sleep and health conditions	Low
	a. <50% response rate,[a] or b. Non-random sampling, or c. Sampling not based on noise exposure, or d. Individuals were excluded based on sleep and health criteria	High
	Insufficient information to make a judgment	Unclear
B. Information bias (exposure assessment)	a. Based on measurements for at least 1 wk, or b. Based on a noise map that was verified by noise measurements, or c. Based on a noise map that was based on actual traffic data	Low
	a. Based on measurements of <1 wk, or measurements were not continuous, or b. Based on a noise map that was not verified by noise measurements, or the predictions were not based on actual traffic data	High
	Insufficient information to make a judgment	Unclear
C. Bias due to confounding	All most-important confounders accounted for in analysis	Low
	No accounting for important confounders	High
	Insufficient information to make a judgment	Unclear
D. Reporting bias	Complete reporting of all outcomes analyzed including nonsignificant results	Low
	Not all outcomes reported, underreporting methods or statistical analysis, not reporting conflicts of interest	High
	Insufficient information to make a judgment	Unclear

Note: WHO, World Health Organization.
[a]The 50% response rate criterion was based on recommendations for cross-sectional studies by the National Institutes of Health.[38]

by two investigators using the Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) criteria.[40] Any differences in the risk of bias assessments for individual studies, or in the quality of evidence across studies for each outcome (GRADE), were resolved by consensus with input from a third investigator if needed.

Meta-Analytic Approach

The primary goal of the meta-analysis was to generate updated exposure–response relationships for the probability of high sleep disturbance for each of the three disturbance types (awakenings, falling asleep, and sleep disturbance) for each traffic mode (air, road, and rail). In line with the WHO review,[19] we also generated a combined estimate for high sleep disturbance across the three different types of disturbance questions, using the following approach: If a study included two or three relevant sleep disturbance questions, the combined estimate was calculated by averaging the responses to those questions for each respondent within a study. This approach was adopted so that each respondent would contribute only a single data point to the analysis of each separate outcome. If a study included only one sleep outcome, the combined estimate and the single study outcome assessed would be the same.

Data for individual studies were provided directly by the authors of each study, binned in 5-dB-wide noise categories. One line of data was created for each sleep disturbance question from each study respondent. For instance, if a study had 500 respondents in the noise category with a 47.5 dB L_{night} midpoint, and 10% were classified as highly sleep disturbed, we generated 450 data lines with non-highly sleep disturbed respondents (binary outcome = 0) and 50 data lines with highly sleep disturbed respondents (binary outcome = 1). Each data line also carried the midpoint of the 5-dB L_{night}-exposure category, a three-level categorical variable for traffic mode (air, road, and rail), a dichotomous variable indicating whether questionnaire data originated from questions that did or did not explicitly mention noise as a source of disturbance in the question for each traffic mode, dichotomous study location indicated a European or non-European study, and a study identification number.

Statistical Analysis

Exposure–response relationships were generated with the following approach: Random study effect logistic regression models with L_{night} (midpoint of the noise exposure category) as the only explanatory variable were performed with the NLMIXED procedure in SAS (version 9.4; SAS Institute, Inc.). This approach accounts for the fact that respondents were clustered within studies, and the weight of a study increases with its sample size. Analyses were restricted to levels between 40 and 65 dB L_{night} because of inaccuracy in predicting noise levels <40 dB and that the highest exposure limit common to all three traffic modes was 65 dB L_{night}. Separate regression models were run stratified by the three traffic modes (air, road, or rail), four sleep disturbance outcome (awakenings, falling asleep, sleep disturbance, or combined estimate of all questions within a study), and the dichotomous noise-specificity of the disturbance question (noise mentioned or noise not mentioned), yielding a total $3 \times 4 \times 2 = 24$ separate regression analyses. Estimate statements were used to generate point estimates and 95% confidence intervals (CIs). Data are reported as dose–response curves and as ORs per 10-dB increase in L_{night}.

To investigate whether a response differed depending between European and non-European studies location, we added study location as a covariate to the logistic regression model and repeated the analysis for the combined estimates of sleep disturbance. These analyses were restricted to the four outcomes where both European and non-European data were available.

We performed a sensitivity analysis to investigate the risks of exposure bias on sleep disturbance. We repeated the logistic regression for the combined estimates of sleep disturbance, restricted between 40–65 dB L_{night}, and stratified analysis by studies that were judged to have a low or high risk of bias in the exposure assessment.

Results

Study Selection

Study identification, screening and selection are summarized in Figure 1. All 25 studies in the WHO review were included.[19] Twenty-one studies published between January 2014 and June

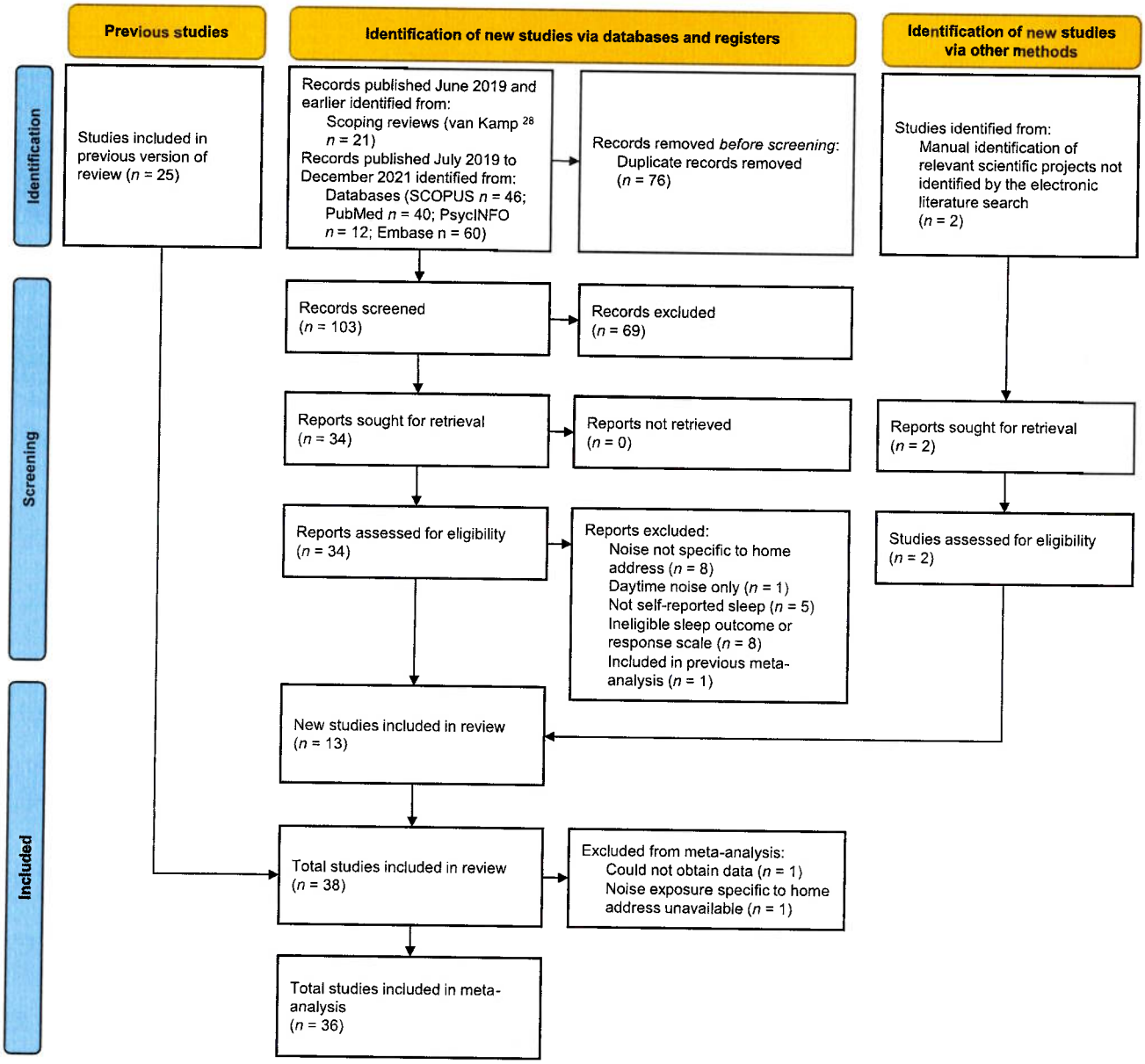

Figure 1. Flow diagram of study identification, screening, and selection. "Study" refers to a data collection campaign including a defined group of participants and one or more outcomes. In one instance, a study was reported in multiple articles[41,42] and is counted as $n = 1$ study. "Report" is a journal article, preprint, conference abstract, study register entry, clinical study report, dissertation, unpublished manuscript, government report, or other document supplying relevant information about a particular study or studies.

2019 were identified by van Kamp et al.[28] Our electronic search additionally identified 82 studies published later than June 2019, after excluding duplicates. After assessing the abstracts and, if needed, the full texts, 11 new studies eligible for the meta-analysis were identified. We also manually identified a further 2 studies that were not identified by the electronic literature searches [the UK Survey of Noise Attitudes (Civil Aviation Authority)[41] and German Noise-Related Annoyance, Cognition and Health (NORAH)[42] projects]. We manually extracted the study documents from project webpages[41,42] and judged both studies to be eligible for inclusion after undergoing the standard screening protocol.

Two studies initially deemed eligible could not be included in the meta-analysis[43,44] because data could not be obtained or noise exposure specific to the home address was unavailable (Table S3). We therefore identified 11 studies in total published since the WHO review to include in the meta-analysis,[20,21,32,34,35,37,41,42,45–47] in addition to the 25 studies included in the original review[19] (Tables 2–4).

Comparison with Previous WHO Review

The effective sample size for each sleep outcome and for each traffic mode, determined using all data in the updated analysis (responses from the WHO analysis plus the 11 newly identified studies) is compared against the sample sizes from the WHO analyses in Figure 2. Sample sizes for the combined estimates where responses to multiple questions were averaged within

Table 2. Studies on exposure to aircraft noise and self-reported sleep outcomes (adapted from the WHO review[19]).

Outcome	Study	N	Location	Disturbance question and responses	Noise metric, (level range)
Awakenings: noise mentioned (total $N = 4{,}613$)	Nguyen et al.[32,a]	559	Hanoi, Vietnam	In daily life, how much do you feel disturbed when an aircraft passes by in the following cases: When you are awakened in your sleep? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (32.5–62.5 dB)
	Nguyen et al.[48]	1,093	Hanoi, Vietnam	In daily life, when an aeroplane passes by, to what degree are you disturbed in the following cases: When you are awakened in your sleep? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (37.5–57.5 dB)
	Yano et al.[49]	776	Hanoi, Vietnam	Same as above	L_{night}, 2200–0600 hours (37.5–57.5 dB)
	Nguyen et al.[50]	511	Da Nang City, Vietnam	Same as above	L_{night}, 2200–0600 hours (37.5–57.5 dB)
	Nguyen et al.[51,52]	804	Hanoi, Vietnam	Same as above	L_{night}, 2200–0600 hours (37.5–52.5 dB)
	Nguyen et al.[53]	870	Ho Chi Minh City, Vietnam	Same as above	L_{night}, 2200–0600 hours (42.5–62.5 dB)
Falling asleep: noise mentioned (total $N = 27{,}869$)	Nguyen et al.[32,a]	545	Hanoi, Vietnam	In daily life, how much do you feel disturbed when an aircraft passes by in the following cases: When it makes it difficult for you to fall asleep? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (32.5–62.5 dB)
	NORAH[42,a]	3,505	Frankfurt, Germany 2011	In the last 12 months aircraft noise has disturbed you when falling asleep? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (32.5–57.5 dB)
	NORAH[42,a]	3,502	Frankfurt, Germany 2012	Same as above	L_{night}, 2200–0600 hours (32.5–67.5 dB)
	NORAH[42,a]	3,505	Frankfurt, Germany 2013	Same as above	L_{night}, 2200–0600 hours (32.5–62.5 dB)
	NORAH[42,a]	5,527	Berlin, Germany 2012	Same as above	L_{night}, 2200–0600 hours (32.5–52.5 dB)
	NORAH[42,a]	2,947	Cologne-Bonn, Germany 2013	Same as above	L_{night}, 2200–0600 hours (32.5–67.5 dB)
	NORAH[42,a]	1,970	Stuttgart, Germany 2013	Same as above	L_{night}, 2200–0600 hours (32.5–52.5 dB)
	Nguyen et al.[48]	1,095	Hanoi, Vietnam	In daily life, when an aeroplane passes by, at what degree are you disturbed in the following cases: When it makes it difficult for you to fall asleep? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (37.5–57.5 dB)
	Yano et al.[49]	780	Hanoi, Vietnam	Same as above	L_{night}, 2200–0600 hours (37.5–57.5 dB)
	Nguyen et al.[50]	512	Da Nang City, Vietnam	Same as above	L_{night}, 2200–0600 hours (37.5–52.5 dB)
	Nguyen et al.[51,52]	805	Hanoi, Vietnam	Same as above	L_{night}, 2200–0600 hours (37.5–52.5 dB)
	Nguyen et al.[53]	868	Ho Chi Minh City, Vietnam	Same as above	L_{night}, 2200–0600 hours (42.5–62.5 dB)
	Schreckenberg et al.[54]	2,308	Germany	How much has aircraft noise in the last 12 months disturbed falling asleep? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (37.5–57.5 dB)
Sleep disturbance: noise mentioned (total $N = 27{,}773$)	Rocha et al.[45,a]	396	Atlanta, Georgia, USA	Thinking about the last 12 months or so, when you were at home, how much was your sleep disturbed by noise from aircraft? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2300–0700 hours (37.5–52.5 dB)
	Brink et al.[46,a]	2,925	Switzerland	Thinking about the last twelve months at your home, during nighttime when you want to sleep, how much did aircraft noise bother, disturb, or annoy you? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2300–0700 hours (22.5–62.5 dB)
	Civil Aviation Authority[41,a]	1,200	United Kingdom	Thinking about the summer, when you were here at home, what number from 0 to 10 best shows the degree to which your sleep was disturbed by noise from aeroplanes? Not at all disturbed (0) to Extremely disturbed (10) (HSD 8,[b] 9,[b] 10[b])	L_{night} 2300–0700 hours, summer (37.5–62.5 dB)
	NORAH[42,a]	3,505	Frankfurt, Germany 2011	In the last 12 months aircraft noise has disturbed you when sleeping in the night? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (32.5–57.5 dB)

Table 2. (*Continued.*)

Outcome	Study	*N*	Location	Disturbance question and responses	Noise metric, (level range)
	NORAH[42,a]	3,502	Frankfurt, Germany 2012	Same as above	L_{night}, 2200–0600 hours (32.5–67.5 dB)
	NORAH[42,a]	3,505	Frankfurt, Germany 2013	Same as above	L_{night}, 2200–0600 hours (32.5–62.5 dB)
	NORAH[42,a]	5,519	Berlin, Germany 2012	Same as above	L_{night}, 2200–0600 hours (32.5–52.5 dB)
	NORAH[42,a]	2,939	Cologne–Bonn, Germany 2013	Same as above	L_{night}, 2200–0600 hours (32.5–67.5 dB)
	NORAH[42,a]	1,973	Stuttgart, Germany 2013	Same as above	L_{night}, 2200–0600 hours (32.5–52.5 dB)
	Schreckenberg et al.[54]	2,309	Germany	How much has aircraft noise in the last 12 months disturbed sleeping during the night? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (37.5–57.5 dB)
Awakenings: noise not mentioned (total $N = 3,726$)	Rocha et al.[45,a]	309	Atlanta, Georgia, USA	How often did the following occur during the past month: You had trouble sleeping because you wake up in the middle of the night or early morning? Not during the past month, Less than once a week, Once or twice a week, Three or more times a week[b]	L_{night}, 2300–0700 hours (37.5–52.5 dB)
	Basner et al.[20,a]	39	Philadelphia, Pennsylvania, USA	During the past month, how often have you had trouble sleeping because you wake up in the middle of the night or early morning? Not during the past month, Less than once a week, Once or twice a week, Three or more times a week[b]	L_{night}, 2300–0700 hours (47.5–57.5 dB)
	Carugno et al.[35,a]	400	Bergamo, Italy	Frequent nocturnal awakening (last month)? No, Yes[b]	LVA, 2300–0600 hours (57.5–72.5 dB)
	Brink et al. (2003 study)[55]	1,450	Switzerland	How often do you have the following symptoms: Problems with sleeping through? Never, Rarely, Sometimes, Often, Very Often,[b] Always[b]	L_{night}, 2200–0600 hours (27.5–62.5 dB)
	Brink et al. (2001 study)[55]	1,528	Switzerland	Same as above	L_{night}, 2200–0600 hours (27.5–62.5 dB)
Falling asleep: noise not mentioned (total $N = 4,375$)	Smith et al.[21,a]	33	Atlanta, Georgia, USA	Please evaluate last night's sleep: falling asleep was Very easy (0) to Very difficult (10) (HSD 8,[b] 9,[b] 10[b])	L_{night}, 2300–0700 hours (37.5–57.5 dB)
	Nguyen et al.[32,a]	620	Hanoi, Vietnam	Do you have any trouble with your sleep? Difficult to fall asleep? Rarely/not at all, Once or twice a week, Three or more times per week[b]	L_{night}, 2200–0600 hours (32.5–62.5 dB)
	Rocha et al.[45,a]	309	Atlanta, Georgia, USA	How often did the following occur during the past month: You had trouble sleeping because you cannot get to sleep within 30 minutes? Not during the past month, Less than once a week, Once or twice a week, Three or more times a week[b]	L_{night}, 2300–0700 hours (37.5–52.5 dB)
	Basner et al.[20,a]	39	Philadelphia, Pennsylvania, USA	During the past month, how often have you had trouble sleeping because you cannot get to sleep within 30 minutes? Not during the past month, Less than once a week, Once or twice a week, Three or more times a week[b]	L_{night}, 2300–0700 hours (47.5–57.5 dB)
	Carugno et al.[35,a]	400	Bergamo, Italy	Long time to fall asleep (last month)? No, Yes[b]	LVA, 2300–0600 hours (57.5–72.5 dB)
	Brink et al. (2003 study)[55]	1,450	Switzerland	How often do you have the following symptoms: Problems falling asleep? Never, Rarely, Sometimes, Often, Very Often,[b] Always[b]	L_{night}, 2200–0600 hours (27.5–62.5 dB)
	Brink et al. (2001 study)[55]	1,528	Switzerland	Same as above	L_{night}, 2200–0600 hours (27.5–62.5 dB)
Sleep disturbance: noise not mentioned (total $N = 195$)	Brink[56]	195	Switzerland	During the last 4 weeks, have you suffered from any of the following disorders or health problems? Difficulty in sleeping or insomnia? Not at all, Somewhat, Very Much[b]	L_{night}, 2200–0600 hours (32.5–52.5 dB)

Note: HSD, highly sleep disturbed; L_{night}, nighttime noise; LVA, Livello di Valutazione del Aeroportuale; NORAH, Noise-Related Annoyance, Cognition and Health; WHO, World Health Organization.

[a]Studies not included in the WHO review.[19]

[b]Response alternatives designated as highly sleep disturbed.

Table 3. Studies on exposure to road traffic noise and self-reported sleep outcomes (adapted from the WHO review[19]).

Outcome	Study	*N*	Location	Disturbance question and responses	Noise metric (level range)
Awakenings: noise mentioned (total $N = 10{,}177$)	Bodin et al.[33]	2,438	Sweden	Do you experience any of the following because of road traffic noise? You wake up? Never, Sometimes, Often[b]	$L_{AEq,24h}$ (37.5–62.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	1,454	Hanoi, Vietnam	How much are you disturbed by awakening during nighttime by road traffic? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (62.5–77.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	1,460	Ho Chi Minh City, Vietnam	Same as above	L_{night}, 2200–0600 hours (67.5–77.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	479	Da Nang, Vietnam	Same as above	L_{night}, 2200–0600 hours (57.5–67.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	680	Hue, Vietnam	Same as above	L_{night}, 2200–0600 hours (52.5–72.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	777	Thai Nguyen, Vietnam	Same as above	L_{night}, 2200–0600 hours (52.5–67.5 dB)
	Sato et al.[59]	1,291	Gothenburg, Sweden	Does the road traffic noise cause the following conditions? Awakening? No, Little Disturbed, Rather Disturbed, Very Disturbed[b]	L_{night}, 2200–0700 hours (42.5–72.5 dB)
	Sato et al.[59]	819	Kumamoto, Japan	Same as above	L_{night}, 2200–0700 hours (47.5–77.5 dB)
	Sato et al.[59]	779	Sapporo, Japan	Same as above	L_{night}, 2200–0700 hours (52.5–67.5 dB)
Falling asleep: noise mentioned (total $N = 13{,}374$)	NORAH[42,a]	3,162	Frankfurt, Germany 2012	In the last 12 months road traffic noise has disturbed you when falling asleep? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (32.5–72.5 dB)
	Bodin et al.[33]	2,444	Sweden	Do you experience any of the following because of road traffic noise? Difficulties falling asleep. Never, Sometimes, Often[b]	$L_{AEq,24h}$ (37.5–62.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	1,471	Hanoi, Vietnam	How much are you disturbed in falling asleep by road traffic? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (62.5–77.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	1,458	Ho Chi Minh City, Vietnam	Same as above	L_{night}, 2200–0600 hours (67.5–77.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	481	Da Nang, Vietnam	Same as above	L_{night}, 2200–0600 hours (57.5–67.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	682	Hue, Vietnam	Same as above	L_{night}, 2200–0600 hours (52.5–72.5 dB)
	Phan et al.[57] Shimoyama et al.[58]	781	Thai Nguyen, Vietnam	Same as above	L_{night}, 2200–0600 hours (52.5–67.5 dB)
	Sato et al.[59]	1,302	Gothenburg, Sweden	Does the road traffic noise cause the following conditions? Difficulty to fall asleep? No, Little Disturbed, Rather Disturbed, Very Disturbed[b]	L_{night}, 2200–0700 hours (42.5–72.5 dB)
	Sato et al.[59]	814	Kumamoto, Japan	Same as above	L_{night}, 2200–0700 hours (47.5–77.5 dB)
	Sato et al.[59]	779	Sapporo, Japan	Same as above	L_{night}, 2200–0700 hours (52.5–67.5 dB)
Sleep disturbance: noise mentioned (total $N = 30{,}590$)	NORAH[42,a]	3,162	Frankfurt, Germany 2012	In the last 12 months road traffic noise has disturbed you when sleeping in the night? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (32.5–72.5 dB)
	Brink et al.[46,a]	5,222	Switzerland	Thinking about the last twelve months at your home, during nighttime when you want to sleep, how much did road noise bother, disturb, or annoy you? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2300–0700 hours (22.5–72.5 dB)
	Evandt et al.[37,a]	12,305	Norway	How disturbed have you been by noise during the night (falling asleep and waking up) from road noise in the last 12 months? Not at all, Slightly, Rather, Very,[b] Extremely[b]	L_{night}, 2300–0700 hours (37.5–67.5 dB)
	Brown et al.[60]	8,841	Hong Kong	How much is your sleep disturbed by road traffic noise? 11 point scale used from 0 (not disturbed at all) to 10 (extremely disturbed) (HSD 8,[b] 9,[b] 10[b])	L_{night} (42.5–67.5 dB)
	Hong et al.[61]	550	Korea	How much have you been disturbed in your sleep by road traffic noise at night when you are sleeping in your house over the last 12 months? 11 point scale used from 0	L_{night}, 2200–0700 hours (50.0–73.0 dB)

Table 3. (*Continued.*)

Outcome	Study	N	Location	Disturbance question and responses	Noise metric (level range)
				(not disturbed at all) to 10 (extremely disturbed) (HSD 8,[b] 9,[b] 10[b])	
	Ristovska et al.[62]	510	Macedonia	Do you think that your sleep was disturbed due to night-time noise or noise events during the night in the last twelve months and more? Not at all, Very little, Moderate, High[b], Very High[b]	L_{night}, 2300–0700 hours (42.5–62.5 dB)
Awakenings: noise not mentioned (total $N = 37,338$)	Martens et al.[34,a]	14,622	The Netherlands	How often during the past 4 weeks did you awaken during your sleep time and have trouble falling asleep again? Never, Seldom, Sometimes, Often,[b] Most of the time,[b] Always[b]	L_{den} (32.5–72.5 dB)
	Evandt et al.[37,a]	12,113	Norway	Have you noticed any of the following in the last 12 months? Repeatedly waking with difficulty falling back to sleep? No/rarely, Less than once a week, 1–2 times per week, 3–5 times per week,[b] Almost every night[b]	L_{night}, 2300–0700 hours, (37.5–67.5 dB)
	Bodin et al.[33]	2,519	Sweden	Do you wake up at night? Rarely/never, A few times per month, A few times a week, Almost every day[b]	$L_{AEq,24h}$ (37.5–62.5 dB)
	Frei et al.[63]	1,231	Switzerland	How often does it happen, that you wake up at night multiple times? Never, Rarely, Sometimes, Often[b]	L_{night}, 2200–0600 hours (27.5–62.5 dB)
	Halonen et al.[64]	6,853	Finland	How many times during the past 4 weeks have you had the following symptoms? Frequently waking up during the night. Never, 1 per month, 1 per week, 2–4 per week,[b] 5–6 per week,[b] nearly every night[b]	L_{night}, 2200–0700 hours (42.5–57.5 dB)
Falling asleep: noise not mentioned (total $N = 39,625$)	Bartels et al.[47]	2,188	Sweden	During the past 12 months, how often have you had problems falling asleep in the evening? Never/seldom, A few times per month, Once per week, Several times per week,[b] Every day[b]	L_{night}, 2200–0600 hours (22.5–72.5 dB)
	Martens et al.[34,a]	14,616	The Netherlands	How often during the past 4 weeks did you have trouble falling asleep? Never, Seldom, Sometimes, Often,[b] Most of the time,[b] Always[b]	L_{den} (32.5–72.5 dB)
	Evandt et al.[37,a]	12,276	Norway	Have you noticed any of the following in the last 12 months? Difficulty falling asleep? No/rarely, Less than once a week, 1–2 times per week, 3–5 times per week,[b] Almost every night[b]	L_{night}, 2300–0700 hours, (37.5–67.5 dB)
	Bodin et al.[33]	2,520	Sweden	Do you have problems falling asleep? Rarely/never, A few times per month, A few times a week, Almost every day[b]	$L_{AEq,24h}$ (37.5–62.5 dB)
	Frei et al.[63]	1,232	Switzerland	How often does it happen, that you cannot fall asleep well? Never, Rarely, Sometimes, Often[b]	L_{night}, 2200–0600 hours (27.5–62.5 dB)
	Halonen et al.[64]	6,793	Finland	How many times during the past 4 weeks have you had the following symptoms? Difficulty falling asleep? Never, 1 per month, 1 per week, 2–4 per week,[b] 5–6 per week,[b] Nearly every night[b]	L_{night}, 2200–0700 hours (42.5–57.5 dB)
Sleep disturbance: noise not mentioned (total $N = 24,093$)	Martens et al.[34,a]	14,619	The Netherlands	How often during the past 4 weeks did you feel that your sleep was not quiet (moving restlessly, feeling tense, speaking, etc.) while sleeping? Never, Seldom, Sometimes, Often,[b] Most of the time,[b] Always[b]	L_{den} (32.5–72.5 dB)
	Frei et al.[63]	1,229	Switzerland	How often does it happen that your sleep is restless? Never, Rarely, Sometimes, Often[b]	L_{night}, 2200–0600 hours (27.5–62.5 dB)
	Brink et al.[56]	8,245	Switzerland	During the last 4 weeks, have you suffered from any of the following disorders or health problems? Difficulty in sleeping, or insomnia? Not at all, Somewhat, Very Much[b]	L_{night}, 2200–0600 hours (32.5–77.5 dB)

Note: HSD, highly sleep disturbed; $L_{AEq,24h}$, air and road traffic noise at 24-h average levels; L_{den}, day-evening-night level; L_{night}, nighttime noise; NORAH, Noise-Related Annoyance, Cognition and Health; WHO, World Health Organization.
[a]Studies not included in the WHO review.[19]
[b]Response alternatives designated as highly sleep disturbed.

Table 4. Studies on exposure to railway noise and self-reported sleep outcomes (adapted from the WHO review[19]).

Outcome	Study	N	Location	Disturbance question and responses	Noise metric (level range)
Awakenings: noise mentioned (total $N = 5{,}311$)	Bodin et al.[33]	2,344	Sweden	Do you experience any of the following because of railway noise? You wake up? Never, Sometimes, Often[b]	$L_{AEq,24h}$ (37.5–62.5 dB)
	Sato et al.[65]	1,418	Hokkaido, Japan	How much are you disturbed by awakening during nighttime by train passing? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0700 hours (27.5–62.5 dB)
		1,549	Kyushu, Japan		L_{night}, 2200–0700 hours (27.5–72.5)
Falling asleep: noise mentioned (total $N = 9{,}786$)	NORAH[42,a]	3,266	Frankfurt, Germany 2012	In the last 12 months railway noise has disturbed you when falling asleep? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (27.5–82.5 dB)
	Bodin et al.[33]	2,342	Sweden	Do you experience any of the following because of railway noise? Difficulties falling asleep? Never, Sometimes, Often[b]	$L_{AEq,24h}$ (37.5–62.5 dB)
	Schreckenberg[66]	1,198	Germany	To what extent have the following outcomes of railway noise occurred in the past 12 months? Railway noise disturbs when falling asleep. Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (42.5–82.5 dB)
	Sato et al.[65]	1,418	Hokkaido, Japan	How much are you disturbed in falling asleep by train passing? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0700 hours (27.5–62.5 dB)
		1,562	Kyushu, Japan		L_{night}, 2200–0700 hours (27.5–72.5 dB)
Sleep disturbance: noise mentioned (total $N = 21{,}094$)	NORAH[42,a]	3,266	Frankfurt, Germany 2012	In the last 12 months railway noise has disturbed you when sleeping in the night? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (27.5–82.5 dB)
	Brink et al.[46,a]	3,543	Switzerland	Thinking about the last twelve months at your home, during nighttime when you want to sleep, how much did railway noise bother, disturb, or annoy you? Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2300–0700 hours (22.5–77.5 dB)
	Evandt et al.[37,a]	12,476	Norway	How disturbed have you been by noise during the night (falling asleep and waking up) from rail noise in the last 12 months? Not at all, Slightly, Rather, Very,[b] Extremely[b]	L_{night}, 2300–0700 hours, (37.5–67.5 dB)
	Schreckenberg[66]	1,199	Germany	To what extent have the following outcomes of railway noise occurred in the past 12 months? Railway disturbs when sleeping during the night. Not at all, Slightly, Moderately, Very,[b] Extremely[b]	L_{night}, 2200–0600 hours (42.5–82.5 dB)
	Hong et al.[61]	610	Korea	How much have you been disturbed in your sleep by railway noise at night when you are sleeping in your house over the last 12 months? 11 point scale used from 0 (not disturbed at all) to 10 (extremely disturbed) (HSD 8,[b] 9,[b] 10[b])	L_{night}, 2200–0700 hours (47.1–70 dB)
Awakenings: noise not mentioned (total $N = 16{,}383$)	Evandt et al.[37,a]	12,577	Norway	Have you noticed any of the following in the last 12 months? Repeatedly waking with difficulty falling back to sleep? No/rarely, Less than once a week, 1–2 times per week, 3–5 times per week,[b] Almost every night[b]	L_{night}, 2300–0700 hours, (37.5–67.5 dB)
	Bodin et al.[33]	2,575	Sweden	Do you wake up at night? Rarely/never, A few times per month, A few times a week, Almost every day[b]	$L_{AEq,24h}$ (37.5–62.5 dB)
	Frei et al.[63]	1,231	Switzerland	How often does it happen, that you wake up at night multiple times? Never, Rarely, Sometimes, Often[b]	L_{night}, 2200–0600 hours (27.5–57.5 dB)
Falling asleep: noise not mentioned (total $N = 16{,}553$)	Evandt et al.[37,a]	12,745	Norway	Have you noticed any of the following in the last 12 months? Difficulty falling asleep? No/rarely, Less than once a week, 1–2 times per week, 3–5 times per week,[b] Almost every night[b]	L_{night}, 2300–0700 hours, (37.5–67.5 dB)
	Bodin et al.[33]	2,576	Sweden	Do you have problems falling asleep? Rarely/never, A few times per month, A few times a week, Almost every day[b]	$L_{AEq,24h}$ (37.5–62.5 dB)
	Frei et al.[63]	1,232	Switzerland	How often does it happen, that you cannot fall asleep well? Never, Rarely, Sometimes, Often[b]	L_{night}, 2200–0600 hours (27.5–57.5 dB)
Sleep disturbance: noise not mentioned (total $N = 5{,}914$)	Frei et al.[63]	1,229	Switzerland	How often does it happen that your sleep is restless? Never, Rarely, Sometimes, Often[b]	L_{night}, 2200–0600 hours (27.5–57.5 dB)
	Brink et al.[56]	4,685	Switzerland	During the last 4 weeks, have you suffered from any of the following disorders or health problems? Difficulty in sleeping, or insomnia? Not at all, Somewhat, Very Much[b]	L_{night}, 2200–0600 hours (32.5–77.5 dB)

Note: HSD, highly sleep disturbed; $L_{AEq,24h}$, air and road traffic noise at 24-h average levels; L_{night}, nighttime noise; NORAH, Noise-Related Annoyance, Cognition and Health; WHO, World Health Organization.
[a]Studies not included in the WHO review.[19]
[b]Response alternatives designated as highly sleep disturbed.

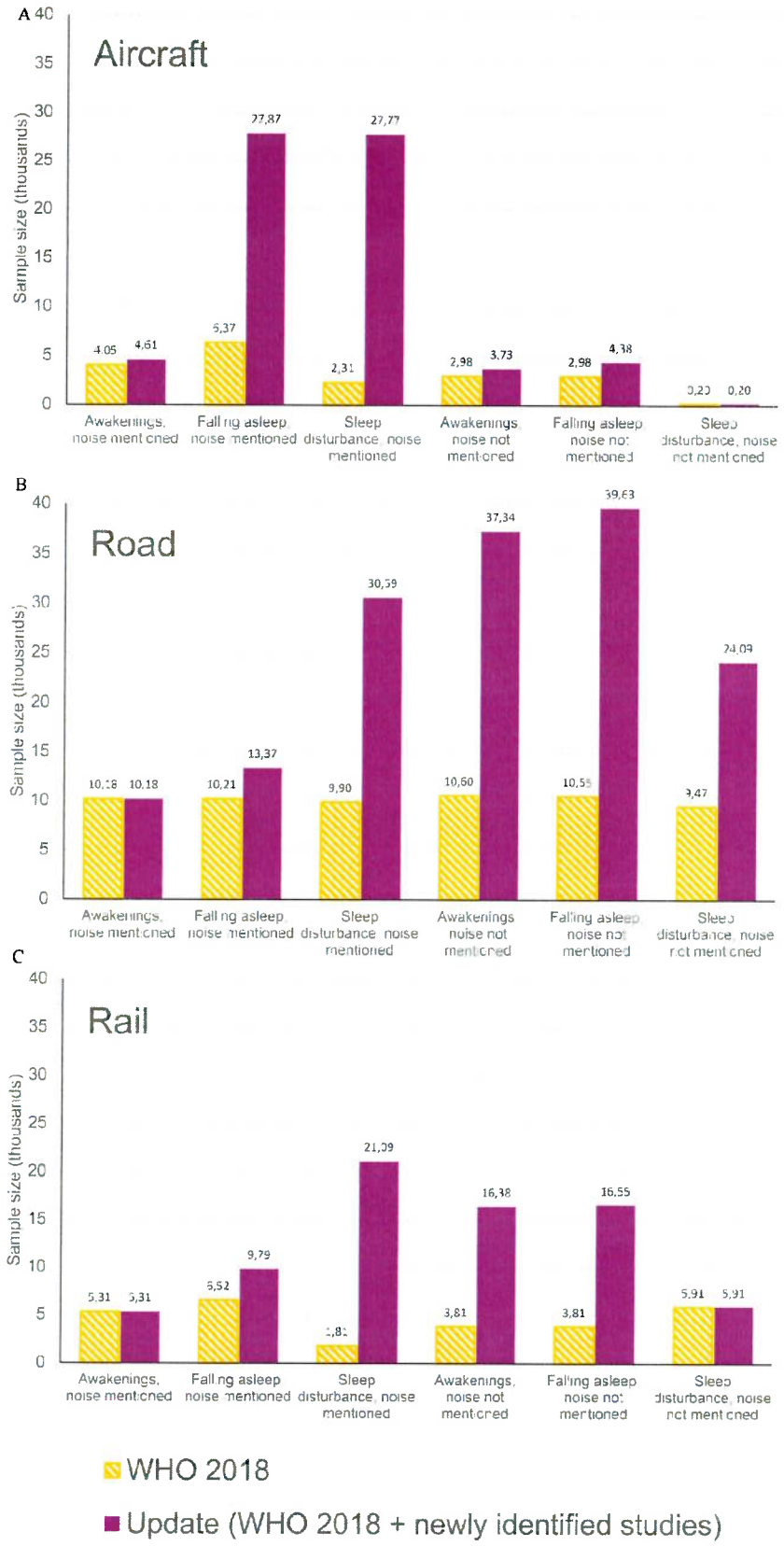

Figure 2. Effective sample sizes for (A) aircraft, (B) road, and (C) rail for each sleep disturbance question in the present updated analysis, determined from the original WHO analysis plus the 11 newly included studies, compared with sample sizes from the WHO 2018 review only.[19] Note: WHO, World Health Organization.

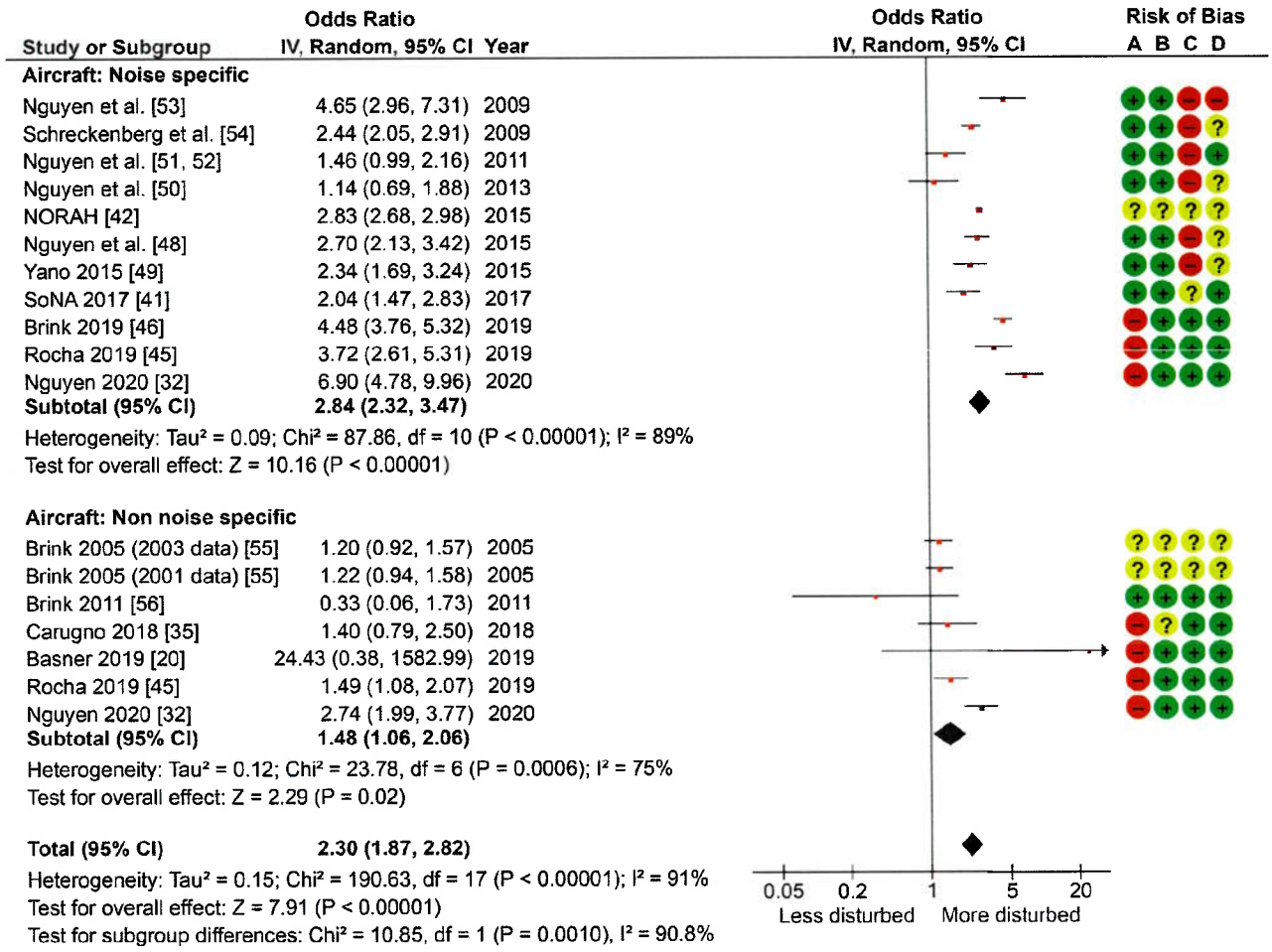

Figure 3. Forest plot for the odds of being highly sleep disturbed by aircraft noise per 10-dB increase in L_{night} (combined estimate derived from all relevant outcomes within studies). Subgroups are presented for questions that mentioned noise as the source of the disturbance, and questions that did not specify noise as the source of the disturbance. Risk of bias: A: selection bias; B: exposure assessment; C: confounding; D: reporting bias. Green (+) denotes low risk of bias, red (–) denotes high risk of bias, yellow (?) denotes unclear risk of bias. Plots were generated using an inverse-variance (IV) random effects method across the full noise range for each individual study (not restricted to 40–65 dB L_{night}). Note: CI, confidence interval; df, degrees of freedom; L_{night}, nighttime noise; NORAH, Noise-Related Annoyance, Cognition and Health.

studies are given in Figure S1. For all three traffic modes, our updated analysis includes a substantially higher number of respondents for all self-reported disturbance questions.

Sleep Disturbance by Noise: Individual Studies

ORs for the probability of being highly sleep disturbed by noise for each study are shown in Figure 3 (aircraft), Figure 4 (road traffic), and Figure 5 (railway). Also shown is the risk of bias assessment for each study (Table S4 for the rationale for each judgment). With a 10-dB increase in L_{night}, there was a statistically significant probability of being sleep disturbed by noise for all three traffic modes. This increased probability was independent of whether noise was specifically mentioned in the sleep question. There were significant differences between the subgroups for each traffic mode, and the ORs were lower in studies that did not specifically mention noise. There was considerable heterogeneity ($I^2 \geq 75\%$) for all three traffic modes when the sleep question mentioned noise. There was substantial heterogeneity ($50\% \leq I^2 \leq 90\%$) between studies of aircraft and road traffic when the sleep question did not specifically mention noise. The heterogeneity between studies of railway noise was deemed unimportant ($I^2 \leq 40\%$) when the sleep question did not specifically mention noise.

Sleep Disturbance by Noise: Overall Analysis

The ORs for the probability of being highly sleep disturbed by nighttime noise, calculated using data from all studies and restricted to 40–65 dB L_{night}, are presented in Table 5. When the question mentioned noise as the source of disturbance, there was a higher probability of being significantly disturbed by noise for all three outcomes, as well as for the combined estimate. When the question did not mention noise, significant relationships were observed only for aircraft and road noise, and for only some of the sleep disturbance outcomes. A substantial proportion of studies into road and railway noise were judged as having a high risk of exposure assessment bias when the question mentioned noise. We decided post hoc to perform a sensitivity analysis for these traffic types, to elucidate the influence of these risks of bias on sleep disturbance. There was a greater probability of being highly sleep disturbed by noise in studies with a low risk of exposure assessment bias compared with studies with a high risk of exposure assessment bias (Table S5).

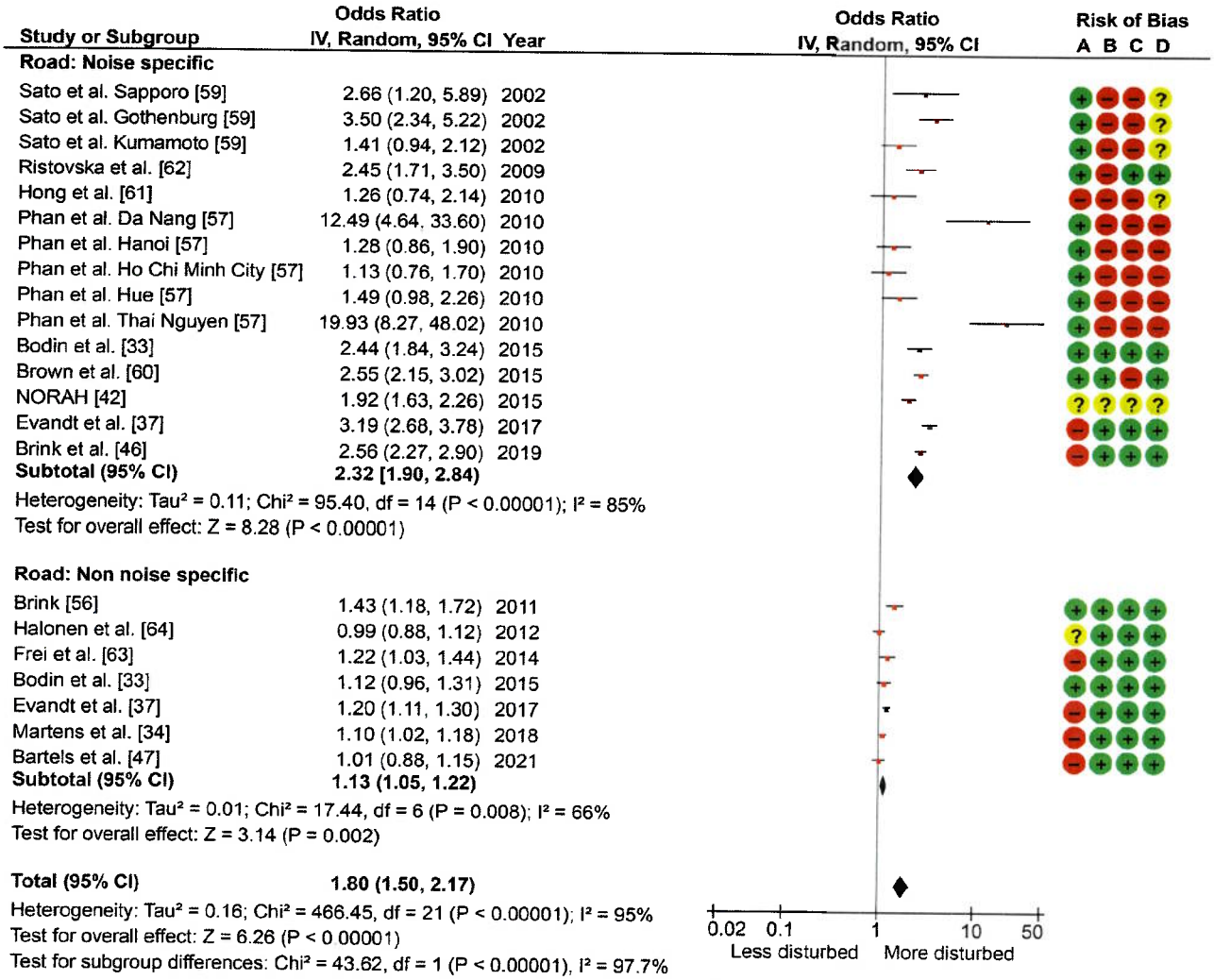

Figure 4. Forest plot for the odds of being highly sleep disturbed by road noise per 10-dB increase in L_{night} (combined estimate derived from all relevant outcomes within studies). Subgroups are presented for questions that mentioned noise as the source of the disturbance, and questions that did not specify noise as the source of the disturbance. Risk of bias: A: selection bias; B: exposure assessment; C: confounding; D: reporting bias. Green (+) denotes low risk of bias, red (–) denotes high risk of bias, yellow (?) denotes unclear risk of bias. Plots were generated using an inverse-variance (IV) random effects method across the full noise range for each individual study (not restricted to 40–65 dB L_{night}). Note: CI, confidence interval; df, degrees of freedom; L_{night}, nighttime noise; NORAH, Noise-Related Annoyance, Cognition and Health.

The ORs for the probability of being highly sleep disturbed, stratified by studies performed in Europe and outside of Europe, are given in Table S6. Analyses were restricted to aircraft, road, and railway traffic when the question mentioned noise, plus aircraft traffic when noise was not specifically mentioned, because these were the outcomes where sleep disturbance data were available for both locations. Non-European study respondents were more highly sleep disturbed by railway traffic when noise was mentioned in the question and by aircraft traffic when noise was not specifically mentioned. Non-Europeans were also less disturbed by road traffic when noise was mentioned. However, none of these effects were significant.

Exposure–Response Curves: Questions Specifically Mentioning Noise

The exposure–response curves for the probability of being highly sleep disturbed, derived using data from questions that specifically mentioned noise, are given in Figure 6. Second-order polynomial equations for each curve are given in Table S7. Disturbance was substantially higher for aircraft noise for all three disturbance questions than for road or railway noise of the same level. Disturbance was similar for road and rail noise at low noise levels, and it was slightly higher for railway noise than road noise at higher noise levels.

We compared the updated exposure–response curves to curves derived using only the 11 new studies published since the WHO review[19] (Figure 7). This was done for the combined estimate only, given that there was a limited sample size for certain sleep questions in these recent studies. For aircraft noise, the recent studies indicated a higher probability of being highly sleep disturbed compared with the analysis incorporating all available data. For road traffic noise, the point estimates were slightly higher at the highest noise levels in the recent studies compared with the overall analysis (2.6% higher at 65 dB L_{night}). For railway noise, the recent studies were essentially identical to the overall analysis.

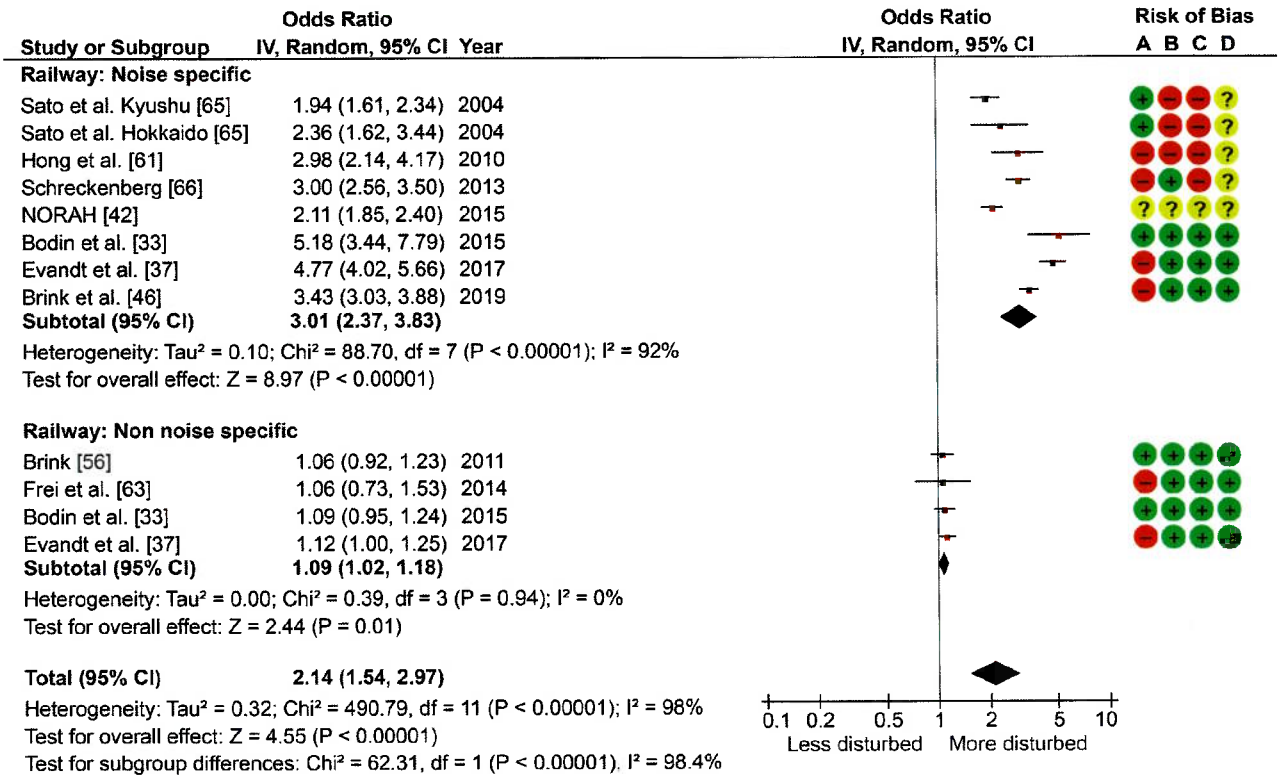

Figure 5. Forest plot for the odds of being highly sleep disturbed by railway noise per 10-dB increase in L_{night} (combined estimate derived from all relevant outcomes within studies). Subgroups are presented for questions that mentioned noise as the source of the disturbance, and questions that did not specify noise as the source of the disturbance. Risk of bias: A: selection bias; B: exposure assessment; C: confounding; D: reporting bias. Green (+) denotes low risk of bias, red (−) denotes high risk of bias, yellow (?) denotes unclear risk of bias. Plots were generated using an inverse-variance (IV) random effects method across the full noise range for each individual study (not restricted to 40–65 dB L_{night}). Note: CI, confidence interval; L_{night}, nighttime noise; NORAH, Noise-Related Annoyance, Cognition and Health.

The exposure–response curves calculated in the original WHO review[19] are given in Figure 6. Relationships for the sleep disturbance question were not calculated in the WHO review due to an insufficient number of studies at the time. Point estimates for aircraft noise are generally slightly higher in the present analyses compared with the previous relationships, particularly at higher noise levels, although they still lie within the 95% CIs of the WHO review. Point estimates for the falling asleep and combined estimate outcomes are almost identical for road and rail traffic in the present analysis compared with the WHO review. For each disturbance question and traffic mode, all of the previous curves lie within the 95% CIs of the updated analyses. As expected, given that no additional studies were included for awakenings by aircraft or road traffic, exposure–response curves for these outcomes were identical to curves in the WHO review.

Table 5. Odds ratios per 10-dB increase in L_{night} for the percent highly sleep disturbed by aircraft, road, and railway traffic noise.

		Noise mentioned as source of disturbance			Noise not mentioned as source of disturbance		
Noise source	Outcome	Studies (n)[a]	Sample size (n)[a]	OR per 10 dB (95% CI)	Studies (n)[a]	Sample size (n)[a]	OR per 10 dB (95% CI)
Aircraft noise	Awakenings	6	4,137	2.34 (1.87, 2.93)	5	2,571	1.11 (0.81, 1.53)
	Falling asleep	8	17,107	2.09 (1.91, 2.28)	7	3,120	1.67 (1.27, 2.19)
	Sleep disturbance	5	15,345	2.28 (2.03, 2.56)	1	153	1.22 (0.08, 18.20)
	Combined estimate	11	19,488	2.18 (2.01, 2.36)	8	3,275	1.52 (1.20, 1.93)
Road noise	Awakenings	8	5,355	1.75 (1.24, 2.47)	5	29,358	1.10 (1.01, 1.20)
	Falling asleep	9	7,754	2.31 (1.85, 2.89)	6	31,136	1.15 (1.08, 1.23)
	Sleep disturbance	6	26,372	2.57 (2.26, 2.93)	3	18,052	1.15 (0.93, 1.43)
	Combined estimate	14	31,738	2.52 (2.28, 2.79)	7	38,380	1.14 (1.08, 1.21)
Railway noise	Awakenings	3	3,576	2.54 (1.49, 4.33)	3	3,197	1.09 (0.78, 1.53)
	Falling asleep	5	6,730	2.70 (2.14, 3.42)	3	3,219	1.27 (0.84, 1.90)
	Sleep disturbance	5	7,262	3.35 (2.75, 4.09)	2	1,168	1.27 (0.11, 15.15)
	Combined estimate	8	10,846	2.97 (2.57, 3.43)	4	4,326	1.17 (0.91, 1.49)

Note: ORs were calculated in logistic regression models with L_{night} included as the only fixed effect and study included as a random effect, restricted to the noise exposure range 40–65 dB L_{night}. Models were run separately for each traffic mode and for sleep questionnaire outcomes that did or did not mention noise. The combined estimate was calculated using average responses of the awakening, falling asleep, and sleep disturbance questions within studies. CI, confidence interval; L_{night}, nighttime noise; OR, odds ratio.
[a]In the L_{night} range 40–65 dB for which ORs were calculated.

Exposure–Response Curves: Questions Not Specifically Mentioning Noise

The exposure–response curves for the probability of being highly sleep disturbed, derived using data from general sleep questions that did not specifically mention noise, are given in Figure 8. Second-order polynomial equations for each curve are given in Table S7. With increasing L_{night}, there was a small increase in disturbance for all questions, although the gradient of the exposure–response curves was generally smaller compared with questions that mentioned noise (Figure 6). The differences between the three traffic modes were also less clear compared with questions mentioning noise (Figure 6).

Quality of Evidence for Being Highly Sleep Disturbed by Noise

Funnel plots of the combined estimate for each traffic mode are given in Figure S2. The plots were approximately symmetrical, indicating a low likelihood of publication bias.

The GRADE profile for the assessment of the quality of evidence across studies is given in Table 6. In the assessment, we deemed that for the majority of studies to be considered high quality (study limitations domain), there should be a low risk of selection bias and also a low risk of exposure assessment bias. If there was a high risk for one or both of these biases in the majority of studies, then overall study quality was deemed low. The overall quality of evidence for nighttime noise from aircraft, road, and railway traffic was rated as moderate when the question mentioned noise. When the question did not mention noise, the quality of evidence was low for aircraft and road traffic noise and very low for railway noise.

Discussion

Noise-Specific Sleep Disturbance

In an update to the latest WHO evidence review and meta-analysis for the effects of traffic noise on self-reported sleep disturbance,[19] we found significant exposure–response relationships for being highly sleep disturbed by nighttime aircraft, road, and railway traffic when the sleep questions explicitly mentioned noise. With increasing nighttime noise levels, and for all three traffic modes, there were increased probabilities of reporting awakenings, having difficulties falling asleep, or having disrupted or disturbed sleep. When the sleep disturbance outcomes were combined for each traffic mode separately, the resulting exposure–response curves for road and railway noise were very similar to those calculated in the WHO review (Figure 6). The similarity in the exposure–response curves improves confidence in the earlier estimates, which informed recent WHO recommendations for nighttime noise from road (45 dB L_{night}) and rail (44 dB L_{night}).[15] For aircraft noise, our updated estimates show a higher probability of being highly sleep disturbed at high L_{night} levels. At 40 dB L_{night}, however, which is the WHO recommendation for nighttime aircraft noise,[15] our updated estimates closely match the point estimates from the previous evidence review.[19]

The ORs for aircraft noise were lower than for both road and railway noise. This is a consequence of the properties of ORs as a relative measure, given that a much higher proportion of people were sleep disturbed by aircraft noise at low reference noise levels. The exposure–response curves show that aircraft noise was in fact more disturbing than road or rail noise of the same level. This finding, although also seen in the original WHO review,[19] is superficially surprising in light of experimental studies showing that aircraft noise is less disruptive to physiological sleep than road or rail traffic.[67] The reasons for higher self-reported disturbance by aircraft are unclear but could result from the timing of aircraft noise events. Nighttime noise levels from aircraft are typically dominated by passenger plane takeoffs and landings that occur at the very start and the very end of the night period (2300–0700 hours). The early night is a period when many people are trying to fall asleep, and the end of the night is a period when people may be awakened by noise more easily, or have greater difficulty falling back asleep after awakenings, because sleep pressure has been dissipated over the preceding night. Noise around these times could therefore have a greater impact on self-reported disturbance than at other times of night. Such an explanation is supported by the higher disturbance for specific questions on awakenings and difficulties falling asleep owing to aircraft noise.

It is also possible that the higher disturbance by aircraft is a result of exposure misclassification. In most studies, noise was assessed at the most exposed façade, and the exposure levels specifically in the bedroom are not known. Noise levels in the bedroom for road and railway traffic are most likely lower, on average, than at the most exposed façade, because bedrooms may be located on quieter sides of the building. There is probably less exposure misclassification for aircraft noise, especially for homes that lie under flight paths, given that the positions of aircraft as noise sources are more dynamic relative to the home. Finally, it is possible that particular characteristics of air traffic are somehow more disturbing than road or rail noise of the same level. Aircraft noise events have a much longer duration than the other traffic modes, and so there are longer windows to become cognizant of the noise and attribute it as a source of sleep disturbance. However, each of these explanations cannot be thoroughly explored without additional temporal, spatial, and acoustical data for the noise sources.

Non-Noise-Specific Sleep Disturbance

The probability of being highly sleep disturbed was less clear when studies used general sleep questions that did not mention noise. For those sleep outcomes, all ORs were in the same direction and >1.0, suggesting potentially increasing disturbance with noise level. However, the effect sizes were smaller compared with noise-specific questions, and they were significant for only a minority of outcomes (5 of 12) assessed across all traffic modes.

Differences in sleep disturbance between studies employing general sleep questions and studies that specifically mention noise could result from heterogeneity between studies generally, which is discussed in detail later. When a question mentions a particular traffic source, a respondent may be better able to correctly attribute noise-induced sleep disturbance to that source, which could also explain the higher effect sizes in studies mentioning noise. Misattributing noise as the reason for an endogenous sleep is also possible, for instance, if respondents awaken spontaneously in the absence of noise, and a noise event that is later recalled coincidentally occurs during the awakening bout. A further important effect modifier could be noise sensitivity. Because noise-sensitive individuals may be more likely to report sleep disturbance than their less-sensitive counterparts,[68–70] they might rate themselves as more sleep disturbed to questions explicitly mentioning noise.

Risk of Bias, Quality of Evidence, and Study Heterogeneity

Most newly included studies were rated as having a high risk of selection bias. In most cases, this was due to response rates being <50%. Low survey response rates in public health research are becoming increasingly common,[71] something that can increase

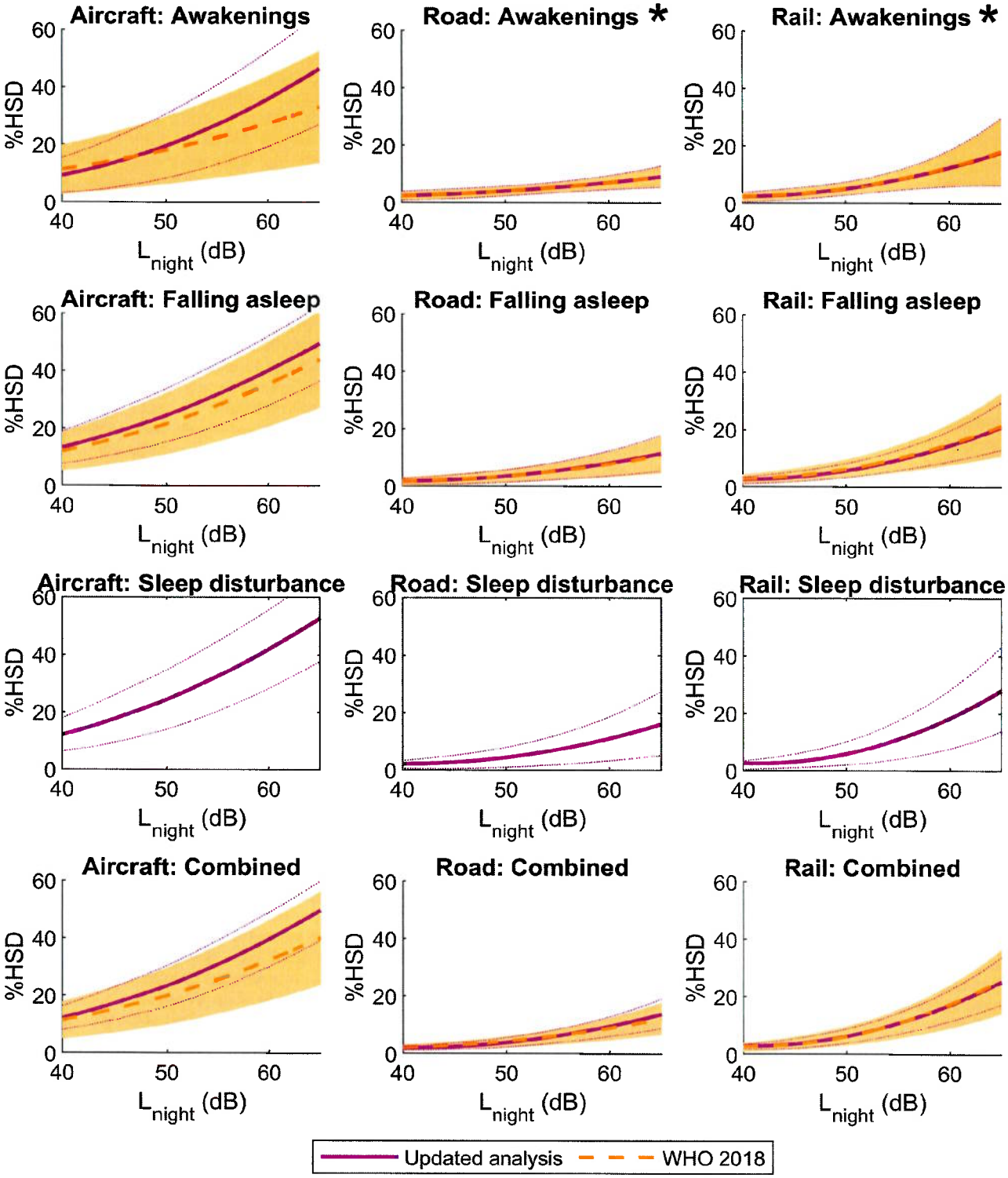

Figure 6. Probability of being highly sleep disturbed (%HSD) by nighttime noise, determined via questions that mention noise as the source of disturbance, stratified by disturbance question and traffic mode. Exposure–response relationships were derived using all available data, from the original WHO review[19] and the 11 newly identified studies. Results of the present updated analysis (solid purple lines with dotted 95% CIs) are compared against results of the 2018 WHO review[19] (dashed orange lines with shaded 95% CIs). Relationships for the sleep disturbance questions were not calculated previously. Asterisks (*) indicate sleep outcomes for which no new studies have been published since the WHO review. Parameter estimates were calculated in logistic regression models with L_{night} included as the only fixed effect and study included as a random effect, restricted to the noise exposure range 40–65 dB L_{night}. Models were run separately for each traffic mode and disturbance question. The combined estimate was calculated using average responses of the awakening, falling asleep, and sleep disturbance questions within studies. Note: CI, confidence interval; L_{night}, nighttime noise; WHO, World Health Organization.

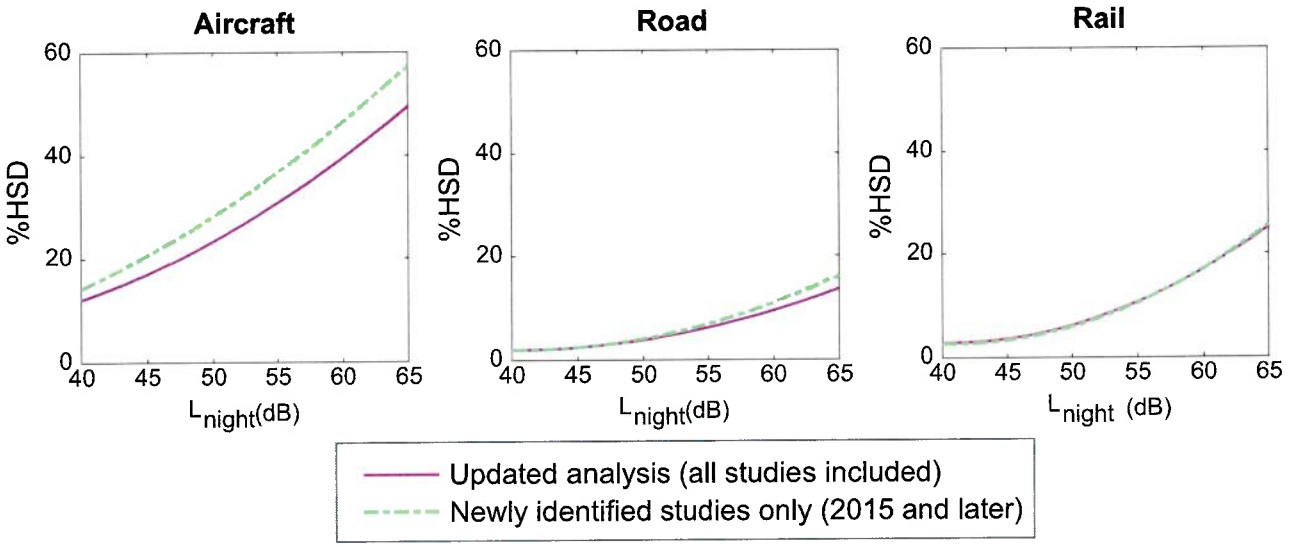

Figure 7. Exposure–response relationships for the probability of being highly sleep disturbed (%HSD) by nighttime noise for questions that mention noise. Curves are shown for the updated analysis that includes all available data (solid purple lines), and for analysis including only newly identified studies published after the WHO review[19] (dashed green lines). Data are calculated as the combined response using average responses of the awakening, falling asleep, and sleep disturbance questions within studies, determined as the within-study average of disturbance questions that explicitly mentioned noise as the source of sleep disturbance. Parameter estimates were calculated in logistic regression models with L_{night} included as the only fixed effect and study included as a random effect, restricted to the noise exposure range 40–65 dB L_{night}. Models were run separately for each traffic mode. Note: L_{night}, nighttime noise; WHO, World Health Organization.

the risk of nonresponse bias.[72] However, nonresponse bias can occur in studies with both low and high response rates.[73] More important than response rates is that the survey responses are representative of the target population sampled,[74] and surveys can still be representative even with lower response rates. Lacking nonresponse analyses, we cannot be certain of the representativeness of the exposure–response relationships, although the high risk of selection bias in the included studies does not necessarily mean that the sleep outcomes are unrepresentative of the overall population exposed to noise. Further studies with increased response rates would decrease the likelihood of nonresponse bias.

Sensitivity analysis revealed that sleep disturbance was lower in studies with a high risk of exposure assessment bias. One possible explanation is that road and railway noise exposure in the bedroom was overestimated in studies judged to have a high risk of bias. This would, in effect, shift the exposure–response relationships to the right in these studies. Alternatively, differences in sleep disturbance could be confounded by the fact that all studies with high risk of exposure assessment bias were published between 2002 and 2010, whereas the low risk of bias studies were from published more recently, between 2013 and 2021. It is plausible that the higher probability of high sleep disturbance in newer studies is attributable to nonacoustical factors, such as changes in attitudes to noise. Temporal changes in self-reported response would align with observed trends for increasing annoyance by a given level of traffic noise, although these trends have been observed predominantly for aircraft rather than road or rail traffic.[75] There have also been changes in the acoustical character of noise, with newer vehicles being typically quieter but with noise occurring more often as traffic flows increase, which may negatively influence perceived sleep disturbance.

The overall quality of evidence differed between studies where sleep disturbance questions did or did not mention noise. The assessment of a moderate quality of evidence for sleep disturbance when the question mentioned noise agrees with the assessment in the WHO review.[19] When the question did not specifically mention noise, we graded the quality of evidence for exposure to railway noise as very low, again agreeing with the WHO review, and the quality of evidence as low for aircraft and road traffic noise, which is one level higher than the very low quality assessment in the WHO review. The reason for the upgrade for aircraft and road noise was due to the statistically significant trends for awakenings (road only), falling asleep, and the combined estimates, that were not found previously. Since the previous review, three major cross-sectional studies involving road traffic noise exposure, with a combined sample size of ~29,000 respondents, were published.[34,37,47] The exposure–response relationships for non-noise-dependent disturbance are thus more representative, and with substantially greater power, than previously found.

There was substantial heterogeneity between studies for all outcomes except studies of railway noise that employed general sleep questionnaires. The heterogeneity could result from variations in the specific phrasing of the sleep disturbance question across studies, even when ostensibly measuring the same outcome. There was also a diverse range of response scales, with 11-point numerical and 3-, 4-, or 5-point verbal scales used to assess sleep disturbance, further diversified by assessing either the severity or the frequency of disturbance. These questions were administered in 14 nations, hence, there may be linguistic differences in the interpretation of certain phrases, as well as cultural differences in attitudes to sleep or noise, as well as contextual differences generally across specific studies. Questions also differed in the reference time frame for sleep disturbance, varying from the last 12 months to the last 4 wk to referencing specific noise events or no time frame at all. Finally, self-reported response to noise can be modified by contextual factors separate from noise level alone, including lifestyle, access to green space, access to quiet areas, social interaction, recreational activities, and local economy of the neighbourhood.[76] One or several of these factors could have contributed to study heterogeneity within specific sleep outcomes, across studies of different traffic modes, or across studies that used either general sleep questions or noise-specific disturbance questions.

Study Location

The majority of new studies originated from Europe. All newly included studies of road[34,37,42,46,47] and railway[37,42,46] noise were

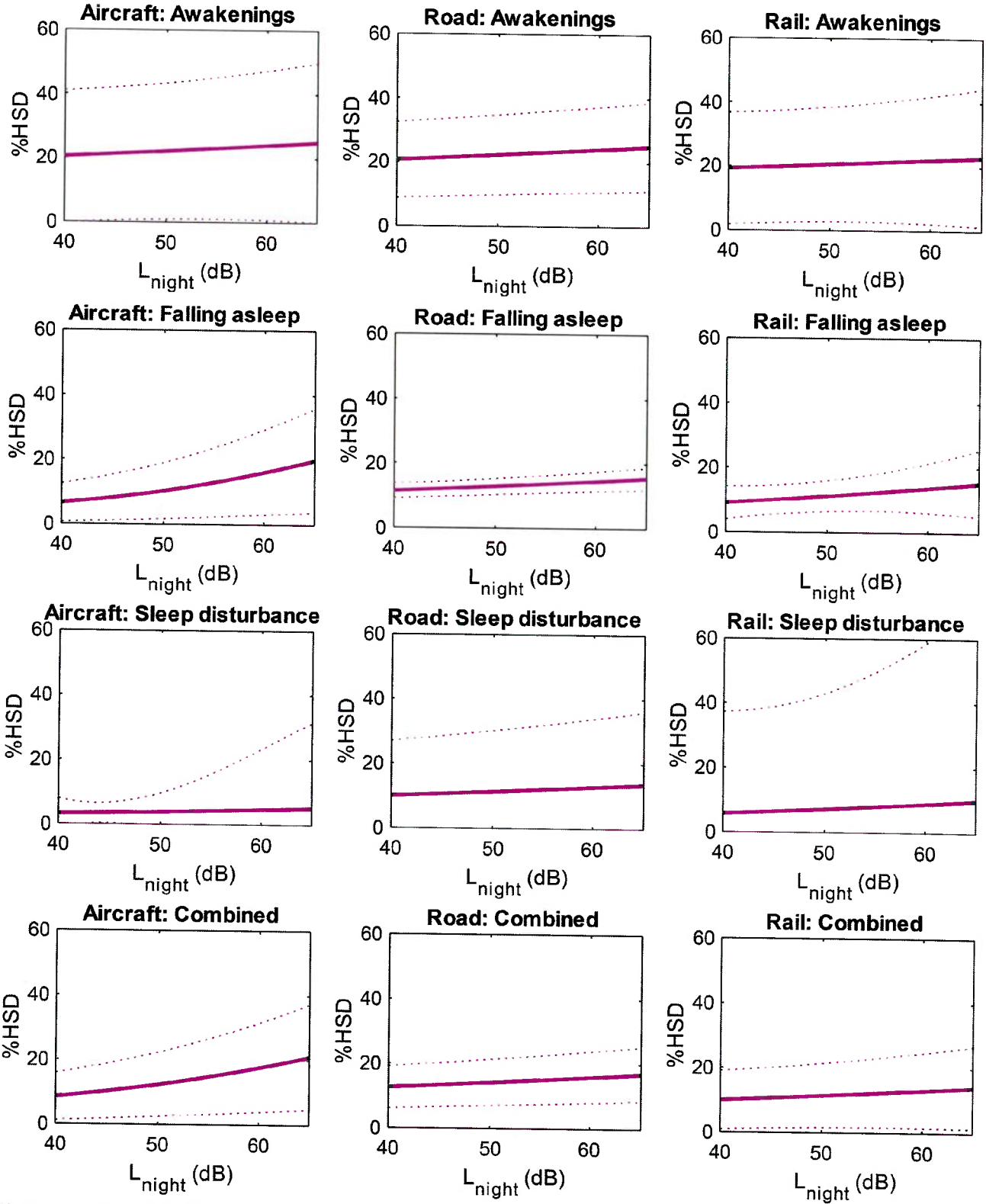

Figure 8. Probability of being highly sleep disturbed (%HSD) by nighttime noise, determined via questions that did not specifically mention noise as the source of disturbance, stratified by disturbance question and traffic mode. Exposure–response relationships were derived using all available data, from the original WHO review[19] and the 11 newly identified studies. Dotted lines indicate 95% CIs. Parameter estimates were calculated in logistic regression models with L_{night} included as the only fixed effect and study included as a random effect, restricted to the noise exposure range 40–65 dB L_{night}. Models were run separately for each traffic mode and disturbance question. The combined estimate was calculated using average responses of the awakening, falling asleep, and sleep disturbance questions within studies. Note: CI, confidence interval; L_{night}, nighttime noise; WHO, World Health Organization.

Table 6. GRADE Evidence profile (adapted from the WHO review[19]).

Domain	Criterion	Assessment	Grade change
Sleep disturbance questions: noise mentioned			
Start level	Longitudinal = high; others = low	All cross-sectional studies	Low quality
Study limitations	Majority of studies low quality	Aircraft and railway: majority of studies have low risk of selection bias (10/19) and exposure assessment bias (14/19)	Aircraft and railway: no downgrade
		Road: majority of studies have low risk of selection bias (11/15) and high risk of exposure assessment bias (10/15)	Road: downgrade one level
Inconsistency	Conflicting results; high I^2	High heterogeneity between studies ($I^2 \geq 85\%$)	Downgrade one level
Indirectness	Direct comparison; same PECO	Same PECO	No downgrade
Precision	CIs contain 25% harm or benefit	CIs narrower than 25% except for few outcomes at high noise levels	No downgrade
Publication bias	Indicated by funnel plot	Symmetrical plots	No downgrade
Judgment after downgrades	—	—	Aircraft, road, and railway: very low quality
Dose–response	Significant trend	Statistically significant trend for all outcomes	Upgrade one level
Magnitude of effect	RR > 2	OR > 2 for 11 of 12 outcomes	Upgrade one level
Confounding adjusted	Effect in spite of confounding working toward the null	Not observed	No upgrade
Overall judgment	—	—	Aircraft, road, and railway: moderate quality
Sleep disturbance questions: noise not mentioned			
Start level	Longitudinal = high; others = low	All cross-sectional studies	Low quality
Study limitations	Majority of studies low quality	Majority of studies have high risk of selection bias (10/18) and low risk of exposure assessment bias (15/18)	Downgrade one level
Inconsistency	Conflicting results; high I^2	Railway: unimportant heterogeneity between studies ($I^2 = 0\%$)	Railway: no downgrade
		Aircraft and road: substantial to considerable heterogeneity between studies ($65\% \leq I^2 \leq 75\%$)	Aircraft and road: downgrade one level
Indirectness	Direct comparison; same PECO	Same PECO	No downgrade
Precision	CIs contain 25% harm or benefit	Wide CIs	Downgrade one level
Publication bias	Indicated by funnel plot	Symmetrical plots	No downgrade
Judgment after downgrades	—	—	Aircraft, road, and railway: very low quality
Dose–response	Significant trend	Aircraft: statistically significant trend for falling asleep and combined estimate	Railway: no upgrade
		Railway: not significant	
		Road: statistically significant trend for awakenings, falling asleep and combined estimate	Aircraft and road: upgrade one level
Magnitude of effect	RR > 2	OR < 2 for all outcomes	No upgrade
Confounding adjusted	Effect in spite of confounding working toward the null	Not observed	No upgrade
Overall judgment	—	—	Railway: very low quality Aircraft and road: low quality

Note: —, not applicable; CI, confidence interval; GRADE, Grading of Recommendations, Assessment, Development, and Evaluations (criteria); OR, odds ratio; PECO, Patient/Problem, Exposure, Comparison and Outcome (framework); RR, risk ratio; WHO, World Health Organization.

European, as were the majority of respondents across the studies of aircraft noise.[35,41,42,46] Although there was one study of aircraft noise from Asia,[32] and three from the United States,[20,21,45] these studies were small, with sample sizes ranging from $n = 33$ to $n = 559$. European studies continue to be overrepresented (Figure S3). However, we found no statistically significant differences in sleep disturbance between European and non-European studies. On one hand, this suggests that there are, in fact, no differences in response between the two locations, that the degree of sleep disturbance by noise is rather global in nature, and that results of the present analyses are relevant outside of Europe. Conversely, the point estimates were rather different between study location for several sleep disturbance outcomes. This could indicate underlying cultural differences in attitudes to noise and perceived sleep disturbance that have not been captured in studies to date. Future investigations outside of Europe may uncover relevant international differences, as well as increasing confidence that existing studies are representative of noise-induced sleep disturbance among these underinvestigated regions.

Considerations on Self-Reported Sleep Disturbance

Our overall findings of self-reported disturbance by noise should be treated with some caution when considering noise-induced effects on sleep. Sleep is, by its nature, an unconscious process, meaning that its subjective evaluation is difficult. Accordingly, there can be substantial differences between self-reported and physiologically derived measures of sleep and noise-induced sleep disturbance.[77–79] Self-report may also suffer from recall bias, particularly when questions relate to the preceding 12 months, as was typical for questions on sleep disturbance in most studies included in our meta-analysis. It is likely that responses to

Table 7. Summary of meta-analytic and evidence quality findings.

Sleep outcome	Noise source	Studies (n)[a]	Respondents (n)[a]	Quality of evidence	Noise metric	OR per 10-dB increase (95% CI)
Self-reported sleep disturbance in adults	Aircraft	11	19,488	Moderate	L_{night}	2.18 (2.01, 2.36)
Noise specified as source of disturbance	Road	14	31,738	Moderate	L_{night}	2.52 (2.28, 2.79)
	Railway	8	10,846	Moderate	L_{night}	2.97 (2.57, 3.43)
Self-reported sleep disturbance in adults	Aircraft	8	2,571	Low	L_{night}	1.52 (1.20, 1.93)
Noise not specified as source of disturbance	Road	7	38,380	Low	L_{night}	1.14 (1.08, 1.21)
	Railway	4	4,326	Very low	L_{night}	1.17 (0.91, 1.49)

Note: ORs were calculated in logistic regression models with L_{night} included as the only fixed effect and study included as a random effect, restricted to the noise exposure range 40–65 dB L_{night}. Models were run separately for each traffic mode and for sleep questionnaire outcomes that did or did not mention noise. Data shown are for the combined estimates calculated using average responses of the awakening, falling asleep, and sleep disturbance questions within studies. L_{night}, nighttime noise; OR, odds ratio.
[a]In the L_{night} range 40–65 dB for which ORs were calculated.

questions on these timescales are driven by noise exposure in the more recent past. However, self-reported sleep outcomes are methodologically convenient and inexpensive to implement in field studies, meaning that we could perform the meta-analysis with a number of studies and sample size that would not have been possible if focusing on physiologic outcomes. As such, we have higher confidence in the accuracy and representativeness of the analysis. A further advantage is that self-reported disturbance is a valuable end point per se, considered by the WHO as a primary health outcome. By focusing our analysis on these outcomes, the results may be useful in future estimates of the disease burden of environmental noise[80] and recommendations for nighttime noise limits,[15] both of which derive from self-reported sleep disturbance. Finally, self-reported outcomes capture habitual sleep quality and disturbance, unlike physiologic measurements that capture only acute effects within single nights. It does, however, remain unclear how long-term self-reported sleep disturbance by noise relates to overall health.

Future large-scale field studies with objective measurements of noise and sleep can offer mechanistic insights linking nocturnal noise, sleep disruption, and epidemiological observations of the development of cardiovascular and metabolic disease associated with exposure to environmental noise in addition to the derivation of exposure–response relationships.[81] A better understanding of the underlying pathophysiological pathways is especially valuable when considering vulnerable populations who may be at increased risk of disturbance. These vulnerable groups include the elderly, who can suffer from age-related declines in sleep quantity and quality[82]; populations who may have already poor sleep quality, such as people with mental health or sleep disorders[83]; and populations with obesity, who are at increased risk of suffering from obstructive sleep apnea, as well as having increased risk for cardiometabolic diseases generally.[84,85] Infants, children, and adolescents can also be considered as vulnerable groups because of the importance of sleep of sufficient quality and duration for development.[80,86,87]

Limitations

Data could not be obtained for two studies that were initially deemed to be eligible for inclusion. It is unlikely that including the study of road traffic noise[44] would have substantially altered the updated relationships because the sample size was low ($n = 225$) compared with the overall sample size for all road traffic studies ($n = 31{,}738$). Including the study of aircraft noise,[43] however, may have altered the sleep outcomes where noise was not mentioned for falling asleep, sleep disturbance, and the combined estimate. Compared with sample sizes of $n = 4{,}379$ for questions on falling asleep and just $n = 195$ for sleep disturbance questions that were included in our analysis, the omitted study had a sample size of $n = 2{,}831$, which would have reflected a substantial proportion of the total data set. The change in effect size that would have resulted from including this study is unclear because the relevant sleep-disturbance questions were single items that formed only part of the insomnia severity index (ISI). Because only overall results from the ISI were published, we do not know whether the relevant items were related to noise exposure, or to what extent.

A limitation of the meta-analysis was that many studies modeled noise exposure at the most exposed façade of the residence, and thus noise levels specifically at the bedroom façade are unknown. This means there is probably some exposure misclassification, with lower noise levels if the bedroom faces away from the noise source. This is more likely for road and railway noise than aircraft noise, with the latter source being less fixed in position relative to the bedroom. This would, in effect, shift the exposure–response curves to the left, leading to an increased probability of disturbance at lower noise levels, given that noise levels at the bedrooms are, on average, probably lower than assuming they are all positioned at the most exposed façade. This was supported by two studies in the meta-analysis that found that a lower proportion of respondents were highly sleep disturbed by road traffic noise[46] or reported insomnia symptoms[37] when the bedroom faced away from the street. Furthermore, disturbance was lower when the difference in noise level between the bedroom and the most exposed façade was greater.[46] A second limitation of the meta-analysis is that we did not adjust for potentially relevant effect modifiers. We adopted this approach so that results would be directly comparable to those in the WHO review, which also did not include such adjustments.[19] Sleep, and its disturbance by noise, may differ depending on age, sex, socioeconomic status, and preexisting sleep disorders. Further, sleep disturbance is not unique to noise exposure and may arise from other environmental stressors, including air pollution,[88–90] vibration (from, for instance, freight trains on railway lines),[91] light,[92] and temperature and humidity.[93,94] Future studies should consider the consequences of exposure to multiple stressors, and their interactions on sleep.

Summary of Evidence

Our main objective was to update the WHO meta-analysis on sleep disturbance by traffic noise with evidence published after 2015.[19] The main findings and quality of evidence are summarized in Table 7. There was a significant probability of being highly sleep disturbed by nocturnal noise from aircraft, road, and railway noise when the disturbance question mentioned noise, and the quality of evidence for these outcomes was moderate. Exposure–response curves were similar to the WHO review for road and railway noise in our updated analysis, and we found an increased probability of being highly sleep disturbed by aircraft noise at high noise levels. Because of the number of studies published since 2015, for the first time, we were able to generate exposure–response relationships for sleep outcomes that did not

explicitly mention noise. Point estimates for these outcomes were smaller than questions mentioning noise, and were often not statistically significant, and the quality of evidence was graded lower, from low to very low. Our findings do not suggest that the recent WHO recommendations for nighttime noise need to be revisited,[15] although quantitative assessments of sleep disturbance by aircraft noise at high exposure levels should consider the implications of our analysis. We did not find significant indications of international differences in sleep disturbance by noise, but future large-scale studies in non-European nations may necessitate a reevaluation of the evidence.

Acknowledgments

Contributions of the authors are as follows—M.G.S.: investigation, formal analysis, data curation, writing (original draft), and visualization; M.C.: investigation and writing (review and editing); and M.B.: conceptualization, methodology, formal analysis, writing (review and editing), supervision, and funding acquisition.

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Editorial

Editorial

The effects on sleep play a critical role in the long-term health consequences of noise exposure

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Sleep is a very dangerous state from an evolutionary perspective as we are unconscious and it takes time to arouse from sleep and react to threats in a meaningful way. Thus, it is not surprising that our full sensory array continues to monitor our environment during sleep, always ready to wake us up (although with different sensitivity depending on sleep stage). In fact, the multiple brief awakenings a healthy sleeper experiences during the night [1] can be thought of as "brief checks" into our sleep environment. As a long-range sensor, the auditory system plays a critical role in monitoring the environment during sleep. It analyzes not only sound levels but also sound content during sleep [2]. The thalamus has a gating function, shielding the cortex from sensory content deemed irrelevant, often associated with a K-complex in the electroencephalogram [3]. We do habituate to noise, but still react to individual noise events during sleep even after long exposure periods (i.e. years), albeit with lower probabilities. Compared to cortical arousals, autonomic arousals habituate to a much lesser degree with likely implications for long-term health consequences (see below) [4].

While the watchman function of the auditory system has served us well on our evolutionary journey, it is less relevant in modern societies where humans sleep in solid housing structures, safe from predators. However, there is no way to switch off biology that has evolved over millennia. Countless studies have unequivocally shown that exposure to traffic noise disturbs sleep and impairs sleep recuperation, without posing any obvious threat [5, 6]. Sensitivity to noise-induced sleep disturbance varies substantially inter-individually [7]. Interestingly, there seems to be a "sweet spot" for the propensity to arouse to internal or external stimuli, with mortality increasing for both very low (e.g. more severe oxygen desaturations in OSA patients?) and very high (e.g. higher degree of noise-induced sleep disturbance?) propensities [8].

At the same time, numerous epidemiological studies have demonstrated associations between environmental noise exposure and long-term health consequences, including cardiovascular disease [9], diabetes [10], cancer [11], and neurodegenerative disease [12]. It is likely no coincidence that short or low-quality sleep has been associated with the same disease endpoints. Recent animal research suggests that intermittent noise exposure during the night is the culprit for the pathophysiological changes that predispose to negative health consequences, while continuous noise exposure or exposure during the day elicited no or much smaller effects [13]. The observed changes include oxidative stress-induced vascular and brain damage, uncoupling of endothelial and neuronal nitric oxide synthase, vascular/brain infiltration with inflammatory cells, and changes in circadian rhythms [14], which all provide biologic plausibility for the associations observed in epidemiological studies. Endothelial dysfunction was also found in human participants after a single night of noise exposure [15], with stronger effects in patients with preexisting cardiovascular conditions [16], and partially mediated by Vitamin C application indicating the involvement of reactive oxygen species in causing vascular dysfunction. Aircraft noise can also trigger acute cardiac events during sleep which constitutes another mechanism of how noise exposure can contribute to cardiovascular mortality [17].

In a Perspectives piece published in this issue of *SLEEP* [18], Ellenbogen et al. discuss the effects of wind turbine noise on sleep, and they do a remarkable job in making their text accessible to laypeople including engineers with limited knowledge of sleep and sleep researchers with limited knowledge on sound measurement and prediction. Noise is defined as unwanted and/or harmful sound [19], stressing that both sound perception and the degree of control over the noise source can affect the reaction to noise. It is thus no surprise that an emotional response to noise mediated by the Amygdala likely plays a key role in major adverse cardiovascular events [20]. The societal discourse about noise is equally emotional, stressing the importance of noise-effects research as a "fact-deliverer" that can inform political and legislative decision-making. The latter is not an easy task and a balancing act, as a noise source typically also generates benefits for a group of individuals or society at large. For example, while aircraft generate noise, airports and airlines also create jobs and revenue, and for many it is very convenient to live close to an

airport. Likewise, clean energy produced by wind turbines is critical in the fight against climate change.

Ellenbogen et al. [18] perform a narrative review of recent studies on the effects of wind turbine noise on sleep and suggest that "noise from wind turbines measured outside the residence, up to 46 dBA (or modeled up to 49 dBA using the new standard), poses no risk to human sleep." One wonders how this suggestion compares to existing "official" limit values? Limits exist in many countries and provinces worldwide, for example, 37-44 dBA in Denmark, 45 dBA in Victoria, Australia, and 40-51 dBA in Ontario, Canada. These limits are often contingent on wind speed, with limits allowing for higher noise levels at higher speeds, and also the area in which wind turbines are sited, with limits demanding lower noise levels in quiet rural areas and areas which are primarily residential compared with more industrial or urban locations. In the United States, however, limits for wind turbine noise do not exist, at least not at the federal level. The Noise Control Act of 1972 (42 U.S.C. § 4901) is supposed to protect Americans from noise that jeopardizes their health and welfare. However, the Office of Noise Abatement and Control at the Environmental Protection Agency was defunded by the Reagan administration in 1982 and continues to be without funding. Since then federal guidance in developing, funding, disseminating, and coordinating information about the serious health impacts of noise has been imperceptible despite a continued congressional mandate. This includes the mandate to "conduct or finance research [. . .] on the effects, measurement, and control of noise, including but not limited to [. . .] investigation of the psychological and physiological effects of noise on humans [. . .] and the determination of dose/response relationships suitable for use in decision making, with special emphasis on the nonauditory effects" (quoted from the Noise Control Act). These dose/response relationships have mostly been generated in Europe and Asia, although other U.S. federal agencies have started to step in (e.g. [21]). Noise policy should be reviewed on a regular basis, include a review of the current literature and various stakeholders (i.e. those affected by noise, those generating noise, health organizations, researchers, and federal agencies). This is even more important as noise is also a justice and equity issue, disproportionally burdening underserved and low-income groups.

The importance of the environment for sleep quality cannot be overstated. In addition to noise, other factors like temperature and air quality play important roles [22]. Studies that inform health impact assessments are critically needed, but we also need to better understand whether noise mitigation strategies work. Noise reduction at the source is the best way of addressing noise effects, but it is sometimes either technically infeasible or too expensive. We therefore need to understand whether simpler and less expensive noise mitigation measures (e.g. sound insulation, white noise [23], and earplugs) are effective in reducing the effects of noise on sleep. Ellenbogen et al. [18] are to be commended for communicating a complex issue to a lay audience, sleep researchers, and engineers alike, and offering a limited value for further discussion.

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