DUBLIN AIRPORT NOISE MEDICAL REPORT

Centre for Cardiology at Johannes Gutenberg University Mainz

Noise medical report
Dublin Airport Relevant Action Planning Application (F20A/0668)
Planning approval to revoke and amend planning conditions attached to the North Runway planning permission

Clients: SMTW Environmental DAC / St Margaret's The Ward Residents Group
Dublin, Ireland

Place, date: Dublin, October 19th, 2021

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Biography: Thomas Münzel MD, is Chief of the Department of Cardiology at the University Medical Center, Johannes Gutenberg University Mainz, Germany. He is an interventionist and also a vascular biologist and got his post doc training as a vascular biologist at the Institute of Applied Physiology, Professor Eberhard Bassenge, Albert Ludwigs University Freiburg and in the Laboratories of David G Harrison, Emory University, Atlanta, GA, USA with focus on endothelial function and oxidative stress. He is co-initiator of the Gutenberg Health Study, one of the largest prospective cohort trials worldwide and was the founding director of the Center for Thrombosis and Hemostasis, CTH in Mainz. Since 2011, his research group is focusing on environmental risk factors for cardiovascular disease with a focus on aircraft noise and air pollution.

Currently he has more than 1000 publications and a Hirsch index of 136, which is one of the highest in Germany

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1. Introduction to report

We were asked for a noise medical report on the health impacts of the Dublin Airport Authority’s (daa) Relevant Action planning application to revoke and amend night-time operation restrictions attached to the planning permission (F20A/0668) for the North Runway at Dublin Airport.

The Proposed Relevant Action, as defined in section 1 of the Environmental Impact Assessment Report (EIAR), “*relates to the night-time use of the runway system at Dublin Airport. It involves the amendment of the operating restriction set out in condition no. 3(d) and the replacement of the operating restriction in condition no. 5 of the North Runway Planning Permission, as well as proposing new noise mitigation measures.*

The proposed Relevant Action, if permitted, would remove the numerical cap on the average number of flights permitted between the hours of 23:00 and 07:00 that is due to come into effect in accordance with the North Runway Planning Permission, replacing it with an annual night-time noise quota between 23:30 and 06:00 and also to allow flights to take off from and/or land on the North Runway (Runway 10L 28R) for an additional two hours i.e. 23:00 to 00:00 and 06:00 to 07:00. Overall, this would allow for an increase in the number of flights taking off and/or landing at Dublin Airport between 23:00 and 07:00 over and above the number stipulated in condition no. 5 of the North Runway Planning Permission, in accordance with the annual night-time noise quota”.

The Proposed Relevant Action seeks to amend Condition 3(d) as follows:

> ‘Runway 10L-28R shall not be used for take-off or landing between 0000 hours and 0559 hours
>
> 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 or where Runway 10L-28R length is required for a specific aircraft type.’

The Proposed Relevant Action seeks to amend Condition 5 as follows:

> ‘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.'

This Relevant Action is being considered under the Aircraft Noise (Dublin Airport) Regulation Act 2019 (https://www.irishstatutebook.ie/eli/2019/act/12/enacted/en/html). This Act is the enactment of EU regulation 598/2014 (https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX%3A32014R0598) into Irish Law.

Under this Act, the Aircraft Noise Competent Authority (ANCA) must ensure that the Balanced Approach is adopted in respect of aircraft noise management at Dublin Airport where a noise problem has been identified. This includes through:

- Using the most cost-effective measures to address the issue; and
- Not applying operating restrictions as a first resort, but only after consideration of the other measures of the Balanced Approach.

This Relevant Action seeks to revoke and amend existing operating restrictions at Dublin Airport and by doing so will lead to an increase in noise primarily at night. As a result, ANCA must deliberate on the noise problem using the Balanced Approach.

The report also takes into account more recent aspects of noise impact research, which have demonstrated, in particular, the serious negative effects of night-time aircraft noise on health. This report builds on the research conducted since the publication of the WHO 2018 Noise Guidelines for Europe.

Dublin Airport currently has no operating restrictions in place. The planning conditions imposed in 2007 for the North Runway only come into force when the Runway is operational in 2022. In addition, there are no noise limits in Irish Law governing Aircraft Noise.

Based on new research on noise impacts, and due to the existing high levels of night-time air traffic at Dublin Airport, it seems highly advisable to use this Relevant Action application as an opportunity to enforce protection and ideally authorise for nocturnal traffic flight restrictions, to protect the health of residents

The consideration of the effect of continuous sound levels in comparison to the individual / maximum levels at night with regard to health must remain open, since the number of studies carried out or their results do not allow any clear interpretations.

This report also analyses the noise results from the Relevant Action planning application, in particular the Environmental Impact Assessment Report (EIAR), and examines if the proposal will lead to a worsening of the noise situation and whether the proposed mitigation measures curtail the noise and whether a complete ban on night-time aircraft movements to protect the health of residents is justified.

This report also references the paper '*Environmental risk factors and cardiovascular diseases: a comprehensive review*' (https://academic.oup.com/cardiovascres/advance-article/doi/10.1093/cvr/cvab316/6381568). This paper includes all the most recent studies on transport noise and associated health impacts and in particular cardiovascular disease.

2. Noise and public health

In particular on the basis of a number of current, methodologically high-quality experimental and epidemiological studies, noise is now to be regarded as a manifest risk factor for public health. The World Health Organization (WHO) estimates that environmental noise in the member states of the European Union (EU) and other Western European countries causes the loss of 22,000 healthy years of life due to tinnitus, 45,000 healthy years of life due to cognitive impairment in childhood, and 61,000 healthy life Years of life due to ischemic heart disease, 654,000 healthy years of life due to noise annoyance reactions and 903,000 healthy years of life due to sleep disorders, with at least 1 to 1.6 million healthy years of life lost annually due to traffic noise exposure in western regions of Europe (1). Thus, noise-related sleep disturbances and noise annoyance have the greatest adverse health effects in terms of the burden of disease. The European Commission (EC) assumes that the social costs (also caused by increased mortality and morbidity) of noise and air pollution, an important co-factor for the assessment of the health effects of traffic noise, in the EU amount to 1 trillion euros per year (2). The social costs that arise, for example, from alcohol consumption or smoking, the most important risk factor for coronary heart disease, are comparatively low at 50-120 billion and 544 billion, respectively. According to further estimates by the European Environment Agency (EEA), exposure to aircraft, road and rail traffic causes noise annoyance in 53 million, and sleep disorders in 34 million adults, of which 21 million are severely noisy and 14 million are severely sleep-disturbed (3). In this context, environmental noise is responsible for 1.7 million additional cases of high blood pressure annually, associated with 80,000 additional hospitalizations and 18,000 premature deaths from coronary heart disease and stroke. In addition, it has been estimated that 270 million people in Europe exceed the WHO night-time noise directive (40 dB Lnight). A current study of the disease burden caused by traffic noise in Germany alone comes to the conclusion that aircraft noise exposure is responsible for the loss of 98,810 healthy years of life (4).

3. Protection concepts and legal regulations

3.1 WHO guidelines on environmental noise

The current WHO guidelines on environmental noise for the European Region formulated specific recommendations for protecting human health from environmental noise from various sources (5). It is important to mention that the development process of the WHO guidelines followed a strict methodology using the GRADE approach (Grading of Recommendations Assessment, Development and Evaluation) and that the recommended noise levels can also be used in other regions, because not only evidence from European studies regarding noise-related health effects (including effects on the cardiovascular system and metabolism, annoyance, sleep, cognitive impairment, hearing damage and tinnitus, miscarriages, as well as quality of life, mental health and well-being), but also studies from America, Asia and Australia were considered. With regard to aircraft noise, the guideline development group formulates the following recommendations and guidelines (strength of recommendation: **strong**) for noise levels that are specified for the exposure on the <u>exterior</u> of the most exposed façade (5):

• *"For the average noise exposure, the guideline development group strongly recommends reducing noise levels caused by air traffic to less than* ***45 dB****, because aircraft noise above this value is associated with harmful health effects.*

• *For night-time noise exposure, the guideline development group strongly recommends reducing noise levels caused by air traffic to less than* ***40 dB Lnight****, because nighttime aircraft noise above this value is associated with negative effects on sleep.*

• *In order to reduce the health effects, the guideline development group strongly recommends that* ***policymakers take appropriate measures to reduce noise pollution*** *from air traffic for the population whose noise exposure exceeds the guideline values for average and nocturnal noise exposure. As far as concrete measures are concerned, the guideline development group recommends making appropriate changes to the infrastructure. "*

3.2 WHO night noise guidelines

In the WHO night noise guidelines for the European Region, external noise levels of less than 40 dB Lnight are recommended because no significant biological effects that could be harmful to human health or moderate effects have been observed (6). External noise levels of more than 40 dB Lnight lead to measurable health restrictions, especially in sensitive and vulnerable groups, and with external noise levels above 55 dB Lnight the risk of cardiovascular diseases increases measurably.

5.6 RECOMMENDATIONS FOR HEALTH PROTECTION

Sleep is an essential part of healthy life and is recognized as a fundamental right under the European Convention on Human Rights[1](European Court of Human Rights, 2003). Based on the systematic review of evidence produced by epidemiological and experimental studies, the relationship between night noise exposure and health effects can be summarized as below. (Table 5.4)

Table 5.4
Effects of different levels of night noise on the population's health[2]

Average night noise level over a year $L_{night, outside}$	Health effects observed in the population
Up to 30 dB	Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed. $L_{night, outside}$ of 30 dB is equivalent to the NOEL for night noise.
30 to 40 dB	A number of effects on sleep are observed from this range: body movements, awakening, self-reported sleep disturbance, arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill and the elderly) are more susceptible. However, even in the worst cases the effects seem modest. $L_{night, outside}$ of 40 dB is equivalent to the LOAEL for night noise.
40 to 55 dB	Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected.
Above 55 dB	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

40 dB Lnight is defined by the WHO in Table 5.4 of their NNG as the Lowest Observed Adverse Effect Level (LOAEL).

In Chapter 7 Table 7-1 of the EIAR (Population and Human Health), the applicants assign absolute noise values and their associated impact criteria for residential receptors.

This table is repeated in Appendix 13A.6.6 and in section 13A.6.9 it states that "*for the night period the value of 45 dB Lnight has been assigned to low impact. This follows from the approach in the UK where the Government proposed the value as the Lowest Observed Adverse Effect Level, and this received broad support*".

Table 7-1 incorrectly assigns 45 dB Lnight as the LOAEL, instead of the WHO recommendation of 40 dB Lnight. The WHO clearly state that between 40 to 55 dB Lnight, "*Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected*".

Selecting 45 dB Lnight as the LOAEL is a serious flaw in the applicant's interpretation of the health impacts of night-time noise

Table 7-1 Noise Impact Criteria (absolute) – Residential

Scale Description	Annual dB L_{den}	Annual dB L_{night}
Negligible	<45	<40
Very Low	45 – 49.9	40 – 44.9
Low	50 – 54.9	45 – 49.9
Medium	55 – 64.9	50 – 54.9
High	65 – 69.9	55 – 59.9
Very High	≥70	≥60

Table 13-4 combines the absolute and relative impacts into a magnitude of effect table.

Table 13-4: Summary of magnitude of effect – air noise

Absolute Noise Level Rating	*Change in Noise Level Rating*					
	Negligible	*Very Low*	*Low*	*Medium*	*High*	*Very High*
Negligible	Imperceptible	Imperceptible	Imperceptible	Not Significant	Slight	Moderate
Very Low	Imperceptible	Imperceptible	Not Significant	Slight	Moderate	Significant
Low	Imperceptible	Not Significant	Slight	Moderate	Significant	Significant
Medium	Not Significant	Slight	Moderate	Significant	Significant	Very Significant
High	Slight	Moderate	Significant	Significant	Very Significant	Profound
Very High	Moderate	Significant	Significant	Very Significant	Profound	Profound

Exhibit 2-4 (Change in Lnight Noise Level Ratings) of the ‘Dublin Airport North Runway, Regulation 598/2014 (Aircraft Noise Regulation) Forecast Without New Measures and Additional Measures Assessment Report (Revision 2 – September 2021)’ document from Ricondo)), combines the absolute noise levels and the change in noise levels (https://northrunway.exhibition.app/assets/pdf/documents/7_Regulation_598_Aircraft_Noise_Regulation_Assessment_Report.pdf):

EXHIBIT 2-4 CHANGE IN Lnight NOISE LEVEL RATINGS

	CHANGE IN LNIGHT NOISE LEVEL RATING					
ABSOLUTE LNIGHT NOISE LEVEL RATING	NEGLIGIBLE (<1.0dB)	VERY LOW (1.0 – 1.9dB)	LOW (2.0 – 2.9dB)	MEDIUM (3.0 – 5.9dB)	HIGH (6.0 – 8.9dB)	VERY HIGH (≥9.0dB)
Negligible (<40.0dB)	Imperceptible	Imperceptible	Imperceptible	Not Significant	Slight	Moderate
Very Low (40.0 – 44.9dB)	Imperceptible	Imperceptible	Not Significant	Slight	Moderate	Significant
Low (45.0 – 49.9dB)	Imperceptible	Not Significant	Slight	Moderate	Significant	Significant
Medium (50.0 – 54.9dB)	Not Significant	Slight	Moderate	Significant	Significant	Very Significant
High (55.0 – 59.9dB)	Slight	Moderate	Significant	Significant	Very Significant	Profound
Very High (≥60.0dB)	Moderate	Significant	Significant	Very Significant	Profound	Profound

SOURCE: Bickerdike Allen Partners LLP., Dublin Airport North Runway, *Noise Information for the Regulation 598/2014 (Aircraft Noise Regulation) Assessment*, Section 3.3 “Significant Effects under the Scenarios,” November 2020.

In exhibit 2-4, the LOAEL is set as 45 dB Lnight and not 40 dB as per the WHO NNG.

An important aspect of the Relevant Action proposals is that the night-time insulation scheme only applies to dwellings >55 dB Lnight and those dwellings that are > 50 dB and suffer a >+9 dB change in noise levels in the first full year when the Relevant Action comes into operation compared with the current permitted operation in the same equivalent year.

In the Residential Sound Insulation Grant Scheme (RSIGS) overview document from Anderson Acoustics (https://northrunway.exhibition.app/assets/pdf/documents/13_Proposed_Sound_Insulation_Grant_Scheme.pdf), dwellings >50 dB Lnight and with a +9dB criteria are deemed to have a “**very significant**” rating.

- Dwellings are considered eligible if they meet either of the following noise related criteria:
  1. Dwellings forecast to be exposed to “high” night-time noise levels in 2025 - at least 55dB L_{night}.
  2. Dwellings with a “very significant” rating arising from forecast noise levels of at least 50dB L_{night} in the first full year when the Relevant Action comes into operation, with a change of at least +9dB when compared with the current permitted operation in the same equivalent year.

Criteria 1 demonstrates that the daa intends to insulate those dwellings suffering a high level of night-time noise >55 dB Lnight which according to the WHO is "*considered increasingly dangerous to public health*". The population experiencing between 40-55 dB Lnight will not be insulated and the vulnerable group will be at most risk.

Criteria 2 demonstrates that the daa also intends to insulate those dwellings >50dB Lnight and with a change of +9dB deemed "very significant". The population experiencing "significant" and 'moderate" effects are excluded. Excluding those significantly affected is contrary to the EPA EIAR Guidelines (https://www.epa.ie/publications/monitoring--assessment/assessment/EPA_EIAR_Guidelines.pdf).

Table 13-51 of the EIAR shows that comparing 2025 Proposed vs 2025 Permitted, 11350 people will be significantly adversely affected with 104 very significantly affected and 40 profoundly impacted.

Table 13-64 of the EIAR compares 2025 Proposed and 2025 Permitted and shows that 10474 people will be significantly adversely impacted by residual effects after allowing for the benefit of the existing and proposed insulation schemes.

Both these tables show the large number of people that this proposed Relevant Action will significantly adversely affect and who are being left out of mitigation measures and whose health and well-being will suffer as a result. These figures also underestimate the population affected as the EIAR fails to identify the correct LOAEL value of 40 dB Lnight for night-time noise as per the WHO Guidelines.

4. Findings from noise impact research

The health effects of noise are still significantly underestimated by the public, although more recent results of noise impact research emphasize noise as an important health risk factor, especially for cardiovascular diseases and also for mental illnesses. From a scientific point of view, it can be clearly stated that noise exposure can trigger health-damaging effects; the results of epidemiological and experimental studies suggest that traffic noise exposure can significantly increase the risk of various clinically relevant phenomena (see review article Münzel, Basner, Babisch (11)).
Large-scale and methodologically reliable studies show that aircraft noise in particular significantly increases the risk of cardiovascular diseases such as high blood pressure, coronary heart disease, myocardial infarction and stroke (12, 13). The underlying pathophysiological mechanisms are multifactorial and the subject of current research. In Babisch's noise effects model, it is postulated that in addition to the direct auditory effects of noise (excessively high or short-term extreme noise levels over long periods of time that lead to noise-induced hearing loss or hearing loss via damage to the hearing organ), the non-auditory, indirect noise effect act as a trigger for a stress hormonal cascade, which ultimately promotes the development of cardiovascular diseases, but, as more recent results show, psychological diseases such as depression and anxiety disorders and metabolic diseases such as diabetes mellitus (14-16). In this sense, long-term exposure to noise that is far below the trigger threshold for noise-induced hearing loss can lead to impairment of sleep, communication and everyday activities and, as a result, trigger reactions to anger or noise. The associated cognitive and emotional stress reactions lead to the activation of the autonomic and endocrine system and, as a result, changes in blood pressure regulation as well as in cholesterol and glucose metabolism to increased development of cardiovascular risk factors, i.e. blood pressure is increased, blood sugar and cholesterol levels are increased, blood viscosity is increased and blood clotting is activated.

It is important to mention that sleep disorders and noise pollution are central mechanisms in the mediation of noise-induced cardiovascular diseases and other clinically relevant symptoms, so that exposure to noise at night represents a particular risk constellation (17). Sleep disorders per se represent a significant cardiovascular risk factor, as was shown in a meta-analysis of 15 prospective studies in which a short sleep duration was associated with an increased risk of coronary heart disease and stroke (18). Chronic exposure to noise as well as persistent stress reactions over the years lead to an increased risk of developing cardiovascular diseases such as high blood pressure, myocardial infarction, coronary heart disease and stroke (Figure 2).

4.1 Epidemiological evidence on the relationship between aircraft noise and cardiac Circulatory diseases

Epidemiological research on the effects of aircraft noise has increased more and more in quantity and quality in recent years, with improved methodology with regard to the calculation of continuous noise levels, consideration of larger and more diverse population-based samples and more extensive controls for confounding variables. Furthermore, phenomena such as exhaustion, conditioning, habituation and sensitization are taken into account in epidemiological designs by considering everyday living conditions and longer observation periods, whereby the recording of these phenomena under experimental conditions with short-term and acute noise exposure proves to be more complex.

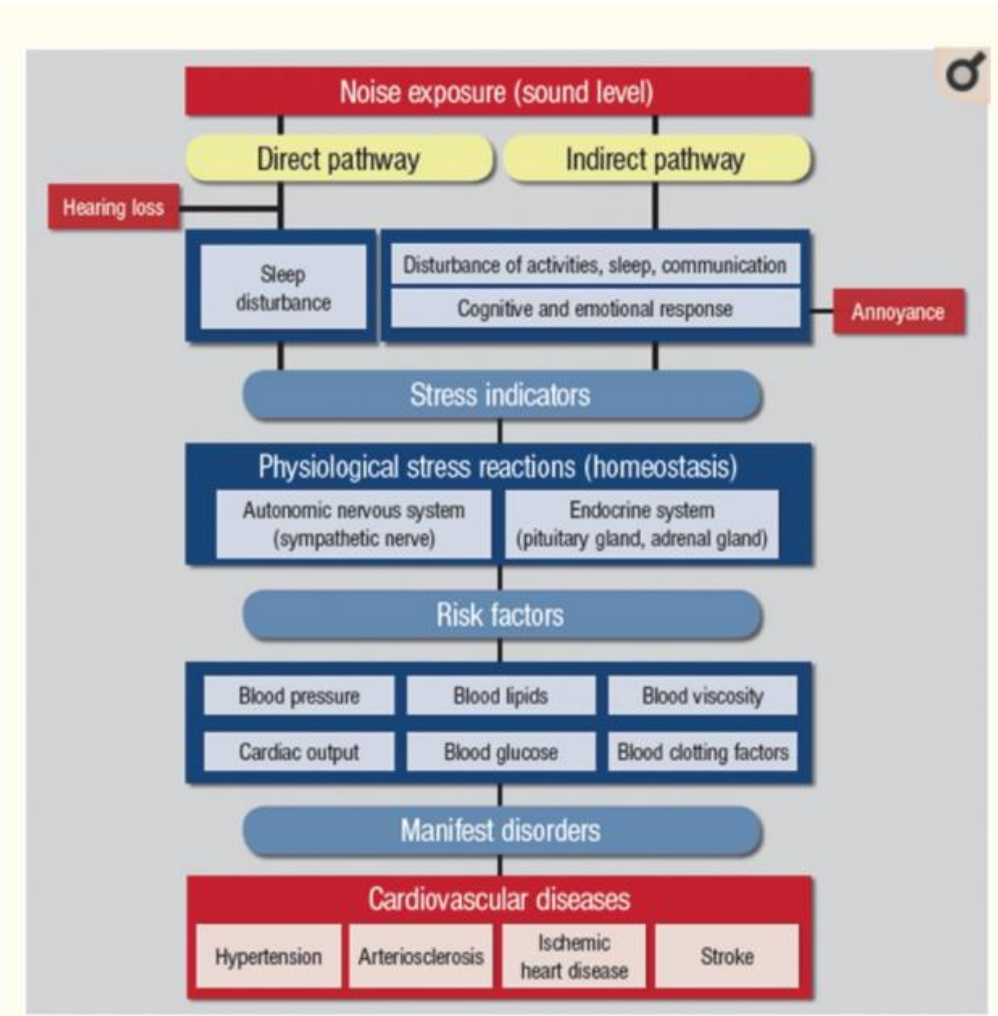


Figure 2. Noise reaction model according to Babisch (14, 15) adapted according to and modified from Münzel et al. (11).

4.1.1 Coronary heart disease

A current meta-analysis from 2018, which was carried out on behalf of the WHO, showed that aircraft noise increases the relative risk (RR) of incident coronary heart disease by 9% [95% confidence interval (CI) 1.04; 1.15] per increase of 10 dB Lden (19).

In another meta-analysis by Vienneau et al. studies on the relationship between aircraft and road traffic noise were summarized and assessed with regard to the risk of incident coronary heart disease (20). A pooled RR of 1.06 [95% CI 1.03; 1.09] with an increase of 10 dB Lden starting from 50 dB.

4.1.2 Hypertension

A meta-analysis by Babisch van Kamp found a 13% higher risk of high blood pressure (OR 1.13 [95% CI 1.00; 1.28]) per increase of 10 dB (A) Ldn in the range of 45-70 dB (A) (21).

A prospective study from Sweden including male subjects examined the risk of incident high blood pressure from aircraft noise exposure (22). An RR of 1.10 [95% CI 1.01; 1.19] with an increase of 5 dB (A) LAeq24h. A comparison of test persons with lower vs. higher noise exposure (<50 vs. ≥ 50 dB (A) LAeq24h) resulted in a RR of 1.19 [95% CI 1.03; 1.37], with an RR of 1.29 [95% CI 1.11; 1.50] could be observed. In a follow-up study in a subgroup analysis for test persons who stated that they felt annoyed by aircraft noise, a RR of 1.42 ([95% CI 1.11; 1.82] ≥ 50 dB (A) Lden)) for the incidence of high blood pressure can be determined (23). It can be concluded that people exposed to noise may represent a vulnerable group with regard to the harmful effects of aircraft noise.

As part of the HYENA study (Hypertension and Exposure to Noise Near Airports in the vicinity of various airports from six European countries) in the Greek population, an almost 3-fold increased risk of incident high blood pressure (odds ratio (OR) 2, 63 [95% CI 1.21; 5.71] were found for an increase of 10 dB Lnight (24), whereas the aircraft noise over the day and evening hours (OR 1.46 [95% CI 0.89 ; 2,39] per increase of 10 dB LAeq16h) and road traffic noise were associated with less pronounced effects on blood pressure While no connection with aircraft noise during the day was observed (25), another study based on HYENA data found an increase of 6 mmHg in systolic and an increase of 7 mm Hg of the diastolic blood pressure if a night flight event with a sound level of> 35 dB (A) had taken place within the last 15 minutes (26).

4.1.3 Heart attack

A nationwide study from Switzerland on the influence of noise exposure from various traffic sources showed an increased risk of aircraft noise-related mortality due to myocardial infarction (Hazard Ratio (HR) 1.027 [95% CI 1.006; 1.049] with an increase of 10 dB Lden starting from 30 dB) (27).

Similar results were observed in a study from France for the aircraft noise-related mortality risk due to myocardial infarction with a risk increase of 28% (Mortality Rate Ratio (MRR) 1.28 [95% CI 1.11; 1.46] per increase of 10 dB (A) Lden (28).

4.1.4 Stroke

In a large-scale English study, exposure to aircraft noise was associated with an increased risk of hospitalization after a stroke (29). A higher risk of hospitalization for night (RR 1.29 [95% CI 1.14; 1.46] at> 55 vs. ≤ 50 dB Lnight) than for daytime aircraft noise exposure (RR 1.24 [95- % CI 1.08; 1.43] at> 63 vs. ≤ 51 dB LAeq16h) can be observed. The outcome pattern for stroke-related mortality was comparable, with this analysis showing broader CI due to the lower number of cases.

4.1.5 Sudden deaths

In a recent analysis from Zurich Airport in Switzerland, acute night-time aircraft noise (2 hours before the event) was associated with an increased risk of nocturnal death from cardiovascular disease (consisting of coronary heart disease, myocardial infarction, heart failure, high blood pressure, stroke and cardiac arrhythmia) tied together. A 44% higher risk of death was observed when comparing people with higher vs. lower aircraft noise exposure (OR 1.44 [95% CI 1.03; 2.04] at> 50 dB vs. <20 dB LAeq) (30).

In a large German case-control study, the influence of aircraft noise on the risk of heart failure or hypertensive heart disease was investigated (31). A risk increase of 1.6% [OR 95% - CI 1.003; 1.030] can be observed per increase of 10 dB LAeq24h starting from 35 dB.

Another German case-control study was able to confirm the influence of aircraft noise on the risk of high blood pressure in connection with hypertensive heart disease (OR 1.139% [95% CI 1.090; 1.190] per increase of 10 dB LAeq24h (32).

Results of the HYENA study showed that night-time aircraft noise in people who have lived in the same place for ≥ 20 years had a 25% higher risk of a combined endpoint of angina pectoris, myocardial infarction or stroke (OR 1.25% [95% CI 1 , 03; 1.51] associated with an increase of 10 dB (A) Lnight) (33).

In a study from France, the maximum interior noise levels in connection with aircraft overflights were associated with a higher heart rate amplitude during sleep (34).

Overall, it can be said that increased risk of cardiovascular diseases can already be observed at noise levels from around 40 to 50 dB Lden (Figure 3) and are thus far below the limit at which direct damage to the hearing organ is to be expected.

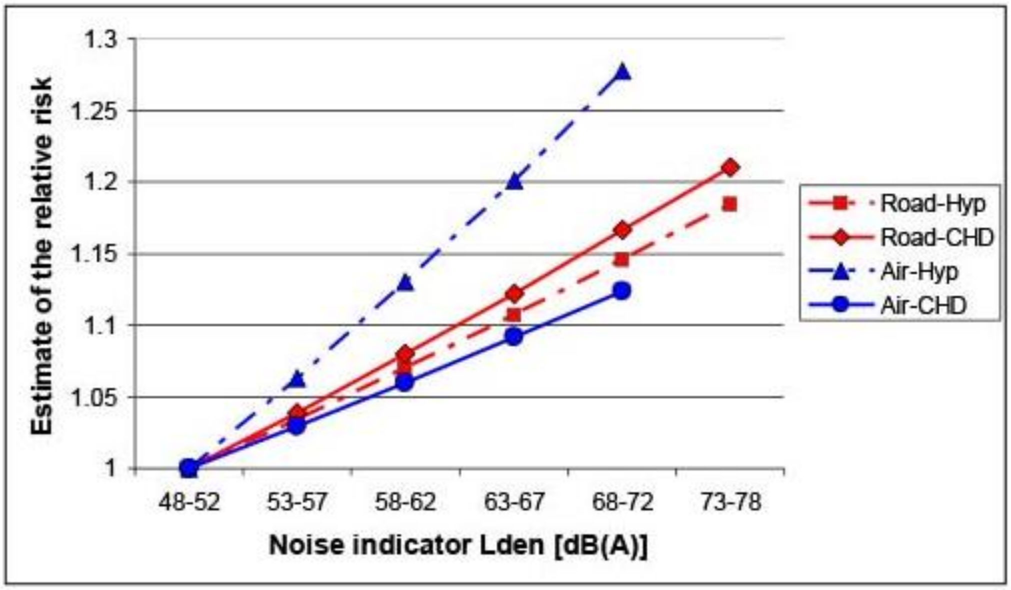


Figure 3. Dose-effect relationship for the relationship between flight and road traffic noise and cardiovascular diseases. Flight: aircraft noise, road: road traffic noise, blood: high blood pressure, CHD: coronary heart disease. Figure and Data taken from Babisch (35).

4.1.6 Aircraft noise pollution

Noise annoyance is the most common reaction of noise-exposed populations, which is promoted by the emotional and cognitive representation of noise exposure in the form of negative feelings and thoughts such as exhaustion, irritability, agitation and distress (36, 37). In the already mentioned HYENA study it could be shown that the annoyance caused by aircraft noise has increased sharply in the past few years well above the EU standard curves, whereby no change in annoyance to road traffic noise could be determined, so that aircraft noise appears to play a special role with regard to the resulting noise annoyance (38). This could also be demonstrated in a current meta-analysis commissioned by the WHO, in which the results indicate that aircraft noise is more annoying than road and rail traffic noise (39) (Figure 4). The large-scale Gutenberg Health Study (GHS) from Germany (carried out in Mainz and Mainz-Bingen) showed for the first time that increasing noise pollution from various sources during the day and during night sleep is associated with an increased prevalence of atrial fibrillation, with overall noise pollution more pronounced at night than during the day (40). The annoyance from aircraft noise at night was associated with a higher risk of atrial fibrillation than during the day (day: OR 1.04 [95% CI 1.00; 1.08]; night: OR 1.09 [95- % CI 1.05; 1.13 per point increase in noise pollution).

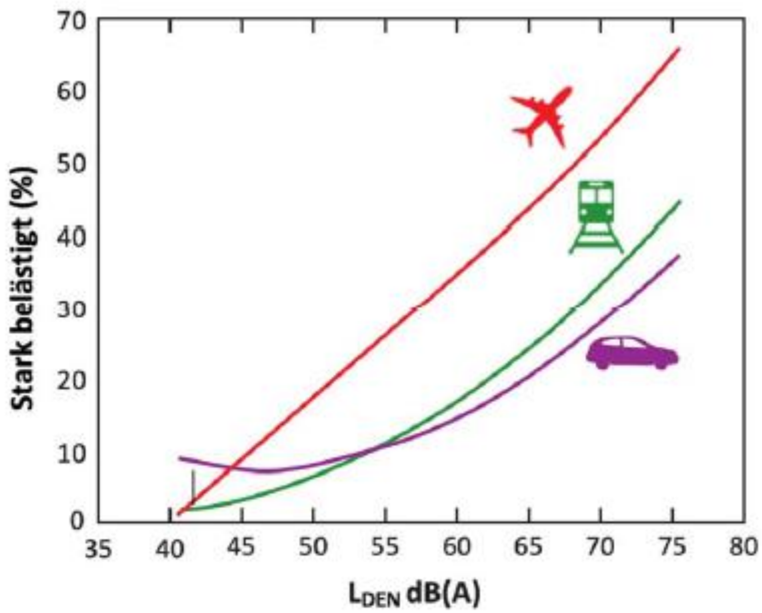


Figure 4. Percentage of people severely annoyed by aircraft, road and rail traffic noise (given in Lden dB (A)) based on data from Guski et al (39).

In addition, the authors were able to prove that the night flight ban introduced in 2011 at the neighboring Frankfurt am Main airport between 11 p.m. and 5 a.m. did not lead to a decrease in aircraft noise, but rather to a significant increase in aircraft noise when comparing the annoyance before and after 2011. The explanation given here was the runway introduced at the same time in 2011, which led to increased flight movements during the day and especially between the off-peak times (10 p.m. to 11 p.m. and 5 a.m. to 6 a.m.). The authors also concluded that the more negative attitudes and increased sensitivity to the topic of aircraft noise due to the increased media and social attention with regard to the current environmental debate could have led to increased aircraft noise annoyance, despite the introduction of the night flight ban.

In another study based on GHS data, aircraft noise annoyance recorded during the day (beta weight 0.016 [95% CI 0.0070; 0.025]) and during sleep (beta weight 0.020 [95% CI 0.010; 0.030]) is associated with increased co-regional pro-atrial natriuretic peptide levels (MR-proANP), a cardiac hormone that is produced to an increased extent as a result of overloading the heart and that secondary serves to relieve the volume and thus pressure in the heart (41). Increased MR-proANP could predict the incidence of cardiovascular disease and death from cardiovascular disease 5 years later.

Furthermore, Babisch et al. based on a case-control study from Germany that night-time aircraft noise annoyance is associated with a 28% higher risk of heart attack in women (OR 1.28 [95% CI 1.01; 1.63] per point increase in noise annoyance) (42) .

Another study explored the relationship between traffic noise exposure and the activation of the limbic system (especially the amygdala nuclei), which plays an important role in the control of functions such as drive, learning, memory and emotions, as well as in the development of cardiovascular diseases. In this study it could be shown that in test persons exposed to traffic noise (aircraft and road traffic noise), in whom activation of the amygdala was detected at the same time, more inflamed vessels and a poorer prognosis, i.e. more cardiovascular events, could be observed. The cardiovascular events here included cardiovascular death, myocardial infarction, unstable angina pectoris, stroke, cardiac insufficiency, and coronary or peripheral revascularization (Figure 5) (43).

This makes it clear that noise pollution and the subsequent annoyance, i.e. the negative emotional processing of traffic noise, is very decisive in conveying cardiovascular events. This association remained statistically significant even after controlling for other confounding factors, including cardiovascular risk factors, air pollution, socio-economic factors, and access to health care. These findings give noise pollution a new and additionally important meaning, in the sense that the neurobiological stress correlate (activation of the amygdala) is directly related to vascular inflammation and cardiovascular events (see also previously mentioned WHO statistics: 654,000 healthy life years lost due to noise annoyance).

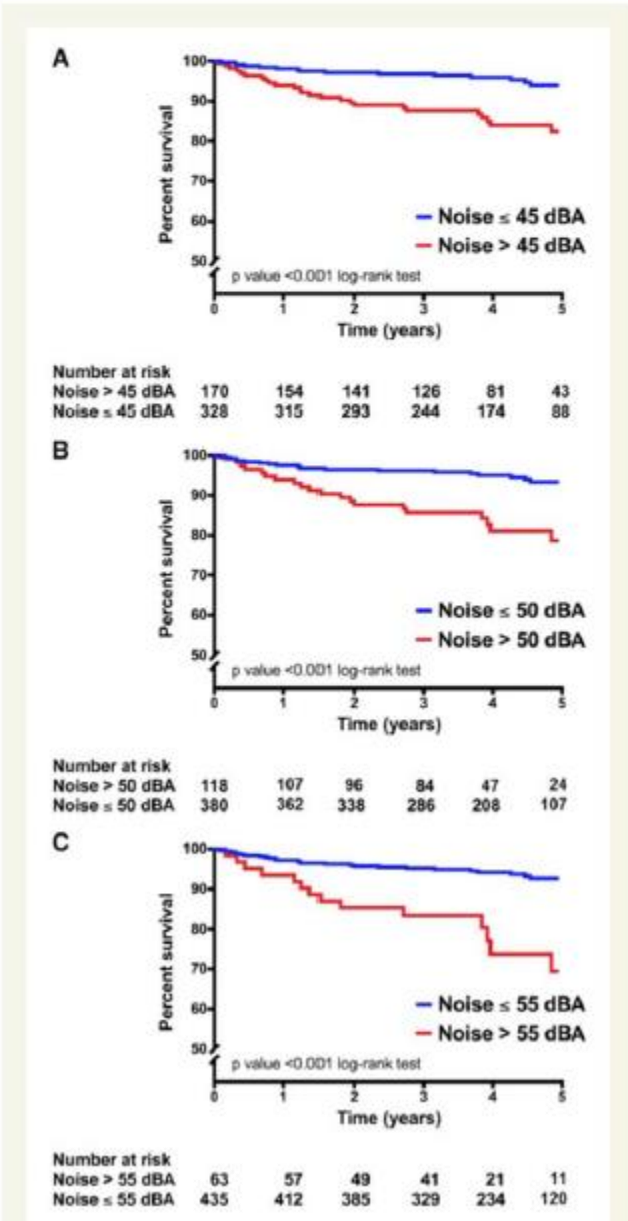


Figure 5. Figure 5. Kaplan-Meier survival curves. Event-free probability of survival in relation to undesirable cardiovascular diseases from traffic noise exposure. Survival probability for test persons when comparing LAeq24h traffic noise levels ≤ (blue line) vs.> (red line): A. 45 dB (A) (upper tertile), B. 50 dB (A) (upper quartile) and C. 55 dB (A (WHO cut-off). **It can be seen from the figure that if traffic noise exposure is above the WHO cut-off of 55 dB (A), the event-free probability of survival decreases by 30% within 5 years.** Figure taken from Osborne et al. (43).

4.2 Aircraft noise and translational studies on healthy and heart disease subjects

In two experimental field studies, Schmidt et al. as part of a field study, the effects of **simulated night flight noise** on vascular function (endothelial function determined by the flow-mediated vasodilation (FMD) of the brachial artery, which is an important early risk marker for the development of cardiovascular diseases) in healthy volunteers (44) or patients with existing coronary heart disease (45). Co-authors were Dr. Babisch (at that time still working at the Federal Environment Agency) and Prof. Basner (University of Pennsylvania Perelman School of Medicine, Philadelphia, USA). At the same time, the stress hormone levels before and after the aircraft noise exposure were measured and the change in sleep quality was determined with the help of questionnaires.

The healthy subjects were exposed to three different noise scenarios (0 overflights (control group) or 30 and 60 overflights with peak sound levels of 60 dB (A) Leq and mean sound pressure levels of 43.12 and 46.28 dB (A) Leq) over a total of three nights and a control scenario (normal ambient noise with a mean sound pressure level of 35.44 dB (A) Leq) randomly exposed (44). The aircraft noise was produced by an MP3 player in the house of the subjects studied and the noise per se was given by Prof. Basner (that time DLR Bonn), an original registration of a night-time flight from the airport Cologne-Bonn. Since the noise was applied in the house of the subjects these studies can be considered as field studies.
The night aircraft noise exposure surprisingly led to a worsening of the FMD (control group: 10.4%; noise 30: 9.7%; noise 60: 9.5%), to reduced sleep quality (Pittsburgh sleep quality index for the control group: 6.70; noise 30: 5.20; noise 60: 4.37) and an increased adrenaline level (control group: 28.3 ng / L; noise 30: 33.2 ng / L; noise 60: 34.1 ng / L). In addition, the pulse wave transit time was reduced after exposure to noise (control group: 271.8 ms; noise 30: 270.9 ms; noise 60: 264.9 ms), a parameter that is associated with increased blood pressure, vascular tone and stiffness. The noise-related deterioration of the FMD was particularly pronounced when the test persons were exposed to the noise scenario 30 and then 60 simulated overflights, so that noise-related sensitization rather than habituation was observed at the vascular level (priming effect). Interestingly, the worsening of FMD in a small subgroup of test persons (n = 5) who were exposed to noise scenario 60 could be improved by a single dose of vitamin C. This suggests that an increased formation of reactive oxygen species and the resulting increased oxidative stress in the vessels is the cause of the endothelial dysfunction. We were able to find this vitamin C effect in another current noise study (train noise) (46). In this study, too, night train noise led to pronounced vascular damage and due to the high statistical significance of the vascular damage, which was already achieved after the inclusion of 70 test persons, the recruitment of further test persons could be stopped, although the inclusion of > 100 test persons was planned.

In a follow-up study by Schmidt et al. 60 test persons with an existing or an increased risk of coronary heart disease, two different noise scenarios (60 night flyovers with an average sound pressure level of 46.9 dB (A) Leq) and a control scenario (normal ambient noise with an average sound pressure level of 39.2 dB (A) Leq) were randomly exposed (45). The observed effects of night aircraft noise on vascular function were more pronounced in the patients with coronary heart disease than in the healthy

subjects (FMD control group: 9.6%; FMD noise 60: 7.9%) (Figure 6), the quality of sleep was reduced, and the systolic Blood pressure rose significantly (control group: 129.5 mmHg; noise 60: 133.6 mmHg).

In a third current study by Schmidt et al. the question of whether the intensity (loudness) or the frequency (number) of nocturnal aircraft noise events is more important for the assessment of aircraft noise-related vascular damage, with the same average sound level being the basis in both scenarios (47). It was demonstrated that 60 simulated night flights with peak sound levels of 60 dB (A) Leq and 120 simulated night flights with peak levels of 57 dB (A) Leq with an identical mean sound level of 45 dB (A) Leq cause comparable vascular damage in 70 test persons with an existing or increased risk of cardiovascular disease. This means that many quieter aircraft noise events have the same negative significance for vascular function as fewer but louder aircraft noise events. In this respect, the mean sound pressure level determines the extent of the deterioration in vascular function. In addition to impaired sleep quality and vascular function (FMD), it was also shown for the first time that the diastolic pump function of the heart is significantly impaired after exposure to noise compared to the control scenario (37 dB (A) Leq). This could explain why the NORAH study (Noise-Related Annoyance, Cognition, and Health) from Germany identified more patients with heart failure as a result of noise (31).

In a cohort study from Switzerland, the SAPALDIA study (Study on Air Pollution and Lung Disease in Adults), the relationship between traffic noise exposure and arterial stiffness (measured using the pulse wave speed), which is an important marker of vascular function and risk factor for cardiovascular diseases, was investigated (48). It could be demonstrated that the number of nocturnal traffic noise events was strongly associated with arterial stiffness (beta weight 1.77 [95% CI 0.45; 3.09] when comparing quartile 1 against quartile 4). In addition, a direct connection between experimental sleep restriction and endothelial dysfunction has been demonstrated (49). In this context, it must be mentioned that the vascular stiffness is a reliable predictor for the development of future cardiovascular diseases or all-cause mortality (50).

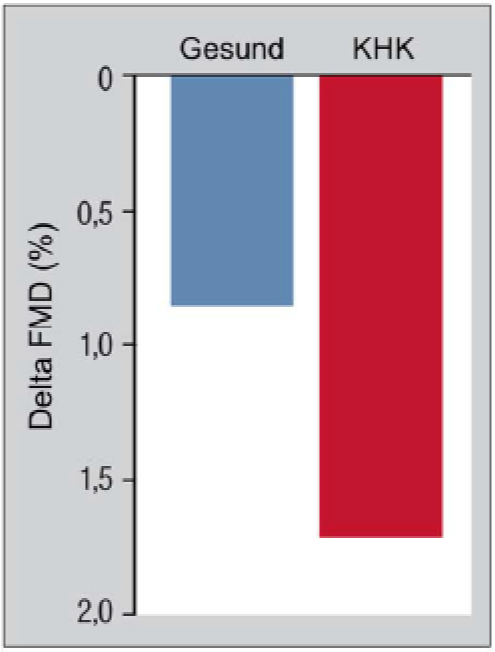


Figure 6. Delta of the endothelial function of the brachial artery (determined with the help of flow-mediated vasodilation or flow-mediated dilation (FMD)) after 60 simulated aircraft noise events at night in healthy subjects (44) compared to patients with established coronary heart disease (45). The graphic shows that in the case of an already existing coronary heart disease and thus pre-damaged endothelium, the extent of endothelial damage is significantly increased after exposure to aircraft noise.

4.3 Aircraft noise and the results of animal noise research

In animal experiments, a newly developed mouse model was used to identify the molecular mechanisms of vascular dysfunction caused by aircraft noise (51, 52). These studies also represented the first investigations ever, in which the cardiovascular consequences of aircraft noise were specifically investigated in animal experiments. Earlier research usually used white noise as a source of noise and employed drastically higher noise levels of white noise. In the first study, mice were exposed to aircraft noise for four days (69 noise events with mean sound pressure levels of 72 dB (A) Leq and peak sound levels of 83 dB (A) Leq) (51). Here, the aircraft noise surprisingly led to pronounced endothelial dysfunction (vascular damage) within 24 hours and, over the course of the 4-day exposure, to increased blood pressure and increased stress hormone levels of norepinephrine, angiotensin II, cortisol and dopamine. In this context, it is important to point out the striking parallelism of human field studies and animal experiments, namely that verifiable vascular damage can occur within 24 hours.

Exposure to white noise with identical mean sound pressure levels in the control scenario, on the other hand, did not

lead to the effects described above, suggesting that in addition to the simple consideration of quantitative characteristics such as volume or sound pressure level, above all qualitative characteristics of the noise exposure such as frequency and complexity as well as their corresponding cognitive and emotional representation as disruptive or impairing when conveying cardiovascular effects are decisive.

In a follow-up study by Kröller-Schön et al. Using a similar test protocol, it was possible to show that especially simulated night aircraft noise, i.e. H. noise during the sleep phase of the mice, but not during the wake phase, was associated with oxidative stress and neuroinflammation in the brain, accompanied by an increased systemic inflammatory reaction, the formation of free radicals in the vessels and even vascular damage (endothelial dysfunction) (52). In addition, it could be shown within the scope of this study that a dysregulation of the transcription factor forkhead box protein O3 (FOXO3) led to a disturbed circadian gene expression, which can promote the occurrence of sleep disorders, a disruption of the circadian rhythm and stress reactions.

In another current study, the combination of experimentally induced high blood pressure by infusion of angiotensin II and aircraft noise exposure at a peak sound pressure level of 85 dB (A) Leq and an average sound pressure level of 72 dB (A) Leq over 7 days caused additive negative cerebral and vascular changes compared to the individual exposure models (53).

These results strikingly go along with the human experimental aircraft noise studies described above and other epidemiological studies that imply that aircraft noise and classic cardiovascular risk factors such as high blood pressure can work together to additively increase the risk of cardiovascular diseases.

4.4. Aircraft noise and cognitive developmental disorders in children

With the help of noise impact research, it was also possible to demonstrate in animal experiments that aircraft noise quickly downregulates the important enzyme, neural NO synthase, in the brain, an enzyme that is responsible for learning and memory and therefore explains the findings of the researcher Stansfeld (54) according to which aircraft noise leads to a delay in cognitive development in children, a finding that has also been proven in the NORAH study (55). The NORAH study produced the following specific results in this regard:

• **Reading skills:** The results show that with a continuous increase in the sound level (LAeq, 08-14h) of 10 dB (A) the acquisition of reading competence deteriorates by an average of one month. The children with an aircraft noise-associated noise exposure of

59 dB (A) are therefore about two months behind the children in their schools with an average aircraft noise-associated noise exposure of 39 dB (A).

• **Anterior Skills:** No connection was found between aircraft noise and the linguistic precursor skills of reading, such as speech perception and auditory memory.

• **Well-being:** The students rate their physical and psychological well-being less positively as the continuous noise level rises.

4.5 Sleep and sleep disorders and cardiovascular risk

Disturbed sleep is a manifest risk factor for cardiovascular and metabolic diseases (18, 56). A current meta-analysis carried out on behalf of the WHO showed that exposure to traffic noise is associated with an increased risk of subjectively perceived sleep disturbances, with aircraft noise having a 2-fold increased risk of severe sleep disturbances per increase of 10 dB Lnight (OR 1.94 [95% - KI 1.61; 2, 3]), so that disturbed sleep at night could play an important role in the mediation of aircraft noise-related cardiovascular diseases (57). In addition, the analysis of polysomnographic studies within the scope of this study was able to show that an increase of 10 dB (A) in the maximum interior noise level caused by aircraft noise results in a 35% higher risk (OR 1.35 [95% CI 1.22; 1, 50 Lmax) for a change in the waking or sleeping phases (from deep sleep phases to the waking state or phase 1) or with a higher probability of nocturnal awakening.

Another recent study from France was able to show that both the increasing number of nocturnal aircraft noise events and increasing aircraft noise levels worsen various objective parameters of sleep quality, determined by actigraphy (58). In another study by the authors on the subjectively assessed sleep quality, night-time aircraft noise exposure was significantly associated with a short total sleep time (≤ 6 h) and the feeling of tiredness when waking up in the morning (59). An increase in the aircraft noise level at night by 10 dB (A) was 63% more likely to have a short total sleep time (OR 1.63 [95% CI 1.15; 2.32) and a 23% higher probability of the feeling of tiredness when waking up in the morning (OR1.23 [95% CI 1.00; 1.54). In a study from the USA, Basner et al. a relationship between the maximum sound pressure level from nocturnal aircraft noise events and the probability of awakening was derived from increased heart rate and body movements (60). The results of the NORAH study, which examined the health effects of aircraft noise triggered by Frankfurt Airport, show that with a background level of 28.8 dB (A) the chance of waking up at night is increased for every 10 dB (A) increase in the maximum level overflight noise increased by 23% (61).

4.6 Mental illness

Because noise exposure and noise annoyance act as a psychological stressor, this fact could represent another important mechanism in the mediation of cardiovascular diseases. In this context, it is interesting that in addition to the numerous studies that were able to highlight noise as a cardiovascular risk factor, there are also indications of an increased risk of noise-related mental disorders (62-64). Mental stress is a significant risk factor for both manifest mental disorders and cardiovascular diseases (65), whereby mental disorders such as depression and anxiety disorders and cardiovascular diseases are in turn in a bidirectional relationship and can be mutually dependent (66). Based on cross-sectional data from the GHS, Beutel et al. demonstrate that the prevalence of depression and anxiety disorders increases in a dose-dependent manner with the level of noise pollution, including aircraft noise (67). Extreme noise pollution resulted in a 2-fold increased risk of depression or anxiety disorder (prevalence rate of 1.97 [95% CI 1.62; 2.39] for depression and 2.14 [95% CI 1, 71; 2.67] for anxiety disorder when compared with no noise pollution). A subsequent prospective study by the authors also showed that (flight) noise pollution can also predict the recurrence of depressive moods, fears and sleep disorders five years later (68). A large-scale case-control study from Germany based on data from the NORAH study showed that aircraft noise can increase the risk of depression (OR 1.23 [95% CI 1.19; 1.28] when comparing <40 to <50 vs. ≥ 50 to <55 dB LAeq24h) (69). A current meta-analysis of aircraft noise studies showed that the risk of depression is increased by 12% for every increase of 10 dB Lden (95% CI 1.02; 1.23) (70). In a smaller case-control study from Italy, people exposed to aircraft noise had an increased risk of generalized anxiety disorder (OR 2.0 [95% CI 1.0; 4.2]) (71). It can therefore be assumed that, in addition to the direct effects of aircraft noise exposure on the cardiovascular risk, indirect effects through the promotion of mental disorders are also conceivable.

4.7 Acute versus chronic noise effects

Based on Wolfgang Babisch's concept of the effects of noise, it was previously assumed that cardiovascular diseases such as coronary heart disease or cardiac insufficiency develop chronically, possibly over years. The work just published by Saucy et al. changed this significantly. As already briefly mentioned, **a nocturnal aircraft noise event at Zurich Airport can cause acute cardiovascular death 2 hours later** (Figure 7) (30). In our opinion, the study is of great importance as it is the first study to investigate the acute effects of night-time aircraft noise on cardiovascular mortality. The selected case crossover design is an innovative approach to analyze the acute side effects of aircraft noise on cardiovascular health. The implications of this study are, and this is also called for by the authors, the introduction of a night flight rest in accordance with the night of 10 p.m. to 6 a.m. defined by the legislator.

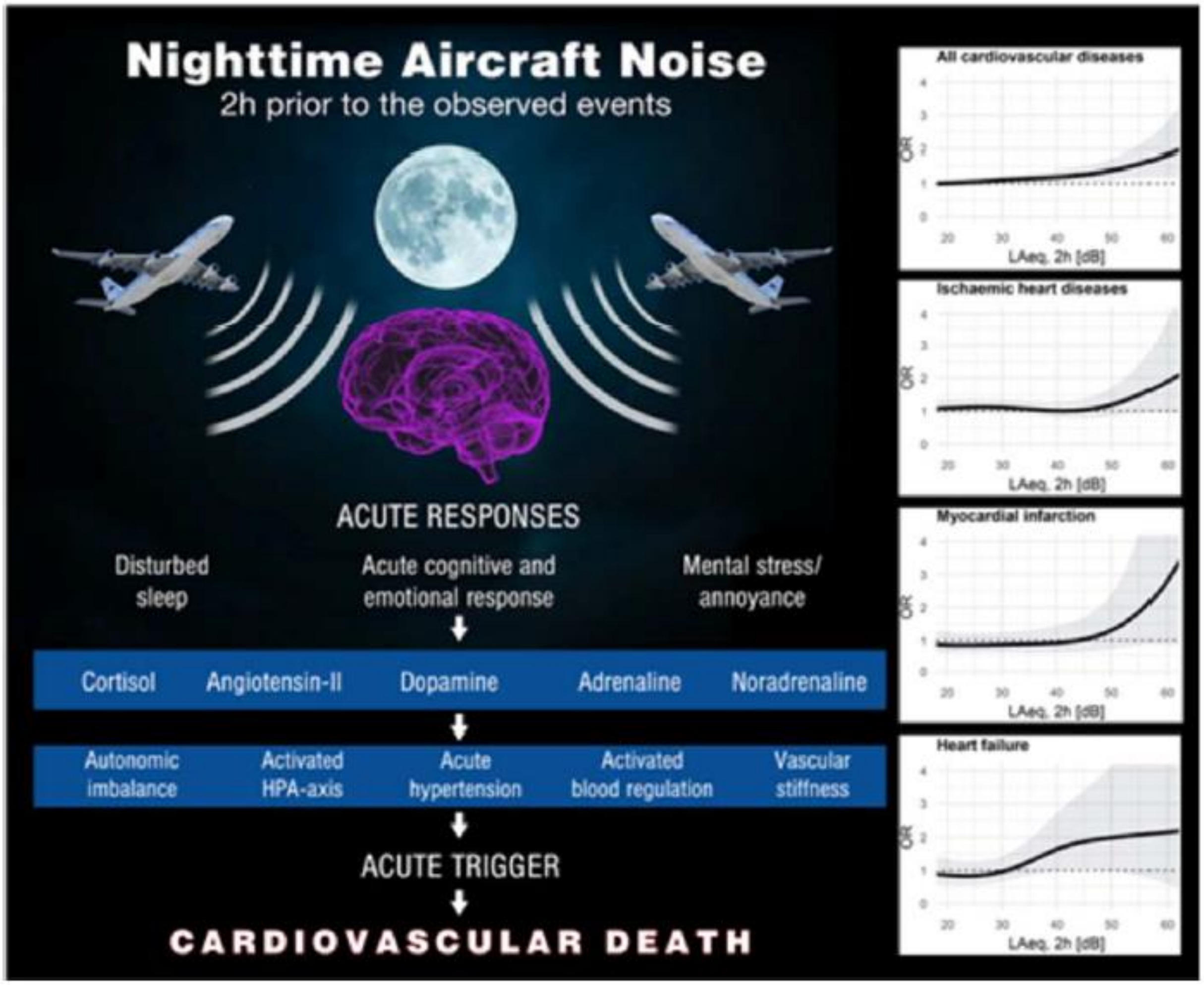


Figure 7. Night aircraft noise causes acute cardiovascular deaths according to Saucy et al. (30).

4.8 Aircraft noise and particulate matter

Aircraft noise or air traffic is usually associated with an increase in particulate matter concentrations (and here in particular ultra-fine dust).

As mentioned above, the EU's “In-Depth Report” showed that the combination of noise and particulate matter in particular causes social costs of up to one trillion euros per year, significantly more than the risk factors alcohol and smoking (2). The problem of ultra-fine dust at Frankfurt Airport was recently discussed. It was found that the measures to combat the COVID-19 pandemic together with the reduction in flight movements reduced the ultrafine dust concentrations by up to 44% (72). Ultra-fine dust is particularly harmful to health, as it can easily be absorbed into the bloodstream and finally into the blood vessels after inhalation via the lungs, where it then triggers inflammations that lead in the long term to heart attacks, strokes, cardiac insufficiency and also cardiac arrhythmias. It is important to note that it must be assumed that the negative effects of aircraft noise are increased by the fine dust and that the adverse effects on health are further intensified by the combination of these environmental stressors.

Air and noise pollution have many of the same sources, such as heavy industry, aircraft, railways and road vehicles. 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.

Air pollution and noise pollution have negative health impacts on all socioeconomic groups, rich and poor. However, the risks may not be evenly shared; it is often society's poorest who live and work in the most polluted environments. Furthermore, these same people may be more impacted by pollution's damaging effects than more advantaged groups of society.

5. Relevant Action Noise Data

Under the Aircraft Noise (Dublin Airport) Regulation Act 2019, the Aircraft Noise Competent Authority (ANCA) shall ensure that the Balanced Approach is adopted where a noise problem at the airport has been identified, taking account of the Noise Abatement Objective at the airport. Dublin Airport currently does not have a Noise Abatement objective and ANCA is required to create one where a noise problem has been identified. As part of planning application F20A/0668, ANCA carried out a screening process to identify whether the Proposed Development may give rise to a noise problem (https://www.fingal.ie/sites/default/files/2021-02/20210209-aspects-of-a-potential-noise-problem-assoc-with-f20a-0668-.pdf).

The 5 key summary observations from the screening study were:

- The harmful effects of aircraft noise in the future with the Proposed Development will be worse than without, particularly at night. As such the Proposed Development will increase aircraft noise rather than reduce it.
- Some people will experience elevated levels of night-time noise exposure for the first time which may be considered harmful to human health.
- The Proposed Development gives rise to significant adverse night-time noise effects as reported within the EIAR. This indicates that the noise effects of the Proposed Development are a material consideration.
- Mitigation in the form of a night-time noise insulation scheme is proposed by the Applicant. The provision of such mitigation is an indicator that the Proposed Development may give rise to a Noise Problem.
- The nature of the Proposed Development is to enable a form of operation which was not considered by ABP in their original decision to grant consent for the North Runway. Such a change will attract significant third-party interest, particularly from communities, who may perceive there to be a noise problem

Following this screening study, ANCA determined “that the proposed development may significantly influence the evolving noise climate at Dublin Airport to the extent that presents a noise problem that requires detailed assessment” (https://www.fingal.ie/sites/default/files/2021-02/20210210-anca-recommendation-report-.pdf) and recommended the following:

1. The determination of a noise problem at Dublin Airport, in the context of the 2019 Act and the Aircraft Noise Regulation, arising from the Application for a Relevant Action ref. F20A/0668.
2. The establishment of a Noise Abatement Objective for Dublin Airport.
3. The commencement of the process of aircraft noise regulation prescribed by Section 34C of the Planning and Development Act of 2000 including the application of the ICAO Balanced Approach.

From section 2.1.4 onwards, the EIAR discusses the Noise Abatement Objective. To support their application the daa have developed a candidate NAO (cNAO). The summary objective of the cNAO is:

“To limit and reduce the adverse effects of long-term exposure to aircraft noise, including health and quality of life, so that long-term noise exposure, particularly at night, does not exceed the situation in **2018**. This should be achieved through the application of the Balanced Approach”.

Section 2.1.8 states that 2018 was chosen as it was the most recent year with full data available when the relevant action assessment process commenced. It was also the first year of the 2018-2023 Dublin Airport Noise Action Plan (NAP). However, the NAP only considered data up to 2016, from the 3rd Round of the END, and data from 2017 and 2018 was not considered. Therefore the 2018-2023 NAP did not consider the most up to date data available to it when it was approved in December 2019 by members of Fingal County Council.

The selection of the baseline year to compare noise against for the NAO is of paramount importance to protect the health and well-being of residents. In the noise problem screening document (https://www.fingal.ie/sites/default/files/2021-02/20210209-aspects-of-a-potential-noise-problem-assoc-with-f20a-0668-.pdf), from section 6.4 a discussion of the historic noise situation at Dublin Airport is given using the data from the 3 Rounds of the Environmental Noise Directive (END) in 2006, 2011 and 2016 and compare with 2018 and 2019. Table 5 shows the Lnight comparison.

Table 5 Reported Night-time Noise Exposure (L_{night}) for Dublin Airport

Noise Band Lnight dB(A)	Population Exposed				
	2006	2011	2016	2018	2019
50 - 54.9	1,800	1,200	6,200	11,600	12,300
55 - 59.9	200	200	400	700	1,400
60 - 64.9	0	0	0	0	100
65 - 69.9	0	0	0	0	0
>=70	0	0	0	0	0

Section 6.7 states that “Over the period 2006 to 2019 the population reported to be exposed to night-time noise above 50 dB Lnight had increased by a multiple of **seven**”. 2018 was the noisiest year on record where the 32m passenger cap wasn’t breached (In 2019 the Airport handled 32.9m exceeding its planning permission).

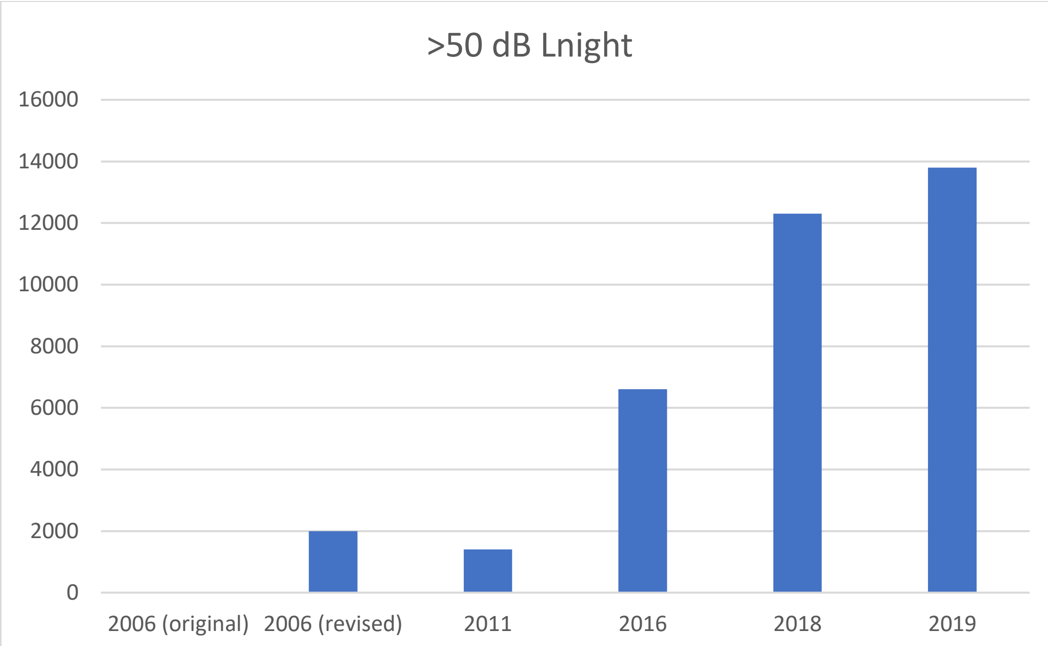
It is also worth noting that the 2006 Lnight figures used in the noise screening document (Table 5 a) were not the figures presented in the 2006 NAP. The figures presented in the screening document are revised figures based on the 2016 census. The population of Fingal is given as 296214 in the 2016 census, 273051 in the 2011 census and 239992 in the 2006 census. As a result, using the 2016 census data for the 2006 Lnight calculation will inflate the figures as the population grew by 56k or 23% in that timeframe.

The original statistics from the 2006 NAP show zero people affected <50 dB Lnight.

Noise Action Plan for Dublin Airport 2019 - 2023

Table 7 Population within Noise Level Band Data for Total Area L_{night}

Noise Band L_{night} dB(A)	2006 (original)	2006 (revised)	2011	2016
50 – 54.9	0	1,800	1,200	6,200
55 – 59.9	0	200	200	400
60 – 64.9	0	0	0	0
65 – 69.9	0	0	0	0
>= 70	0	0	0	0



The chart above clearly shows an escalating noise problem over the 3 Rounds of the END.

Comparing the >45 dB Lden and >40 dB Lnight contour sizes for 2016 and 2018 using the Reporting Templates https://www.fingal.ie/sites/default/files/2021-06/20210618-reporting-template-update.xlsx and https://www.fingal.ie/sites/default/files/2021-08/20210827-anca-reporting-template-update-2016-end.xlsx, it's very clear that the size of the contours increased significantly in 2018 compared to 2016.

Year	Population				Area (km^2)	
	>45dB Lden	>40dB Lnight	>55dB Lden	>50dB Lnight	>45dB Lden	>40dB Lnight
2016			20300	6600	370.5	212.8
2018	716726	307458	35482	12316	703.2	304.4

Comparing the populations exposed to >55 dB Lden and >50 dB Lnight between 2016 and 2018, shows a significant increase in numbers affected. From the area contours above, it is evident that the increase in the populations affected is due to the increase in the contours and not encroaching developments.

2018 was the noisiest year on record at Dublin Airport where the passenger limit wasn't breached. There are no figures provided for 2016 for the lower contours of >45 dB Lden and >40 dB Lnight beyond which the WHO states lead to adverse health effects.

For 2018:

- 716k people >45 dB Lden and 307k people >40 dB Lnight.
- Over 12k people affected >50 dB Lnight
- Over 35k people exposed to >45 dB Lden.

These levels cannot be used as acceptable baseline levels to compare against. Using 2018 for the NAO is detrimental to health of residents. The Local Authority and Competent Authority have allowed levels of noise, deemed unsafe by the WHO, to be inflicted on a significant number of residents.

It is worth noting that the members of Fingal County Council approved new noise zones for planning purposes on December 9th 2019, via Variation No.1 of the Fingal Development Plan 2017-2023 (https://www.fingal.ie/sites/default/files/2020-01/adopted-fdp-variation-1.pdf). Variation No.1 took on board the growing scientific evidence that night-time noise is detrimental to health and included Lnight metrics in the definition of the zones.

Table 7.2 Aircraft Noise Zones

Zone	Indication of Potential Noise Exposure during Airport Operations	Objective
D	≥ 50 and < 54 dB $L_{Aeq, 16hr}$ and ≥ 40 and < 48 dB L_{night}	**To identify noise sensitive developments which could potentially be affected by aircraft noise and to identify any larger residential developments in the vicinity of the flight paths serving the Airport in order to promote appropriate land use and to identify encroachment.** *All noise sensitive development within this zone is likely to be acceptable from a noise perspective. An associated application would not normally be refused on noise grounds, however where the development is residential-led and comprises non-residential noise sensitive uses, or comprises 50 residential units or more, it may be necessary for the applicant to demonstrate that a good acoustic design has been followed.* *Applicants are **advised** to seek expert advice.*
C	≥ 54 and < 63 dB $L_{Aeq, 16hr}$ and ≥ 48 and < 55	**To manage noise sensitive development in areas where aircraft noise may give rise to annoyance and sleep disturbance, and to ensure, where appropriate, noise insulation is incorporated within the development** *Noise sensitive development in this zone is less suitable from a noise perspective than in Zone D. A noise assessment **must** be undertaken in order to demonstrate good acoustic design has been followed.*

	dB L_{night}	*The noise assessment must demonstrate that relevant internal noise guidelines will be met. This **may** require noise insulation measures.* *An external amenity area noise assessment **must** be undertaken where external amenity space is intrinsic to the development's design. This assessment should make specific consideration of the acoustic environment within those spaces as required so that they can be enjoyed as intended. Ideally, noise levels in external amenity spaces should be designed to achieve the lowest practicable noise levels.* *Applicants are **strongly advised** to seek expert advice.*
B	≥ 54 and < 63 dB $L_{Aeq,\ 16hr}$ and ≥ 55 dB L_{night}	**To manage noise sensitive development in areas where aircraft noise may give rise to annoyance and sleep disturbance, and to ensure noise insulation is incorporated within the development.** *Noise sensitive development in this zone is less suitable from a noise perspective than in Zone C. A noise assessment **must** be undertaken in order to demonstrate good acoustic design has been followed.* *Appropriate well-designed noise insulation measures **must** be incorporated into the development in order to meet relevant internal noise guidelines.* *An external amenity area noise assessment **must** be undertaken where external amenity space is intrinsic to the developments design. This assessment should make specific consideration of the acoustic environment within those spaces as required so that they can be enjoyed as intended. Ideally, noise levels in external amenity spaces should be designed to achieve the lowest practicable noise levels.* *Applicants **must** seek expert advice.*
A	≥ 63 dB $L_{Aeq,\ 16hr}$ and/or ≥ 55 dB L_{night}	**To resist new provision for residential development and other noise sensitive uses.** *All noise sensitive developments within this zone may potentially be exposed to high levels of aircraft noise, which may be harmful to health or otherwise unacceptable. The provision of new noise sensitive developments will be resisted.*

Notes:

- 'Good Acoustic Design' means following the principles of assessment and design as described in ProPG: Planning & Noise – New Residential Development, May 2017;
- Internal and External Amenity and the design of noise insulation measures should follow the guidance provided in British Standard BS8233:2014 'Guidance on sound insulation and noise reduction for buildings'

Noise Zone D includes a night noise contour band as low as >=40dB Lnight and <48 dB Lnight.

The new noise zones were adopted in December 2019 to take account of night-time noise from a planning perspective. Immediate mitigations plans should have been introduced to limit the health impacts to the populations exposed to such night-time noise levels.

Objective DA07 was included in Variation No.1. It states:

"Objective DA07: Strictly control inappropriate development and require noise insulation where appropriate in accordance with table 7.2 above within Noise Zone B and Noise Zone C and where necessary in Assessment Zone D, and actively resist new provision for residential development and other noise sensitive uses within Noise Zone A, as shown on the Development Plan maps, while recognising the housing needs of established families farming in the zone. **To accept that time based operational restrictions on usage of a second runway are not unreasonable to minimize the adverse impact of noise on existing housing within the inner and outer noise zone**."

Objective DA07 facilitates the use of operating restrictions to mimise the adverse effects of noise

Oral Hearing

Planning permission was granted for the North Runway in 2007 following an Oral Hearing. The original planning decision by Fingal County council was appealed to the Appeals body, An Bord Pleanala (ABP), and they proceeded to conduct an Oral Hearing. They employed Mr Rupert Thornely-Taylor as their independent noise consultant. Mr Thornely-Taylor provided a report to the Oral Hearing and using data provided in Table 1 of additional request number 3, he concluded that the number of dwellings in the 63dB LAeq16 contour increased from 112 to 185 between 2007 and Option 7b 2025 High Growth and the number of people increased from 336 to 439. Mr Thornely-Taylor added that the number of dwellings in the 57dB LAeq16 contour increased from 1801 to 3225 from 2007 to Option 7b 2025 High Growth and the number of people increased from 5403 to **7431**.

Using the Reporting Template (https://www.fingal.ie/sites/default/files/2021-06/20210618-reporting-template-update.xlsx) provided to ANCA, the population >57dB LAeq in 2018 was **9177** and **9706** for 2019. These figures are far higher than the 7431 value for Option 7b 2025 High Growth which were deemed unacceptable by Mr Thornely-Taylor.

(It is also worth noting that the original EIS for the North Runway used High Growth projections of 348k movements and 43m passengers which far exceed the 32m projections used in this Relevant Action.)

Mr Thornely-Taylor further stated that the "*proposed development will result in an extension of the significant effects of noise as indicated by the population counts given…This conclusion is predicated on confinement of the use to Option 7b and a ban on the use of the proposed new runway between the hours of 2300 and 0700. This will be partially offset by the noise mitigation scheme as a result of the extension to the noise insulation programme, the buy-out scheme and the scheme for noise insulation of schools, but outside the limits of these schemes there will be an increase in noise exposure for the people affected.".*

As stated in section 2.4 above, this Relevant Action only applies to dwellings >55dB Lnight and those judged by the daa to be very significantly affected (>50dBLnight plus +9dB change in noise levels).

- Dwellings are considered eligible if they meet either of the following noise related criteria:
  1. Dwellings forecast to be exposed to "high" night-time noise levels in 2025 - at least 55dB $L_{night.}$
  2. Dwellings with a "very significant" rating arising from forecast noise levels of at least 50dB L_{night} in the first full year when the Relevant Action comes into operation, with a change of at least +9dB when compared with the current permitted operation in the same equivalent year.

It was pointed out in section 2.4 above that the daa's significance levels underestimate the population significantly affected by noise. By restricting their mitigation to this narrow cohort of the population, Mr Thornely-Taylor's comments still apply that "***outside the limits of these (insulation) schemes there will be an increase in noise exposure for the people affected***".

Forecast noise levels less than 2018 levels were not acceptable to Mr Thornely-Taylor and ABP when granting permission for the North Runway and therefore 2018 should not be acceptable as a baseline reference year, now or at any time in the future.

6. Proposed versus Permitted Scenarios

The Proposed Relevant Action, as defined in section 1of the EIAR, “relates to the night-time use of the runway system at Dublin Airport. It involves the amendment of the operating restriction set out in condition no. 3(d) and the replacement of the operating restriction in condition no. 5 of the North Runway Planning Permission, as well as proposing new noise mitigation measures.

The proposed Relevant Action, if permitted, would remove the numerical cap on the average number of flights permitted between the hours of 23:00 and 07:00 that is due to come into effect in accordance with the North Runway Planning Permission, replacing it with an annual night-time noise quota between 23:30 and 06:00 and also to allow flights to take off from and/or land on the North Runway (Runway 10L 28R) for an additional two hours i.e. 23:00 to 00:00 and 06:00 to 07:00. Overall, this would allow for an increase in the number of flights taking off and/or landing at Dublin Airport between 23:00 and 07:00 over and above the number stipulated in condition no. 5 of the North Runway Planning Permission, in accordance with the annual night-time noise quota”.

The Proposed Relevant Action seeks to amend Condition 3(d) as follows:

> ‘Runway 10L-28R shall not be used for take-off or landing between 0000 hours and 0559 hours
>
> 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 or where Runway 10L-28R length is required for a specific aircraft type.’

The Proposed Relevant Action seeks to amend Condition 5 as follows:

> ‘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 EIAR focuses on 2018 and Permitted and Proposed scenarios for 2022, 2025 and 2035. The Permitted scenario is the scenario with the existing planning restrictions remaining in place, namely no night-time flights on the new North Runway between 23:00-07:00 and only 65 flights between 23:00-07:00 on the existing South Runway.

The Proposed scenario represents the situation with the proposed Relevant Action in place. It assumes that the North Runway becomes operational but the airport is not constrained by the restrictions on night-time use of the runway system at Dublin Airport, namely the restriction on the number of flights permitted between the hours of 23:00 and 07:00 which limits the number of flights to an average of 65 between these hours (i.e. conditions no. 3(d) and no. 5).

The Permitted and Proposed scenarios for 2022, 2025 and 2035 also assume that the 32m passenger cap remains in place.

Highly Annoyed / Highly Sleep Deprived

The formulae to calculate HA and HSD were discussed in the WHO 2018 Guidelines and were added to Annex III of Directive 2002/49/EC via Directive 2020/367.

HA:

$$AR_{HA,air} = \left(-50.9693 + 1.0168 * L_{den} + 0.0072 * {L_{den}}^2\right) \Big/ 100 \qquad \text{(Formula 6)}$$

HSD:

$$AR_{HSD,air} = \left(16.7885 - 0.9293 * L_{night} + 0.0198 * {L_{night}}^2\right) \Big/ 100 \quad \text{(Formula 9)}$$

$$N_{x,y} = \sum_j \left[n_j * AR_{j,x,y}\right] \text{ (Formula 12)}$$

Where:

— $AR_{x,y}$ is the AR of the relevant harmful effect (HA, HSD), and is calculated using the formulas set out in point 2 of this Annex, calculated at the central value of each noise band (e.g.: depending on availability of data, at 50,5 dB for the noise band defined between 50-51 dB, or 52 dB for the noise band 50-54 dB),

— n_j is the number of people that is exposed to the j-th exposure band.

The number of people highly annoyed in 2018, 2022 / 2025 / 2035 Permitted scenarios are listed in Table 13-23 of the EIAR:

Table 13-23: Number of people highly annoyed – 2018 and Permitted Scenarios

Scenario	No. People Highly Annoyed	
	Excluding Consented Developments	Including Consented Developments
2018	110,238	120,205
2022 Permitted	50,603	58,880
2025 Permitted	64,241	73,209
2035 Permitted	33,437	41,234

The number of people highly sleep disturbed in 2018, 2022 / 2025 / 2035 Permitted scenarios are listed in Table 13-29 of the EIAR:

Table 13-29: Number of people sleep disturbed – 2018 and Permitted Scenarios

Scenario	No. People Highly Sleep Disturbed	
	Excluding Consented Developments	Including Consented Developments
2018	42,260	48,062
2022 Permitted	18,789	23,729
2025 Permitted	22,500	27,806
2035 Permitted	11,374	15,551

The comparison between the number of people highly annoyed and highly sleep disturbed between ‘2025 Proposed’ and ‘2025 Permitted’ are given in tables 13-45 and 13-50 of the EIAR:

Table 13-45: Number of people highly annoyed – 2025

Scenario	No. People Highly Annoyed	
	Excluding Consented Developments	Including Consented Developments
2025 Proposed	79,405	88,950
2025 Permitted	64,241	73,209

‘2025 Proposed’ leads to an additional **15164** people highly annoyed compared to ‘2025 Permitted’.

Table 13-50: Number of people highly sleep disturbed – 2025

Scenario	No. People Highly Sleep Disturbed	
	Excluding Consented Developments	Including Consented Developments
2025 Proposed	37,080	43,179
2025 Permitted	22,500	27,806

‘2025 Proposed’ leads to an additional **14580** people highly sleep disturbed compared to ‘2025 Permitted’.

Significant Effects

A comparison of the significant effects due to changes in day-time noise levels between '2025 Proposed' and '2025 Permitted' is given in Table 13-46 of the EIAR:

Table 13-46: Air Noise (L_{den}) People by Magnitude of effect – 2025 Proposed vs 2025 Permitted

Magnitude of effect	No. people with Beneficial Effect	No. people with Adverse Effect
Imperceptible	922	438,000
Not Significant	198	38,352
Slight	12	21,653
Moderate	0	12,598
Significant	0	67
Very Significant	0	0
Profound	0	0

No people assessed as having a significant beneficial benefit going from '2025 Permitted' to '2025 Proposed' but **67** people assessed as suffering a minimum of a significant adverse effect.

A comparison of the significant effects due to changes in night-time noise levels between '2025 Proposed' and '2025 Permitted' is given in Table 13-51 of the EIAR:

Table 13-51: Air Noise (L_{night}) People by Magnitude of effect – 2025 Proposed vs 2025 Permitted

Magnitude of effect	No. people with Beneficial Effect	No. people with Adverse Effect
Imperceptible	42	198,375
Not Significant	68	17,197
Slight	69	26,688
Moderate	5	14,578
Significant	0	11,350
Very Significant	0	104
Profound	0	40

No people assessed as having a significant beneficial benefit going from '2025 Permitted' to '2025 Proposed' but **11494** people assessed as suffering a minimum of a significant adverse effect.

Residual Effects

The residual effects, after allowing for the benefit of the residential sound insulation schemes are listed in Table 13-64 of the EIAR for both Lden and Lnight:

Table 13-64: Summary of Residual Air Noise Effects, Proposed vs Permitted

Year	L_{den} Residual Effects			L_{night} Residual Effects		
	Significant Beneficial	Significant Adverse	Not Significant	Significant Beneficial	Significant Adverse	Not Significant
2022	79	10	368,727	151	8,985	166,605
2025	8	54	511,742	86	10,560	257,813
2035	0	20	255,657	12	4,284	131,432

46 people suffer a significant adverse effect in terms of Lden and **10474** suffer a significant adverse effect in terms of Lnight between ‘2025 Permitted’ to ‘2025 Proposed’.

7. Noise Study

The client, SMTW Environmental DAC, conducted a noise study on 3 sample houses in the St Margarets area. The selected houses had been previously insulated by the daa as part of the planning conditions for the North Runway. The purpose of the study was to determine the indoor LAmax noise values after insulation.

The WHO 1999 Community Guidelines gives guideline values in chapter 4. For dwellings, it states:

"*The effects of noise in dwellings, typically, are sleep disturbance, annoyance and speech interference. For bedrooms the critical effect is sleep disturbance. Indoor guideline values for bedrooms are 30 dB LAeq for continuous noise and **45 dB LAmax** for single sound events. Lower noise levels may be disturbing depending on the nature of the noise source. At night-time, outside sound levels about 1 metre from facades of living spaces should not exceed 45 dB LAeq, so that people may sleep with bedroom windows open. This value was obtained by assuming that the noise reduction from outside to inside with the window open is 15 dB*".

The WHO 2009 Night Noise Guidelines reference the 1999 Guidelines and comment that new studies had become available since 1999 and that the thresholds are now known to be lower than 45 dB LAmax for a number of effects. It reiterates that the advice in the 1999 Guidelines are still valid and that the 2009 NNG for Europe are complimentary to the 1999 guidelines.

Based on the WHO Guidelines the aim of the study was to detect the number of occurrences of indoor noise values exceeding 45 dB LAmax. The study was carried out by MLM Consulting Engineers and the conclusions state:

"*It was found that two of the three locations have a number of exceedances over the guideline =<10 events > 45 dB L_{Afmax}. It should be noted that these events are likely to increase once restrictions ease following the COVID-19 pandemic and Dublin Airport returns to operating at normal capacity*".

At location number 1, there were 20 events that exceeded the 45 dB LAmax limit. At location number 2, there were 17 events exceeding 45 dB LAmax. There was just one occurrence of an exceedance of 45 dB LAmax at location number 3.
These 3 properties fall within the Residential Noise Insulation Scheme (RNIS) and have been insulated. The recorded noise levels exceed the WHO 1999 Guidelines for single noise events.

45 dB LAmax is also set as a reference limit in the ProPG Guidelines. It recommends not to exceed it more than 10 times a night.

ACTIVITY	LOCATION	07:00 – 23:00 HRS	23:00 – 07:00 HRS
Resting	Living room	35 dB $L_{Aeq,16\,hr}$	-
Dining	Dining room/area	40 dB $L_{Aeq,16\,hr}$	-
Sleeping (daytime resting)	Bedroom	35 dB $L_{Aeq,16\,hr}$	30 dB $L_{Aeq,8\,hr}$ **45 dB $L_{Amax,F}$** (Note 4)

Based on the results of this study, it's evident that the insulation provided fails to maintain an internal safe environment and that the noise levels are detrimental to the owners. It is worth noting that aircraft activity at Dublin Airport at the time of the study was severely restricted because of Covid-19. It is a safe assumption that more violations of the 45 dB LAmax limit would be expected when normal airport activity resumes. An immediate restriction on night-time flights needs to be imposed to maintain safe levels of habitation for the residents affected.

This study is of extreme importance in the context of the work just published by Saucy et al. **A nocturnal aircraft noise event at Zurich Airport can cause acute cardiovascular death 2 hours later** (Figure 7) (30).

For the night-time deaths (Figure 5b), average LAmax was 57dB with events up to 85dB. The median NAT55 ranged between 0 and 75 flights for the 2h exposure window preceding the time of case and control events.

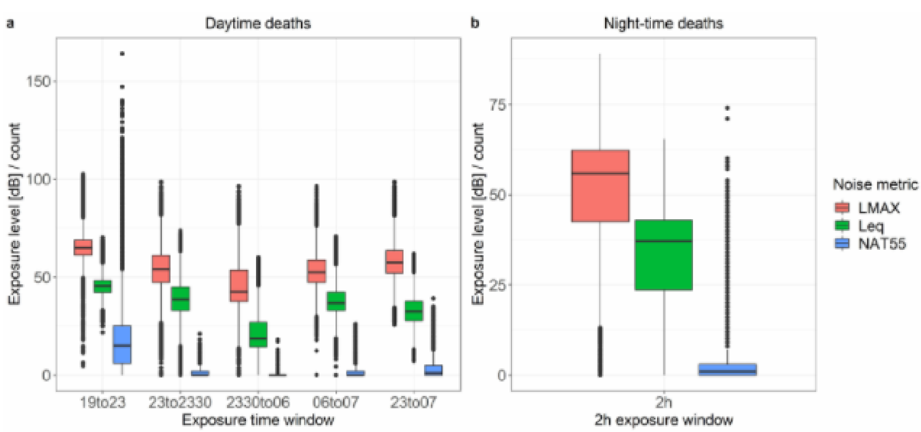


Figure 5. (a) Distribution of the noise exposure levels L_{Amax} and L_{Aeq} (in dB) as well as NAT $_{55}$ (count) for the different time windows among all events (case and control) for daytime deaths, years 2000–2015. (b) Distribution of the noise exposure levels L_{Amax}, L_{Aeq} and NAT_{55} for the 2 h exposure window among the events (case and control) for nighttime deaths, years 2000–2015. The horizontal line of the box-plot represents the median value, the squares the interquartile range (IQR), and the whiskers the lower and upper limits (lower IQR value—1.5*IQR/upper IQR value + 1.5*IQR).

8. Population and Human Health

Chapter 7 of the EIAR is titled Population and Human Health. It discusses the relevant legislation applicable to the proposed Relevant Action. It refers to the WHO 2018 Guidelines and states that "*the guidelines provide robust public health advice underpinned by evidence*". It reiterates the strong recommendations from the WHO regarding aircraft noise:

- For average daytime noise exposure, it is strongly recommended that daytime noise levels produced by aircraft are below **45 dB Lden**;

- For average night noise exposure, it is strongly recommended that noise levels that night noise levels produced by aircraft are below **40 dB Lnight**; and

- To reduce health effects, policy makers should implement suitable measures to reduce noise exposure where the population is exposed to levels above the guideline values for average daytime and night noise exposure.

Section 7.4 focuses on a literature review on the potential health impacts of the Relevant Action on human health and well-being. It discusses the key health outcomes: annoyance, sleep disturbance, cardiovascular health, mental health and cognitive learning in children. This review of the key health outcomes is less than 2 pages in length. No metrics or statistics are provided from the review of the literature, as are provided in this report under 'Findings from noise impact research'. In that regard the literature review is of poor quality and doesn't help to inform or help quantify the impact of the adverse health outcomes on the populations affected.

Chapter 7 also details the findings of an assessment of the likely effects on population and human health as a result of the proposed Relevant Action.

Table 7-16 presents the number of people assessed to have a residual significant beneficial and residual significant adverse effect comparing the Proposed vs Permitted scenarios for day (Lden) and night-time (Lnight).

For Lnight, net **10474** people will be **significantly adversely affected** by the Proposed scenario compared to the Permitted scenario in 2025.

Table 7-16 Residual Air Noise and Vibration Significant Effects, Proposed vs Permitted

Year	L_{den} 24-hour period metric				L_{night} Overnight Metric			
	Significant Beneficial	Percentage of population proportion	Significant Adverse	Percentage of population proportion	Significant Beneficial	Percentage of population proportion	Significant Adverse	Percentage of population proportion
2022	79	<0.01%	10	<0.01%	151	<0.01%	8,985	3.0%
2025	8	<0.01%	54	<0.01%	86	<0.01%	10,560	3.6%
2035	0	0.0%	20	<0.01%	12	<0.01%	4,284	1.5%

Table 7-24 presents the number of people highly annoyed by aircraft noise for 2022, 2025 and 2035 Proposed and Permitted scenarios.
There is an increase of **15164** (+23.6%) people **highly annoyed** in 2025 Proposed compared with 2025 Permitted.

Table 7-24 Number of people highly annoyed by air noise and vibration (Permitted vs Proposed scenarios)

Scenario	Number of people highly annoyed			
	Excluding consented developments	Percentage of population highly annoyed	Including consented developments	Percentage of population highly annoyed
2022 Permitted	50,603	17.1%	58,880	19.9%
2022 Proposed	52,713	17.8%	61,161	20.7%
2022 Permitted vs Proposed Change	+2,110 (+4.2%)	-	+2,281 (+3.9%)	-
2025 Permitted	64,241	21.7%	73,209	24.7%
2025 Proposed	79,405	26.8%	88,950	30.0%
2025 Permitted vs Proposed Change	+15,164 (+23.6%)	-	+15,741 (+21.5%)	-
2035 Permitted	33,437	11.3%	41,234	13.9%
2035 Proposed	39,693	13.4%	47,963	16.2%
2035 Permitted vs Proposed Change	+6,256 (+18.7%)	-	+6,729 (+16.3%)	-

Table 7-25 presents the number of people highly sleep disturbed by aircraft noise for 2022, 2025 and 2035 Proposed and Permitted scenarios.

Table 7-25 Number of people highly sleep disturbed by air noise and vibration (Permitted vs Proposed scenarios)

Scenario	*Number of people highly sleep disturbed*			
	Excluding consented developments	*Percentage of population highly sleep disturbed*	*Including consented developments*	*Percentage of population highly sleep disturbed*
2022 Permitted	18,789	6.3%	23,729	8.0%
2022 Proposed	19,188	6.5%	23,885	8.1%
2022 Permitted vs Proposed Change	+399 (+2.1%)	-	+156 (+0.7%)	-
2025 Permitted	22,500	7.6%	27,806	9.4%
2025 Proposed	18,789	6.3%	23,729	8.0%
2025 Permitted vs Proposed Change	-3,711 (-16.5%)	-	-4,077 (-14.7%)	-
2035 Permitted	11,374	3.9%	15,551	5.3%
2035 Proposed	18,711	6.3%	23,567	8.0%
2035 Permitted vs Proposed Change	+7,337 (+64.5%)	-	+8,016 (+51.5%)	-

There is a major error in this table as the 2025 Proposed figures are incorrect and appear to be a copy of the 2022 Permitted values.

Table 13-50 compares the number of people highly sleep disturbed between 2025 Permitted and 2025 Proposed and the figure for 2025 Proposed is given as 37080 excluding consented developments. Section 13.4.49 discusses these figures and asserts that 2025 Proposed leads to a +65% increase in people highly sleep disturbed.

Table 13-50: Number of people highly sleep disturbed – 2025

Scenario	No. People Highly Sleep Disturbed	
	Excluding Consented Developments	Including Consented Developments
2025 Proposed	37,080	43,179
2025 Permitted	22,500	27,806

13.7.49 Comparing the 2025 Proposed Scenario with the 2025 Permitted Scenario, the number of people exposed to aircraft noise is forecast to increase, for all contour levels. Consequently, the number of people assessed as highly sleep disturbed by aircraft noise increases by 65% from 22,500 to 37,080 (excluding consented developments). The number of people exposed to at least a high level of noise (i.e. 55 dB L_{night} or above) increases from 280 to 1,059.

There is an increase of **14580** (+65%) people **highly sleep disturbed** in 2025 Proposed compared with 2025 Permitted.

As a result of this error, the EIAR has underestimated the number of people highly sleep disturbed by 18291. Therefore, the EIAR has not adequately assessed the health impact of these 18291 people due to this error.

9. HSE Environmental Health submission

It is noted that the Project Team listed in Table 1-2 of the EIAR does not contain any experts in the medical profession. The serious adverse effects of aircraft noise highlighted in this report are:

- Coronary heart disease
- High blood pressure
- Heart attack
- Stroke
- Sudden deaths
- Cognitive developmental disorders in children
- Sleep and sleep disorders
- Mental illness

Expert opinion from the relevant domain areas are required and should inform the decision-making process.

It is worth highlighting a submission to the original planning application dated the 28th of January 2021 from the Environmental Health section of the Health Service Executive (File ref 686993).

It states that the HSE's assessment was based on the WHO Environmental Noise Guidelines 2018, as endorsed by the European Commission through Directive 2020/367.

The submission comments on the number of people highly annoyed:

> While the EHS welcomes the significant reduction in the people exposed to airline noise between the 2018/2019 baseline and the 2022/2025 forecast baseline scenario it still acknowledges that a significant proportion of people, namely 63,316 people assessed as highly annoyed and 128 people exposed to at least a high noise level based on the 2025 baseline scenario, will still be exposed to airline noise above the WHO recommendations of 45Lden.
>
> The WHO 2018 Noise Guidelines strongly recommends reducing night noise exposure levels produced by aircraft during night time below 40 dB Lnight, as it states aircraft noise above this level is associated with adverse effects on sleep.

And on the number of people highly annoyed:

> **While the EHS welcomes the significant reduction in the people exposed to airline noise between the 2018/2019 baseline and the 2022/2025 forecast baseline scenario it still acknowledges that a significant proportion of people, namely 19,464 people assessed as highly annoyed and 281 people exposed to at least a high noise level based on the 2025 baseline scenario, will still be exposed to airline noise above the WHO recommendations of 40Lnight.**

The HSE submission lists the WHO's critical health outcomes associated with aircraft noise and reiterated the WHO's strong recommendations to reduce noise levels <45dB Lden and <40dB Lnight.

- Cardiovascular disease
- Annoyance
- Cognitive Impairment
- Hearing impairment and tinnitus
- Adverse birth outcomes
- Quality of life, well-being and mental health
- Metabolic outcomes

The HSE further states "**that the WHO levels of 45 dB Lden and 40 dB Lnight should be used when assessing eligibility for schemes such as the sound insulation improvement works**".

The conclusion from the HSE submission is very strong stating all efforts should be made by the DAA to ensure as many people as possible are protected from the adverse health effects associated with aircraft noise. And that this **must** include reducing aircraft noise levels to <45dB Lden and <40dB Lnight.

> **Conclusion:**
> The EHS makes the following observations in relation to this proposed development:
>
> - **All efforts should be made by the DAA to ensure as many people as possible are protected from the adverse health effects associated with aircraft noise as outlined above in this report. This must include reducing aircraft noise levels to below 45 dB Lden, and for night noise exposure to below 40 dB Lnight.**
>
> - **The EHS if of the opinion that The World Health Organisation's Environmental Noise Guidelines of 45dB Lden and 40 dB Lnight should have been used for ground noise assessments.**

10. Summary

As part of the planning approval procedure for the Relevant Action planning application to amend and revoke operating restrictions at Dublin Airport, we were asked to prepare a noise medical report.

It is important to note that a new level of noise impact research has been achieved since 2007, which proves that the mitigation measures under the planning conditions for the North Runway are not sufficient to exclude negative health effects and, above all, do not cover the area of noise prevention.

In particular, significant progress has been made in the past 8-10 years with regard to the knowledge of the health consequences of night-time aircraft noise based on epidemiological studies, translational aircraft noise research on healthy subjects and patients with established coronary heart disease, as well as based on translational animal research, which have made it possible for us to identify the causes of possible damage for our cardiovascular system and brain to be better understood and to be able to take countermeasures accordingly.

Clinical studies, in particular the work of Röösli and colleagues, have dealt with the health consequences of night-time aircraft noise and have come to the following results:

- First and foremost, night aircraft noise leads to increased stiffness of the vessels (48).
- Nocturnal aircraft noise leads to increased deaths from heart attack (27).
- Night-time aircraft noise triggers acute cardiovascular death 2 hours after the flight noise event (30), independent of other traffic noise sources such as road outdoor and rail transport.

Another important point is the annoyance response from aircraft noise. In large epidemiological studies, but also in laboratory studies, it was repeatedly measured that aircraft noise > road traffic noise > rail noise triggers an annoyance reaction (39). This in turn has multiple negative effects on health and has been shown to be associated with an increased frequency of cardiac arrhythmias (40), activates neurohumoral systems that play an important role in heart failure (41), and is responsible for cognitive developmental disorders in children (54, 62). The importance of the publication by the working group from Harvard University is outstanding, since it was able to prove that an emotional stress reaction as a result of road and aircraft noise can lead to increased vascular inflammation and secondary to more cardiovascular events (43). Among other things, this supports the research results of Wolfgang Babisch (73), so that noise

pollution must be viewed as an effect modifier, i.e. the more one feels annoyed by aircraft noise (resulting in activation of the amygdala or the limbic system), the sooner one has to expect cardiovascular events such as cardiovascular death or death from heart attack.
.

Translational research on the topic of night aircraft noise, vascular function and stress reactions in humans have been reported in particular by the Johannes Gutenberg University Mainz. Field tests in collaboration with Prof. Basner (formerly DLR) and Dr. Babisch can prove that simulated night aircraft noise (aircraft noise Cologne / Bonn)

1) Triggers a vascular dysfunction that leads to increased stiffness of the vessels.
2) This can primarily be explained by an increased release of stress hormones.
3) The increase in rigidity is explained by the formation of free radicals in the vessel wall
4) The finding is more pronounced in patients who have already been diagnosed with coronary heart disease and thus already have vascular damage.
5) The increases in blood pressure caused by night-time flight noise are also more pronounced in heart patients
6) A stress activation of the body could also be measured with the help of polygraphic measurements.
7) For the first time it has been shown that post-flight noise can trigger a diastolic form of heart failure.
8) The mean sound level and not the peak sound level is decisive with regard to the extent of the deterioration in vascular function.

A series of **translational animal studies** resulted in models that examined the negative health effects observed in humans and gave the following results. Aircraft noise, and here in particular night-time aircraft noise (52), leads

1) to increases in blood pressure
2) an increase in stress hormone levels
3) to a vascular dysfunction (endothelial dysfunction)
4) to the increased formation of free radicals in the vessel wall and in the brain
5) to a down-regulation of the neuronal NO synthase, an enzyme that
controls the function of memory and learning
6) leads to drastic inflammatory reactions in the brain and in the blood vessels
7) disrupts the circadian rhythm
8) “White noise” as a control showed no negative effects on the cardiovascular system, despite the same mean sound level.

What are the consequences of these negative health effects of aircraft noise?

- 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 Relevant Action proposal only aims to mitigate those ‘very significantly’ affected by night-time noise, leaving a large proportion of the population subjected to noise levels beyond the WHO recommended safe limits.

- The noise data presented for the Relevant Action proposal shows a significant increase in the population highly annoyed, highly sleep disturbed, significantly adversely affected and an increase in those suffering residual effects after mitigation.

- The noise data presented for 2018 shows 716k people exposed to noise levels >45dB Lden and 307k people exposed to >40dB Lnight. These extremely high numbers of the Irish population exposed to adverse health effects of noise cannot be accepted as a baseline and the authorities have an opportunity through the Noise Abatement Objective to safeguard and protect their heath.

- 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.

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12. Appendix A: Environmental risk factors and cardiovascular diseases: a comprehensive review (Abstract, Chapter 1: Introduction, Chapter 2: Noise and cardiovascular risk)

Thomas Münzel, MD, Omar Hahad, PhD, Mette Sørensen, PhD, Jos Lelieveld, PhD, Georg Daniel Duerr, MD, Mark Nieuwenhuijsen, PhD, Andreas Daiber, PhD

Abstract:
Noncommunicable diseases (NCDs) are fatal for more than 38 million people each year and are thus the main contributors to the global burden of disease accounting for 70% of mortality. The majority of these deaths are caused by cardiovascular disease. The risk of NCDs is strongly associated with exposure to environmental stressors such as pollutants in the air, noise exposure, artificial light at night and climate change, including heat extremes, desert storms and wildfires. In addition to the traditional risk factors for cardiovascular disease such as diabetes, arterial hypertension, smoking, hypercholesterolemia and genetic predisposition, there is a growing body of evidence showing that physicochemical factors in the environment contribute significantly to the high NCD numbers. Furthermore, urbanization is associated with accumulation and intensification of these stressors. This comprehensive expert review will summarize the epidemiology and pathophysiology of environmental stressors with a focus on cardiovascular NCDs. We will also discuss solutions and mitigation measures to lower the impact of environmental risk factors with focus on cardiovascular disease.

1. Introduction
Cardiovascular diseases (CVDs), besides chronic respiratory and metabolic diseases, constitute a large part of noncommunicable diseases (NCDs), including acute and chronic coronary artery disease, heart failure and arrhythmia, stroke and arterial hypertension. Importantly, 70% of annual global deaths (around 40 Mio people) can be attributed to NCDs and this share will further increase by 10% according to the World Health Organization (WHO) projections for the year 2030 [1]. NCDs account for 80.6% [95% confidence interval (CI) 78.2–82.5] of age-standardized years lived with disability in 2016, as indicated by data of the Global Burden of Disease (GBD) study [2]. CVDs are responsible for the majority of deaths that are caused by NCDs [3]. In the GBD study (2019 update), the contribution of CVDs to overall global mortality continuously increased from 12.1 million in 1990 to 18.6 million in 2019 [4]. Interestingly, low- and middle-income countries have the highest share (86%) of premature deaths triggered by NCDs [5, 6]. The economic burden caused by NCDs are severe, and may amount to global economic costs of $47 trillion within the coming 20 years [7]. Risk factors for NCDs are mostly originating from the environment, which is supported by observations that up to 25% of all ischemic heart disease are related to an unhealthy environment, especially to air pollution [8]. Nevertheless, the environmental share to NCDs is notoriously ignored as reflected by the failure to mention environmental risk factors in the 2013 WHO NCD Global Action Plan [6]. In addition, research on, prevention of, and treatment of environmentally triggered NCDs are severely underfunded, relative to their disease

burden in the general population [9]. This dramatic gap is now paid more attention by the emerging “exposome” research field, investigating the life-long effects of all environmental exposures on biochemical pathways and health effects (**Figure 1**) [10, 11] as well as “healthy cities” campaigns [12, 13].

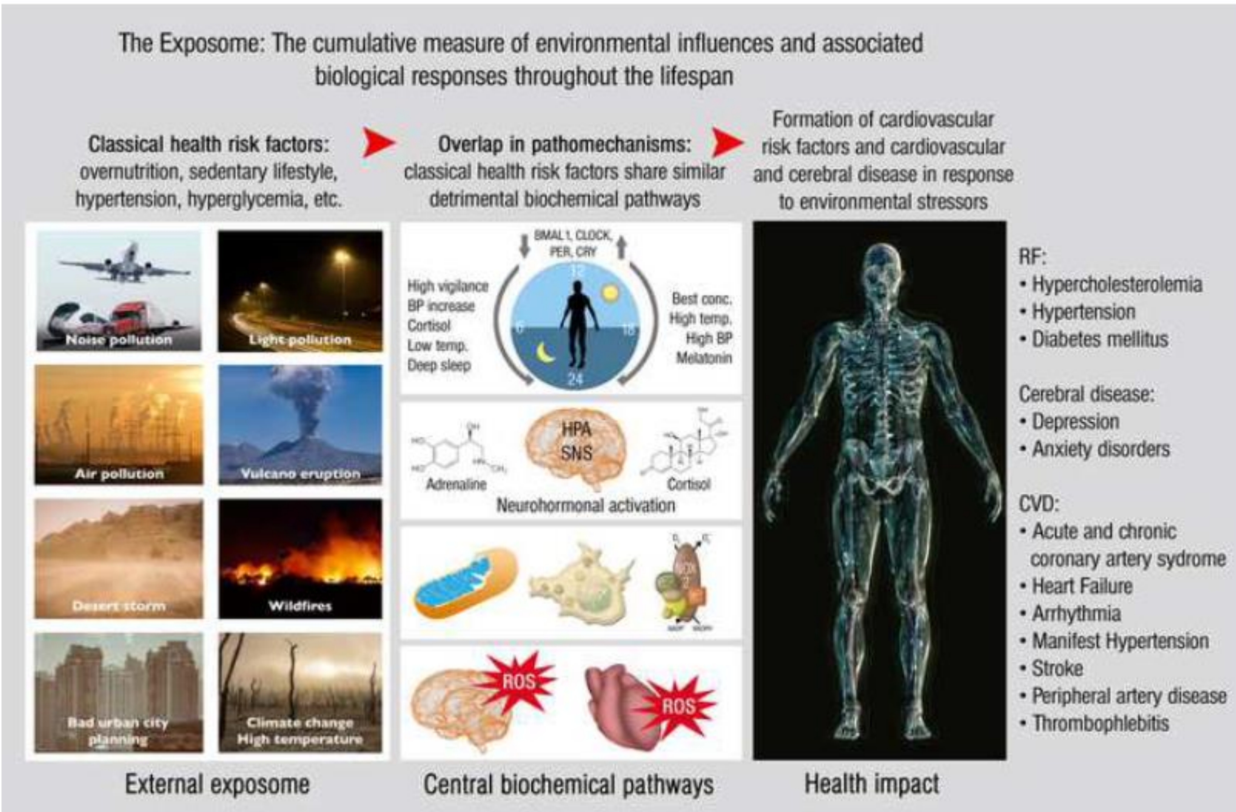

Figure 1. The exposome concept. Exposure to environmental risk factors (=external exposome) leads to changes of central biochemical pathways with associated health impact. The central biochemical pathways comprise changes in circadian clock genes leading to impaired rhythmicity and phase-shifts, stress hormone release (cortisol and catecholamines), production of reactive oxygen species by mitochondria and NADPH oxidase in activated immune cells, inflammation with tissue infiltration of activated immune cells, and oxidative damage in different organs. Because classical health risk factors share similar pathomechanisms, people with existing classical health risk factors or disease (e.g. diabetes or hypertension) may experience additive adverse health effects upon exposure to environmental risk factors. HPA, hypothalamic-pituitary-adrenal axis; SNS, sympathetic nervous system; NOX-2, phagocytic NADPH oxidase (isoform 2); ROS, reactive oxygen species. Merged and redrawn from previous reports 172, 267 with permission; Copyright © 2020, The Authors; Published by Elsevier B.V.

The exposome concept comprises a multi-exposure perspective [14]. Besides external environmental risk factors (e.g. traffic noise and air pollution), our lifestyle and environmental factors on the whole (e.g. socioeconomic status and climate) also

characterize the exposome of an individual [15, 16], the assessment of which requires a multidisciplinary approach using smart sensor devices, multi-OMICs techniques and big data handling using bioinformatics and systems biology approaches [17]. In order to better address these multi-exposure conditions, the refined "envirome" concept was developed, which is defined by three consecutively nested domains, consisting of natural, social, and personal environments that are monitored in parallel and connected to biochemical changes and health effects using "enviromics" [18]. Based on the increasing awareness of the major impact of environmental risk factors the term was coined "Genetics load the gun, but the environment pulls the trigger" [19].
This comprehensive expert review will summarize the epidemiology and pathophysiology of environmental stressors on NCDs, however without considering the contribution of other important environmental health risk factors, e.g. mental stress [20] and ionizing radiation (either by anticancer therapy [21] or ionospheric and geomagnetic exposures [22]). We will also discuss solutions and mitigation measures to lower the adverse health effects by environmental stressors with focus on CVDs

2. Noise and cardiovascular risk

2.1 Epidemiological evidence for adverse effects of noise on our health

Noise pollution from traffic is an increasing public health problem. Road traffic noise is the dominant source of transportation noise-associated health effects, and mapping of the European Union (EU) in 2019 showed that 113 million Europeans (20%) are subjected to a burden of road traffic noise that exceeds the limit of 55 dB(A) (L_{DEN}: day-evening night average) as suggested by the EU guideline [23]. This estimate is most likely underestimated, as the Environmental Noise Directive is not ubiquitously applied in all urban areas and roads in entire Europe [23].
In 2018, a WHO expert panel stated that there was high quality of evidence to conclude that road noise was associated with ischemic heart disease (IHD) [24]. Based on a meta-analysis, the group of experts calculated that per 10 dB increase in road noise the relative risk (RR) for IHD was 1.08 (95% confidence interval: 1.01-1.15), starting at chronic exposure levels of >50 dB where significant health effects were observed. For noise from trains and aircrafts in relation to IHD, the expert panel ranked the quality of evidence as very low and low, respectively, due to few high-quality studies. However, recent studies covering Switzerland, the Rhine-Main region and the island of Montreal have suggested that these noise sources may also be risk factors for myocardial infarction, although results are not consistent, and more evidence is needed [25-27].
For all other cardiovascular health effects excluding IHD, the WHO group of experts found very low, low or moderate evidence due to lack of high-quality studies [24]. **However, high-quality studies have subsequently emerged together with studies on new CVD outcomes and risk factors that were not studied in a noise context in the past, which we will summarize in the following suppl. Table S1**.
Numerous studies addressed whether traffic noise is a risk factor for hypertension, but unfortunately using a cross-sectional design in most cases [24]. The WHO group of

experts found >35 cross-sectional studies on traffic noise and hypertension, with a joined RR for prevalent hypertension of 1.05 (1.02-1.08) for road noise, but the quality level was judged as “very low” due to the inherent problem of the cross-sectional design [24]. Later studies on noise and hypertension incidence have reported inconsistent results [28-30]. However, there is a large variation between the different studies with regard to the way of hypertension was defined, which complicates reliable conclusions and warrants for more studies.

The quality of evidence for stroke incidence was by the WHO judged as moderate based on a single study that reported road noise to increase risk of stroke [24]. Subsequently, five studies on road traffic noise and incident stroke have been published: three large population-based studies that cover an entire region or country (London, Frankfurt and Denmark) found road noise to aggravate stroke risk [31-33], whereas smaller classical cohort studies from Sweden, Norway and UK with a limited number of cases (900-1900) found no association [34, 35]. Effects of noise on incident heart failure were not evaluated by WHO, but the few recent studies conducted have consistently showed transportation noise to increase the risk [25, 26, 36-38]. In contrast, the few studies investigating the impact of noise on atrial fibrillation have reported inconsistent results [37, 39].

Studies investigating transportation noise as a risk factor for cardiovascular death have been summarized in a meta-analysis from 2021 [40]. This study reported a pooled RR for road traffic noise per 10 dB of 1.02 (0.97-1.08) for IHD mortality and 1.06 (0.94-1.20) for stroke mortality (based on cohort and case-control studies) suggesting that road noise is associated with a slightly increased risk of cardiovascular mortality. However, the quality level of evidence was judged as moderate and more longitudinal high-quality studies are required. **Importantly, a study from 2021 investigating acute effects of aircraft noise led further support to noise from all sources of transportation as a risk factor of cardiovascular mortality [41]. The authors report that high aircraft noise exposure two hours preceding death was found to trigger night-time cardiovascular deaths, with an odds ratio of 1.44 (1.03-2.04) when comparing exposures >50 dB with <20 dB. As the first of its kind, this novel study needs to be reproduced**.

Epidemiological studies suggest associations of transportation noise, mainly from road traffic, with several cardiovascular risk factors (**suppl. Table S2**). One of these is disturbance of sleep, which is hypothesized to be a key pathway through which noise is thought to impair the cardiovascular system [42, 43]. A pooled analysis of polysomnographic studies on the adverse health effects of acute noise, found that the awakening probability was increased with greater exposure to road, rail and aircraft noise [44]. The study also found an association of nighttime noise with severe sleep disturbance (self-reported questionnaires).

A cardiovascular risk factor consistently found associated with road noise is metabolic disease. A 2019 meta-analysis found a RR of 1.11 (1.08-1.15) per 10 dB higher road noise for incident diabetes based on five high-quality longitudinal studies [45].

In support of noise as an important metabolic risk factor, several studies have found road noise associated with adiposity markers and obesity [46-49]. Of note, results demonstrating that central obesity and waist circumference are associated with noise are more consistent than results on BMI, which perfectly agrees with the concept that noise increases cortisol (stress hormone), which is known to cause mainly central obesity.

Some studies have reported on noise from all forms of transportation as a risk factor for an unhealthy lifestyle. According to two studies road noise exposure was associated with reduced physical activity, mainly with any leisure time sport and not intensity, implying that noise may influence whether people exercise at all [50, 51]. Furthermore, a study suggested that road noise may potentially be associated with alcohol consumption and smoking [52]. More studies investigating noise-induced changes in health behaviour are important as these may represent an important link between noise and CVDs.

Lastly, studies have suggested that road noise may cause higher risk of depression [25, 53, 54]. However, a complicating factor in these studies is that they use different definitions of depression, e.g. interviews, self-reports, use of antidepressants, and hospital admissions, making between-study comparisons difficult, and a 2020 review judged that the evidence for an association may be insufficient for an overall conclusion [55].

2.2 Mechanistic insights into noise-induced pathophysiology by clinical studies

The cognition of noise and the resulting cortical and sympathetic activation causes the generation of stress hormones (e.g. cortisol and catecholamines), with subsequent activation of the renin-angiotensin-aldosterone system. If chronically present this pathway may first lead to development of cardiovascular risk factors (e.g. hyperglycemia and hypercholesterolemia), blood clotting factor activation and high blood pressure, ultimately leading to myocardial infarction, heart failure, arterial hypertension, arrhythmia and stroke (**Figure 2A**) [56-58]. Moreover, noise causes sleep disturbance, interferes with activities and impairs communication, all of which can trigger annoyance and increased CVD risk.

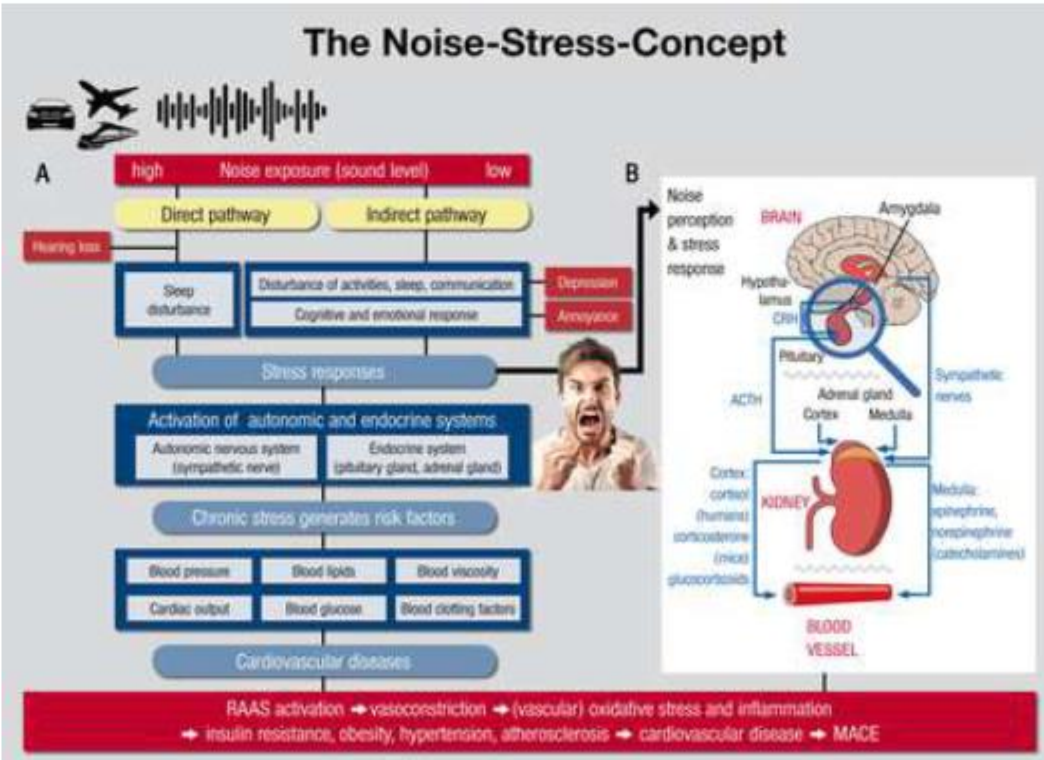

Figure 2. Noise-stress concept and the adverse health consequences in humans. (**A**) Noise reaction model for the direct (auditory) and indirect (non-auditory) effects of noise exposure. Adapted from 268 with permission; Copyright © 2014, Oxford University Press. (**B**) Neuronal activation (arousals), e.g. by noise exposure, causes signalling via the hypothalamic-pituitary-adrenocortical (HPA) axis and sympathetic nervous system (SNS) via corticotrophin-releasing factor (CRF) in the pituitary gland and adrenocorticotropic hormone (ACTH) in the adrenal gland leading to activation of other neurohormones (e.g. the renin-angiotensin-aldosterone system), inflammation and oxidative stress. The adverse effects of cortisol (or corticosterone) and catecholamines on cardiovascular function and molecular targets are well characterized. Adapted from 269 with permission; Copyright © 2013, Campos-Rodríguez et al.; Creative Commons Attribution License (CC BY).

Recently, it was established that the limbic system, more precise the amygdala nuclei, becomes activated in response to transportation noise caused by cars and aircraft [59]. In this study, around 500 patients underwent a ^{18}F-fluorodeoxyglucose positron emission tomography/computed tomography imaging investigation and the authors demonstrated that noise “dose-dependently” increased amygdala activity, with coronary inflammation and major adverse cardiovascular events (e.g. CVD death, myocardial infarction, stroke and coronary / peripheral revascularization) (**Figure 2B**) [59]. In a subsequent investigation the authors found that more pronounced resilience to chronic socioeconomic or environmental stressors such as transportation noise was clearly associated with lower risk for CVD events [60].

Translational field studies found adverse effects of simulated noise from aircrafts and trains on vascular function, stress hormone release, sleeping quality and inflammation markers in healthy subjects and coronary artery disease patients [43, 61, 62].

Furthermore, flow-mediated dilation (FMD) was found impaired by noise in an exposure dose-dependent manner, and the antioxidant vitamin C (2g p.o.) significantly improved FMD, pointing to an important role of reactive oxygen species in this phenomenon (**Figure 2C**) [43, 62]. Proteomic analysis of plasma proteins revealed that redox, pro-thrombotic and proinflammatory pathways were significantly affected in noise-exposed subjects as compared with unexposed controls [62], The impairment of cardiac function seemed to be aggravated by the number of noise events despite preserved average sound pressure level [63], which may provide an explanation for the heart failure risk by transportation noise [64].

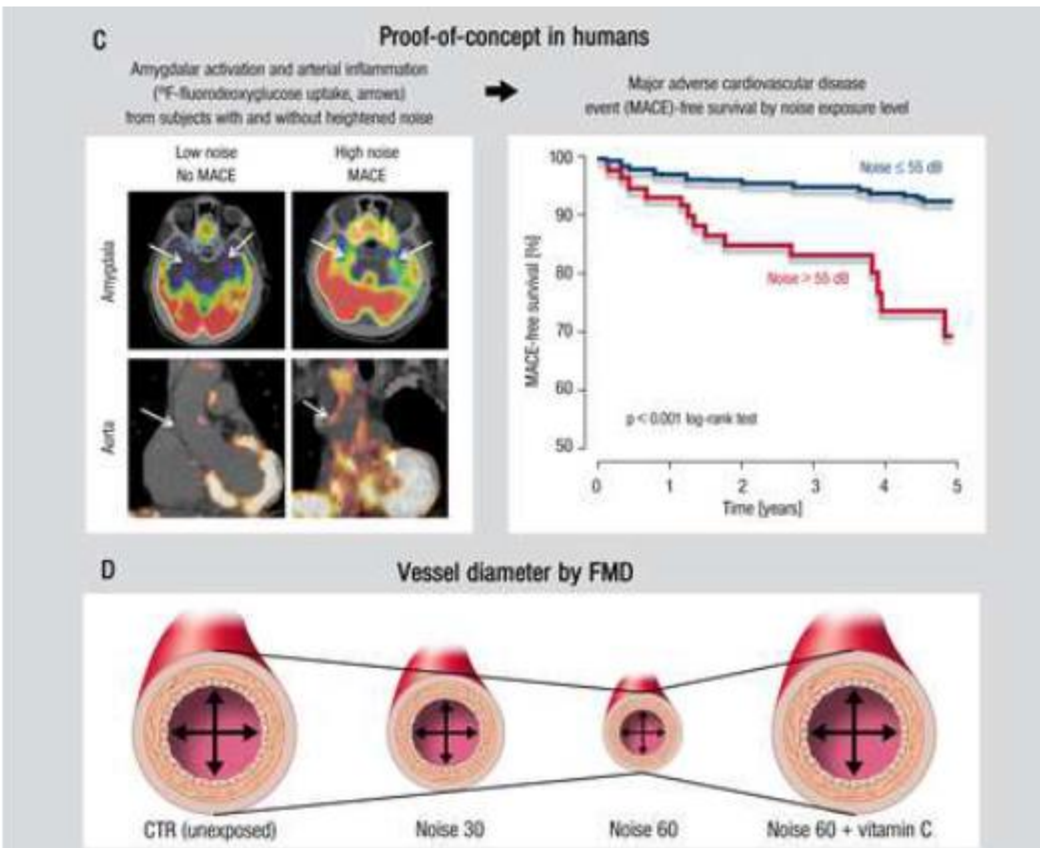

Figure 2. Noise-stress concept and the adverse health consequences in humans. (**C**) Neuronal activation (arousals) and subsequent atherosclerosis with a higher cardiovascular risk by noise exposure was proven in subjects by 18F-PET scans indicating an association of amygdala activation, coronary inflammation and increased incidence of major adverse cardiovascular events (MACE). Adapted from 59, 270 with permission; Copyright © 2019, Oxford University Press. (**D**) Flow-mediated dilation (FMD) is measured by high resolution B-mode ultrasound. Schematic presentation of adverse effects of simulated nighttime aircraft or train noise (either 30 or 60 events for one night) versus unexposed control group (CTR) on FMD of the brachial artery in response to postischemic hyperaemia and the beneficial acute effects of the antioxidant vitamin C. Results of own studies 43, 62.

It has been found that these noise-induced adverse health effects correlate with higher circulating cortisol levels and more pronounced noise sensitivity [65, 66]. A Swiss cohort study (SAPALDIA) demonstrated that traffic noise and air pollution were associated with alterations of epigenetic DNA changes priming the tissues for altered inflammatory cascades and changes of immune responses [67]. The SAPALDIA consortium also found that intermittent nighttime railway and road noise may affect arterial stiffness as shown by measurement of pulse wave velocity [68]. These data were supported by results of a German cohort study, which found an association between nighttime traffic noise and subclinical atherosclerosis [69, 70]. Altogether these studies support the concept that psychological stress in general and noise exposure in particular promotes the release of stress hormones, the activation and recruitment of immune cells and impairs cardiovascular function in men. This concept is also in accordance with the observation that the severity of immunological changes in response to psychological stressors correlates with the number of cardiovascular events [71, 72].

2.3 Cardiovascular effects of transportation noise exposure: mechanistic insights from animal studies

Early animal studies demonstrated that chronic noise exposure (85 dB(A) for 4 weeks to 9 months) caused a persistent increase in blood pressure in monkeys [73] or rats [74]. When rats were exposed to white noise (100 dB(A) for 1 to 4 weeks) an impaired endothelium-dependent vasodilation of the thoracic aorta [75] and the mesenteric artery [76] could be observed. These previous landmark studies are in accordance with strong evidence suggesting that background noise levels ≥42 dB(A) in animal housing buildings may induce a significant pathophysiology based on hypertension, impaired vascular function, endocrine stress responses, but also modulation of the immune system, slower wound healing, impaired fertility and reproduction [77]. More animal studies on noise effects (≤100 dB(A)) can be found in **suppl. Table S3**.

Mouse studies conducted by Münzel and coworkers showed dysregulation of vascular gene networks by noise (revealed by RNAseq) and downstream impairment of endothelial/vascular signaling [78]. Their data also clearly showed that noise exposure of sleeping mice but not during their activity phase causes more pronounced cardiovascular complications via major pathomechanisms comprising endothelial dysfunction, oxidative stress and inflammation in the vasculature as well as in the brain and by dysregulated *Foxo3*/circadian clock signaling (identified by RNAseq) [79]. These adverse effects of noise were mostly normalized by *Nox2* knockout, supporting a major role of phagocytic cells. They also reported normalization of noise-induced microvascular dysfunction (in dorsal and cerebral arterioles), proinflammatory changes of the plasma proteome and endothelial adhesion of leukocytes in Nox2 deficient mice [80]. This proposed concept was confirmed using a mouse model with lysozyme M (LysM)-specific overexpression of an inducible diphtheria toxin receptor ($LysM_{iDTR}$ mice) allowing specific removal of LysM-positive myelomonocytic cells by diphtheria toxin treatment [81]. Detailed flow cytometric analysis demonstrated that genetic ablation of

LysM-positive monocytes/macrophages prevented vascular inflammation and oxidative stress but also impaired endothelium- dependent relaxation and increased blood pressure in the peripheral circulation but failed to prevent neuroinflammation and stress hormone release in the brain as activation of microglia by noise was not suppressed in $LysM_{iDTR}$ mice. Aircraft noise also caused lower expression and uncoupling of the neuronal nitric oxide synthase, which may explain at least in part the impaired cognitive development of noise-exposed children [79]. Of note, noise-dependent development of inflammation and oxidative stress, impairment of endothelial function and onset of hypertension were all improved by heme oxygenase-1 induction (using hemin) and NRF2 activation (using dimethyl fumarate) [82].

As the pathomechanisms of noise-induced cardiovascular damage show large overlap with traditional risk factors for cardiovascular events, such as diabetes [83], hypertension [84] and hypercholesterolemia [85], it may be speculated that noise exposure on top of an established CVD or risk factor contributes to accelerated vascular/cerebral atherosclerosis and neurodegenerative disease and adds to the severity of these disease in an additive manner. In line with this concept noise exposure has been found to aggravate arterial hypertension and all associated cardiovascular as well as cerebral complications in a mouse model of angiotensin-II infusion [86]. A similar observation was made regarding the more pronounced impairment of endothelial dysfunction by nighttime aircraft noise in coronary artery disease patients in comparison with healthy controls [43, 61].

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14. Supplementary Material

Table S1. Epidemiological/observational evidence for an association between traffic noise and cardiovascular disease, events, and mortality with focus on recent studies.

First author / year	Population / cohort	Noise sources	Major outcomes	Ref
Roca-Barceló, 2021	21,936 CVD deaths	Aircraft noise	CVD and CHD mortality risk tended to increase with increasing levels of aircraft noise (L_{dn}), while no linear trend was found for stroke mortality.	1
Kupcikova, 2021	502,651 subjects	Road traffic noise	Road traffic noise exposure (L_{den} >65 vs. ≤55 dB(A)) led to 0.77% (95% CI 0.60-0.95) higher SBP, 0.49% (95% CI 0.32-0.65) higher DBP, 0.79% (95% CI 0.11-1.47) higher triglycerides, and 0.12% (95% CI –0.04-0.28) higher glycated hemoglobin.	2
Yankoty, 2021	1,065,414 subjects	Total environmental / transportation noise	The HRs for incident MI were 1.12 (95% CI 1.08-1.15), 1.11 (95% CI 1.07-1.14), and 1.10 (95% CI 1.06-1.14) per 10 dB(A) increase in L_{Aeq24}, L_{den}, and L_{night}, respectively.	3
Gilani, 2021	909 subjects	Road traffic noise	An OR of 2.25 (95% CI 1.38-3.67) for the prevalence of CAD per 5 dB(A) increase in road traffic noise (L_{den}) was found.	4
Saucy, 2021	24,886 CVD deaths	Aircraft noise	Acute increases in aircraft noise 2 hours preceding death were associated with total CVD mortality (OR 1.44, 95% CI 1.03-2.04) for the highest group of exposure (L_{Aeq} >50 vs. <20 dB).	5
Baudin, 2021	5,860 subjects	Aircraft noise	Aircraft noise levels per 10 dB(A) increase in L_{night} increased the odds of antihypertensive medication by 43% (OR 1.43, 95% CI 1.19-1.73).	6
Osborne, 2020	498 subjects	Combination of road traffic and aircraft noise	Higher noise exposure per 5 dB(A) increase in L_{Aeq24} predicted major CV events (HR 1.341, 95% CI 1.147-1.567).	7
Bai, 2020	37,441 cases of incident acute MI and 95,138	Road traffic noise	Road traffic noise (L_{Aeq24}) per IQR increase was associated with an elevated risk of incident acute MI (HR 1.07, 95% CI 1.06-1.08) and CHF (HR, 1.07 95% CI 1.06-1.09).	8

	cases of incident CHF			
Thacher, 2020	52,758 subjects	Road traffic noise	At the most exposed façade, road traffic noise per IQR increase was associated with a 13% (HR 1.13, 95% CI 1.06-1.19) and 11% (HR 1.11, 95% CI 0.99-1.25) higher CVD and stroke mortality, respectively. At the least exposed façade, road traffic noise remained to be associated with CVD (HR 1.09, 95% CI 1.03-1.15), IHD (HR 1.10, 95% CI 1.01-1.21), and stroke (HR 1.06, 95% CI 0.95-1.19) mortality.	9
Thacher, 2020	52,053 subjects	Road traffic noise	There was no association between road traffic noise and filled prescriptions for antihypertensive drugs.	10
Andersson, 2020	6,304 men	Road traffic noise	The HRs were 1.08 (95% CI 0.90-1.28) for CV mortality, 1.14 (95% CI 0.96-1.36) for IHD incidence, and 1.07 (95% CI 0.85-1.36) for stroke incidence in response to road traffic noise (L_{Aeq24} >60 vs. <50 dB.	11
Shin, 2020	Subjects without a history of hypertension (701,174) or diabetes mellitus (914,607)	Road traffic noise	An increase in L_{Aeq24} per 10 dB(A) was associated with an 8% increase in incident diabetes mellitus (HR 1.08, 95% CI 1.07-1.09) and a 2% increase in incident hypertension (HR 1.02, 95% CI 1.01-1.03). Similar estimates were obtained for L_{night}.	12
Baudin, 2020	6,105 subjects	Aircraft noise	An increase per 10 dB(A) in L_{night} was associated with an increased risk of hypertension (RR 1.03, 95% CI 1.01-1.06$_t$). An association was also found between aircraft noise annoyance and hypertension risk (RR 1.06, 95%CI 1.00-1.13 for highly annoyed vs. not highly annoyed).	13
Pyko, 2019	20,012 subjects	Road traffic, railway, aircraft noise	In subjects exposed to all three traffic noise sources at ≥45 dB L_{den}, risks of IHD were elevated with a HR of 1.57 (95% CI 1.06-2.32), and a comparable observation for stroke (HR 1.42, 95% CI 0.87-2.32).	14
Héritier, 2019	4.4 million subjects	Road traffic, railway, aircraft	MI mortality was increased in response to road traffic (HR 1.034, 95% CI 1.014-1.055), railway (HR 1.020, 95% CI	15

		noise	1.007-1.033), and aircraft noise (HR 1.025, 95% CI 1.005-1.046) per 10 dB increase in L_{den}.	
Héritier, 2018	4.41 million subjects	Combination of road traffic, railway, aircraft noise	For the core night, the highest HR was observed for IHD mortality (1.025, 95% CI 1.016-1.034), while this association was lower for the daytime (1.018, 95% CI 1.009-1.028). HF mortality and daytime noise was associated with the highest HR (1.047, 95% CI 1.027-1.068).	16
Pyko, 2018	4,854 subjects	Road traffic, railway, aircraft noise	Aircraft noise increased the incident risk of hypertension by 16% (HR 1.16, 95% CI 1.08-1.24) per 10 dB increase in L_{den}. Road traffic and railway noise were not associated with incidence of hypertension.	17
Yang, 2018	663 subjects	Road traffic noise	Road traffic noise per 5 dB(A) increase was associated with the prevalence of CVD (OR 2.23, 95% CI 1.26-3.93).	18
Cai, 2018	21,081 incident CVD cases	Road traffic noise	No associations were found between road traffic noise and incident CVD, IHD, or CBVD in the total population.	19
Hahad, 2018	14,639 subjects	Road traffic, railway, aircraft noise	Traffic-related noise annoyance is associated with increased prevalence of AF.	20
Héritier, 2017	4.41 million subjects	Road traffic, railway, aircraft noise	HRs for MI mortality were per 10 dB increase in L_{den} 1.038 (95% CI 1.019-1.058) for road traffic, 1.018 (95% CI 1.004-1.031) for railway, and 1.026 (95% CI 1.004-1.048) for aircraft noise.	21
Zeeb, 2017	137,577 cases and 355,591 controls	Road traffic, railway, aircraft noise	There was no association between any of the traffic noise sources and incident hypertension. Likewise, no association between nighttime noise levels and hypertension was found. For the group of subjects with newly diagnosed hypertension followed by hypertensive heart disease, the ORs were elevated.	22
Fuks, 2017	41,072 subjects	Road traffic noise	A weak relationship between road traffic noise and incident self-reported hypertension was found, whereas no conclusive association with measured hypertension was established.	23

Pitchika, 2017	2,552 subjects	Road traffic noise	No association between road traffic noise (L_{Aeq24}) and prevalent hypertension was found.	24
Roswall, 2017	50,744 subjects	Road traffic noise	Road traffic noise was associated with a higher risk of MI, with a HR of 1.14 (95% CI 1.07-1.21) per IQR increase in L_{den}.	25
Evrard, 2017	1,244 subjects	Aircraft noise	Only in men, a 10 dB(A) increase in aircraft noise (L_{night}) was associated with risk of hypertension (OR of 1.34, 95% CI 1.00-1.97).	26
Dimakopoulou, 2017	780 subjects	Aircraft noise	A 10 dB increase in L_{night} resulted in an OR of 2.63 (95% CI 1.21-5.71) for hypertension and of 2.09 (95% CI 1.07-4.08) for doctor-diagnosed cardiac arrhythmia.	27
Sørensen, 2017	57,053 subjects	Road traffic noise	An IRR of 1.14 for HF (95% CI 1.08-1.21) per IQR increase in L_{den} road traffic noise was found.	28
Seidler, 2016	19,632 cases and 834,734 controls	Road traffic, railway, aircraft noise	A 10 dB increase in L_{Aeq24} was associated with higher odds of MI in response to road traffic (2.8%, 95% 1.2-4.5) and railway noise (2.3%, 95% CI 0.5-4.2), but not aircraft noise. Aircraft noise levels of 60 dB and above were associated with increased MI risk (OR 1.42, 95% CI 0.62-3.25).	29
Recio, 2016	Cohort of subjects ≥65 years	Road traffic noise	Short-term road traffic noise increased the risk of death from IHD, MI, and CBVD.	30
Monrad, 2016	57,053 subjects	Road traffic, railway noise	A 10 dB increase in L_{den} road traffic noise was associated with a 6% increased risk of AF (IRR 1.06, 95% CI 1.00-1.12), which was weaker after further adjustment for air pollutants. AF risk was not related to railway noise.	31
Sørensen, 2011	57,053 subjects	Road traffic noise	An IRR of 1.14 for stroke (95% CI 1.03-1.25) per 10 dB increase in L_{den} road traffic noise was found.	32
Beelen, 2009	120,852 subjects	Road traffic noise, traffic intensity	Traffic intensity was associated with CV mortality, with highest RR of 1.11 (95% CI 1.03-1.20 per increase in 10,000 motor vehicles/24 h). Road traffic noise (>65 dB(A)) was associated with increased risk of IHD (RR 1.15, 95% CI 0.86-1.53) and HF mortality (RR 1.99, 95% CI 1.05-3.79), which was attenuated after further adjustment air pollution and traffic intensity.	33

CVD: Cardiovascular disease, CHD: Coronary heart disease, L_{dn}: Day-night noise levels, SBP: Systolic blood pressure, DPB: Diastolic blood pressure, HR: Hazard ratio, MI: Myocardial Infarction, $L_{Aeq(time\ period)}$: Noise levels over a certain period of time, L_{night}: Night noise levels, IHD: Ischemic heart disease, CHF: Congestive heart failure, IQR: Interquartile range, CBVD: Cerebrovascular disease, dB: Decibel, OR: Odds ratio, CI: Confidence interval, CAD: Coronary artery disease, L_{den}: Day-evening-night noise levels, AF: Atrial fibrillation, IRR: Incidence rate ratio, RR: Relative risk

Table S2. Human studies on the association of atherosclerosis, vascular (endothelial) dysfunction, inflammation, or oxidative stress with ambient air pollution or traffic noise with focus on recent studies.

First author / year	Population / cohort	Noise sources	Major outcomes	Ref
Traffic noise				
Schmidt, 2021	70 subjects with CVD	Aircraft noise	Acute aircraft noise exposure at night impaired endothelial function (flow-mediated dilation) and cardiac diastolic function.	66
Hahad, 2021	5,000 subjects	Aircraft, railway noise	Aircraft and railway noise annoyance were associated with increased midregional pro atrial natriuretic peptide, which predicted incident CVD.	67
Biel, 2020	46 subjects	Total environmental noise (traffic noise included)	Acute increases in both air pollution and noise were associated with endothelial function and heart rate variability.	68
Eze, 2020	1,389 subjects	Road traffic, railway, aircraft noise	Both air pollution and traffic noise were associated with DNA methylation, with both distinct and shared enrichments for pathways linked to cellular development, immune responses, and inflammation.	69
Thiesse, 2020	26 subjects	Road traffic noise	After sleeping with highly intermittent road traffic noise, evening cortisol levels were elevated.	70
Herzog, 2019	70 subjects	Railway noise	Acute railway noise exposure was associated with impaired flow-mediated dilation. Proteomic analysis indicated substantial changes of plasma proteins in response to noise centered on proinflammatory, redox, and pro-thrombotic pathways.	71
Cai, 2017	144,082 subjects	Road traffic noise	An IQR increase in L_{day} road traffic noise was associated with 0.7% (95% CI 0.3-1.1) higher triglycerides, 1.1% (95% CI 0.02-2.2) higher C-reactive protein, and 0.5% (95% CI 0.3-0.7) higher high-density lipoprotein, with only the latter being robust to further control for air pollution.	72
Foraster, 2017	2,775 subjects	Road traffic, railway, aircraft noise	A 0.87% (95% CI 0.31-1.43) increase in brachial-ankle pulse wave velocity per IQR increase in L_{den} railway noise was observed. Total number of noise events at night, but not at day, was related to brachial-ankle pulse wave velocity.	73
Lefèvre, 2017	1,244	Aircraft noise	Aircraft noise was associated with cortisol in the evening.	74
Halonen, 2017	2,592 subjects	Road traffic noise	A 9.1µm (95% CI –7.1-25.2) increase in carotid intima-media thickness per 10 dB(A) increase in L_{night} was observed.	75
Schmidt, 2015	60 subjects at increased risk of CVD	Aircraft noise	Acute aircraft noise exposure at night impaired endothelial function and increased systolic blood pressure.	76
Sørensen, 2015	39,863 subjects	Road traffic noise	Slightly higher cholesterol may be linked to road traffic noise.	77
Schmidt, 2013	75 subjects	Aircraft noise	Acute aircraft noise exposure at night was associated with impaired flow-mediated dilation, which was attenuated by the administration of Vitamin C. Adrenaline was increased and pulse transit time decreased in response to noise.	78

CVD: Cardiovascular disease, $PM_{(diameter\ size)}$: Particulate matter, NO_2: Nitrogen dioxide, IQR: Interquartile range, L_{night}: Night noise levels, OR: Odds ratio, CI: Confidence interval, O_3: Ozone, NO_x: Nitrogen oxides, HR: Hazard ratio, BC: Black carbon, CO: Carbon monoxide, PAHs: Polycyclic aromatic hydrocarbons, NO: Nitrogen monoxide, CAD: Coronary artery disease, L_{den}: Day-evening-night noise levels, L_{day}: Day noise levels

Table S3. Animal in vivo studies on non-auditory noise effects on cardiovascular and endothelial dysfunction, inflammation, or oxidative stress. Only articles that are not mentioned in the main article text and used <100 dB average sound pressure level are listed here.

First author / year	Animals and model	Noise scenario	Major outcomes	Ref
Borg, 1981	Sprague-Dawley, spontaneously hypertensive rats (SHR)	80 - 100 dB (noise type unknown), 10 h/d for entire lifespan	Noise exposure caused a shorter lifespan and higher frequency of CVD in spontaneously hypertensive rats as compared to normotensive rats.	79
Peterson, 1984	Rhesus monkeys	85 dB (realistic noise sequence), 24 h/d for 6 months	Noise exposure caused a substantial increase in blood pressure as well as disruption of the diurnal rhythm of heart rate, blood pressure, and caused "pauses" in cardiac rhythm.	80
Peterson, 1984	Macaque monkeys	87 - 90 dB (construction noise), 4 - 8 h/d for 97 d	Noise exposure caused an increase in blood pressure by 8.2% (4 h/d scenario) and 16.5% (8 h/d scenario). Whereas blood pressure increases persisted after noise cessation, the heart rate returned to baseline.	81
Kirby, 1984	Macaque monkeys	95 dB (broadband noise), 30 min	Noise exposure caused a more pronounced increase in blood pressure in the offspring of hypertensive monkeys, whereas heart rate was significantly changed. Also the resting blood pressure in the offspring of hypertensive monkeys was higher than offspring of normotensive monkeys.	82
Wu, 1992	Rats	85 - 95 dB (unknown noise type), 12 - 16 h/d for 4 - 8 weeks	Noise exposure impaired endothelium-dependent vasodilation as determined by acetylcholine (ACh)-response in the isolated thoracic aorta. Noise also increased the sensitivity to the vasoconstrictor serotonin, but not phenylephrine or potassium chloride, and increased systolic blood pressure by 31 mmHg.	83
Altura, 1992	Rats	Up to 100 dB (broadband noise), 4 h/d for 2 - 4 weeks	Noise exposure led to increased systolic and diastolic blood pressure (16 mmHg) along with magnesium deficiency and reduced lumen sizes of microvessels.	84
Morvai,19	CFY rats	95 dB (industrial	Noise exposure lowered cardiac output and hepatic blood flow.	85

94		noise), 6 h/d for 3 weeks	Noise also modified the hemodynamic effects of noradrenalin by an alteration of the alpha-adrenergic response.	
Wu, 1994	Rats	Up to 100 dB (broadband noise), 4 h/d for 3 - 4 weeks	Noise exposure increased systolic blood pressure by 25 mmHg (3 weeks noise) and by 37 mmHg (4 weeks noise), which was associated with pronounced endothelial dysfunction in isolated mesenteric arterial rings.	86
Herrmann, 1994	Wistar rats and SHR	65 dBA (low frequency noise, 4 and 250 Hz), 24 h/d for 52 weeks	Noise exposure was associated with significantly increased microvessel wall area, number of microvessels with an outer diameter > 19 microns, the degree of cardiac fibrosis, and the extent of ischemic myocardial lesions in SHR, but not in normotensive rats. Noise did not alter cardiac weights and dimensions, heart rate, and dp/dtmax.	87
Singewald , 2000	Wistar-Kyoto rats and SHR	95 dB (noise type unknown), 3 min	Noise exposure led to a tetrodotoxin-sensitive increase in glutamate release in the amygdala of SHR, but not normotensive rats. Also pressor response to noise was enhanced in SHR, all of which indicates an exaggerated stress response of glutamatergic neurons in the amygdala of SHR as compared to normotensive rats.	88
Baldwin, 2007	Rats	90 dB (noise type unknown), 15 min/d for 3 – 5 weeks	Noise exposure impaired the microvascular integrity (mesenteric arteries) in rats as revealed by significantly more leaks per venule length and greater leak area per venule length. Co-treatment with vitamin E plus a-lipoic acid or Traumeel (a homeopathic anti-inflammatory-analgesic) partly prevented these adverse effects of noise.	89
Antunes ,2013	Rats	90 dB (low frequency noise, ≤ 500 Hz) for 3 months	Noise exposure caused significant myocardial fibrosis (increased collagen deposition between the cardiomyocytes) in rats. Also connexin43/muscle ratio was decreased by noise. Transmission electron microscopy also revealed noise-induced changes of cardiomyocyte ultrastructure, e.g. altered interstitial collagen deposits and changes in mitochondria and intercalated discs of the cardiomyocytes.	90-92
Gannouni, 2013	Wistar rats	70 – 80 dB (octave-band noise (8-16	Noise exposure increased corticosterone levels, affected various parameters of the endocrine glands and cardiac function. Markers of	93

		kHz), 6 h/d for 3 months	oxidative stress (catalase, superoxide dismutase and lipid peroxidation) were also increased by noise. In summary, noise enhanced physiological function related to neuroendocrine modulation and oxidative imbalance.	
Ersoy, 2014	Albino rats	Noise type and protocol unknown	Noise exposure significantly decreased superoxide dismutase expression in the cerebral cortex but increased malondialdehyde levels in the brainstem and cerebellum. Rosuvastatin increased superoxide dismutase expression in the cerebral cortex and brain stem, but significantly decreased malondialdehyde values in the brain stem.	94
Gannouni, 2014	Wistar rats	70 dB (noise type unknown)	Noise exposure caused time-dependent changes in the morphological structure of the adrenal cortex involving disarrangement of cells and modification in thickness of the different layers of the adrenal gland. These observations are compatible with noise-induced changes of the morphological structure of heart tissue causing irreversible cell damage and leading to necrosis or cell death.	95
Said, 2016	Albino rats	80 - 100 dB (chronic and intermittent octave band noise, 8-16 kHz), 8 h/d for 20 d	Noise exposure adversely affected the cardiovascular system by increased levels of circulating stress hormones (e.g. corticosterone, adrenaline, noradrenaline, endothelin-1). Noise also negatively affected oxidative stress markers (e.g. higher malondialdehyde levels and decreased superoxide dismutase expression). These data are compatible with endothelial dysfunction, which was further supported by impaired nitric oxide metabolism and elevated blood pressure in noise-exposed rats.	96
Cui, 2016	Rats	Up to 100 dB (octave band noise, 0.4-6.3 kHz), 4 h/d for 30 d	Noise exposure caused a transient increase in markers of inflammation, blood glucose, triglycerides, and alterations in the microbiome that returned to baseline at 14 d after noise exposure cessation.	97
Kvandova, 2020	C57BL/6 mice and *Ogg1*$^{-/-}$ mice	72 dB (aircraft noise), 24 h/d for 4 d	Noise exposure induced oxidative DNA damage that was associated with enhanced leucocyte oxidative burst activity and other markers of inflammation (e.g. cyclooxygenase-2 as well as oxidative stress (e.g. 4-hydroxynonenal, 3-nitrotyrosine levels and NOX-2 expression). Noise impaired endothelial function (ACh-response) but not endothelium-independent relaxation (nitroglycerin-response). Genetic deficiency in 8-oxoguanine glycosylase knockout (*Ogg1*$^{-/-}$) further aggravated most of these adverse noise effects and induced a significant impairment of the endothelium-independent relaxation (nitroglycerin-response).	98

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