

Report on the modelling of dioxins and furans at Ringaskiddy

Qualifications and experience of author

My name is Dr Gordon Reid. I recently retired as Senior Lecturer in Physiology at University College Cork. Previously I was Professor of Physiology at the University of Bucharest (Romania) and Visiting Professor at the University of Bristol (UK). I have over 30 years' experience in physiological research and teaching, specialising in the fields of neuroscience and neuropharmacology. I have published in most of the high-impact journals in my field over these 30 years.

My research included the first description of the neuronal cold and menthol receptor that is responsible for cutaneous temperature sensation, which also happens to be implicated in the development of prostate cancer. The finding was published in "Nature", the most highly regarded journal publishing original scientific research. As well as a personal award (Nicolae Simionescu Prize of the Romanian Academy) this work led to the nomination of my laboratory as an Eastern European Centre of Excellence by the Physiological Society.

I also have considerable experience in mathematical simulation, in particular the development and programming of the first mathematical model to describe the action potential ("nerve impulse") from human nerve fibres. I worked together with Professor Jürgen Schwarz (Hamburg) to make the first intracellular recordings from human nerve fibres, on which this model was based. The model is based on a system of simultaneous differential equations solved using the Euler method of numerical integration, and is capable of reproducing accurately the time course of human nerve action potentials in both healthy and damaged nerve fibres (including in a study of motor neurone disease). The model is thus conceptually and computationally somewhat more complex than the RISC-HUMAN model under consideration here, but gives me a good basis on which to examine it.

Along with this research work, I have also reviewed (at the request of the editors) a large number of articles submitted for publication to these journals (including Nature, among several other high-impact journals). Peer review is the process by which a piece of original research is judged to be valid and worthy of publication (or not). It requires a keen eye for erroneous presentation and misrepresentation of data, for omissions that would make conclusions meaningless or misleading, and for the distinctive ways that numbers behave. On (fortunately) rare occasions, I have used this skill to detect attempted scientific fraud and to alert a journal editor to the fact that a more than usually critical approach is required to a certain piece of work.

I have been asked by CHASE to report on the analysis and modelling of dioxins and furans at Ringaskiddy, submitted as part of the application by Indaver Ireland for a proposed waste incinerator. Since 2016 I have examined a number of versions of this analysis compiled for the proposed Ringaskiddy incinerator, and have already reported on these earlier versions to An Bord Pleanála, the High Court, and the oral hearing on this application held in 2016. The current version of the modelling is presented in Appendix 6.3 of the EIS, and draws on soil sampling in Appendix 6.2, and air dispersion modelling in Appendix 8.1 and 8.4. All sections are compiled by AWN Consulting.

Summary of conclusions

My conclusions from the present analysis are:

1. Even at baseline, with no incinerator, the dioxin and furan intake modelled for the hypothetical human family in Appendix 6.3 exceeds the EU's permitted limit, the Tolerable Weekly Intake. Appendix 6.3 cites an obsolete higher value for the EU TWI, and uses this to argue that the site is safe for the proposed incinerator. Appendix 8.1 makes a similar mistake.

2. Furthermore, the dioxin and furan intake values modelled in Appendix 6.3 are grossly underestimated. This is partly owing to some remarkable omissions from the model (e.g. assuming a starvation-level diet for the hypothetical family whose intake is modelled, thus understating their food-related dioxin and furan intake, which is by far the main route of exposure), partly to deviations from the standard methodology that the Appendix states is being used (e.g. failing to consider the dioxin and furan intake of a child, which is far greater than that of an adult), and partly to failure to compare like with like (modelling only dioxins and furans, but then comparing with an EU intake limit that also included dioxin-like PCBs). Correct application of the model would result in an intake value that is very substantially above the permitted limit.

3. Comparison with earlier (2008) modelling by the same consultant at the same site, with a modelled incineration plant operating at the same dioxin and furan output concentration and with similar quantities being emitted, shows a remarkable reduction in the stated effect of the incinerator on the family's modelled intake in 2025 compared to 2008. (Lest it be thought that this is due to improved technology, it should be noted that the model simply uses the maximum permitted output concentration, which was exactly the same in 2008 as it is in 2025, so changes in technology do not enter the model at all.)

In the case of the most toxic of the dioxins, the reduction in intake is an incredible 560-fold. Given the similarity in the data on which the modelling was based – the modelled incinerator output, the terrain, and the soil – this discrepancy raises questions about the credibility and reproducibility of the modelling which urgently require clarification.

4. I am also concerned that, although I understand the Directive requires data in the EIS to be the most recent available, substantial parts of the present Appendices I have inspected are based on old data, and other parts that were present in previous versions of the EIS are simply missing now. Although new air dispersion modelling was performed in 2025, this was not used in the dioxin and furan intake modelling, which is simply a re-submission of the 2019 EIA Report for the EPA licence application. It is based on soil samples that were collected in 2015. The air dispersion modelling, although it is new for this EIS, lacks the cumulative analysis of all dioxin and furan sources that was included in previous versions; the apparent reason for this is quite bizarre.

Detailed report

Description of the dioxin and furan model

The report presented in Appendix 6.3 of the EIS is intended to show that the site proposed for the incinerator is safe, in that the soil concentrations of dioxins and furans (dioxin-related compounds) are low enough that food could be grown on the site or closely adjacent land without endangering human health. It does this by modelling the dioxin and furan intake that would result from eating food grown at the site, at baseline and with the addition of the proposed incinerator.

The modelling begins with eight soil samples taken from locations around the site and the wider Cork Harbour area. One of these sampling locations is chosen as being nearest to the expected point of maximum dioxin deposition from the incinerator (modelled in the Air Quality chapter of the EIS). A self-sufficient farming family is then assumed to be living on a plot of land at that point. They eat only vegetables grown on that plot of land, milk from a cow grazing on it, and meat from the same cow. Because they are living near the point of maximum deposition, each member of this family is considered to be a 'maximum at risk individual' (abbreviated as MARI).

Using the soil concentrations of dioxins and furans at the chosen sampling location, the model calculates the movement of these compounds through the food chain – from soil to grass and

vegetables, and from grass into the cow. This allows the dioxin and furan content of the MARI family's food to be worked out, and, knowing the amounts they eat, allows their dioxin and furan uptake to be calculated. This is the baseline intake, with no incinerator. Then the modelled deposition of dioxins and furans from the incinerator is added to the existing concentrations in the soil, and the process repeated, to obtain the total predicted dioxin and furan intake of the MARI family if the incinerator is in operation.

One may wonder why a subsistence farmer is the model, when subsistence farmers are not commonly found in Ringaskiddy; some have criticised the model as unrealistic for that reason. It is important to realise why the MARI model is based on a subsistence farmer. It is not because there are many real subsistence farmers around the incinerator, but because the MARI model is intended to answer the questions: is the land around the proposed incinerator a safe environment to grow food? If all your food were grown there, would it keep you healthy? The words 'all your food' are the crucial ones here. If the proposed incinerator would make the land around it so toxic that a person could not safely live solely on food grown there, then either food grown there should not go into the human food chain, or else, the incinerator should not be built. This is a crucial matter, because the land around the proposed incinerator, and the area potentially affected by its emissions (which is a large area, as the EIS shows: see my Appendix 5), is one of the most important for Irish agriculture, especially dairy farming. It is important to know whether the quality of the land would be harmed by the proposed incinerator.

Analysis of the results of the model

1. In the model as presented in Appendix 6.3, the stated MARI intake is above the EU permitted level, both at baseline and with the modelled incinerator

The baseline dioxin and furan intake for an adult member of the MARI family is stated in Appendix 6.3 as 2.0826 pg/kg/week TEQ (Appendix 6.3, Table 5.1; TEQ is 'toxic equivalency', a derived quantity that compensates for the different degrees of toxicity of different dioxins and furans). With the proposed incinerator added to the model, this increases to 2.2443 pg/kg/week TEQ. This is described as 'well below applicable limit values for PCDD/F¹ intake' (Appendix 6.3, page 16). The applicable limit here is the Tolerable Weekly Intake (TWI) limit recommended by the European Food Safety Authority (EFSA), acting on behalf of the European Commission.

The EU's TWI is stated in the Appendix as 14 pg/kg/week TEQ, citing an EU publication dating from the year 2000 (Appendix 6.3, page 11). The calculated intakes mentioned in the paragraph above are indeed well below that level.

The first comment that needs to be made is that *the currently applicable EU TWI is not 14 pg/kg/week!* What Appendix 6.3 inexplicably fails to recognise is that the EU TWI was updated in 2018. In response to a request from the Commission, the EFSA carried out a detailed risk assessment based on the best and most recent available data, and in 2018 lowered the TWI from 14 pg/kg/week TEQ to 2 pg/kg/week TEQ². This means that, *even if the calculations in Appendix 6.3 were correct, the resulting dioxin and furan intake of the MARI family would exceed the EU tolerable weekly intake*. Since the purpose of the modelling was to establish that the proposed site is a suitable one for an incinerator that generates dioxins and furans, the implication would be that *the*

¹The abbreviation 'PCDD/F' is equivalent to 'dioxins and furans'; it stands for 'polychlorinated dibenzo-p-dioxins / polychlorinated dibenzofurans'

²EFSA Panel on Contaminants in the Food Chain (CONTAM), EFSA Journal, 16(11), 2018.

<https://doi.org/10.2903/j.efsa.2018.5333>. See also press release at

<https://www.efsa.europa.eu/en/press/news/dioxins-and-related-pcbs-tolerable-intake-level-updated>.

site is already contaminated to an extent that no additional generation of dioxins and related compounds can be allowed.

If this were all that needed to be said, this submission would be very short. It would indeed be short, if the modelling in Appendix 6.3 were correct and well founded. But the modelling is very far from that, as the following section will show.

2. Omissions, distortions and methodological deviations in the baseline modelling in Appendix 6.3

In what follows, I will show that the baseline intakes presented in Appendix 6.3 are grossly underestimated, and that a correct application of the model it uses would result in far higher values, greatly exceeding the currently applicable EU tolerable weekly intake, and even considerably exceeding the pre-2018 TWI that is mistakenly cited in Appendix 6.3.

Appendix 6.3 states that the modelling approach follows the methodology recommended by the United States EPA, the internationally recognised HHRAP (Human Health Risk Assessment Protocol) methodology³ (Appendix 6.3, page 6). In fact, some aspects of the modelling deviate from the HHRAP method in essential ways; other aspects deviate from what the Appendix states should be done; and other aspects deviate from simple common sense. The only theme linking these deviations is that they all reduce the modelled dioxin intake. In summary, the faults in the modelling are as follows:

(a) The diet given to the MARI family members is grossly inadequate. The modelled food intake is drawn from a research study on the average Irish diet, but, because the MARI diet consists only of vegetables, milk, and meat, only these food groups have been included, and the rest of the average Irish diet apart from vegetables, liquid milk and meat is ignored. The MARI adults drink the same amount of milk as an average Irish adult, but they consume no other dairy product: they eat no butter, no cheese, no yogurt, etc. They eat no grain at all: no bread, no pasta, etc. The result is that the calorie intake of the MARI adult is below 1000 calories per day, and that of the six-year-old MARI child below 500 – this compares to the real food intake of a six-year-old child, which is 1400-1600 calories per day, and a real adult of 2000-2600 calories. Since the vast majority of human dioxin and furan intake is from food, the MARI family's intake of these compounds is thus reduced to below half of the intake that would result from eating a truly adequate diet.

The choice of such a bizarrely restricted starvation diet is inexplicable, because to know how much a person eats does not require specialist knowledge; it is a matter of everyday experience. It is hard to see how the consultant implementing the model could fail to see that the food intake is ridiculously low; but of course, this very low food intake also makes the modelled dioxin intake very low.

Because detailed data tables are given, it is possible to calculate what the dioxin and furan intakes *would* be, if the MARI diet included a similar intake of carbohydrates and dairy fats to the average Irish diet. This analysis is presented in my Appendix 1.

(b) The HHRAP of the US EPA, which Appendix 6.3 states is being followed, explicitly considers the intakes of the adult and child MARI separately, and states that for a non-carcinogenic endpoint this is the approach that should be adopted. The endpoints used in setting both the old and the current EU TWI are non-carcinogenic in every case.⁴ In contrast to the HHRAP recommendation,

³ 'Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities' US EPA, 2005. Available from <https://nepis.epa.gov/Exe/ZyPDF.cgi/P10067PR.PDF?Dockey=P10067PR.PDF>

⁴Some of the principal effects of dioxins at low concentrations are on the reproductive system during early development, and these among others were the basis for the earlier intake limits: 'based on ... those effects

adult and child are lumped together in the Appendix as a 'lifelong' exposure for MARI, based on 6 years of childhood and 64 years as an adult. Obviously this weighted average biases the 'lifelong' intake strongly towards the adult intake. A child's dioxin and furan intake from food, *relative to their body weight*, is inevitably greater than that of an adult, because children eat more than adults relative to their body weight. This fact would be hard for the modeller to overlook, as the tables in Attachments D and J of Appendix 6.3 explicitly show all three values for child, adult and lifelong intakes, and the relation between the values is obvious even to a casual reader (an example is given in my Appendix 2). The lack of mention of the child's intake in the main body of the report is especially surprising, because in his EIS report for the Indaver incinerator at Carranstown, Co. Meath, the same author did correctly report child and adult intakes separately.⁵

The effect of presenting only the 'lifelong' intake in the main body of Appendix 6.3 in the present EIS (Tables 5.1 and 7.1) is to obscure the much higher intake of the MARI child.

(c) Appendix 6.3 makes no mention of the dioxin and furan intake of a breastfed baby. In addition to the child, the HHRAP requires that the intake of dioxins and related compounds for a breastfed baby be considered: "We also recommend evaluating infant exposure to PCDDs and PCDFs via the ingestion of their mother's breast milk as an additional exposure pathway at all recommended adult exposure scenario locations. Chapter 2 and Appendix C further describe the ingestion of breast milk exposure pathway." (footnote 3, page 4-14).

This is of particular concern because, as already noted (footnote 4), the adverse effects of dioxins that occur at the lowest concentrations are largely in early development, so foetuses and babies are at particularly high risk. Research has shown that, for well understood physiological reasons, the smallest humans unfortunately tend to receive the highest concentrations of dioxins; and this makes their inclusion in HHRAP and their omission from the modelling in the EIS particularly significant.

(d) The model in Appendix 6.3 includes only dioxins and furans, and compares the modelled intake of these compounds for the MARI with the EU tolerable weekly intake (TWI). Leaving aside for the moment the fact that the Appendix uses an outdated value for the TWI, it is also imperative to point out that the TWI (in both its old and new guises) *is not a limit only for dioxins and furans*. It also includes a *third* component of toxicity, the dioxin-like polychlorinated biphenyls (DL-PCBs). The EU TWI of 2 pg/kg/week TEQ is a permitted limit for the *total* intake of these three components: dioxins, furans, and DL-PCBs. Obviously the inclusion in the EIS of only dioxins and furans falsely underestimates the modelled intake relative to the EU TWI, and makes a nonsense of the comparison, because it is not comparing like with like.

The exclusion of DL-PCBs also contradicts the explicit recommendation of the HHRAP, which says PCBs should be included, especially in cases where waste oils and/or variable waste streams such as municipal or commercial waste will be burned as at Ringaskiddy (HHRAP, page 2-61). Similarly, and surprisingly, the exclusion of DL-PCBs directly contradicts the previous practice of the author of Appendix 6.3: in his 2002 report for Indaver for the EPA licence application at Ringaskiddy, he correctly *did* include the DL-PCB component in the baseline dioxin-like toxicity, and stated that it

considered to be the most sensitive in experimental animals, namely endometriosis, developmental neurobehavioural effects, developmental reproductive effects and immunotoxicity' (Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment, 2001, Ref COT/2001/07. <https://cot.food.gov.uk/sites/default/files/cot/cot-diox-full.pdf>)

The revised EU TWI of 2 pg/kg/week is based on effects on human semen quality, as well as hormonal effects on newborns and developmental enamel defects in teeth (<https://www.efsa.europa.eu/en/press/news/dioxins-and-related-pcbs-tolerable-intake-level-updated>)

⁵http://www.epa.ie/licences/lic_eDMS/090151b2800547ee.pdf, page 22; the child's intake, which appears to be at baseline, is reported as twice the adult's and rather more than twice the current EU TWI, at 0.675 pg/kg/day (4.72 pg/kg/week).

contributes about 45-55% of total dioxin-like toxicity, so he appears to know it should be included and is important.⁶

It is easy to assess how big an effect the exclusion of DL-PCBs will have, because they are tested regularly by the EPA in Irish milk, including at Ringaskiddy. This testing shows that DL-PCBs since 2012 added on average around 57 % of the toxicity of dioxins and furans alone (see my Appendix 3 for the EPA data). The effect of omitting DL-PCBs is thus to reduce the modelled intake of dioxin-like toxicity by around one third. This omission is corrected in my Appendix 3.

(e) The sample site on which the modelling is based is stated in Appendix 6.3 as being the one closest to the point of maximal dioxin deposition from the proposed incinerator, as modelled in the Air Quality section of the EIS. The logic of this is weak, for two reasons. Firstly, the point of maximum deposition, or concentration, from the proposed incinerator moves widely around the Ringaskiddy area and beyond (as will be seen in my Appendix 5). The point of maximal deposition one year will probably not be maximal in another year. Secondly, the dioxin intake that matters for human health is not that from one individual source (our bodies cannot distinguish where the dioxins come from), but the total intake based on the existing baseline soil concentrations plus the modelled incinerator output. Given that the maximal deposition has always been within the area around Ringaskiddy, within which there are four sampling sites in Appendix 6.2, a practical approach would be to assume that maximal deposition could occur at or near any of them, and to use the one with the highest baseline concentration.

But even the site with the lowest concentration of the four at Ringaskiddy would give a MARI intake well above the permitted EU TWI, if the modelling is performed correctly, including an adequate diet, the contribution of DL-PCBs, and reporting the child's intake.

The effects of the deficiencies described above are not small, and rectifying them is mostly not optional:

(a) If the MARI adult were given a normal diet instead of one with fewer than 900 kilocalories per day, their dioxin intake would approximately double (see Appendix 1). The present food intake in the model is completely unrealistic.

(b) Because the MARI child's food intake relative to body weight is far greater than that of the MARI adult, their dioxin intake relative to body weight is also far greater: the child's intake is well over twice the 'lifelong' intake (Appendix 2). It is required by the HHRAP methodology that the child be considered explicitly.

(c) The effect on a breastfed baby is harder to quantify in the model, but the HHRAP methodology recommends it be included in the assessment.

(d) Since DL-PCBs contribute about half the level of toxicity of dioxins and furans, inclusion of DL-PCBs would increase the modelled intake of dioxin-like toxicity by about half (Appendix 3). Their inclusion is not optional, because the EU TWI includes DL-PCBs, so the model cannot be compared with it unless they are included.

(e) If the sample site with the highest overall concentration is used for the modelling, the resulting intake would be increased by a little more than 50 % (Appendix 4). AWN's logic underlying site selection is shaky, and this choice would be more secure and robust, so that the conclusions of the modelling would be better founded.

Since each of the factors (a), (b), (d), and (e) above is independent of the others, they combine multiplicatively. Appendix 2 shows the calculation as well as explaining each choice in more depth.

⁶https://epawebapp.epa.ie/licences/lic_eDMS/090151b2800980fb.pdf, page 16. The baseline dioxin/furan intake is given as 0.284 pg.kg.day, and, when PCBs are assumed to contribute 45-55% of total dioxin-like toxicity and added to this, the resulting total intake is given as 0.516-0.631 pg/kg/day, roughly twice the current EU TWI.

The result in the child considerably exceeds even the obsolete value for the EU TWI of 14 pg/kg/week TEQ, and for both child and adult it is far in excess of the currently permitted EU TWI of 2 pg/kg/week.

3. The contribution of the incinerator to the MARI family's intake – credibility and year-to-year variability

The 2008 application by Indaver to build an incinerator at the same site contained similar modelling to determine the dioxin and furan intake of the MARI family. The modelling was done in exactly the same way as for the present application in 2016 and 2019 (the 2019 modelling being re-used in the present 2025 EIS). In all these years, the incinerator was modelled as operating at maximum output, 24 hours a day, with maximum permitted emission of dioxins and furans as prescribed by the relevant EU Directives (this level did not change between 2008 and 2019). The quantity of dioxins and furans emitted from the modelled incinerator was similar in the 2008 and 2019 modelling: 6.7 ng/sec in 2008, 5.9 ng/sec in 2019.

However, the 2019 modelling in the 2025 EIS shows a considerably reduced impact of the facility on the predicted dioxin and furan intake for the MARI, compared to the 2008 model - in fact, it is reduced to one sixth (see my Appendix 5). In terms of total dioxin and furan intake, the facility contributes 0.97 pg/kg/week TEQ in 2008, but only 0.1617 pg/kg/week TEQ in 2019. This is puzzling, given that the incinerator and its Cork Harbour environment differ very little between the two years.

The MARI intake from the incinerator, like the baseline MARI intake, depends on the soil concentrations of dioxins and furans. The contribution from the incinerator thus depends on its effect on these soil concentrations. This depends on the deposition of dioxins and furans on the soil surface, and on the rate at which they disappear from the soil (called 'soil loss' and due to erosion, washout, evaporation etc). Without getting into the mathematics, which are explored in my Appendix 5, this process is simple to understand: the deposition represents the quantity of dioxins and furans landing on the ground, soil loss tells us the rate at which they disappear, and the balance determines the soil concentration.

The sixfold reduction in MARI intake from the incinerator is, as expected, paralleled by a similar reduction in the total modelled soil concentration of dioxins and furans (expressed as TEQ). But they are all affected to a different degree, as shown in my Appendix 6. It is perhaps best to concentrate on only one, the most toxic of the dioxins, TCDD⁷. To understand the reduced soil concentration in 2019 compared to 2008, since we know that it depends on only two factors in the model (deposition and soil loss), we need to know which of the two has changed.

In the case of TCDD, it appears that both have changed. The soil concentration of this most toxic of the dioxins is also the one that is reduced to the greatest extent (by far) in 2019 compared to 2008. As shown in my Appendix 6, although the MARI intake from the incinerator in 2019 is reduced by 6-fold overall, the reduction in TCDD soil concentration is a remarkable 560-fold. There is a roughly 14-fold fall in deposition, and a roughly 40-fold increase in the rate of soil loss (multiplying 14 by 40 gives 560).

Of all of the dioxins and furans, only TCDD has changed so radically in its modelled disappearance from the soil. The others show only a slight increase in the rate of soil loss in 2019 compared to 2008 (ranging from 1.2 to 1.5 times faster). But by far the biggest change is reserved for the most toxic, TCDD, which is modelled in 2019 as disappearing from the soil 40 times faster than in 2008.

⁷Its full name is 2,3,7,8 tetrachlorodibenzodioxin, which is why I am calling it TCDD.

To say that this requires explanation would be to state the obvious. Where is the evidence for such a radical change in soil loss for only one of the dioxins? Why is it not mentioned in Appendix 6.3 with a reference to the scientific literature as evidence?

With regard to the rate of deposition, all dioxins and furans show reductions in 2019 compared to 2008, to widely varying degrees. The overall deposition, in terms of the total amount of all dioxins and furans landing on the ground at the point of maximal deposition, falls by more than 95 % from 2008 to 2019, which is surprising, in view of the only 12 % fall in incinerator output of dioxins and furans (from 6.7 ng/sec in 2008 to 5.9 ng/sec in 2019, as mentioned above). The fact that deposition can change by a factor of 20 from one year to another, with only small changes in the output of the incinerator itself, raises questions about the credibility of the modelling – either there is extreme year-to-year variation, or something is wrong with the calculation. The very marked and unexplained change in soil loss for TCDD (see above) raises suspicions of the latter.

If the 2019 modelling of the incinerator contribution is indeed unreliable, and if the modelling from 2008 is more robust, we have to face the fact that the modelled contribution of the incinerator to MARI intake in 2008 (0.97 pg/kg/week) was *almost half* of the baseline intake currently modelled by AWN (2.0826 pg/kg/week). If we allow for the roughly 12 % reduction in incinerator output in 2025 compared to 2008, other things being equal, the contribution of the incinerator to MARI intake would still be 0.85 pg/kg/week, a more than 40 % increase above baseline.

Considering that the baseline is already considerably above the EU TWI, the fact that modelling from 2008 shows a further 40 % increase above baseline is alarming. It is imperative that this issue be clarified, because at present, when we consider the contribution of the incinerator to MARI intake of dioxin-like toxicity, we have the choice between a low value in 2019 that raises very serious doubts about its credibility, and an apparently more robust value from 2008, adjusted for the reduced output of the proposed incinerator, that would I suspect be considered unacceptable when added to the already high baseline.

4. Old analyses, and missing analyses in the 2025 EIS compared to earlier versions for the same project

It will be clear from the foregoing that the modelling in Appendix 6.3 is not new, but simply re-uses the 2019 version. The samples on which it is based are not new either, and were not new even in 2019; they are from 2015, and were used in the 2016 EIS. The excuse used is that levels of dioxins and similar compounds in Ringaskiddy milk have not greatly changed in the time since the samples were collected, so the old samples can be re-used. Obviously the modelling from the 2016 EIS cannot be re-used, for reasons that will be familiar to everyone who attended the 2016 oral hearing and are explored in the 2016 Inspector's Report. A total of 96 pages of data tables were presented that purported to be based on the Ringaskiddy 2015 samples; but part of the data was not from 2015, and the rest was not from Ringaskiddy, but the first few lines were different, which made it appear that the data was from Ringaskiddy in 2015. This is why we have later modelling in the 2025 EIS, carried out by the same author in 2019. The air modelling that feeds into the model has, however, been carried out anew in 2025, at least in part (see Appendices 8.1 and 8.4).

It is obvious that the air dispersion modelling shows very marked variation from year to year, in the location of the point of maximum deposition (as will be obvious from the aerial photos in my Appendix 5), and in the level of deposition at that point (as is explored in (3) above). In every Indaver EIS or EIAR at this site up to now, from 2002 to 2019, both air dispersion modelling and MARI intake modelling have been carried out, and the deposition calculated from the air dispersion modelling has fed into the MARI intake modelling for the same year. In 2025 one would have expected the same procedure as every year. But instead, the unusually low deposition modelled in

2019 has been 'frozen in time' in the MARI model to keep the MARI intake low, which avoids the possibility that the 2025 air modelling might produce a higher value, more similar to that in 2008.

In fact, although new air dispersion modelling does appear in the 2025 EIS, it is only partial modelling. It appears that the emissions for the Indaver facility alone were modelled as usual, but what is missing for other pollutants (including dioxins and furans) is the cumulative analysis of the effect of the Indaver facility along with existing sources in the area. That cumulative analysis has been done only for NO₂.

The reason no cumulative analysis was done except for NO₂ appears at first sight quite bizarre, and is a matter of the most serious concern with regard to the integrity of the EIS. In fact, the text of the paragraph introducing the cumulative analysis has been edited, and the edit makes it *appear* that it is not necessary in 2025.

In 2019, and indeed in every year since 2008, this paragraph has stated, *'The USEPA does not require a full cumulative assessment for a particular pollutant when emissions of that pollutant from a proposed source would not increase ambient levels by more than the significant ambient effect level (annual average of 1 µg/m³). A similar approach has been applied in the current assessment. A significance criterion of 2% of the ambient air quality standard or guideline has been applied for all non-criteria pollutants. These releases consist of NO₂, SO₂, HCl, HF, Dioxins, Cd, PAHs, As and Ni.'*

However in 2025, the sentence *'A significance criterion of 2% of the ambient air quality standard or guideline has been applied for all non-criteria pollutants.'* has been removed. This apparently small edit changes the meaning dramatically. Instead of stating that *'A significance criterion of 2% of the ambient air quality standard or guideline has been applied for all non-criteria pollutants. These releases consist of NO₂, SO₂, HCl, HF, Dioxins, Cd, PAHs, As and Ni.'*, it now states *'The USEPA does not require a full cumulative assessment for a particular pollutant when emissions of that pollutant from a proposed source would not increase ambient levels by more than the significant ambient effect level (annual average of 1 µg/m³). A similar approach has been applied in the current assessment. These releases consist of NO₂, SO₂, HCl, HF, Dioxins, Cd, PAHs, As and Ni.'*

The 2019 version tells us that the significance criterion for that list of pollutants is 2 % of the relevant air quality guideline. The 2025 version, in contrast, tells us that the significance criterion for these pollutants, including dioxins, is 1 µg/m³.

I need hardly state that if the concentration of dioxins in air were ever to reach 1 µg/m³, one would indeed not pause to carry out a cumulative impact analysis; one would immediately evacuate a wide area surrounding what must have been a very serious accident. That criterion of 1 µg/m³ is an absolutely ridiculous level to use for dioxins. The author knows that, so perhaps he did not read what he had written and think about what it meant.

But what has disappeared along with the apparently careless deletion of that sentence is the cumulative analysis of the dispersion of dioxins in air that was also in the 2019 EIS and not in the 2025 one. Using a criterion of 1 µg/m³, of course it could not possibly be necessary.

In truth, even the 2 % criterion for dioxins cannot be used, despite what the sentence said before it was deleted, because there is no statutory limit in air. It appears that in past years, although a criterion of 2 % has always been stated, the actual criterion that caused cumulative analysis to be carried out was the presence of other significant dioxin sources in the area, which have always appeared in a table in the relevant appendix (in 2019 this was Appendix 8.4). The presence of these

dioxin sources has not changed, though the table showing the other significant sources (present in every EIS and EIAR since 2008) has also disappeared.

The editing out of these two items from the EIS, that would have made clear the need for a full cumulative analysis, is of the most serious concern with regard to the integrity of the EIS; the reader gains the impression that there was no need for such analysis in 2025. The absence of the analysis itself is of course concerning for a different reason, because it makes adequate environmental impact assessment in this regard impossible.

If a decision had been made on evidence-based grounds that cumulative analysis of the other pollutants, including dioxins, was no longer considered necessary, that fact would have needed to be stated, along with the evidence. There is no indication that such analysis is less necessary now than it was in 2008 and 2016, and indeed, for dioxins it is *more* necessary, in view of the revision of the tolerable weekly intake (TWI) to reflect the recognition that dioxins are harmful at lower levels than previously thought.

Whatever the reason, it is clear that the omission of the cumulative analysis for dioxins and other pollutants apart from NO_x (oxides of nitrogen, including NO₂) was not a mistake. This fact is made explicit by the fact that, after the text in italics above, the following sentence has been added: 'For the facility, the only significant cumulative pollutant was NOX emissions.'