

Modelling of dioxins and related substances in the NIS

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Qualifications and experience of authors

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 very much more complex than the very simple 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.

Dr. Dara Fitzpatrick: My background is as a Senior Lecturer in Analytical Chemistry at University College Cork and a long time member and volunteer for Birdwatch Ireland carrying out the Wetland Bird Surveys of Cork Harbour for over 15 years. My activities with the Tracton Biodiversity Group have been recognised through a national Pride of Place Award (Nov 2025) for climate action and biodiversity.

I obtained a Ph.D. on the topic of transdermal drug delivery from Trinity College Dublin and went on to a postdoctoral research position on a European Framework V project (EDETTOX) modelling the skin permeability of compounds. Project partners included TNO in Amsterdam with modelling training based in HSE Sheffield also. This led to a book chapter in 'Dermal Absorption and Toxicity Assessment' (2nd edition, Informa Healthcare, Vol 177, Ch 15, p287). A paper titled 'Modelling skin permeability in risk assessment – the future' was also published in Chemosphere with over 100 citations. I went on to an academic lecturing position in UCC (Analytical Chemistry) where I developed novel instrumentation with multiple global patents and a spin-out company.

On behalf of CHASE we have compiled this report on the analysis and modelling of dioxins and furans at Ringaskiddy in the Natura Impact Statement, submitted as part of the application by Indaver Ireland for a proposed waste incinerator. The current version of the modelling is presented in Appendix 15 of

the NIS, which models dioxin intakes for species in the SPA and SAC in Cork Harbour, and draws on beach sediment sampling in Appendix 14, and air dispersion modelling in Appendix 11. All sections are compiled by AWN Consulting.

Description of the model in Appendix 15

The aim of the modelling, as for the human dioxin intake modelling in the EIS, is to show that the proposed site for the incinerator is safe, by modelling the concentrations of dioxins and furans in the eggs of fish-eating birds, and in the fish eaten by otters (which is then interpreted in terms of the otters' dioxin intake). These concentrations are then compared with experimentally derived limit values, described as No Observed Adverse Effect Limit (NOAEL) or Lowest Observed Adverse Effect Limit (LOAEL).

Unlike the model used in the EIS to investigate human intake of dioxins and related compounds, and described in my submission on the EIS modelling, the model used in the NIS is described in very sketchy terms. One equation is given, which gives us some understanding of it; the equation is derived from a US EPA publication.¹ The concentration in a bird's egg is given by the concentration in sediment (from the soil and sediment sampling in Appendix 14), divided by the organic fraction in the sediment, and multiplied by the biota-sediment accumulation factor, an overall term that describes how much of the dioxins in the sediment is accumulated in the bird's egg, then multiplied by the lipid fraction in the egg, to give the concentration of each congener in the egg. This is converted to toxicity equivalent (TEQ) using standard toxicity equivalence factors, which adjust for the different toxicity of each congener.

As for the modelling of the otters' intake, no information is given at all. It may be concluded, from reading the EPA publication that was the source for the bird model, that the equation used was probably the same, but nowhere is this stated in the Appendix. The total dioxin and furan content in fish is calculated, but how one arrives at the otters' dioxin and furan intake from this is left mysterious, as is the question of how one judges whether or not it is safe, though we are confidently assured that it is.

The diagram below (from Footnote 1) shows the general scheme of the model, as described by the US EPA, as it was intended to be used. It may help with orientation in reading this report. The relevant parts for this report are the pathway from sediment to birds' eggs via fish, and from sediment to fish to otters.

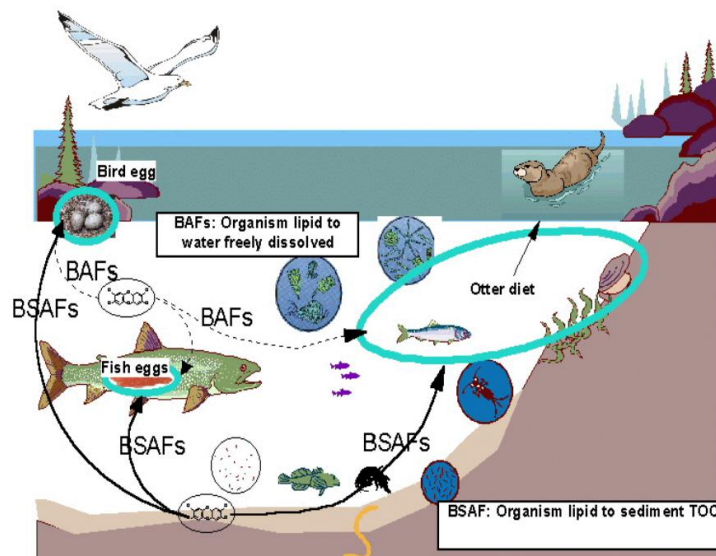


Figure 6. Estimating chemical concentrations in eggs and diet by applying BAFs and BSAFs for PCDDs, PCDFs, and PCBs.

¹ 'Framework for Application of the Toxicity Equivalence Methodology for Polychlorinated Dioxins, Furans, and Biphenyls in Ecological Risk Assessment', US EPA, 2008.

Detailed comments on the model and the results

1. The modelling is supposedly concentrating on cormorant and common tern. But when the concentration in eggs obtained from the modelling is compared to the value given for the no observed adverse effect level (NOAEL), we find that the NOAEL is not derived from cormorant or tern. It comes from two different species, 'black-eyed gull' (actually black-headed gull) and herring gull, cited from an Australian report.² The NOAEL value we are given is 50,000 pg per gram of egg. Interestingly, the Australian report that is the source for this NOAEL reports that these two gull species are the *least* sensitive among the SPA species or their close relatives. Another reason the NOAEL given is so high is that the 'adverse effect' for the gulls was embryo death! (It is well known that dioxins cause more subtle harms at concentrations very much lower than the lethal dose, as will be shown below.) The reason for choosing the lethal dose in very insensitive species as the criterion here is not stated.

This choice is especially puzzling because the same Australian report that presents the very high gull values used in the modelling, also presents values in cormorant and tern that could have been used instead. These are much lower than the gull values used in the modelling. The values in the Australian report are for lowest observed adverse effect levels (LOAEL) in eggs from double-crested cormorant and Foster's tern. They are more appropriate for risk assessment, not only because these are close relatives of the tern and cormorant in the SPA that are the focus of the modelling, but also because the LOAEL values relate to developmental abnormalities and not to death.

To understand how different the LOAEL or NOAEL values are for the two endpoints of embryo damage vs death, we can compare the values in eggs of double-crested cormorant in the Australian report. Developmental abnormalities are observed at 0.8 – 2 pg/g in cormorant eggs, and death at 4,000 pg/g, at least 2,000 times higher. In Foster's tern the LOAEL for developmental abnormalities is 8 pg/g egg (p. 39-40 of footnote 2; note that although that table also gives values in common tern, they are not in eggs, and only Foster's tern has egg values that are comparable to the modelling).

2. On a related point, we are particularly concerned that the applicant was provided by the Department of Arts, Heritage and the Gaeltacht with an LOAEL value in eggs in Foster's tern, and recommended to use it for common tern, but failed to do so (this is evident from the letter in Appendix 2). No reason for that is given. The LOAEL they were given in Foster's tern was 90 pg/g, *over 500-fold lower* than the LOAEL in gulls that they actually used in the modelling (But as mentioned in (1) above, the Australian report cites a still lower LOAEL value in Foster's tern from other research.)

3. The modelling in Appendix 15, as performed by AWN, results in a baseline value of 2.23 pg/g in eggs of a 'fish-eating bird' (Table 2.2 or Appendix 15). It will be remembered that the LOAEL in double-crested cormorant is 0.8 – 2 pg/g, meaning that ***the modelling in Appendix 15 already produces a value for egg dioxin concentration above the upper end of the range of LOAEL for developmental abnormalities in the cormorant.***

4. The report covers only dioxins and furans, but completely fails to consider the impact of other substances, notably dioxin-like polychlorinated biphenyls (DL-PCBs), as well as other persistent organic pollutants such as the per- and polyfluoroalkyl substances (PFAS).

The omission of DL-PCBs is a particularly serious failing, because the modelling is based on fish consumption by birds and mammals, and DL-PCBs constitute a large fraction of dioxin-like toxicity in fish. Data from the Food Safety Authority of Ireland (FSAI) indicates that in wild mackerel and farmed

²'National Dioxins Program Technical Report No. 11: Ecological Risk Assessment of Dioxins in Australia', Australian Government, Department of the Environment and Heritage, 2004

salmon, for example, including DL-PCBs in the analysis would add 60 % or more to the toxicity that comes from dioxins and furans alone.³

Although DL-PCB concentrations were not used in the modelling, they were nevertheless measured by the labs used, and make a brief appearance in Appendix 14. Insert 4.1 of that Appendix presents DL-PCB values, which will immediately appear to the reader as strikingly low in view of the FSAI research mentioned in the paragraph above. The reason they are low is that the concentrations given are ‘lower bound’ values, meaning that if a substance is present at a level below the detection threshold for the test, its concentration is reported as zero. Obviously this can underestimate the true concentration. It is safer and more usual in present-day practice to give the ‘upper bound’ values, meaning that if a substance is not detected, its concentration is reported as the limit of detection of the test. This may give an overestimate of the true concentration, but is a more precautionary approach when dealing with environmental toxins.

In the case of DL-PCBs, the difference between lower bound and upper bound value is very large indeed. The table below makes the difference clear; the second column shows the lower bound values, given in the report in Appendix 14, and the third column the upper bound values which are *not* shown and are only to be found in the raw lab reports. The upper bound values for DL-PCBs that are not presented in the text of the report are 18 – 155 times higher than the values that *are* presented in Insert 4.1.

Sample code	DL-PCB concentration (ng/kg TEQ), lower bound (from Insert 4.1)	DL-PCB concentration (ng/kg TEQ), upper bound (missing from text of report)
1A	0.0064	0.12
2A	0.0031	0.48
3A	0.0046	0.47
4A	0.0042	0.10

It may be noted here that lower bound values are also the ones chosen for the dioxins and furans presented in Inserts 4.1, 5.2, and 5.5, and we are told this is to allow ‘comparison with earlier measurements’ (which would justify the use of lower bound values in one comparative table, but not in *all* tables, omitting the upper bound values completely). In any case, this rationale cannot apply for DL-PCBs, because no earlier PCB measurements are presented.

We can see no good reason to conceal the upper bound values for DL-PCBs (or indeed for dioxins and furans), but the effect is to make the environment around the proposed incinerator and the SPA appear cleaner than it really is. This is really unacceptable in view of the importance of DL-PCBs in a marine context, and the known very large contribution they make to total dioxin-like toxicity.

Comparison of these upper bound DL-PCBs values with the dioxin and furan values in Appendix 14 shows that the DL-PCB concentration can vary from about 20 % of the dioxin and furan values up to over 200 %. This variability makes any single sample unreliable as a basis for estimating the DL-PCB contribution to overall dioxin-like toxicity in Cork Harbour, but emphasises the importance of DL-PCBs in a marine context. It is clearly unacceptable to ignore DL-PCBs in the modelling of risk to the species in the SPA.

5. We should now consider the effect that inclusion of DL-PCBs would have on the modelling in Appendix 15. This is hard to quantify based on the highly variable Cork Harbour samples, but as mentioned in (4) above, FSAI data shows that their inclusion increases total dioxin-like toxicity by

³ ‘Investigation into Levels of Dioxins, Furans, Polychlorinated Biphenyls and Brominated Flame Retardants in Fishery Products in Ireland’, Food Safety Authority of Ireland, April 2013; see Table 5.

about 60 % or more in real fish samples, compared to that estimated from dioxins and furans alone. Using that to guide our estimate, ***the value of 2.23 pg/g of dioxins and furans presented in the NIS (see item 3 above) would therefore increase to around 3.57 pg/g (2.23 pg/g x 1.6), which is well above the LOAEL for cormorant.***

6. Only piscivorous (fish-eating) bird species are considered in the modelling in Appendix 15, and only intake via fish is modelled. Other species protected in the SPA, that feed on worms, crustaceans and molluscs on the beach at low tide, are completely ignored – these include at least ten of the species protected in the SPA⁴. ***This omission makes assessment of possible adverse effects of the proposed incinerator on these species impossible.*** It is of particular concern that the accumulation of dioxins from sediment into living creatures (the BSAF value, explained in the description of the model above) is reported to be around ten times greater in molluscs (bivalves) than in fish⁵, raising the likelihood that species such as oystercatcher, feeding on molluscs and other creatures buried in the beach, could receive a much larger dioxin intake than fish-eating birds. Indeed a recent report from Sweden⁶, which focuses on oystercatcher, tern and cormorant in environmental monitoring, reports a trend towards increases in PCBs and a highly significant increase in dioxin and furan concentrations in oystercatchers from 2011-2018, which is not evident in the other species. It should be noted in this connection that the NIS shows that most of the sediment samples taken in Cork Harbour in 2009 and 2015 also show a marked increase in dioxin and furan concentrations over the period 2009 – 2015 (Appendix 14, Insert 5.5). This is a strong indication that modelling in piscivorous species does not tell us the situation of other species, and that oystercatchers, for one, appear to be more vulnerable, and, (based on the NIS data just mentioned), possibly increasingly vulnerable. This suggests that it is essential that the NIS include species such as those mentioned in footnote 4, and to include modelling of the dioxin-like toxicity in molluscs and other seabed-living species. Serious doubt is raised about the protection of the species that are ignored in the modelling in Appendix 15.

7. It is fundamental to any peer review that one should be able to follow what the authors of a report have done, in order to be able to verify that it was done in a valid way. But when one tries to follow the method used for the modelling here, it turns out to be very difficult. At baseline, we are told that the Whitegate sample is used, but in Insert 4.1 which gives the sample concentration, this appears as sample 1A, while in Inserts 5.2 and 5.5 it appears as sample 4A. As if this were not bad enough, the dioxin and furan concentration at Whitegate appears as 0.480 ng/kg in Insert 4.1, and 0.193 ng/kg in Insert 5.5. In fact, every value in Inserts 5.2 and 5.5 is different in Insert 4.1, whether one compares them based on the stated sample location, or the sample code – not only are they in the wrong order, but completely different.

When we consider the effect of the incinerator, we find that basic information in the modelling, such as how the change in sediment concentration was calculated, is completely missing. This information was given in the human modelling, and needs to be here too, as the reader may justifiably suspect that the physical processes determining sediment concentration are not the same as those determining soil concentration on dry land, and should thus be modelled differently.

8. The confusion about the identity of the baseline samples has implications for the conclusions of the modelling. If Inserts 5.2 and 5.5 identify the samples correctly, and Whitegate is sample 4A, then its concentration is rather low; sample 1A at Ringaskiddy has a concentration of dioxins and furans that is about 2.5 times higher than sample 4A at Whitegate (as well as obviously being closer to the proposed incinerator). In this case, the concentration of dioxins, furans and DL-PCBs in cormorant eggs ***would be increased from 3.57 pg/g (see 5 above) to 8.925 pg/g egg (i.e. 3.57 x 2.5). This is far above the LOAEL***

4 These species include Curlew, Whimbrel, Redshank, Greenshank, Black-tailed Godwit, Turnstone, Oystercatcher, Common Sandpiper, Knot, and Dunlin.

5 'National Dioxins Program Technical Report No. 11: Ecological Risk Assessment of Dioxins in Australia', Australian Government, Department of the Environment and Heritage, 2004; see Figs. 3.5 and 3.6

6 Soerensen AL, Faxneld S, 'The use of common tern, Eurasian oystercatcher, and great cormorant as indicator species for contaminant monitoring', Swedish Museum of Natural History, report no. 5, 2020.

for cormorant eggs of 0.8 – 2 pg/g egg, and is now also above the LOAEL for tern eggs of 8 pg/g egg. Obviously the error in sample identification, and the uncertainty about which of the tables may be correct, introduces doubt about the modelling which is not conducive to the welfare of the protected species in the SPA, even those that were modelled.

9. On inspecting the sediment concentration values in Tables 2.2 and 2.4 of Appendix 15 of the NIS, one finds that when the incinerator is added to the model, the modelled dioxin and furan concentrations in marine sediments change by a similar or identical amount to the modelled soil concentrations in the EIS.⁷ This is a serious concern because, as mentioned in the report by one of us (GR) on the human modelling in the EIS, the change in modelled soil concentration and dioxin uptake caused by the incinerator was reduced sixfold between 2008 and the latest 2019 modelling – despite only a small change in the dioxin and furan output of the proposed incinerator. It thus seems likely that the problems highlighted in the report on the human modelling – unexplained and extreme changes in deposition rate and soil loss, that cannot be explained by the small change in incinerator output – have been carried over into the modelling in the NIS. This needs to be clarified. Until the detail of the calculations is given, and the issues relating to soil concentration in my report on the EIS are clarified, we must keep in mind the possibility that the 2008 report (where these problems do not arise) is a better representation of the effect of the incinerator than the present model, and that its effect may be about six times greater than shown in Tables 2.2 and 2.4.

10. Turning now to otters, the risk assessment is presented in terms of the dioxin content of the fish they are assumed to eat. But information about the model used to obtain the dioxin content of fish is completely missing. The only equation in the Appendix relates to birds' eggs, and we are left guessing as to whether the same equation is used for the fish in the otters' diet. The reader may suspect that the equation might be the same, but this is nowhere made clear, nor is the source of the parameters in the model given (e.g. lipid content and BSAF). This makes the method difficult to follow and validate.

11. As an outcome of the model, we are given a value for dioxin content of fish, and that is all. ***The model does not arrive at a dioxin intake value for the otters.*** There is no way even for the reader to work out the dioxin intake, because we are not told how much fish an otter is assumed to eat or its body weight, which are the two pieces of information that would allow us to work out the dioxin intake.

12. We are not told the species of fish being modelled, which makes it impossible to compare the modelled dioxin content of fish with real measured values in fish (there is great variation between fish species in their dioxin content). The value for fish dioxin content that emerges from the modelling is low, and far lower than reported values in real fish (e.g. mackerel and salmon) from the FSAI report⁸. There must therefore be a degree of doubt about whether the model is truly representing the uptake of dioxins into fish.

13. As with the modelling of birds' eggs, mentioned in (4) above, DL-PCBs are completely ignored. This is particularly unfortunate in view of the data in fish from the FSAI, which shows that including DL-PCBs for salmon and mackerel would increase dioxin-like toxicity by 60 % or more.

14. No NOAEL or LOAEL is given for otters, which is odd, since such values for *Mustelidae* based on food intake are not difficult to find. For this reason, even if we had been given information on the otter's dioxin intake, it would be impossible to make any comparison between that intake and the level that might be safe.

15. The above shortcomings do not prevent the author of the Appendix giving us their conclusion from the otter modelling, which is that baseline dioxin concentrations in fish-eating birds and otters are 'considered to be low and well within limit values for the eggs of fish eating birds.' This is doubly odd,

⁷ See Appendix 6.3 of the EIS and the report by GR on it, which accompanies this one.

⁸ 'Investigation into Levels of Dioxins, Furans, Polychlorinated Biphenyls and Brominated Flame Retardants in Fishery Products in Ireland', Food Safety Authority of Ireland, April 2013; see Table 5.

given that we know absolutely nothing of the dioxin concentration in otters that results from the modelling, as mentioned in (11) above, and that LOAEL values for birds' eggs are not necessarily applicable to otters, owing to the known physiological differences between sea mammals and eggs.

16. The predicted change in dioxin concentrations due to the incinerator is considered to be insignificant for otters, based on exposure to forage fish, but again we are not told (and, as far as we can tell, the modeller does not know) what the change in otters' dioxin intake or concentration is, nor how its significance or insignificance can be assessed in the absence of any LOAEL or NOAEL value.

Conclusions

We would point to four major themes in our observations on the modelling of dioxin-related toxicity intake in the NIS.

1. Based on published lowest observed adverse effect level (LOAEL) data and the existing modelling, the concentrations of dioxins and furans in cormorant eggs are likely to be above the upper range of the LOAEL for developmental abnormalities. This is even more so when dioxin-like PCBs are included.
2. There are serious gaps and shortcomings in the modelling, the most serious being the modelling only of piscivorous (fish-eating) birds, and the complete omission of all the birds that feed on molluscs, worms and crustaceans on the mudflats; especially in view of the higher biota-sediment accumulation factors (BSAF) in bivalves than fish, leading to possible higher intakes for such species. It is impossible to be sure what their dioxin intake might be, at baseline or with the proposed incinerator.
3. The confusion about sample identification and sample concentrations makes it difficult to have trust in the integrity of the data. Only by going to the original lab reports has it been possible to check on some aspects of the data to verify some of the findings.
4. Beyond the confusion just mentioned, there are also deliberate decisions that are really inexplicable in a scientific report. For instance, the choice of a no observed adverse effect level (NOAEL) in gulls, the least sensitive species in the SPA, based on embryo death rather than developmental abnormalities; the failure to use the 500-fold lower LOAEL value that the investigators were given by the Department of Arts, Heritage and the Gaeltacht; the use of lower bound values to report PCBs, when the upper bound values were up to 150 times higher; and the complete omission of dioxin-like PCBs, which is very hard to justify, especially in a marine context.

In summary, the report is not one on which we could base a confident judgement that the protected bird species in the SPA, or the otters protected under the Habitats Directive, would not come to harm if the proposed incinerator were to be built. On the contrary, it suggests that even at baseline, levels of dioxin-like toxicity are already at or over the limits where adverse effects may be observed.