Strategic Infrastructure Development
Application to An Bord Pleanála
Reg. no. PL04.PA0045

Proposed municipal and hazardous waste incinerator at Ringaskiddy, Co. Cork

Report on the integrity and validity of the modelling of dioxin-like toxicity intake carried out by Dr Fergal Callaghan (AWN) for Indaver

Dr Gordon Reid
19 July 2017

1. Qualifications and experience

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 and other epithelial cancers. 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, the learned society that supports and oversees the profession of physiology.

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. 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.

2. Summary

In addition to a number of technical documents, Indaver has submitted a letter (document 01 of the Further Information) and an addendum to one of the reports (document 03 of the Further Information), giving its explanation of the discrepancies in the original Appendix 6.4 that we revealed at the oral hearing.

The claims made in these documents can be grouped under the following headings; our response to each is given briefly, and will be considered in detail in the next section.

2.1. Indaver states that it has now enclosed "the correct print-outs of attachments D and J that are referenced in the report" (i.e. appendix 6.4 of the EIS). This appendix originally contained version of attachments D and J that bore no relation to the rest of its contents. These attachments should link the measured or predicted soil concentrations of dioxins and furans with tables 5.1 and 7.1 of appendix 6.4. Indaver's claim is that they now do, apart from "minor transcription errors".

Response: While I agree that the submitted attachment D is broadly able to link the soil sample data (appendix 6.3 of the original EIS) with the calculated dioxin/furan intake for MARI, the maximum at risk individual (table 5.1 of appendix 6.4 in the original EIS), I find that the submitted attachment J is *not* able to link the predicted soil levels of dioxins/furans with the predicted dioxin/furan intake for MARI (table 7.1 of appendix 6.4 in the original EIS). Of 17 congeners, *every single one* has a different value. I do not believe that this can credibly be explained as a "minor transcription error" (see also 2.3 below).
In addition, I note that as well as having submitted new versions of attachments D and J, the applicant has also replaced the main body of attachment 6.4 (document 02 of the Further Information). It was maintained during the oral hearing that the modelling results (tables 5.1 and 7.1 of the original appendix 6.4) were correct, and that the only error had been the copying and pasting of the wrong attachments D and J. We were told the correct versions of attachments D and J would be submitted if required. This has not happened: it seems it is no longer the case that both original tables 5.1 and 7.1 are correct, as the values given for the predicted contribution to MARI intake from the incinerator (table 7.1) are now different from those presented during the oral hearing. One thing that has not changed is that the air concentration of dioxins and furans (page 9 of document 02, the modified appendix 6.4) is still reported as 0.0014 pg/m^3 TEQ. This error was noticed by Joe Noonan, and I pointed it out on the last day of the oral hearing; the correct value should be 0.014 pg/m^3. The error results from simple arithmetical mistakes in transferring the data from tables A8.10, A8.11 and A8.12 to table A8.23: 13.5 femtograms is converted to 0.0013 picograms (it is actually 0.0135 picograms); and 0.0013 is added to 0.001 to give 0.0014. These are basic errors that do not belong in an EIS in the first place, and not to have corrected them after they had been pointed out is surprising and disappointing.

2.2. Indaver states that an independent expert, Dr Paul Johnston, has examined the "modelling report" submitted by Indaver.

Response: Comparison of the filenames and values referred to in Dr Johnston's report leads me to conclude that the documents reviewed and reported on by Dr Johnston are

not the same documents as those that have now been placed on the Indaver website (and presumably submitted by Indaver to An Bord Pleanála in its response to the request for further information). This will be detailed in section 3 below.

2.3. Indaver states (document 01, page 1, point 3) that Dr Johnston has "reconfirmed the robustness of the model, methodology, inputs and outputs", apart from "a number of *minor transcription errors* in the modelling report" (my italics).

Response: In reconfirming the "robustness of the model, methodology, inputs and outputs" Dr Johnston has overlooked very substantial problems in the modelling by Dr Callaghan, the consultant acting for Indaver. These omissions, and deviations from the stated methodology, have the effect of artificially reducing the apparent MARI intake of dioxin-like toxicity, and will be considered in more detail in relation to point (7) below. Section 4 is devoted to a detailed analysis of these omissions and deviations.
The "minor transcription errors" between attachment J and table 7.1 of appendix 6.4 are simply not credible as transcription errors. Of 17 values, *all* are wrong, and the majority are wrong by *exactly the same proportion*. Eleven of 17 values "copied down wrongly" and now "corrected" are within a range of 95.40 - 95.67 % of the "correct" values (i.e. indistinguishable, within rounding).
People simply *do not make that kind of mistake* in copying numbers from the screen. I conclude that the numbers that were copied from the screen to make table 7.1 of appendix 6.4 in the original EIS were not from the same dataset as now presented in attachment J. Indaver still has not provided a version of attachment J that accounts for the data presented in the original table 7.1. Furthermore, if the parameters of the model that produced the new versions of attachments D and J are compatible, it follows that the original table 7.1 must have resulted from the use of *different* model parameters from those used to produce the original table 5.1. This difference has not been explained.

2.4. In addition to these "minor transcription errors", the addendum states that "one value out of 58 input fields was entered as a higher concentration than the actual measured value on-site" and that this was corrected.

Response: Prof Johnston noted more than one error in the input values. He lists errors in the baseline values for 1,2,3,4,7,8 HxCDD, 2,3,7,8 TCDF and 2,3,7,8 TCDD entered into attachment D, and has overlooked an error in the baseline value of 1,2,3,7,8,9 HxCDF. He also lists errors in the predicted values for 2,3,4,7,8 PeCDF and "2,3,4,6,7,8 HpCDF" (this is actually 2,3,4,6,7,8 HxCDF) entered into attachment J, has overlooked an error in the predicted value of 1,2,3,7,8,9 HxCDF, and apparently used a different value in the comparison for 2,3,4,6,7,8 HxCDF from the value that appears in the EIS. This is a total of seven input values for dioxin/furan concentration that show discrepancies, from a total of 34 input values (baseline and predicted values, for each of 17 congeners). This tells a rather different story from "one value out of 58 input fields" as claimed by Dr Callaghan.

2.5. Dr Callaghan proposes that the "minor transcription errors" occurred because the RISC-HUMAN model does not produce an output in a form that can be read directly into the Excel spreadsheet used to generate the tables given in the EIS, thus requiring the modeller to write them down and enter them manually into Excel.

Response: It is not necessary to write the numbers from the screen, and it is surprising that any professional engineer would use such an error-prone approach. We are told that RISC-HUMAN produces a text format file that contains the numbers that need to be entered into the Excel spreadsheet. With such a file available, it is a simple matter to edit it for direct import into Excel, thus avoiding any possibility of error, and even simpler to copy and paste the numbers. I have discussed this matter with other colleagues who are used to dealing with the analysis of large datasets in our academic work, and none would use such an unreliable approach as to copy values from the screen when the text file is available. Even if the choice is made to copy values from the screen, it is irresponsible not to proofread them, when dealing with a matter such as this that has profound implications for human health. The multiple errors made possible by this approach raise serious doubts about the competence of the consultant who produced the data, and thus about its reliability as a basis for a sound environmental impact assessment.

2.6. Indaver and Dr Callaghan offer a narrative to explain why, as we pointed out at the oral hearing, attachments D and J of appendix 6.4 bore no relation to the other data in the appendix and were, in fact, identical to attachments D and J of Indaver's 2008 application (apart from two lines at the beginning of the text). No explanation is offered to clarify why attachment J was also the same as in a 2008 planning application by College Proteins, County Meath, as we also pointed out at the oral hearing.

Response: The narrative we are offered could explain why a *complete* document from 2008 could have been imported wrongly into appendix 6.4, but is unable to account for the fact that the second and third lines of the file, which indicate the filename and the date and origin of the model data, are different between the attachments in the 2015 EIS and the 2008 originals. The 2008 files appear to have been manually edited either before or after being converted to Word, so as to make them look as if they were from 2015. It would be impossible to do such a manual edit without noticing that the original file being edited was from 2008.
Furthermore, there is no explanation for the fact that data in attachment J of Indaver's 2016 and 2008 applications at Ringaskiddy is also identical to attachment J of another 2008 application, prepared by the same consultant, for a different site - a biomass CHP plant at College Proteins, Nobber, Co. Meath. Strangely, data in attachment J in the 2016 EIS is also identical to attachment D of the College Proteins application, because, in the 2008 College Proteins EIS, attachment D (pre-CHP plant) and attachment J (predicted with CHP plant) are in fact *identical to each other*. So we have four attachments with identical data:
Nobber, 2008, attachment D (pre-CHP plant)
Nobber, 2008, attachment J (predicted post- CHP plant)
Ringaskiddy, 2008, attachment J (predicted post-incinerator)
Ringaskiddy, 2016, attachment J (predicted post-incinerator)
- where these differ at all, it is only in the edited lines at the beginning that make them appear to be from different places in different years. In so far as we still have not been told how data files were mixed up, not only between different years at the same site, but between different sites at opposite ends of the country, Indaver has not fully complied with the Board's request to explain the discrepancies we revealed at the oral hearing.

If it is suggested that these are *all* copy-and-paste errors, the degree of carelessness involved is almost beyond belief, and this again raises very serious doubts about the competence of the consultant who did the work and thus the reliability of the data that form the basis for Indaver's claim that the proposed incinerator will be safe.

2.7. Indaver continues to maintain that "the proposed development will have no significant impact on dioxin and furan intake for even the theoretical maximum at risk individual" (abbreviated as "MARI").

Response: The documents provided (both the original EIS, and the further information) do not offer sufficient detail to allow that claim to be assessed: no basis is given for the values of dioxin/furan deposition that appear n attachment H and are inserted into the model for the run shown in attachment J. In view of the fundamental unreliability of the data we have already been given by Dr Callaghan in the original EIS, and its only partial improvement in the further information now submitted to the Board, I would suggest that the Board cannot accept at face value the dioxin deposition values, without verifiable detail on how they were arrived at.
I pointed out during the oral hearing, in questioning to Dr Edward Porter, the inconsistency between Dr Porter's modelling of the spread of emissions from the incinerator and independent modelling carried out for CHASE. This discrepancy is still unresolved and unexplained, and adds to the sense of uncertainty about the contribution the incinerator would make to dioxin-like toxicity levels in soil.
I have a particular and very serious concern over the data given in attachment H of appendix 6.4, which shows the predicted dioxin/furan deposition rates, and their addition to existing soil concentrations to give a predicted soil concentration with the proposed incinerator in operation. The measured soil concentrations are in mass units (ng/kg soil), but the predicted deposition is stated as being in toxicity equivalent units (ng/kg TEQ). The TEQ is equal to the mass multiplied by a toxicity equivalence factor (TEF), which varies from 1 for the most toxic congeners, to 0.0003 for the least toxic. After this addition, the resulting values are entered into the model to calculate dioxin intake for MARI, which is ostensibly in mass units. These mass units are then converted again, using the TEF values, into TEQ values.
This would under-represent the added toxicity from the incinerator for most congeners, by a factor of between 10 and 3000. This is because the conversion from mass to TEQ units is being made *twice* for the predicted dioxin/furan deposition for the incinerator. If the addition of mass units to TEQ units is a mistake, it is an astonishing mistake for an expert in the field to make - it is meaningless to add values in completely different units.
I note that attachment H is not among the documents that have been replaced in the Further Information. I take it from this that Indaver and Dr Callaghan consider that attachment H is correct in its present form.

Because, as mentioned above, we have no way to check the predicted deposition rates, we are therefore able to check only the calculation of the background intake of dioxin-like toxicity for MARI (i.e. the existing level, without the proposed incinerator). On examination of this, I find that the MARI diet, on which the intake is based, is below 1000 calories per day (MARI is a subsistence farmer, doing 16 hours per day of manual work in the fields!) and omits many of the food groups that MARI would be most likely to eat. The criterion given for the choice of soil sample site would actually lead a different site to be chosen as the basis for the modelling. Assumptions are made

(e.g. lifetime averaging of dioxin/furan intake, ignoring the intake of a child which would be higher; the omission of PCBs from the predicted intake) that differ from those made by Dr Callaghan in previous modelling, and from the HHRAP methodology that Dr Callaghan tells us he is following. In every case, these assumptions, deviations and omissions are in such a direction as to reduce the apparent MARI intake of dioxin-like toxicity. When they are corrected, a more accurate estimate of the MARI child's intake of dioxin-like toxicity is over 30 times as high as the lifetime average value stated by Dr Callaghan, and is far above the EU and WHO "tolerable" intake (this is examined more fully in section 2 below). This would lead to the conclusion that no further sources of dioxin-like toxicity (such as the proposed incinerator) should be approved at Ringaskiddy until soil levels of dioxins and furans are well below their current values.

I note in this connection the comments of a previous An Bord Pleanála inspector, dealing with an earlier application by Indaver at Ringaskiddy: "It would therefore be reasonable to conclude that it is the view of all international and regulatory authorities that PCDD/F are considered highly toxic and the policies of WHO, Stockholm Convention, and EU are in the direction of reduction of these substances with ultimate aim of elimination. Having regard to unequivocal references to 'protection of human health' and the objectives of reduction and elimination referred to above, I am not satisfied that conclusion drawn by the applicants and in particular the by the HIA that 'there would be no impact on human health' is plausible. In my view, notwithstanding non-ratification of the Stockholm Convention, it would also be reasonable to conclude that any operation which would give rise to increase in such substances no matter how small would be contrary to these policies, in principle. In this regard, Regulation EC 850/2004 of particular relevance."[1]

The points mentioned above will be considered in more detail below, under two broad headings: firstly, the consistency of the data offered by Indaver and reported on by Dr Johnston (section 3), and secondly, the methods used to model dioxin uptake by MARI; whether the methods are consistent with the stated HHRAP or COT methodologies, and whether the resulting uptake is realistic (section 4).

3. Consistency of data

The further information supplied by Indaver includes a report by Prof Paul Johnston, entitled "Report on Data Consistency in Modelling of Risk Assessment". Dr Johnston describes his task thus: "... to check the data consistency between the inputs for modelling risk assessment, as given in the Baseline Reports that formed part of the EIS submitted to An Bord Pleanála with the application for planning permission for the Indaver Waste to Energy facility, Ringaskiddy in January 2016.
Specifically, I was asked to confirm that the data chain used in modelling analysis as reported in the Modelling Report (Modelling of PCDD/F intake for Ringaskiddy Resource Recovery Centre 2015, AWN Technical Report FC/14/8104SR02), submitted with the EIS, was complete and consistent. The soil and modelled data given in the body of the report was checked against the printed

[1] PA0010 inspector's report, 2009, page 334

input/output files from the computer model as printed in the relevant attachments to the report."

(a) Baseline soil concentrations (section 5(1) of Dr Johnston's report)

The first point to be checked was the entry of the soil dioxin and furan concentrations from sample site 4A. These are given in the lab report from Scientific Analysis Laboratories Ltd (SAL; page 56 of 62 in appendix 6.3) and are transcribed into table 2 (page 30 of the same appendix). Dr Johnston tells us that "The baseline soil dioxin concentrations ... are reported in Table 2 ... and presented in an Excel spreadsheet (attached)." At this point it should be mentioned that no Excel spreadsheet is attached to Dr Johnston's report on the Indaver website, thus we (and the Board) are lacking this important piece of information.

Dr Johnston does not mention that, although the soil concentrations in mass units (ng/kg) in table 2 correspond with the lab reports, the TEQ values do not. There is a similar lack of correspondence between the TEQ values in table 2 and those in insert 4.1 of appendix 6.3, which presents a summary of the soil concentrations in TEQ units. Insert 4.1 agrees with the lab report for sample 4A (and for sample 3A, which will feature later in this report).

The discrepancy between table 2 on the one hand, and insert 4.1 and the lab reports on the other, appears to result from the use of a different system of toxicity equivalence factors (NATO-CCMS I-TEF) in table 2 that is not used in other parts of the appendix. Why this different system was used here is not clear. It contributes nothing and causes confusion.

Dr Johnston tells us that "These concentration data ... were checked against the computer model input data file (BASE2015.LOC) as reproduced in attachment D1 of the modelling report (Report FC/14/8104SR02)." Report FC/14/8104SR02 is the original appendix 6.4 of the EIS, dated 17th December 2015, and submitted to An Bord Pleanála in January 2016. The baseline computer model input data file in attachment D of this report has the filename RINBSL4.loc, not BASE2015.LOC. A revised version of attachment D was submitted with the further information supplied to the Board by Indaver; the data in this attachment relate to the model input file FBAS2015.LOC. Nowhere in the original EIS or the additional information is there a computer model input file with the name BASE2015.LOC, which was the file checked for consistency by Dr Johnston.

Moving on to the numbers themselves, the two anomalies noted by Dr Johnston (for 1,2,3,4,7,8 HxCDD and 2,3,7,8 TCDF) are not to be found in the version of Attachment D presented to the Board. I have also been unable to trace the following: "For 2378 TCDD, however, an observed value of 0.061 ng/kg in Table 2 was identical to the 'site soil' input value of 6.10×10^{-8} mg/kg in Attachment D1 but a different component value of 2.61×10^{-7} mg/kg for 'soil in open surface areas' appears in the computer data input file." I can find no value of 2.61 x 10-7 mg/kg (which would appear as 2.61E-07) in the entirety of attachment D, and no entry for 'soil in open surface areas'. I am thus in the dark (as is the Board) as to what Dr Johnston saw in the data presented to him by Indaver; whatever it was, it is not in the data presented to the Board.

Dr Johnston does not mention the difference between the soil concentration of 1,2,3,7,8,9 HxCDF measured by SAL and reported in Table 2 of Appendix 6.3 (<0.13 ng/kg), and that entered in the computer model input data file and shown in the revised Attachment D (1.03×10^{-7} mg/kg, i.e. 0.103 ng/kg).

Taken together, the three facts noted above:
(i) the filename of the computer input data file inspected by Dr Johnston in the Attachment D he inspected (BASE2015.LOC) is not the same as that supplied to the Board in Attachment D of Indaver's further information (FBAS2015.LOC);
(ii) differences noted by Dr Johnston between Table 2 of Appendix 6.3, and the data contained in the version of Attachment D that he saw, are not present in the version of Attachment D supplied to the Board;
(iii) Dr Johnston's report does not mention a discrepancy for 1,2,3,7,8,9 HxCDF between Table 2 of Appendix 6.3 and the version of Attachment D supplied to the Board;
lead me to conclude that the version of Attachment D inspected and reported on by Dr Johnston is not the same document that has now been supplied to the Board. This fact is not acknowledged in Indaver's submission. It is claimed by Indaver that "errors have been corrected", but this does not explain why new errors have apparently come into being since Prof Johnston's report, or why there is no numerical value at all for "soil in open surface areas" in the Attachment D presented to the Board.
I would question the value of a consistency report that does not relate to the documentation that the Board has been given, and fails to mention the inconsistencies that it contains.

(b) Predicted soil concentrations including incinerator emissions (section 5(2) of Dr Johnston's report)

Dr Johnston states that "The WtE model output data (file INT2015.LOC, Ringaskiddy intake 2015) for the change in PCDD dose using an air dispersion model, is given in Attachment J : Model output file for change in PCDD/F dose. These data represent inputs to the risk assessment model, RISC HUMAN, as presented in a separate spreadsheet (attached). These data in Attachment J were checked against the spreadsheet table of input values for calculating the MARI."

The first point that must be made is that the attachment J supplied to the Board does not contain the data from file INT2015.LOC; it contains data from a different file, FINT2015.LOC. Secondly, the "separate spreadsheet (attached)" has not been attached. Once again, Dr Johnston is reporting on documentation that the Board does not have. Both points echo those already made for the baseline data.

The predicted soil concentrations entered in attachment J are derived from the table in attachment H of the EIS. No amended version of attachment H has been submitted, from which I conclude that the original is thought to remain valid. I have compared the predicted values for each congener in attachment H with those now submitted to the Board in the amended attachment J.

The values for 2,3,4,6,7,8 HxCDF and 1,2,3,7,8,9 HxCDF are different between attachment H and the amended attachment J.

Dr Johnston does not mention the discrepancy in the value for 1,2,3,7,8,9 HxCDF between attachment J and attachment H. Attachment H gives a predicted soil concentration of 3.40 x 10-7 mg/kg, while the value in attachment J is 1.22 x 10-7 mg/kg.

He does, however, note a difference for 2,3,4,6,7,8 HxCDF (wrongly cited as "234678 HpCDF" in his report, and also in Dr Callaghan's documentation[2]), but the difference he reports is not the difference to be found in the documentation submitted to the Board.

Dr Johnston reports that the value for "234678 HpCDF" (i.e. 2,3,4,6,7,8 HxCDF) is 5.63 x 10^{-7} mg/kg in attachment J (with which I agree), but that the "predicted value reported as input to RISC HUMAN" - possibly from the "attached spreadsheet" that is not attached - is 5.60 x 10^{-7} mg/kg, a value that is not to be found in the documentation submitted to the Board. The value in the documentation we and the Board have is instead 4.69 x 10^{-7} mg/kg, in attachment H.

Dr Johnston notes a small difference for 2,3,4,7,8 PeCDF (2.71 x 10^{-7} vs 2.70 x 10^{-7}), which is not present in the documentation submitted to the Board; the entries for this compound in attachment H and attachment J agree (both are 2.71 x 10^{-7}).

Dr Johnston does not comment on the fact that the predicted deposition due to incinerator emissions is reported in attachment H as being in TEQ units, while soil and total concentrations are reported as being in mass units (already mentioned above in point 7, page 4, of my report).

(c) Predicted dioxin/furan uptake for MARI including incinerator emissions (section 5(4) of Dr Johnston's report)

Dr Johnston gives a table of predicted dioxin/furan uptakes for MARI, comparing the values from attachment J and table 7.1. The value for 2,3,7,8 TCDD given for attachment J in this table does not agree with the value in the attachment J supplied to the Board, for reasons that are not clear. The discrepancy is apparently not explained by the replacement of an earlier wrong value by the correct value, because the predicted soil concentration for this congener in attachment H of the original EIS (6.13 x 10^{-8} mg/kg) agrees (within rounding error) with the input value in attachment J (6.12 x 10^{-8} mg/kg) - i.e. the input value has not changed. This input value gives a predicted uptake for MARI of 3.32 x 10^{-11} mg/kg/day, as is evident from the version of attachment J suppled to the Board. But Dr Johnston's report shows a value of 5.04 x 10^{-11} mg/kg/day for 2,3,7,8 TCDD in "attachment J". Clearly this is another

[2] The abbreviation "HpCDF" is short for "heptachlorodibenzofuran", and "HxCDF" is "heptachlorodibenzofuran", referring to molecules containing seven and six chlorine atoms respectively. The numbers (e.g. 2,3,4,6,7,8) refer to the positions of the chlorine atoms. A list of six numbers would inevitably be followed by "HxCDF" and not "HpCDF". I find this mistake an odd one to be made by an expert in the field of dioxin chemistry, as we are told Dr Callaghan is, and it is also odd that Dr Johnston did not correct it. It is apparently not a simple typo on Dr Callaghan's part, as all mentions I can find of 2,3,4,6,7,8 HxCDF in every attachment to appendix 6.4 in the original and amended documentation refer to "2,3,4,6,7,8 HpCDF"; only tables 5.1 and 7.1 in the main body of appendix 6.4 refer to it correctly.

manifestation of the fact that Dr Johnston was given a different ersion of attachment J from that given to the Board and to us (see 1(b) above).

The other values in Dr Johnston's table (page 5 of his report) agree with those in the documentation supplied to the Board. What is striking about this table is that *every number* in the left-hand column is different from the corresponding number in the right-hand column. The numbers (as found in the documentation submitted to the Board) are given in the following table. Attachment J (second column) in the table below refers to the document now laid before the Board. Table 7.1 (third column) refers to appendix 6.4 of the original EIS, submitted with the planning application in January 2016. The table is not identical to the version in Dr Johnston's report, because of the discrepancy noted above for 2,3,7,8 TCDD.

Congener	Attachment J mg/kg/day	Table 7.1 mg/kg/day	Ratio
2378TCDD	3.32E-11	5.20E-11	0.638
12378PeCDD	8.72E-11	8.41E-11	1.037
123478HxCDD	7.79E-11	7.44E-11	1.047
123678HxCDD	1.65E-10	1.58E-10	1.044
123789HxCDD	1.18E-10	1.13E-10	1.044
1234678HpCDD	1.57E-09	1.50E-09	1.047
OCDD	1.23E-08	1.17E-08	1.051
2378TCDF	3.36E-11	3.48E-11	0.966
12378PeCDF	6.44E-11	6.32E-11	1.019
23478PeCDF	6.88E-11	6.67E-11	1.031
123478HxCDF	2.93E-10	2.81E-10	1.043
123678HxCDF	2.18E-10	1.99E-10	1.095
234678HxCDF	3.68E-10	3.51E-10	1.048
123789HxCDF	7.83E-11	7.48E-11	1.047
1234678HpCDF	1.55E-09	1.48E-09	1.047
1234789HpCDF	2.09E-10	1.99E-10	1.050
OCDF	1.68E-09	1.60E-09	1.050

The addendum submitted by Indaver along with the amended dioxin report (document 03 of the Further Information) describes these differences thus: "Dr Paul Johnston's review of the correct appendices and the modelling found that the data in the appendices matched the model inputs and outputs with some minor transcription errors, these errors were related to how the data was transcribed and were minor in nature."

The "transcription errors" are stated to have happened during this process: "The modeller then takes the output from this model, again from the screen, and inputs that to the Word Document Model and Excel Report also and then writes up the Intake Model Report." So we are being told that the 17 numbers in attachment J (left column in the table above) were copied from the screen and entered into table 7.1 (right column in the table above).

It is *simply not credible* that anyone copying these 17 numbers from the screen would copy them *all* wrongly! Furthermore, the "transcription errors" are not random. With

only two exceptions, the numbers entered into table 7.1 are smaller than those in attachment J, and a majority of them are smaller by almost exactly the same proportion. As shown in the rightmost column of the above table, eleven of the 17 numbers are smaller by 4.4 - 5.0 % (i.e. they are the same, within the error of rounding to two decimal places).

I conclude that these are *not* transcription errors, and that the claim that they are is simply not credible. It is clear that the numbers being copied from the screen to create table 7.1 were *not* the numbers in the version of attachment J that has been submitted to the Board.

This is a matter of concern, and not only because the explanation offered by Indaver is so obviously not the correct one. The modelling we have been given is meant to link soil concentrations to modelled dioxin/furan uptakes for MARI. Between the baseline and incinerator conditions, only the input values should differ. For the baseline condition, the input value should be the soil concentration (appendix 6.3), and for the incinerator condition, it should be the soil concentration plus predicted deposition (attachment H). Apart from these, all parameters (i.e. input conditions) of the model should be identical. But the differences between the attachment J now provided, and the original table 7.1, indicate that this is not the case.

When the measured soil concentrations are entered into the model (in the attachment D given in the Further Information), the resulting intakes are largely those shown in table 5.1 of the original appendix 6.4 (apart from the errors noted by Dr Johnston and the additional errors I have noted above). However, when the predicted soil concentrations including the deposition from incinerator emissions (attachment H) are entered into the same model (in attachment J in the Further Information), the modelled uptakes are *not* those we were given in table 7.1 of the original appendix 6.4 I conclude that there is an unidentified difference in the modelling parameters that produced tables 5.1 and 7.1 in the original appendix 6.4; like is not being compared with like; something is causing the model output to differ by about 5 % between the two conditions. This difference requires explanation, but no explanation has been offered by Indaver; instead the difference has been explained away as a “transcription error”, which it is clearly not.

During the oral hearing, we were told that the modelling that produced tables 5.1 and 7.1 of the original appendix 6.4 was correct, and only the wrong model output files (attachments D and J) had been inserted. We were assured that the correct model output files could and would be provided, to link the soil samples and calculated deposition to the “correct” data shown in tables 5.1 and 7.1 of appendix 6.4.

This has clearly not happened. Indaver has not explained why.

4. Modelling of dioxin-like toxicity uptake

The modelling offered by Indaver of dioxin/furan uptake by the theoretical maximum at risk individual (MARI) is rife with omissions and deviations from the stated

methodology, that all tend in the direction of underestimating the true level of uptake. We will analyse each of these in turn.

The report's author, Dr Callaghan, tells us in the EIS that he follows the internationally recognised HHRAP (Human Health Risk Assessment Protocol) methodology (Appendix 6.4, section 3.0): "The Conceptual Site Model (CSM) was developed, using the methodology presented in the relevant US EPA Modelling Guidance[3]. The US EPA Methodology uses the concept of the MARI (Maximum at Risk Individual) ... The US EPA Methodology was chosen as it includes a mathematical model which allows calculation of average dioxin and furan concentrations over the lifetime of the facility, taking into account the natural processes which affect dioxin and furan concentrations in the soil over time, such as leaching, volatilisation and degradation."

In questioning during the oral hearing, Dr Callaghan modified this somewhat, to say that he had not followed the HHRAP methodology in all respects, because approaches differ slightly in the EU. He mentioned in particular the UK Committee on Toxicity (COT) methodology as an example of the EU approach, and this will be detailed below.

However, Dr Callaghan's approach deviates in important ways both from the HHRAP methodology and the slightly different EU/COT approach he told us he was using, as well as from his own description of the CSM. The points to be considered are:
(a) Choice of soil sample site for baseline modelling: we are told in the description of the CSM that soil from a site "close to the location of maximum deposition" was used in the modelling, but the sample site used is not in fact the closest to the point of maximum deposition; the modelling ignores a closer sampling site that has a higher dioxin/furan concentration.
(b) Ignoring the MARI child uptake by calculation of a 70-year "lifetime" MARI uptake, contrary to HHRAP recommendation and previous practice of the same author.
(c) Omission of any consideration of uptake of a breastfed baby, which is specified as one of the analyses recommended by HHRAP.
(d) Omission of dioxin-like PCBs from the assessment of dioxin-like toxicity, contrary to HHRAP guidance and previous practice of the same author, and contrary to evidence of their contribution to dioxin-like toxicity both at baseline and in emissions from incinerators.
(e) Seriously deficient diet, allowing fewer than 1000 calories/day for an adult and fewer than 500 calories/day for a child.
(f) Omission of major food groups from the diet of MARI, notably all dairy products except milk, as well as omission of fish (a major source of dioxin-like toxicity, which should be included, according to HHRAP).
(g) Lack of consideration of the body burden of a young mother, resulting from the choice of a 70-year "lifetime" averaging period. To be consistent with the EU/UK

[3] The reference given here is ref. 1 of appendix 6.4, a 1999 report from the US EPA. The updated version of this report is available online as "Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities", published by Office of Solid Waste and Emergency Response (5305W), US EPA; ref. EPA530-R-05-006, September 2005, available from www.epa.gov/osw. I will refer to the up-to-date edition of the report here.

COT methodology (as well as relevant to the breastfed baby above) a more appropriate averaging period would be more like 20 years.

Before considering these corrections, it is worthwhile to consider how realistic the MARI model is capable of being: can it represent at the dioxin-like toxicity intake for a self-sufficient smallholder living beside the site of the proposed incinerator? Now that we have a version of attachment D that (broadly) reflects the 2015 soil concentrations, we can do this, simply by comparing the dioxin/furan content of the milk that MARI is consuming with measured concentrations in real Ringaskiddy milk. This is easy to do, since we are given the milk-based intake of each congener, and we are given the milk intake from which this dioxin/furan intake is calculated. The following table shows the calculation.

Congener	Adult uptake	Weight	Adult uptake	Milk intake	Milk content	WHO TEF	Milk content	Milk content	Milk content
	mg/kg/day	kg	mg/day	g/day	mg/g		mg/g TEQ	pg/g TEQ	pg/g TEQ
	(from att. D)				Whole milk		Whole milk	Whole milk	Milk fat
2378TCDD	4.84E-12	60	2.90E-10	243	1.20E-12	1	1.20E-12	1.20E-03	2.99E-02
12378PeCDD	6.72E-12	60	4.03E-10	243	1.66E-12	1	1.66E-12	1.66E-03	4.15E-02
123478HxCDD	7.44E-12	60	4.46E-10	243	1.84E-12	0.1	1.84E-13	1.84E-04	4.59E-03
123678HxCDD	1.58E-11	60	9.48E-10	243	3.90E-12	0.1	3.90E-13	3.90E-04	9.75E-03
123789HxCDD	1.03E-11	60	6.18E-10	243	2.54E-12	0.1	2.54E-13	2.54E-04	6.36E-03
1234678HpCDD	1.74E-10	60	1.04E-08	243	4.30E-11	0.01	4.30E-13	4.30E-04	1.07E-02
OCDD	1.35E-09	60	8.10E-08	243	3.33E-10	0.0003	1.00E-13	1.00E-04	2.50E-03
2378TCDF	8.77E-12	60	5.26E-10	243	2.17E-12	0.1	2.17E-13	2.17E-04	5.41E-03
12378PeCDF	1.59E-11	60	9.54E-10	243	3.93E-12	0.03	1.18E-13	1.18E-04	2.94E-03
23478PeCDF	1.59E-11	60	9.54E-10	243	3.93E-12	0.3	1.18E-12	1.18E-03	2.94E-02
123478HxCDF	3.09E-11	60	1.85E-09	243	7.63E-12	0.1	7.63E-13	7.63E-04	1.91E-02
123678HxCDF	2.45E-11	60	1.47E-09	243	6.05E-12	0.1	6.05E-13	6.05E-04	1.51E-02
234678HxCDF	3.32E-11	60	1.99E-09	243	8.20E-12	0.1	8.20E-13	8.20E-04	2.05E-02
123789HxCDF	8.15E-12	60	4.89E-10	243	2.01E-12	0.1	2.01E-13	2.01E-04	5.03E-03
1234678HpCDF	1.74E-10	60	1.04E-08	243	4.30E-11	0.01	4.30E-13	4.30E-04	1.07E-02
1234789HpCDF	1.90E-11	60	1.14E-09	243	4.69E-12	0.01	4.69E-14	4.69E-05	1.17E-03
OCDF	1.50E-10	60	9.00E-09	243	3.70E-11	0.0003	1.11E-14	1.11E-05	2.78E-04
Total									2.15E-01
									=0.215 pg/g

This value of 0.215 pg/g milk fat in MARI's cow's milk can be compared easily with the measured concentrations of dioxins and furans in Ringaskiddy milk, as measured by the EPA. Based on the EPA's "Dioxin Report 2012"[4] (page 42), dioxin/furan content in Ringaskiddy milk is 0.21 pg/g milk fat. We can therefore conclude that the transfer of dioxins and furans from soil, to plant, to cow, and finally to milk, is modelled well by the HHRAP approach. (It is worth mentioning at this point, however - and I will come back to this later - that the above table derived from attachment D does not include the dioxin-like PCB content of milk, which is an important component of total baseline dioxin-like toxicity; the EPA's report shows

[4] EPA - "Dioxin Report 2012"
https://www.epa.ie/pubs/reports/other/dioxinresults/Dioxin%20Report%202013_web.pdf

that this is 0.22 pg/g milk fat, slightly more than the dioxin/furan content. PCBs are considered in point 4 below.)

Dr Callaghan was at pains to point out during the oral hearing the unrealistic nature of the MARI model. If that were really the case, one would wonder how it could possibly be of value in assessing the baseline level of dioxin-like toxicity around Ringaskiddy, and why an engineer advising the applicant would have chosen such an unrealistic model. The calculation above shows that, on the contrary, the basic HHRAP approach (as implemented in the RISC-HUMAN software) is robust as a predictor of dioxin/furan content of real Ringaskiddy milk.

Cows' milk is a widely used proxy for environmental dioxin-like toxicity; this is the reason why the EPA's long-standing programme of milk sampling was set up. This also implies that the dioxin/furan content of Ringaskiddy beef is probably also well represented in this model, since both it and the milk dioxin content reflect the animal's body burden. Similarly, the plant dioxin/furan content is probably also accurate, since that is what determines the cow's body burden. These pathways constitute by far the greatest fraction of the total dioxin-like toxicity to which a person is exposed.

Although the HHRAP model is apparently a robust predictor of the dioxin/furan contents of the food groups in MARI's diet, this does not mean that Dr Callaghan's predicted baseline human uptake of 0.29 pg/kg/day is in any way accurate as an estimate of MARI's intake of dioxin-like toxicity. This is to a large degree because (as I will show below) MARI is effectively starving! It is also because Dr Callaghan's adult MARI is not the *maximum* at risk individual; he has not given us the intake of a child, of a breastfed baby, or of a young mother whose foetus is the basis for internationally accepted judgements of tolerable weekly intake. Dr Callaghan also ignores PCBs on this occasion, although on a previous occasion at the same site he did consider that PCBs should be included in the baseline intake. In summary, the MARI intake reported by Dr Callaghan is wildly inaccurate because his work is not consistent with basic human physiology, with the HHRAP or COT methodologies, or even with his own previous practice.

I will present below a re-calculation of the MARI uptake of dioxin-like toxicity, correcting the multiple underestimates in Dr Callaghan's calculation. My starting point is the value given for "lifetime" MARI dioxin/furan intake in the revised version of appendix 6.4, which is **0.29 pg/kg/day TEQ** (table 5.1 of the revised appendix 6.4, which is document 02 of the further information).

(It should be noted that I questioned Dr Callaghan on some of the points below on the last day of the oral hearing, and presented values for dioxin/furan intake that were based on the 2008 attachment D, which was the only reliable model file we had at the time. The calculation below is based on the revised attachment D and therefore the numbers involved are different. The points below are also more complete, because I have now had a little more time for a more detailed analysis than was possible before and during the oral hearing.)

(a) Choice of sample site

The sample site chosen for the baseline of the MARI modelling was intended to be "close to the site of maximum deposition", as stated in Appendix 6.4 of the EIS: "A monitoring survey conducted by AWN found the background soil dioxin concentration in the immediate vicinity of the Ringaskiddy Waste to Energy site in the area likely to be the close to the location of maximum deposition was Sampling Site E, which is located on the high ground adjacent to the Ringaskiddy Waste to Energy Plant site. The measured PCDD/F Concentration for this site was 0.3 ng/kg I-TEQ. It was proposed to use this concentration to define the baseline dioxin exposure for the MARI."

It should be noted that the last time a soil sample was actually taken from a sampling site identified as "Sampling Site E" was in 2001, when the eight sample sites used were labelled A - H. In 2015 the sample sites were designated with numbers followed by the letter "A". Appendix 6.3 of the EIS (Insert 2.1) shows the correspondence between the sample site designations used in 2001 and 2015: sampling site E (2001) corresponds to site 4A (2015). The soil concentration stated in the 2016 EIS at this site is 0.310 ng/kg TEQ PCDD/F (appendix 6.3, insert 4.1), which is given as 0.3 ng/kg TEQ in Appendix 6.4 (page 9).

Aerial photographs in appendix 6.3 show the positions of the sampling sites; site 4A is shown on page 37. It is within the circular wall bounding the Martello Tower, on the hilltop overlooking the proposed incinerator site. It covers about 2/3 of the area enclosed by the wall, to the west and south of the tower.

There is another sample site immediately adjacent to this one: sample 3A (aerial photo on page 38 of appendix 6.3). It is also within the wall surrounding the Martello Tower, to the north-east of the tower, and its soil concentration is 0.680 ng/kg TEQ PCDD/F (appendix 6.3, insert 4.1), about 2.2 times higher than the soil concentration at site 4A.

The aerial photographs taken from appendix 6.3 of the EIS are attached as Fig. 1.

Importantly, sample site 3A (the site with the *higher* concentration) is *closer* to the point of maximum dioxin/furan deposition than site 4A; but site 4A, the more distant site with the lower soil concentration, was the one that was used in the MARI modelling.

The point of maximum deposition is given in appendix 8.4 of the EIS (Table A8.89) as (547900, 5742150). The note below the table says that these are National Grid coordinates but they are not. During the oral hearing I had some difficulty in establishing what coordinate system had been used, because Dr Edward Porter, who had prepared appendix 8.4, gave a wrong answer to my question on the topic. Only when Dr Porter was himself told by another questioner that the coordinate system was UTM (Universal Transverse Mercator) did he confirm that this was in fact the coordinate system that had been used. I have converted the UTM coordinates for this point to latitude and longitude using a standard online tool[5] to allow it to be plotted

[5] http://www.engineeringtoolbox.com/utm-latitude-longitude-d_1370.html

using Google Earth, and have measured the distances from that point to the estimated midpoints of the two sampling sites 3A and 4A (Fig. 2). This establishes that site 4A (as used in the MARI model) is not in fact the closest sample site to the point of maximum deposition, and that site 3A is closer.

No justification is given for choosing the more distant site, and this choice contradicts what we are told in the description of the site model. The only obvious advantage (from Indaver's point of view) in choosing the more distant site is that it gives a substantially lower value for the MARI dioxin/furan uptake. The logic of the site model would require that the site closer to the point of maximum deposition be chosen, i.e. site 3A.

An additional reason to choose site 3A over site 4A would be simply that the methodology requires that we assess the uptake of the *maximum* at risk individual in the presence of the modelled deposition from the incinerator. Given a choice between two sampling sites for the baseline measurement, the maximum at risk individual will be the one living with the higher baseline concentration. This is important because, contrary to what was suggested by Dr Callaghan at the oral hearing, dioxin and furan levels are not diffusely and evenly distributed around Ringaskiddy (or anywhere else). A quick look at insert 4.1 of appendix 6.3, which gives the soil concentrations of dioxins/furans and of dioxin-like PCBs around the harbour, will show how wide this variation can be. For meteorological reasons explained by other observers at the oral hearing, it is not surprising that the highest levels tend to be recorded at elevated sites.

It should be noted that, although the values in insert 4.1 of appendix 6.3 differ by a factor of 2.2, this is a difference in the "lower bound" values ("lower bound" means that when a congener is not detected, its concentration is entered as zero). In the MARI modelling the "upper bound" values are used (where a congener is not detected, its concentration is entered as the limit of detection of the analysis method). The upper bound values are 0.480 ng/kg (site 4A) and 0.750 ng/kg (site 3A). The upper bound values thus differ by a factor of 1.56 (i.e. the soil concentration of dioxins and furans at site 3A is 56 % higher than at site 4A).

Modelled dioxin/furan uptake from ingestion (by far the major route) is very closely proportional to the soil concentrations in the HHRAP model, as can be seen from the equations used in the model (ref. 1, equations 5-20, 5-22 and 5-24). We can thus closely approximate the uptakes that would be modelled from sample site 3A by multiplying the reported dioxin/furan intake based on site 4A (0.29 pg/kg/day) by the ratio of the soil concentrations at the two sites (1.56).

If the sampling site closer to the point of maximum deposition (3A) had been used in the MARI modelling, as provided for in the site model described in the EIS, instead of the more distant site 4A, the calculated dioxin/furan intake based on 70-year lifetime average would thus be increased by 56 %, from 0.29 pg/kg/day to **0.45 pg/kg/day** TEQ.

However, the 70-year lifetime average is not the one recommended by either the HHRAP or the COT methodology, as we will show in the next sections.

(b) Inappropriate choice of 70-year lifetime exposure as the exposure duration: disregard of child's intake

The choice of a 70-year lifetime average is inappropriate for two reasons. Firstly, for a non-carcinogenic endpoint (on which the EU and WHO "tolerable" intakes of dioxin-like compounds are based), the average should be taken over the period of exposure, and for a child that is the duration of childhood, considered in HHRAP to be six years. Secondly, the UK COT (and EU) approach considers the exposure of a foetus via its mother's body burden, which is related to an average exposure over her lifetime so far. Since it is unusual to have one's first child at age 70, the "lifetime" exposure period for this purpose needs to be shorter.

The first of these will be considered here, and the second under point 7 below.

It is obvious from attachments D and J that the MARI child's uptake of dioxins and furans is far higher than that of the MARI adult (ranging from 2.2 - 2.8 times higher). The reported values are neither those of the child nor those of the adult, but are a weighted "lifetime average" based on a 70-year lifespan, 6 years of which are as a child. This gives reported values that are strongly weighted to those of the MARI adult.

The choice of exposure duration and of whether to use the lifetime average or the average over the exposure duration is a topic dealt with in the HHRAP manual (ref. 1). Section 2.3.10 (page 2-66) deals with the choice in relation to dioxins and furans. When we are concerned with cancer risk, it is appropriate to use a lifetime average (section 2.3.10.1). Where we are concerned with non-cancer health hazards, the relevant averaging period is the period of exposure: "The pertinent exposure estimate would be the ADD, or Average Daily Dose, experienced over the course of the exposure duration, rather than the LADD, which is this ADD averaged over a lifetime." (ref. 1, section 2.3.10.2).

Exposure duration for different MARI scenarios is considered in ref. 1, section 6.5 and table 6.3. For the "Farmer Child" (the term used for the MARI child in the HHRAP manual), the relevant exposure period is 6 years, the assumed duration of childhood. When the child's uptake is constant, as it is in the MARI model, the averaged uptake over the 6-year period will clearly be identical to the single value given for the child's intake, which can be read from attachment D.

Importantly, the currently accepted WHO and EU guidelines for limits on tolerable intake of dioxins and furans are based on non-carcinogenic effects, principally effects on the reproductive system during early development:
"The WHO consultation recommended a TDI for dioxins and dioxin-like PCBs of 1-4 pg WHO-TEQ/kg based on the NOAEL/LOAELs of those effects considered to be the most sensitive in experimental animals, namely endometriosis, developmental neurobehavioral effects, developmental reproductive effects and immunotoxicity ... Based on the LOAEL from a study showing developmental effects in male rat offspring following repeated subcutaneous administration of TCDD, the SCF established a tolerable weekly intake (TWI) of 14 pg WHO-TEQ/kg bw."[6]

[6] Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (2001)

Because we are dealing with a non-carcinogenic endpoint, it is clear from this that the HHRAP methodology would require the *child's* intake of dioxin-like toxicity to be given priority, because the biological processes that are affected at the lowest levels of dioxin-like toxicity are those happening during early development.

Dr Callaghan, who produced this report for Indaver, also produced a similar report[7] for their EPA licence application at Carranstown in County Meath. Figs. 3a and 3b show pages taken from that report. Curiously, in that report Dr Callaghan *does* report the MARI child's intake separately from the adult, and does not mention a lifetime average. The inconsistency of his approach is worrying, and makes it hard to see why the child's intake was not also considered in the current report - although it has to be acknowledged that the much lower lifetime average value is more conducive to Indaver's aims.

The table below shows in columns 3-5 the values given in the uptake tables of the revised attachment D (document 04 in the further information). Columns 6-8 show these values converted to TEQ. The rightmost column shows the values reported in table 5.1 of the revised appendix 6.4 (document 02 in the further information). As noted in section 3, these values differ from the version of table 5.1 presented at the oral hearing. (Of course these values are derived from sample site 4A.)

"Statement on the tolerable daily intake for dioxins and dioxin-like polychlorinated biphenyls" Ref COT/2001/07. https://cot.food.gov.uk/sites/default/files/cot/cot-diox-full.pdf Note that "NOAEL" and "LOAEL" stand for "no observable adverse effect level" and "lowest observable adverse effect level", and "SCF" is the Scientific Committee on Food of the EU. TDI is tolerable daily intake.

[7] http://www.epa.ie/licences/lic_eDMS/090151b2800547ee.pdf

Congener	WHO TEF	child	adult	lifetime	child	adult	lifetime
		mg/kg/d	mg/kg/d	mg/kg/d	pg/kg/d	pg/kg/d	pg/kg/d
					TEQ	TEQ	TEQ
2378TCDD	1	7.13E-11	2.95E-11	3.31E-11	7.13E-02	2.95E-02	3.31E-02
12378PeCDD	1	1.70E-10	7.65E-11	8.45E-11	1.70E-01	7.65E-02	8.45E-02
123478HxCDD	0.1	1.51E-10	6.59E-11	7.32E-11	1.51E-02	6.59E-03	7.32E-03
123678HxCDD	0.1	3.21E-10	1.40E-10	1.56E-10	3.21E-02	1.40E-02	1.56E-02
123789HxCDD	0.1	2.09E-10	9.12E-11	1.01E-10	2.09E-02	9.12E-03	1.01E-02
1234678HpCDD	0.01	3.14E-09	1.35E-09	1.50E-09	3.14E-02	1.35E-02	1.50E-02
OCDD	0.0003	2.48E-08	1.06E-08	1.18E-08	7.44E-03	3.18E-03	3.54E-03
2378TCDF	0.1	8.12E-11	2.89E-11	3.34E-11	8.12E-03	2.89E-03	3.34E-03
12378PeCDF	0.03	1.46E-10	5.29E-11	6.09E-11	4.38E-03	1.59E-03	1.83E-03
23478PeCDF	0.3	1.46E-10	5.29E-11	6.09E-11	4.38E-02	1.59E-02	1.83E-02
123478HxCDF	0.1	5.28E-10	2.24E-10	2.50E-10	5.28E-02	2.24E-02	2.50E-02
123678HxCDF	0.1	4.20E-10	1.78E-10	1.99E-10	4.20E-02	1.78E-02	1.99E-02
123789HxCDF	0.1	1.39E-10	5.92E-11	6.61E-11	1.39E-02	5.92E-03	6.61E-03
234678HxCDF	0.1	5.78E-10	2.46E-10	2.74E-10	5.78E-02	2.46E-02	2.74E-02
1234678HpCDF	0.01	3.03E-09	1.29E-09	1.44E-09	3.03E-02	1.29E-02	1.44E-02
1234789HpCDF	0.01	4.00E-10	1.75E-10	1.95E-10	4.00E-03	1.75E-03	1.95E-03
OCDF	0.0003	2.92E-09	1.26E-09	1.41E-09	8.76E-04	3.78E-04	4.23E-04
Total					6.06E-01	2.58E-01	2.88E-01

Correcting for the use of the more appropriate sample site 3A instead of 4A, the child's intake would be (0.606 x 1.56) pg/kg/day, i.e. **0.945 pg/kg/day**, and the adult's intake would be (0.258 x 1.56) pg/kg/day, i.e. **0.403 pg/kg/day**

(c) Omission of any consideration of the intake of a breastfeeding baby

Here we can only note that no attempt has been made to assess the dioxin/furan intake of a breastfed baby with MARI as its mother. This is unfortunate for two reasons. Firstly, the basic biology of dioxins and furans dictates that the individual receiving the greatest dose of dioxins and furans is a breastfed baby, because a substantial part of the mother's accumulated body burden of these compounds is transferred into the breast milk. Secondly, the HHRAP protocol, which Dr Callaghan tells us he used, specifically provides that infant exposure via breast milk should be assessed: "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." (ref. 1, page 4-14).

(d) Omission of dioxin-like PCBs from the analysis, contrary to HHRAP guidance and to previous practice of the same author

As well as dioxins and furans, another class of persistent organic pollutants with serious effects on human health is the dioxin-like polychlorinated biphenyls (DL-PCBs). These need to be considered whenever the effects of dioxins are under discussion, because dioxins, furans and dioxin-like PCBs all act on the same receptor, a cell surface molecule called AHR (the aryl hydrocarbon receptor). DL-PCBs are not mentioned at all in the MARI modelling, and when questioned about this on the last day of the oral hearing Dr Callaghan stated that this was because the proposed incinerator would not be a source of PCBs (he cited the US EPA as a source for this claim).

In reality, the US EPA states exactly the opposite of this in their published HHRAP guidance (see below). But even if it were true that waste incinerators do not emit PCBs, these substances form part of the baseline exposure to dioxin-like toxicity so need to be included in the MARI analysis. Dr Callaghan acknowledged this in his earlier report for the EIS of the Indaver Ringaskiddy EPA licence application: "...PCB intake has not been modelled as part of this study, as the US EPA advise that PCBs have not been detected in the emissions from waste to energy facilities and the proposed facility will therefore have no impact on the PCB fraction of PCDD/F and PCDD/F like compounds. However studies have shown that PCB intake can contribute approximately 45-55 % of dietary exposure of PCDD/F like compounds ... (note that there is no predicted PCB emission from the proposed facility and that PCB component of the predicted exposure is the background component only)." A scan of this page is included as Fig. 4.

As mentioned above, the US EPA states in the HHRAP manual (Ref. 1) that waste incinerators *do* emit PCBs. In the section on DL-PCBs (section 2.3.9, pages 2-61 and 2-62), it states:
"Because of evidence that PCBs can be emitted from combustion sources regardless of feed characteristics, and considering the significant toxicity of PCBs, we ... recommend automatically including PCBs as COPCs for combustors that burn ... highly variable waste streams such as municipal and commercial wastes (for which PCB contamination is a reasonable assumption) ... An increasing body of information supports the likelihood that PCBs may be emitted as by-products of burning, regardless of PCB contamination in the combustor feed ... In most cases, PCBs were found in the stack even when there were no PCBs in the combustor feed. Overall, PCB emissions exceeded dioxin and furan emissions by approximately a factor of 20, and this trend appeared to hold over five orders of magnitude in dioxin and furan emissions."

We also have direct evidence from the UK, in the form of an annual environmental report from the Veolia Sheffield incinerator[8]. This shows substantial emission of DL-PCBs, at about two-thirds the level (in bird TEQ terms) or one-sixth the level (in human TEQ terms) of dioxins and furans. In terms of estimating the deposition from the incinerator that ought to be incorporated into the MARI model (attachment H,

[8] http://www.ukwin.org.uk/files/pdf/sheffield_2012.pdf

attachment J, and table 7.1), this is clearly a very significant component and it is not acceptable to simply ignore it or maintain that it will not happen.

(In passing it could be noted that these high DL-PCB levels expressed in terms of bird TEQ may well be relevant in consideration of the state of the Cork Harbour SPA and its protected species, and the Appropriate Assessment of this application. As noted elsewhere in this report (footnote 15), PCB levels in Cork Harbour have been identified as being unusually high. As far as I can tell, PCBs have not been considered in the PCDD/F risk analysis in the NIS (Appendix 15), which may make Appropriate Assessment impossible.)

Not including DL-PCBs in the analysis of incinerator emission, and especially not considering their contribution to baseline dioxin-like toxicity, is thus an important omission; and, as shown, not including it in consideration of background uptake is in contradiction of Dr Callaghan's previous practice.

The contribution of DL-PCBs to dioxin-like toxicity estimated by Dr Callaghan in 2001 was 45-55 %; roughly, we can say that they contribute about the same amount as dioxins and furans combined. This is borne out by EPA analysis of milk from Ringaskiddy[9], which shows (Table 5, page 42) that PCDD/F content in milk was 0.21 pg/g TEQ fat, and that of DL-PCBs was 0.22 pg/g TEQ fat.

We must therefore add to the value already arrived at for the MARI child, 0.945 pg/kg/day for dioxin/furan exposure, an equal amount of 0.945 pg/kg/day for DL-PCB exposure, to arrive at a value of **1.89 pg/kg/day** for total baseline exposure of the MARI child so far. For the adult, the total exposure would be 0.403 pg/kg/day of dioxins/furans plus 0.403 pg/kg/day of dioxin-like PCBs, giving a total of **0.806 pg/kg/day**.

These values must however be modified to correct for the very serious dietary deficiency of MARI, in the next section.

(e) Seriously deficient diet, allowing fewer than 1000 calories per day for an adult and fewer than 500 for a child
(f) Omission of major food groups from the diet of MARI, notably all dairy products except milk, and omission of fish.

The diet of MARI (adult) consists of only the following:
Leafy Vegetables = 0.118 kg/day
Tuberous vegetables = 0.225 kg/day
Meat = 0.179 kg/day
Milk = 0.243 kg/day

The child's diet is considered by Dr Callaghan to be exactly half of the adult's in each category.

[9] EPA - "Dioxin Report 2012"
https://www.epa.ie/pubs/reports/other/dioxinresults/Dioxin%20Report%202013_web.pdf

These food intakes are derived from a national food intake survey[10], and it is easy to reconstruct the food groups that have been used (the complete table is shown in figs. 5a-b). From this we can reconstruct the calorie intake of the MARI adult, as follows. I will use the adult values for the moment, because these were used in the IUNA survey. (The child's intake in Dr Callaghan's model is simply half of the adult's.)

Leafy vegetables:

Food item & code	Mean intake (g/day)	kcal per 100 g	Total kcal
28 - Vegetable & pulse dishes	17	155 (based on baked beans)	26
29 - Peas, beans & lentils	23	98 (average of peas & lentils)	23
30 - Green vegetables	14	15 (based on lettuce)	2
31 - Carrots	15	41	6
32 - Salad vegetables	24	15 (based on lettuce)	4
33 - Other vegetables	25	41 (based on carrots)	10
Total	118		71

Tuberous vegetables:

Food item & code	Mean intake (g/day)	kcal per 100 g	Total kcal
25 - Potatoes	158	77	122
26 - Processed and homemade potato products	7	77 (as potato)	5
27 - Chipped, fried and roasted potatoes	59	312	184
Total	224		311

Meat (food items 43-55 in the IUNA table):

Food item & code	Mean intake (g/day)	kcal per 100 g	Total kcal
43-55 - Meat	179	250 (as beef)	448

[10] IUNA North/South Ireland Food Consumption Survey: Food and Nutrient Intakes, Anthropometry, Attitudinal Data and Physical Activity Patterns, published by the Irish Universities Nutrition Alliance and The Food Safety Promotion Board, Abbey Court, Lower Abbey St, Dublin, 2001; http://www.iuna.net/docs/NSIFCSummary.pdf

Milk (food items 10-12 in the IUNA table):

Food item & code	Mean intake (g/day)	kcal per 100 g	Total kcal
10-12 - Milk	243	64.2	156

The total energy intake based on the above categories is thus:

	kcal/day
Leafy vegetables	71
Tuberous vegetables	311
Meat	448
Milk	156
Total for adult	**986**
Total for child	**493**

According to the UN Food and Agriculture Organisation (FAO), the energy needs of a subsistence farmer would be 3300 kcal/day[11], over three times the intake allowed to MARI. A six-year-old child's normal energy intake is 1600 kcal/day (girls) or 1800 kcal/day (boys)[12], also over three times the MARI child's intake.

It is also noticeable that, of 67 food types in the IUNA survey, the MARI adult diet contains only 25 of them. Among the large contributors of dioxin-like toxicity in the average Irish diet, the likely consumption of butter, cheese and other dairy items has been entirely omitted. It is inconceivable that a subsistence farmer like MARI, with a cow, would consume only the average Irish amount of milk, and no butter, cheese, cream or yogurt at all.

For a subsistence farmer with high energy needs, the omission of food types 1-9 (the carbohydrate group) would also be a serious omission.

In addition, fish is certain to be a part of the diet, for a subsistence farmer who does not want to kill their cow as their major source of protein, but who lives within a few metres of the sea. This is also explicitly recommended by the HHRAP protocol: "We don't usually recommend the ingestion of fish exposure pathway for the Farmer exposure scenario. However, as indicated in the notes to Table 4-1, we do recommend that you consider evaluating the fish ingestion pathway if regional or site-specific exposure setting characteristics (e.g., presence of ponds on farms or ranches that support fish for human consumption) are identified that warrant consideration." We suggest that the presence of the sea next to the site of the proposed incinerator would be an appropriate "site-specific exposure setting characteristic" that would indicate that fish has to be considered as part of the diet. According to a recent study, fish consumption contributes 39 % of total intake of dioxin-like toxicity in the average Irish diet[13].

[11] http://www.fao.org/docrep/007/y5686e/y5686e07.htm

[12]https://www.cnpp.usda.gov/sites/default/files/usda_food_patterns/EstimatedCalorieNeedsPerDayTable.pdf

[13] Tlustos et al "Exposure of the adult population resident in Ireland to dioxins and PCBs from the diet" Food Additives and Contaminants Part A 31(6):1100-1113 (2014)

I will consider below the implications of adding each of these food groups. It should be noted that, for brevity and to save time, this still omits important sources of dioxin-like toxicity in the Irish diet, such as eggs and vegetable oils/nuts. Together these contribute about 11 % of total intake of dioxin-like toxicity (see footnote 13). The final value we will arrive at is therefore underestimated by roughly this proportion.

(i) Dairy products apart from milk

Consumption of these for MARI can be based on the IUNA data (footnote 10).

Food item & code	Mean intake (g/day)	kcal per 100 g	Total kcal	Fat %	Fat (g)
13 - Creams	2	350	7	10	0.2
14 - Cheeses	12	400	48	32	3.8
15 - Yogurts	16	62	10	4	0.64
16 - Ice creams	7	200	14	13	0.91
17 - Puddings/ desserts	16	100	16	2.8	0.45
18 - Milk puddings	6	100	6	2.5	0.15
20 - Butter	6	720	43	80	4.8
21 - Low fat spreads	4	720 (assume MARI would eat butter)	29	80	3.2
22 - Other spreading fats	12	720 (butter)	86	80	9.6
Total	81		259		23.75

In the original MARI modelling, the only dairy product allowed was 243 g of milk per day. Full fat milk contains about 4 % fat, i.e. the MARI adult has an intake of 9.72 g milk fat. As the table above shows, 23.75 g of milk fat has been omitted from the calculation. We can add back the missing fraction by adding (23.75/9.72) of the current dairy-based dioxin/furan intake. Since the child's intake is still assumed to be exactly half of the adult's, the same proportionality factor will apply to the child as to the adult.

(ii) Carbohydrate food group (food types 1-9)

The MARI diet in the EIS contains no grain-based carbohydrates (rice, breads, cereals, biscuits). These provide a large fraction of the calories in the Irish diet. Since MARI is constrained to eat only home-produced food, the most likely replacement (plausible in the context of our history) would be potatoes (growing grains would need more land than MARI has). We can therefore increase the intake of potatoes to replace the calories not obtained from these grains.

Considering these 9 food items in the IUNA survey:

Food item & code	Mean intake (g/day)	kcal per 100 g	Total kcal
1 - Rice/pasta/flours/grains/starches	20	131	26
2 - Savouries	24	470	113
3 - White breads	78	265	207
4 - Wholemeal breads	45	247	111
5 - Other breads	15	265	40
6 - Ready to eat breakfast cereals	19	379	72
7 - Other breakfast cereals	16	379	61
8 - Biscuits	14	474	66
9 - Cakes/pastries/buns	17	436	74
Total	248		770

To replace these 770 calories with potatoes (77 kcal/100 g), MARI would need to eat 1 kg of potatoes per day. The intake of tuberous vegetables thus increases from 224 g/day to 1224 g/day, and of total vegetables from 342 g/day to 1342 g/day.

We are not given a separate analysis of the dioxin content of leafy vs tuberous vegetables, so all we can do is to increase the vegetable fraction of the dioxin-like toxicity intake by (1000/342). Again, the same proportionality factor applies to the child as to the adult, as the child's food intake is assumed for the moment to be half of that of the adult.

Calorie intake for the MARI adult is increased by 259 kcal a day (non-milk dairy) and 770 kcal/day (carbohydrate) to a total of 2015 kcal/day (1008 kcal/day for the child). This is still an inadequate diet, but I will correct this later.

To determine the effect these corrections would have on total uptake, we can simplify by extrapolating from one congener to the others, so I will consider 2,3,7,8-TCDD as an example. The following uptake table is drawn from attachment D.

Exposure per route (mg/(kg.d))

Exposure route	Child	Adult	Lifelong
inhalation indoor air	9.73E-17	2.39E-17	3.02E-17
inhalation outdoor air	1.21E-17	3.81E-17	3.58E-17
ingestion soil	6.10E-13	5.08E-14	9.88E-14
dermal contact soil	2.61E-14	7.78E-14	7.33E-14
inhalation soil	9.64E-16	5.67E-16	6.01E-16
ingestion milk	2.13E-11	4.84E-12	6.25E-12
ingestion meat	3.66E-11	1.82E-11	1.98E-11
ingestion vegetables	1.28E-11	6.37E-12	6.92E-12
Total exposure	7.13E-11	2.95E-11	3.31E-11

To this we should add the missing dairy fat (23.75 / 9.72 x current dairy fat intake), and the missing vegetable intake (1000 / 342 x current vegetable intake):

missing dairy fat	5.20E-11	1.18E-11	1.53E-11
missing vegetable	3.74E-11	1.86E-11	2.02E-11
Revised total exposure	1.61E-10	5.99E-11	6.86E-11
% increase in total dioxin/furan intake	126 %	103 %	107 %

Adding the missing dairy fat and carbohydrate (i.e. tuberous vegetable) consumption has increased the child's total dioxin/furan intake by 126 %. Since this correction will affect all congeners in a broadly similar way, we can approximate by using the same correction factor for them all, and simply add 126 % of the total intake. The total intake for the MARI child thus increases from 1.89 pg/kg/day to **4.27 pg/kg/day**. The adult intake similarly increases by 103 % from 0.806 pg/kg/day to **1.64 pg/kg/day**.

This assumes, of course, that dioxin-like PCBs will behave in the same way and be subject to the same correction factors as the dioxins and furans. Because of the complete absence of any consideration of PCBs in Dr Callaghan's MARI modelling, we have no information on which to base any other approach. These substances are chemically similar to the dioxins and furans, but bioaccumulation can be greater; the above values may therefore underestimate the PCB component of the dioxin-like toxicity.

It should be noted at this stage that the EU tolerable weekly intake corresponds to 2 pg/kg/day; we have already exceeded this by over twofold for the MARI child, and the MARI child is still eating a seriously inadequate diet.

(iii) Fish

The IUNA survey indicates a total consumption of 26 g/day of fish (food items 41 and 42 combined). Because fish is entirely omitted from the EIS, we have to rely on other sources for information on its dioxin/furan content. We can obtain this from a document published by the Food Safety Authority of Ireland, and cited by Indaver's medical expert in appendix 6.2 of the EIS[14].

This report gives (Table 5) the mean levels of dioxins and DL-PCBs in Irish fish from various sources. These range from 1.08 pg/g TEQ (wild salmon) to 4.01 pg/g TEQ (farmed salmon). It should be noted that fish from Cork Harbour would be expected to have a relatively high level of PCBs, as Cork Harbour is listed as being in the highest category of concern for PCBs[15]. Levels are probably closer to those of farmed than wild salmon, given that farmed salmon are kept in calm inlets from the sea like Cork Harbour which may predispose to accumulation rather than washout of pollutants. In view of these considerations, we may use the level quoted for farmed trout (1.36 pg/g) which is probably a conservative estimate; it is quite likely that the level found in farmed salmon is nearer to the truth.

Daily intake of dioxin-like toxicity from fish would therefore be of the order of (1.36 pg/g x 26 g) = 35.4 pg TEQ, i.e. 0.59 pg/kg body weight (MARI's body weight is 60 kg). The MARI child's consumption is half of the adult's, and the child's body weight is assumed to be 15 kg (HHRAP, footnote 3, page 6-6); the child's intake from fish is thus 17.7 pg/day divided by 15 kg = 1.18 pg/kg/day. Total intake for the MARI child including fish is therefore increased from 4.27 pg/kg/day to **5.45 pg/kg/day** and for the adult from 1.64 pg/kg/day to **2.23 pg/kg/day**.

[14] Food Safety Authority of Ireland "Report on waste incineration and possible contamination of the food supply with dioxins" https://core.ac.uk/download/pdf/15573033.pdf
[15] http://www.epa.ie/wfdstatus/TraC/TraC_Chemistry.pdf, table 5

The adult's dioxin-like toxicity intake from fish is 26.5 % of the total, somewhat below the average for Ireland of 39 % (see footnote 13). This may reflect both the rather conservative value used for the dioxin-like toxicity content of fish, and the possibly higher than average intake that MARI already has from vegetables, dairy products and meat.

Calorie intake would be increased by the fish consumption. Taking a value for fish of 305 kcal/100 g (79 kcal for 26 g), the MARI adult energy intake would increase from 2015 kcal/day to 2094 kcal/day (1047 kcal/day for the MARI child).

(iv) Total energy intake

According to the US Department of Agriculture[16], energy intake for an active 6-year-old child is about 1800 kcal/day (male) or 1600 kcal/day (female). We will take the mean of these, as the MARI child is equally likely to be one or the other. For an adult subsistence farmer, the UN Food and Agriculture Organisation estimates energy requirement as 3300 kcal/day[17].

After including the major missing food items in the MARI child's diet, we are still deficient by about 650 kcal/day (intake of 1047 compared to requirement of 1700 kcal/day). As any parent knows, children eat more than would be expected based only on their size, because they are growing and usually more active than adults. It is not surprising that a diet based on half of the adult intake is too low for the MARI child. Assuming that the mix of foods does not differ greatly from the one we have arrived at so far, which is broadly based on the average Irish diet, we should simply increase the total calorie intake (and the total intake of dioxin-like toxicity) by a fraction to reflect the actual energy intake of 1700 kcal/day.

The multiplier is (1700 / 1047) = 1.62, i.e. we should increase all food groups by 62 %. This will increase dioxin-like toxicity intake by 62 %. The MARI child's intake of dioxin-like toxicity will then increase from 5.45 pg/kg/day to **8.83 pg/kg/day.**

For the adult, who up to now has been receiving 2094 kcal/day, the multiplier is (3300/2094) = 1.58, so the MARI adult's total intake would now be increased from 2.23 pg/kg/day to **3.52 pg/kg/day**

(g) Lack of consideration of the body burden of a young mother

During the oral hearing, Dr Callaghan argued against the HHRAP methodology that he stated in the EIS he had used. He stated that he had used a 70-year lifetime average exposure because the EU approach, as expressed in the UK Committee on Toxicity statement on tolerable weekly intake[18], is based on a body burden approach. It is worthwhile to consider the implications of taking that approach.

The COT statement includes this: "80. We recommend that a tolerable daily intake of 2 pg WHO-TEQ/kg bw per day is established, based upon effects on the developing

[16]https://www.cnpp.usda.gov/sites/default/files/usda_food_patterns/EstimatedCalorieNeedsPerDayTable.pdf
[17] http://www.fao.org/docrep/007/y5686e/y5686e07.htm
[18] https://cot.food.gov.uk/sites/default/files/cot/cot-diox-full.pdf

male reproductive system mediated via the maternal body burden." The body burden in humans is considered to be accumulated over a period of 15-30 years (section 62).

It is clear from this that the 70-year lifetime average assumed by Dr Callaghan when arguing in favour of the EU/COT body burden approach is far too long: few women wait until age 70 to have their first child! If we use a lifetime average exposure, as advocated by Dr Callaghan, it should clearly be the lifetime average exposure of a young mother (e.g. 20 years old).

Dr Callaghan calculates the 70-year lifetime average (erroneously stated in appendix 6.4 as a 66-year average) based on 6 years of childhood, which is derived from the HHRAP. The first six months of that is spent as a baby, and it is recommended that babies during this time are exclusively breastfed. So the maternal 20-year lifetime average would be made of 6 months as a breastfed baby, 5.5 years as a child, and 15 years as an adult. The daily intakes for the MARI child and MARI adult are calculated above; the MARI mother's intake as a breastfed baby can only be estimated based on the survey data reported by FSAI (see footnote 14).

Average Irish breastmilk (see footnote 14, table 6):
Median dioxin/furan content 6.91 pg/g fat TEQ
Median dioxin-like PCB content 4.66 pg/g fat TEQ
Total dioxin-like toxicity 11.6 pg/g fat TEQ (at around 1 month from birth)

This value is entered into the following table, in the "Dioxin content" column, column 5. at one month from birth. It is adjusted for the fall in dioxin content of breastmilk after birth, using data from Lorber & Phillips (2002). Other data in the following table are drawn from Mannetje et al (2014)[19] and Lorber & Phillips (2002)[20].

Time point	Body weight	Milk intake	Milk fat (4 %)	Dioxin content	Dioxin intake	Dioxin intake
	kg	ml/day	g/day	pg/g	pg/day	pg/kg/day
Birth	3.3	690	27.6	12.6	348.5	105.6
1 month	4.3	690	27.6	11.6	319.3	74.3
2 months	4.6	690	27.6	10.5	290.6	63.2
3 months	6	770	30.8	9.5	291.7	48.6
4 months	6.7	770	30.8	8.4	259.1	38.7
5 months	7.4	770	30.8	7.4	227.0	30.7
6 months	7.9	770	30.8	6.3	194.5	24.6
Mean						55.1

The mean intake over 6 months of breastfeeding, based on average Irish breastmilk, is thus about 55.1 pg/kg/day.

[19] Mannetje et al "Estimated infant intake of persistent organic pollutants through breast milk in New Zealand" New Zealand Medical Journal 127:56-68 (2014)
[20] Lorber & Phillips "Infant exposure to dioxin-like compounds in breast milk" Environmental Health Perspectives 110:A325-A332 (2002)

The lifetime average for 20 year old mother is thus as follows:
Breastfed baby: 55.1 pg/kg/day x 0.5 years
MARI child (see above): 8.83 pg/day x 5.5 years
MARI adult (see above): 3.52 pg/kg/day x 15 years

Average lifetime exposure = **6.45 pg/kg/day**

It should be noted that this lifetime averaging approach somewhat overestimates the actual body burden, because some of the intake as a baby would have been excreted during childhood and adult life. (The COT estimates the relevant period for accumulation of body burden as about 15-30 years).

However, even if the mother's body burden reflected only her intake as an adult (which would clearly be an underestimate), it will still be well above the tolerable level, because the MARI adult intake is very substantially above the EU and COT recommended tolerable weekly intake.

At this point it is worthwhile to look back to where we came from. Dr Callaghan gave us a figure of 0.29 pg/kg/day, by choosing a sample site more distant from the site of maximum predicted dioxin/furan deposition, with lower soil concentrations of dioxins and furans; by basing the calculation on 70-year lifetime average exposure, rather than that of a child or young mother; by ignoring the contribution of dioxin-like PCBs to the baseline intake of dioxin-like toxicity; and by assuming a diet for MARI that was grossly deficient, and lacking in many of the major contributors of dioxin-like toxicity to the MARI (and average Irish) diet.

By correcting for these distortions, we arrive at a value of 8.83 pg/kg/day for the MARI child, which is over 30 times the value cited by Dr Callaghan and over four times the EU "tolerable intake".

Even the MARI adult's intake of 3.52 pg/kg/day is well above the EU tolerable intake.

The body burden of a young mother would be at a level reflecting a daily dose somewhere between the adult dose of 3.52 pg/kg/day and her lifetime average of 6.45 pg/kg/day.

These are likely to be reasonably accurate representations of the true intakes that would be experienced by a self-sufficient smallholder family living beside the proposed incinerator, because (as shown at the beginning of this section) the HHRAP equations appear to give a good estimate of plant dioxin/furan content, of the uptake into the cow, and of the transfer of these substances into milk and meat, for pooled Ringaskiddy milk.

It should be noted that these are the *baseline* exposures to dioxins, furans and dioxin-like PCBs. The analysis above does not include any contribution from the proposed incinerator. We have not been given sufficient data to critically assess the values given for that. All I can say on that point is - if the contribution from the incinerator is expressed in TEQ units that are being added to mass units (as attachment H shows), and since it only includes dioxins and furans (excluding DL-PCBs, which, according

to the US EPA and to the UK incinerator report mentioned above, should be included) -then whatever the incinerator contributes is likely to be far above the level we have been told in the EIS.

However, I would suggest that the existing baseline level of dioxin-like toxicity is so high in the Ringaskiddy area that it is absolutely out of the question that approval could be given for an additional source of dioxins, furans and PCBs, however small its contribution is claimed to be. This is in accordance with the conclusion of the inspector at the 2009 oral hearing for this site, as quoted at the end of the summary of this report (page 7 and footnote 1).

We should instead urgently be exploring ways to reduce this baseline exposure to levels considered tolerable by the EU, or better, the more cautious value of 1 pg/kg/day recommended by the WHO.

My concern on this point is supported by samples taken for the Indaver EIS and reported in appendix 6.3 (Insert 5.5), which show very steep increases in dioxins and furans in the mudflats of the Cork Harbour SPA in 2015 compared to the previous sampling in 2009. This table is shown in Fig. 6; it caused concern when it was discussed at the oral hearing. These real-world measurements suggest that the frighteningly high levels of dioxin-like toxicity revealed by the proper application of the MARI model are mirrored by real increases in dioxin-like toxicity in the Harbour, in contrast to falling levels nearly everywhere else.

5. Personal note

In my career as a working scientist and educator of future scientists, I have striven to maintain and to nurture in my students the values of critical and honest enquiry, and to encourage the highest standards of integrity and rigour in the presentation and discussion of scientific research. I have never reviewed a piece of work that was so full of errors and omissions, and that presented such a distorted result as this modelling study. In particular the near-starvation diet of MARI, and the omission of important food groups with high dioxin content, suggest a desperate need for physiological expertise and advice that was lacking in the preparation of this report, and a surprising lack of simple thought as to MARI's likely diet. The omission of dioxin-like PCBs, which activate exactly the same receptor (the AHR receptor) and cellular pathways as dioxins and furans, suggests a lack of understanding of the basic physiological processes being modelled. While very occasional errors do creep into any piece of complex scientific work, the sheer number and nature of the errors in this piece of work indicate a high degree of carelessness. But some of the discrepancies explained away as "transcription errors" are not credible as such, and I cannot accept that explanation of their origin.
If this is the standard of one part of the Indaver EIS, that I happen to have examined in some detail because it fits my area of expertise, I am left wondering how confident we can be about the rest of it. I can only hope the sections of the Further Information dealing with helicopter safety demonstrate a higher level of academic rigour and accuracy.