Webinar on Dioxins in food and feed | 11 December 2025 | Questions & Answers

This document contains answers to questions received either during registration or live at the Webinar held 11 December 2025 to explain the draft conclusions and provide insights on the principles and methods applied for the draft risk assessment under public consultation.

Questions

1. **I would like to see the feed to food transfer calculations presented.**

The transfer rates (TRs) and bioconcentration factors (BCFs) reported in the draft Opinion are based on transfer studies reported in the open literature. This is presented in the draft Opinion in ANNEX E. In some cases, TRs/BCFs were calculated by EFSA based on reported levels and assumptions shown in the footnotes. Due to time constrains, the Webinar has not touched upon this section of the draft Opinion.

Follow up question: We have published a study with a model on specific transfer of these compounds from feed to food and I will provide a comment on that via the public consultation tool.

If there are relevant references missing, they will be considered for possible inclusion.

2. **Is the modification of the Tolerable Weekly Intake (TWI) only related with the modified Toxic Equivalent Factors (TEFs) or have new studies been taken into consideration which resulted in a decrease of the TWI?**

New studies in the open literature since the publication of the 2018 EFSA Opinion have been reviewed but none of them was considered as the critical study. The change in the WHO-TEFs had both implications for the derivation of the TWI and for the dietary exposure estimates. The change in the TWI reflects the use of a different critical study, i.e. a study with rats, and application of an uncertainty factor. Following the revision of the TEFs by the WHO, the critical epidemiological study in the 2018 EFSA Opinion was deemed unsuitable as the main basis for setting the current TWI, but it was retained as supportive evidence for the new TWI.

3. **Can a TWI be derived for non-dioxin-like (NDL-) PCBs?**

NDL-PCBs were outside the remit of this assessment, that focused on PCDD/Fs and DL-PCBs.

4. **When do you think the final opinion will be published?**

The deadline for adoption of this draft Opinion is 30 April 2026. Once adopted, it is expected to be published in the EFSA Journal in June 2026.

5. **Which are the countries with the highest reported pollution levels about this? In which support or material?**

EFSA does not report on pollution levels in the environment, however it reports on occurrence data in food sampled in the EU, submitted by national food authorities, research institutions, academia, food business operators and other stakeholders. Highest occurrence of PCDD/Fs and DL-PCBs were found in fish liver. Occurrence data are averaged across reporting countries assuming a common European market.

6. **How does the new TWI relate to the previous one when the new TEFs are taken into account?**

The exposure estimates are lower due to the decrease of the WHO_{2022}-TEFs compared to WHO_{2005}-TEFs. However, the TWI has also been lowered, and to an even greater extent due to the decision to use a rat study as the critical one. Overall, this results in a more pronounced exceedance of the TWI.

Follow up question: Is this a more stringent TWI than the one before and if so, by how much?

Both dietary exposure estimates and the TWI have decreased following the application of the new WHO-TEFs, as some congeners appear less toxic than previously assumed. However, for various reasons, the TWI was reduced by a factor of approximately three, which increases the gap between exposure and TWI. Human milk levels suggest that the European population exceeds the TWI to a lesser extent than when comparing the dietary exposure estimates with the TWI, and warrants further investigation whether this is due to kinetic differences between congeners, not included in the PBK modelling. The applied PBK-model was developed for TCDD. Even when adapting the model for other congeners, the question would be which congener pattern should be modelled. Alternatively, such kinetic differences might be taken into account when establishing the TEFs. In the uncertainty section this issue is discussed, considering the difference in the risk characterisation outcome when comparing human milk levels with the level resulting from exposure at the TWI, as compared to basing it on the estimated exposure.

Follow-up question: Do you think there will be a new re-evaluation of TEFs soon?

In any further evaluation of the TEF values it is worth considering whether this could be approached differently. In establishing the current WHO_{2022}-TEFs, greater weight was given to in vivo animal data than to in vitro human data. Currently, we have a human study that aligns with animal findings, but when applying the new WHO-TEFs for both PCDD/Fs and DL-PCBs, this association is not observed. If the current TEFs are not suitable for humans, continuing to look for associations in human studies will not be meaningful. TEFs that are accurate and appropriate for humans are needed to ensure reliable interpretation of human data. The CONTAM Panel made a recommendation in the current draft Opinion to examine alternative approaches for establishing TEFs that are more suitable for humans.

7. **What do you think the data availability is to actually perform the PBK modelling in a more proper way, in a congener specific fashion? How do you expect the in vitro results to inform this?**

We used a PBK model for TCDD. There have been attempts to adapt kinetic models for other congeners by adjusting parameters related to the half-life, which varies across congeners. For instance, PCB-126 has a shorter half-life than TCDD, partly explaining changes in congener patterns between food and human milk. For example, TCDF contributes to the dietary exposure

but is hardly detected in human milk. These kinetic differences might be considered when establishing TEFs, similar to excluding other non-2,3,7,8-substituted congeners that do not accumulate in animals and humans. If congener-specific PBK models were developed, a key question would be which congener pattern to use: the dietary exposure profile? Finally, we compare long-term exposure levels with the TWI, which relates to potential health risks for the offspring due to accumulation of relevant congeners in the mother. Should this broader perspective on kinetic differences be prioritised rather than feeding congener-specific patterns into models? How to address this remains an open question.

Follow-up question: The TEFs are already set by the WHO. How much kinetics were taken into account in the establishment?

The WHO-TEFs are primarily based on in vivo data from rats, which have a short half-life for these compounds. Long-term accumulation of PCDD/Fs and DL-PCBs in humans was not considered when establishing the current TEFs

Follow-up question: Should we keep the WHO_{2022}-TEFs as they are and then take the accumulation of these compounds into account in the toxicokinetic modelling?

This is a possibility and worth discussing. The recommendations in the draft Opinion emphasise improving the approach for establishing the TEFs for different congeners, as currently less weight is given to human in vitro data. A future review of TEFs may be necessary, also in support of evaluating the results from human studies.

Considering the impact of kinetic differences, in the draft Opinion a table is included that illustrates this point, highlighting discrepancies between dietary exposure levels and human milk levels for Total-TEQ and key individual congeners. For example, PCB-126 contributes on average 37.6% to the dietary exposure. Using this value, one can calculate its dietary exposure in pg TEQ/kg bw per week, transfer this to predicted human milk levels (ng TEQ/kg fat), and compare the outcome to the levels seen in the two human milk datasets considered in the draft Opinion. Interestingly, PCB-126 contributes less in human milk samples than to the dietary exposure, while the reverse is observed for some other congeners. From these data, the fold overestimation of predicted levels can be calculated, which is particularly high for TCDF (up to 39-fold). TCDF appears not to transfer to human milk and seems less stable and persistent in humans. This leads to large overestimations, because in the PBK modelling it is assumed that TCDF behaves like TCDD. For TCDD, the overestimation is 1.4–2.0, which is within acceptable uncertainty. However, uncertainties also exist in the human milk data, as it comes from a limited number of countries and individual data from only one country. Since the current TWI is based on the assumption on persistence, we should consider whether differences in toxicokinetic profiles should be reflected in PBK models or in the TEFs. This is the key message of the table and the relevant section of the draft Opinion, which also questions whether PCB-126 is as potent as suggested by the current TEF and contradicted by data with human cells. Please refer to the draft Opinion for further details.

8. **Should this new assessment lead to new consumer advise for fish?**

EFSA's role is to provide independent scientific advice on matters linked to food and feed safety at a European level, that will provide risk managers, i.e. the European Commission, European Parliament and the Member States, with the sound scientific advice required for them to take the final legislative or regulatory decisions on matters related to food and feed safety. The question posed is more directed to risk managers than for EFSA as a risk assessor.

EFSA has recently received a new mandate to assess the risk–benefit of fish consumption, taking into account the various contaminants that may be present in fish. This work will begin soon and is expected to continue for a couple of years.

9. **You conclude that at current exceedance of TWI there is no evidence of effects in adolescents, adults and older age groups. What is this conclusion based on, and at what exposure levels will dioxin exposure be a problem in these age groups?**

Based on the human data we have identified that there is no evidence for effects at current exposure estimates. There is some evidence for effects on tooth development at slightly higher exposure than we currently have, it was specifically addressed in the 2018 Opinion, but this is still only relevant in infants as a developmental effect. It was not possible for the CONTAM Panel to quantify when the risk of adverse effects starts to increase in adults.

10. **Is it correct that only analytical results on food and feed provided by national authorities were taken into account for the exposure assessment? If yes, were there also data submitted by stakeholders and why weren't they taken into account?**

All available occurrence data in food and feed submitted to EFSA by Member States and other Stakeholders were taken into consideration.

11. **Let's look at specific global locations currently processing fish meal as animal feed, including the US Gulf of Mexico.**

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