USE OF THE TEQ MODEL FOR ASSESSING AHR MEDIATED TOXICITY RISKS TO POPULATIONS OF LAKE TROUT AND OTHER SPECIES IN LAKE ONTARIO

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Introduction

The toxicity equivalence (TEQ) model for assessing aryl hydrocarbon receptor (AHR) mediated toxicity risks associated with polyhalogenated aromatic chemicals structurally similar to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)[1] has been applied to human health risks for more than 15 years. In 1997 the establishment under the World Health Organization of consensus toxicity equivalence factors (TEFs) for mammals, birds, and fish created a general TEQ methodology for ecological risk assessments parallel to that for human health[2]. In a workshop organized by U.S. EPA and U.S. DOI, international experts concluded that the TEF/TEQ methodology is appropriate for ecological risk assessments and reduces uncertainties associated with other options which do not consider the additive impacts of multiple AHR agonists[3]. In addition to endorsing use of TEFs, workshop participants supported evaluation of relative potency data for calculation of relative potency factors (RPFs) as alternatives to the TEFs when species and toxicity endpoint specificity are likely to improve the accuracy of a risk assessment. Finally, the lack of rigorous analyses of associations between TEQ based toxicity predictions and expected population responses for fish, birds, and mammals was described as an important need for validation of the method.

By the middle of the 20th century, the Great Lakes were highly contaminated with TCDD and other AHR agonists for which TEFs are now available. Simultaneously, populations of some fish and bird species declined. Given the number of biological, chemical, and physical stressors present that could affect populations, attribution of a population change over time to a specific stressor like AHR mediated toxicity requires a high degree of accuracy for predictions of exposure, toxicity related mortality, and consequent population response. In the early 1990s determinations of great sensitivity of lake trout (*Salvelinus namaycush*) to TCDD-induced early life stage mortality[4,5] led us to pursue a complete assessment of the contribution of such toxicity to effects on populations of this keystone species in the Great Lakes. The retrospective analysis of the decline of lake trout in Lake Ontario and difficulties in restoring a naturally reproducing population provides a compelling example of the importance and effectiveness of the TEQ model[6].

Material and Methods

Exposures of fish embryos to persistent bioaccumulative toxicants are best measured as concentrations in the whole embryo. Early life stage toxicity data for ten species of fish exposed as freshly fertilized eggs to TCDD demonstrate that fish species sensitivities vary by at least 50-fold with trout being most sensitive[7]. In the case study for lake trout[6] TCDD toxicity data, based on concentrations in the embryo, that were specific for the species, end point, and most sensitive life stage, as well as extensively replicated[4,5,8-10] were used. Similarly, most of the TEFs for fish were based on early life stage mortality in rainbow trout[11,12], a closely related species, and the same TCDD dose metric used for the lake trout early life stage mortality data. Direct measurement of toxicity equivalence concentrations for lake trout eggs (TEC_{egg}s) was only possible for the period after 1978. Correlation with data from herring gull egg contaminant data allowed lake trout egg exposures to be estimated back to 1970. In order to examine the potential impact of AHR mediated toxicity on Lake Ontario lake trout populations it was necessary to trace exposures back to the 1920s when lake trout were abundant. This was accomplished by measuring concentrations of PCDDs, PCDFs, and PCBs in radionuclide dated sediment core sections and calculating TEC_{egg}s with biota sediment accumulation factors (BSAFs)[13] measured for lake trout eggs in the period of 1987-1991. BSAFs were adjusted slightly for conditions prior to 1970 when concentrations in lake waters were relatively greater in comparison to concentrations in surficial sediments due to large chemical loadings to the lake. TEC_{egg}s for different years were calculated as the sum of the products of concentration in sediment from the time period, BSAF, and TEF for each AHR agonist. Complete details of the methods, analytical data, and epidemiological analyses are available in Cook et al.[6].

Results and Discussion

The results of the retrospective analysis of TEC_{egg}s for lake trout from the primary reference sediment core are illustrated in Figure 1. Note that although this plot takes the form of a sediment core analysis, the values plotted are based on concentrations in lake trout eggs so that direct comparisons to toxicity risks can be made. The rise and fall of concentrations of AHR agonists in sediments and biota during the 20th century was a common pattern in many aquatic ecosystems, but TCDD concentrations were exceptionally large in Lake Ontario. Because of this and the great relative sensitivity of fish to TCDD, more than half of each TEC_{egg} after 1940 is attributable to TCDD. This is not the case for TECs calculated analogously for mammals and birds because of significant differences in TEFs and BSAFs. The sediment core based TEC_{egg}s for lake trout are in good agreement with TEC_{egg}s based on herring gull egg data and the measured TEC_{egg}s from lake trout. Although the herring gull egg based TEC_{egg}s indicate that the sediment core based TEC_{egg}s may be underestimating actual TEC_{egg}s when peak exposures occurred, the difference may be attributable to slight differences in food chain effects on bioaccumulation by gulls versus trout over time. The prediction of toxicity impacts on mortality of lake trout fry from the late 1930s into the 1990s is very consistent with the epidemiological records and recent signs of restoration of natural reproduction [6].

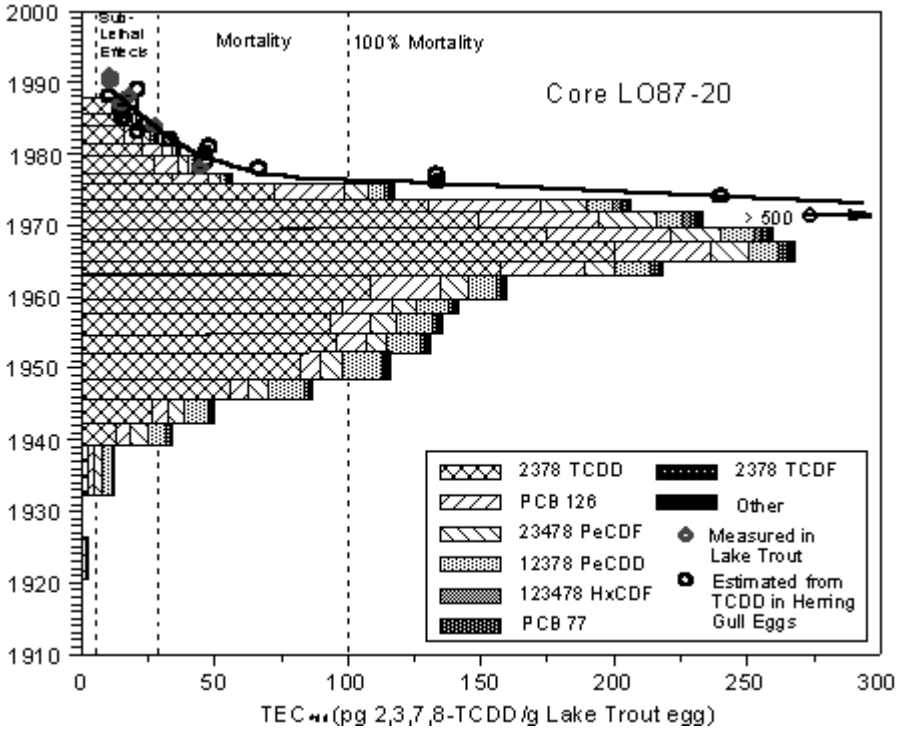

Figure 1. Retrospectively determined lake trout TEC_{egg}s from analysis of a radionuclide dated sediment core collected in eastern Lake Ontario. TEC_{egg}s greater than 30 pg TCDD equivalence/g trout egg (wet) result in overt mortality in laboratory studies. Sub-lethal effects under environmental conditions may cause mortality with TEC_{egg}s < 30 pg/g.

In addition to laboratory studies supporting the TEQ additivity model for lake trout early life stage mortality [14,15], two important validation steps in this study involved testing the plausibility of the toxicity predictions with population response data from Lake Ontario. Figure 2 illustrates the agreement found on the basis of the lake trout commercial catch which documents the historical decline of lake trout to extirpation by 1960 and the incidence of overt mortality with signs of TCDD toxicity observed in the laboratory[16] in sac fry from eggs obtained from stocked Lake Ontario lake trout. The somewhat greater mortality observed for the feral eggs in comparison to the overt mortality prediction (min sac fry mortality) may be attributable to AHR agonists which were not included in the TEC_{egg}

calculations. The predicted max sac fry mortality incorporates sub-lethal toxicity effects in combination with bioenergetic and environmental factors that may exacerbate the impact of AHR mediated toxicity under Lake Ontario conditions.

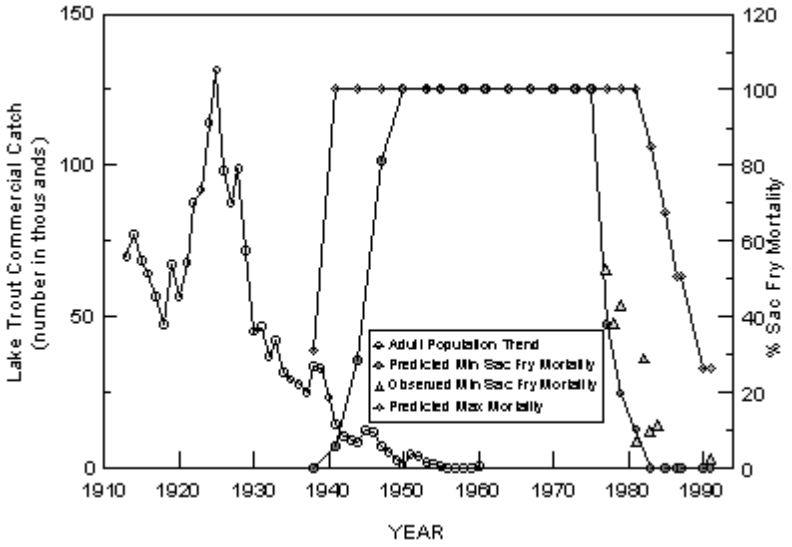

Figure 2. AHR mediated toxicity predictions in comparison to historical lake trout population levels and lake trout sac fry mortality data for eggs collected from stocked trout. Maximum mortality predictions are based on sub-lethal effects and presence of potential AHR agonists that were not include in the TEC_{egg} calculations.

While in retrospect it may seem obvious that the use of the 1997 WHO TEFs[2] based on fish early life stage mortality should increase the accuracy of lake trout mortality predictions in comparison to use of earlier TEFs which were based exclusively on mammalian responses, it is informative to make the comparison. Figure 3 shows that the TEC_{egg} values for lake trout, and consequently toxicity risks, would be at least 3 times greater (several dioxin-like PCB congeners were not included) if mammalian TEFs were applied to eggs (line ME versus line FE). The population response data suggest that this would result in overestimation of the actual ecological risks. This case study also highlights another potential source of error in the application of TEFs that should be avoided. Application of TEFs directly to concentrations measured in effluents, sediments, soils, or other abiotic media commonly results in toxicity equivalence concentrations (TECs) that are unrelated to dose metrics associated with the toxicity data used in ecological risk assessments. As such, they do not account for changes in mixture composition and mass associated with chemical-specific differences in bioavailability, metabolism, and biomagnification. The impact on TEC calculations when TEFs are applied to sediments, rather than an appropriate biological medium, is demonstrated in Figure 3 (lines FS and MS versus line FE).

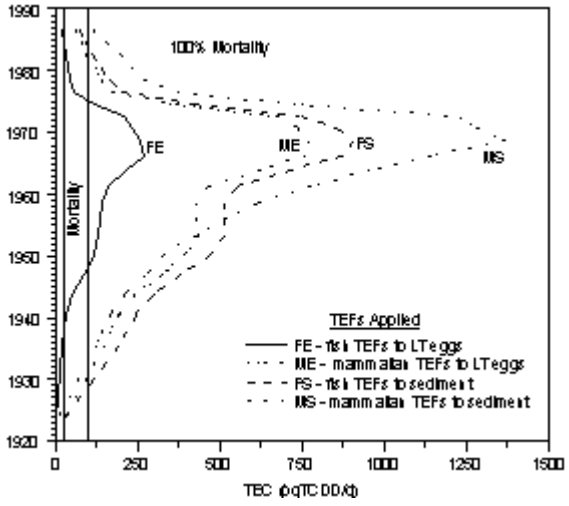

Figure 3. Comparison of Lake Ontario lake trout TEC_{egg}s[6], based on application of fish TEFs to concentrations of AHR agonists in lake trout eggs (FE), to TEC calculations that would result from inaccurate and inappropriate applications of TEFs : (1) application of mammalian TEFs in lieu of fish-specific TEFs to concentrations of AHR agonists in lake trout eggs (ME); (2) application of fish TEFs to concentrations of AHR agonists in sediments (FS); and (3) application of mammalian TEFs to concentrations of AHR agonists in sediments (MS).

RPFs for PAHs have been applied with RPFs or TEFs for PCDDs, PCDFs, and PCBs to calculate TECs based on concentrations in sediments. This has resulted in conclusions that PAHs contribute more to dioxin-like activity than the PCDDs, PCDFs, and PCBs[17]. In some cases similar conclusions are followed with caveats recognizing that PAHs have low bioaccumulation potential in vertebrates[18] and thus are unlikely to contribute to AHR mediated effects of concern. We feel that it would be more appropriate to restrict applications of TEFs and RPFs to concentrations of chemicals in tissues of organisms at risk or their diets in a manner consistent with the TCDD dose metric associated with the toxicity relationship to which the TEC is to be compared.

Conclusions

The convergence of good research and field data, historical records, and development of the TEQ model for ecological risk assessments has allowed the assessment of AHR mediated toxicity risks to lake trout populations over time in Lake Ontario to provide a model case study for planning future risk assessments. The toxicity risks to other species in the Lake Ontario ecosystem may be assessed with these data. For example, TCDD effects data for lake herring embryo exposures[7] and predicted TEC_{egg} values indicate that AHR mediated toxicity may have contributed to the observed population decline for this species after 1960, despite the lower sensitivity of herring to TCDD. This case study indicates that much of the uncertainty for TEQ assessments can be minimized through selection of parameters that maximize species, end point, and dose specificity while applying TEFs or RPFs in a manner that is consistent with the TEQ model constructs and assumptions.

Acknowledgements

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