Perspectives on the Toxicity Equivalency Approach to Quantify the Effects of Coplanar Polychlorinated Biphenyls in Ecological Risk Assessment

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1. Introduction

Traditional approaches to evaluating the toxicity of coplanar polychlorinated biphenyls (PCBs) in wildlife involve calculating their 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity equivalents (TEQs) and critical body burdens in key species. There are 13 PCBs, including 3 non-*ortho*-, 8 mono-*ortho*-, and 2 di-*ortho*-substituted compounds, that are considered "dioxin-like" in their coplanar conformation and mechanism of toxicity. A recent review of the available scientific information indicated that there is sufficient information to recommend TCDD toxic equivalence factors (TEFs) for dioxin-like PCBs, based on evidence suggesting a common receptor-mediated mechanism of action[2]. First proposed by Safe[3], there are currently four TEF schemes proposed for estimation of toxicity and risk assessment that are based on exposure or intake concentrations of these compounds (Table 1).

Table 1. Toxic equivalency factors (TEFs) proposed for PCBs for human health risk assessment.

IUPAC	TEF-system			
	Ahlborg et al. (1994)	Ahlborg (1992)	Safe (1990)	Safe (1994)
77	0.0005	0.0005	0.01	0.01
126	0.1	0.1	0.1	0.1
169	0.01	0.01	0.05	0.05
105	0.0001	0.0001	0.001	0.001
114	0.0005	0.0005	0.001	0.002
118	0.0001	0.0001	0.001	0.001
123	0.0001	0.0001	0.001	0.00005
156	0.0005	0.001	0.001	0.0004
157	0.0005	0.001	0.001	0.0003
167	0.00001	-	0.001	-
189	0.0001	-	0.001	-
170	0.0001	-	0.00002	-
180	0.00001	-	0.00002	-

There is, however, an insufficient scientific basis for inferring that the TEQ approach proposed by the U.S. Environmental Protection Agency[1] is a valid method for estimating the toxicity of so-called "dioxin-like" PCBs in non-mammalian species. For example, there are conflicting data suggesting that TCDD toxic equivalency factors (TEFs) for many coplanar PCBs may over- or underestimate toxicity in some fish and avian species. Preliminary results of fish bioassays have suggested that the EROD induction potency of certain penta- and hexa-CBs may be comparable to or greater than that of TCDD. For these reasons, it appears that the relative ecological risks associated with coplanar PCBs versus dioxins in fish and wildlife do not parallel the potential health risks in humans.

A WHO/IPCS commission recently concluded that separate TEFs for body burdens and ecotoxicology should be explored for various fish and wildlife[2]. For example, there is sufficient data suggesting differential sensitivity to several coplanar PCB compounds for both fish and avian species[4,5,6,7,8,9,10,11]. In addition, some TEFs that have been reported for coplanar PCBs and PCDD/Fs in rainbow trout based on EROD activity are substantially higher than those reported for mammals[8,12,13]. Thus, it is increasingly evident that reliance on current TEFs derived from mammalian systems are imprecise for determining relative toxicities in avian and fish species because the physicochemical properties affecting environmental persistence, distribution, and bioavailability differ widely among congeners[11].

2. Ecological Risk Issues

Notwithstanding the inherent conservatism of the TEF approach, there are aspects of the approach that must be considered separately in risk assessments for humans and ecological receptors. Coplanar PCB TEQs calculated from body burdens in Great Lakes fish suggest generally higher values for fish than mammals, indicating that coplanar PCBs may be more bioavailable and, may pose a greater risk in aquatic systems than in terrestrial systems[2,5,6,7]. Because coplanar PCBs or TCDD TEQs appear to occur at higher concentrations in aquatic environments, fish and fish-eating birds are likely at greater risk than humans from these compounds.

For ecological receptors, it appears that the endpoints of these compounds are clearly different than those of human receptors, and may be species-specific. For example, Tillitt et al.[14] demonstrated that there is a linear correlation between total PCB TEQs in eggs and reproductive toxicity in double-crested cormorants from the Great Lakes. However, Williams and Giesy[9] did not find significant correlations between PCB TEQs and reproductive toxicity in chinook salmon from the same system. Other fish species, including many that are closely related, appear to have varying sensitivities to TCDD TEQs. For instance, within the salmonidae family, both Walker et al.[15] and Mac et al.[6] have demonstrated that lake trout may be more sensitive to the effects of these compounds than are rainbow trout or chinook salmon.

For these reasons, it appears that the potential toxicity of TCDD TEQs should be evaluated on a species- and endpoint-specific basis. According to Tillet et al.[16], the absolute value of TCDD-TEQs in Foster Tern eggs vary as much as a factor of 40, depending on which set of TEF values are used. Total TEQ in eggs were shown to range between 2292 pg/g using mammalian TEFs by Safe[3] to 314 based on avian LD_{50}s, and 57 using H411E-EROD induction as the relevant endpoint. TEFs should be developed for key organisms in a risk assessment rather than using those derived from mammalian studies. From a regulatory perspective, it may be necessary to use the most conservative TEFs (i.e., those derived for the most sensitive species and endpoint) that are available for a phylogentic group of organisms. In addition to species-specific concerns, the endpoints for each key organism should be considered. For, instance, in fish, it appears that early life stage mortality is the most sensitive endpoint to TCDD TEQs, while in avian species reproductive toxicity is the most sensitive endpoint. A TEF scheme that is specific to each of these endpoints for the respective organisms should be developed for use in risk assessment.

3. Summary of Conclusions

The scientific evidence suggests that it is not appropriate to use mammalian TEFs for ecological risk assessments where fish and avian species are the key organisms that may be at risk; such an approach may over- or under-state the risks to these organisms. The potential toxicity of TCDD TEQs should be evaluated on a species-specific basis, and TEFs should be developed for key organisms in a risk assessment rather than using those derived for a surrogate organism that may be more or less sensitive to these compounds.

4. References

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