Exposure Toxicity Equivalents (ETEs): A Plea for more Environmental Chemistry in Dioxin Risk Assessment

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2,3,7,8-Cl_4DD toxicity equivalents (TEs) are a convenient tool to estimate the toxicity of complex mixtures of the 210 different chlorinated dibenzo-p-dioxin and dibenzofuran congeners. They allow the risk manager to work with a single number instead of a complex array of analytical results. They also enable the analyst to concentrate his efforts on a small subset of PCDD/F congeners.

The TE approach has gained widespread acceptance, both in the scientific community and with regulatory agencies. Analytical results are commonly quoted in terms of TEs and we tend to interpret these TEs as a measure of the relative hazard associated with the sampled matrix. This has led to the incorporation of TEs as a regulated quantity in various countries. In Germany for instance there is a legal limit of 0.1 ng TE/m^3 for waste incinerator emissions and 100 ng TE/kg DW for agricultural use of sewage sludge. There is however a fundamental problem with this approach: It does not consider the congener specific environmental chemistry of the PCDD/F.

Toxicity equivalent factors (TEFs) are determined from *in vivo* and *in vitro* studies of the relative toxicity of PCDD/F congeners. With *in vitro* studies and those *in vivo* studies where the compound is administered through injection the relative toxicity of comparable tissue concentrations is determined. The fact that all *in vivo* studies are not normalized for tissue concentration is a shortcoming that has been addressed elsewhere. There are other problems with the TE approach such as different dose response curves for different compounds that will not be further dealt with here. In general though the TEFs represent the relative toxicities of PCDD/F congeners as present in tissue.

The crux of the problem is that one cannot treat emissions samples or soil samples as if they were present in human tissue. Many things happen to these compounds on their way to human tissue. The fact that some compounds are more successful than others in reaching

this target is apparent from the very different PCDD/F patterns measured in humans and PCDD/F sources. The environment is a PCDD/F filter, and this filter alters the relative concentrations of the PCDD/F homologues significantly. Thus the TEs of an environmental sample have little meaning, as the relative concentrations of the PCDD/F in human tissue due to the sampled matrix will be different that those measured in the sample itself.

This problem can be overcome by expanding the TE system to include the relative congener specific transfer along the critical exposure pathway from the measured matrix to the target (eg. humans). The multiplication of the environmental transfer factors (TF) with the toxicity equivalent factor (TEF) yields an exposure toxicity equivalent factor (ETEF). These are multiplied with the sample concentrations just as if calculating TEs to give a quantity that I have called the exposure toxicity equivalent (ETE). This quantity can then serve as the basis for comparison of the risk arising from a specific matrix.

Consider for example an agricultural soil. The critical human exposure pathway is likely soil-cow-milk-man. A good estimate of the transfer of PCDD from soil ingested by the cow to the milk would be 35% for 2,3,7,8-Cl_4DD and 2% for Cl_8DD. If we assume that the intestinal resorption of these compounds from milk in humans is similar, we can calculate as follows:

$$ETEF(soil\text{-}milk\text{-}man) = TF(Soil/Milk) \times TF(Milk/Man) \times TEF$$

$$ETEF(2,3,7,8\text{-}Cl_4DD) = 0.35 \times 0.35 \times 1$$

$$= 0.1225$$

$$ETEF(Cl_8DD) = 0.02 \times 0.02 \times 0.001$$

$$= 0.0000004$$

The usefulness of this more complicated calculation can be shown with the following example. Consider two hypothetical soils where only the two PCDD/F congeners above were detected. In the one soil the concentrations are 10 and 1000 ng/kg DW respectively (from a combustion source for instance), in the second 1 and 10,000 (perhaps from a pentachlorophenol source). The I-TEs and ETEs are compared in Table 1.

Table 1: Comparison of I-TE and ETE Value for Two Soils

	Concentration (ng/kg)		I-TE	ETE
	2,3,7,8-Cl_4DD	Cl_8DD		
Soil #1	10	1000	11	1,2
Soil #2	1	10000	11	0,12

According to the I-TEs the risk arising from the soils is comparable, whereas the more appropriate ETEs indicate that the risk arising from soil 1 is a factor of 10 larger. The use of I-TEs in this case could result in a serious misinterpretation of the relative risk and inappropriate allocation of resources to reduce this risk.

This example illustrates the dangers associated with the current use of TEs. ETEs are essential for the determination of relative risk. Furthermore, ETEs are the suitable quantity for regulating PCDD/F emissions and levels in the environment. Short term biological assays will not help us in this regard, as they too will predict the toxicity of a mixture in human or other tissue but will not include the environmental filter effects. Environmental chemistry plays and will continue to play an important role in the risk equation. This should not be neglected in our search for knowledge and consensus.