Evaluation of the U.S. EPA's "Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities", with Emphasis on PCDD/PCDFs

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Introduction:

In 1998, the United States Environmental Protection Agency (EPA) published draft guidance for estimating both direct and indirect exposures to emissions from hazardous waste incinerators. The "Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities" (HHRAP), a three volume, 2000 page document, provides the approach, equations, and parameters necessary to calculate the concentrations of various chemical emissions in various environmental media and the resulting risks from their uptake. The Protocol requires that the 17 carcinogenic PCDD/PCDFs be included in the evaluation whether or not they are detected in the emissions. Although not entirely unexpected in such a massive document which tackles a fairly complex issue, we have concluded that there are some significant flaws with both the general guidance and with the parameters used in dealing with the PCDD/PCDFs. This paper presents an overview of the Protocol and a number of the changes that should be incorporated since the current methods yield inaccurate estimates of the concentrations and risks for the carcinogenic dioxins and furans.

Figure 1 shows the conceptual site model described by the Protocol. The Industrial Source Complex Short-Term Model (ISCST) is used to model point and area source emissions as both vapors and particles. Chemicals of Potential Concern (COPCs) are partitioned between the particle and vapor phases as functions of their vapor pressures. At each receptor, there are five modeled values for each chemical – vapor and particle concentrations, wet vapor deposition rates, and dry and wet particle deposition rates. The COPCs enter the soils, plants, and water bodies. Beef cattle, dairy cows, pigs, and chickens bioaccumulate the chemicals due to ingestion of impacted soil and plants. The receptors include adult and child residents, subsistence farmers, and subsistence fishermen. The residential exposure pathways are via ingestion of soil, home grown vegetables, and drinking water and direct inhalation of vapors and particles. The subsistence fisher has the same pathways plus ingestion of fish. The subsistence farmer has the same pathways as the resident plus ingestion of home grown beef, pork, milk, chicken, and eggs. In addition, the uptake rate of PCDD/PCDFs via mother's breast milk is included for infants.

Issues:

The partitioning of chemicals between the particle and vapor phases is based on ambient particle concentrations rather than site-specific particle emission rates. The fractions in the vapor phase, Fv, and the particle phase, 1-Fv, are assumed constant for each chemical, regardless of the mass, size, and rate of the particles emitted from the source. They are calculated per the Junge equation:

$$Fv = 1 - \frac{c \cdot St}{Pl + c \cdot St}$$

where, c = 1.74E-4 atm-cm, St = surface area of particles per unit volume in cm^2/cm^3, and Pl = liquid vapor pressure of COPC in atm. The EPA has fixed the value of St at 1.1E-5 cm^2/cm^3, the background level found in air in metropolitan areas. Since this approach leaves out the particles emitted from the source, it will over estimate the concentrations of chemicals in the vapor phase and under estimate the amounts in the particle phases for most facilities.

In previous guidance, the EPA estimated chemical concentrations in soils based on inputs from particle and vapor deposition and losses due to erosion, runoff, leaching, degradation, and volatilization. In their latest guidance, the EPA eliminated erosion and volatilization. For PCDD/PCDFs, erosion is the only loss pathway of significance. The result is that dioxin and furan concentrations in soils will continue to increase almost indefinitely until the source ceases. The EPA eliminated erosion stating that erosion simply caused chemicals to move from one soil area to another, with no net loss or gain. However, they then use erosion as a source of input to surface water bodies, apparently creating mass.

As has been known for several years, concentrations of PCDD/PCDFs in plants are the predominant source of risk in these assessments since they are the major source for foraging animals whose products are in turn the major sources for uptake by humans. The uptake into plants is via the root, particle deposition, and vapor partitioning. Using the EPA's Fv values, the vapor pathway controls the concentrations in plants. The EPA assumes that equilibrium is established between the vapor phase, Cv ($\mu g/m^3$), and the plant, Pv (mg/kg):

$$Pv = \frac{Cv \cdot Bvag \cdot VGag}{\rho a}$$

where, Bvag is the air to plant transfer factor (mg/g in plant per mg/g in air), VGag is an empirical correction factor, and ρa is the density of air. The EPA first calculates the Bvag value as a function of the octanol water partitioning coefficient, Kow, and Henry's constant, H. For PCDD/PCDFs, it then divides the value by 100 to bring them into line with other experimental results. For plants eaten by humans, the VGag value is 0.01, and for plants eaten by foraging animals, it is 1.0. The EPA contends that this is to account for the difference in uptake of plants grown for consumption and grasses eaten by animals. Thus, it appears that the plant concentrations are based on a combination of the results of several experiments that have been modified to accommodate the results of one or two other studies. Since this equation and the factors so critically influence the results, they need to be revisited and probably changed.

As shown in more than 20 prior assessments, the primary source of dioxins for most humans is the ingestion of animal products. Using the EPA methodology, the PCDD/PCDF concentrations in beef, pork, milk, chicken and eggs are calculated by multiplying the daily animal uptake (mg/day) from ingestion of soils and plant material by animal and chemical specific bioaccumulation (Ba) factors (day/kg). For chicken and eggs, the modeled concentrations in soils, not feed, drive the risks. The Ba values were calculated by multiplying the bioconcentration factors (BCF, pg/g in tissue per pg/g in feed) from Stephens et al (date) by the ingestion rate of feed and soils (0.02 kg/day). It is fairly clear that the EPA's Ba values are too low by a factor of 2,500 since the BCFs should have been divided by the feed rate, not multiplied. Besides this calculation error, there are some other problems with the chicken and egg pathways. For example, Stephens et al used two soil levels, 42 and 460 pg/g I-TEQ, and found two sets of BCFs. By definition, the BCFs should be the same for the low and high doses. However, the BCFs for the higher dosed chickens and eggs were generally higher than those in the low dose group. The EPA chose the higher BCF values although the typical modeled soil concentrations are generally much closer to those for the lower dose group. Additionally, while there may be a few farmers whose

chickens are free to roam, and perhaps some farmers who eat them on occasion, the vast majority of chickens are kept indoors and fed grain, preventing chickens from contact with any impacted soil. Thus, although it is perhaps worthwhile to perform the calculations, it is important to properly characterize the size of the potentially impacted population in the risk assessment.

Since the milk, beef, and pork risks are driven by the plants eaten by the animals, these calculations need to be quite accurate. The Ba values for PCDD/PCDFs in milk are important factors in the equation. They are based on the BCFs from a study by McLachlan et al in which they traced the concentration in milk over time for one cow fed known levels of dioxins and furans. The EPA determined the Ba values for beef by multiplying the Ba values in milk by the ratio of the fat content in beef to that in milk, 5.43 (19%/3.5%). Similarly, the EPA determined the Ba values for pork by using the fat in pork to fat in milk ratio of 6.57 (23%/3.5%). Thus, the EPA derived the risks for milk and beef, the two major indirect pathways, from the results of one study of the milk of only one cow. The validity of this approach is worsened by assuming that pigs and cows have the same metabolisms (an unlikely assumption).

Due to the large number of assumptions made throughout the HHRAP, the EPA has discussed the possibility of performing field sampling near an incineration facility in order to validate their exposure assessment model. Although an important exercise, due to the high cancer slope factors for PCDD/PCDFs, and the low concentrations in the environment which could produce risks in the range of one to ten per million., we wonder whether this plan is viable. The authors obtained reasonable minimum estimated detection limits (EDLs) for the ten media of concern and used these values to calculate the risks. Figure 2 shows the risks due to the uptake of each media and the total risk. Interestingly, the risks at the EDLs for soil and air are between 1E-7 and 1E-6, with the risks for the other media at their EDLs between 1E-5 and 1E-4. The sum of all risks is 3E-4. Thus, it appears that validation would not be possible at most realistic sites, since most of the concentrations in the various media would be below their EDLs.

Conclusions:

In conclusion, there are numerous shortcomings and computational errors in the current version of the HHRAP methodology, and these need to be addressed before reliance is placed on it for purposes of risk decision making. Until more exacting data are available to support the use of point estimates for multimedia modeling on such a large scale, we suggest that the EPA evaluate the thoughtful use of other approaches, such as looking at realistically exposed receptors and monte carlo techniques when conducting these analyses.

References:

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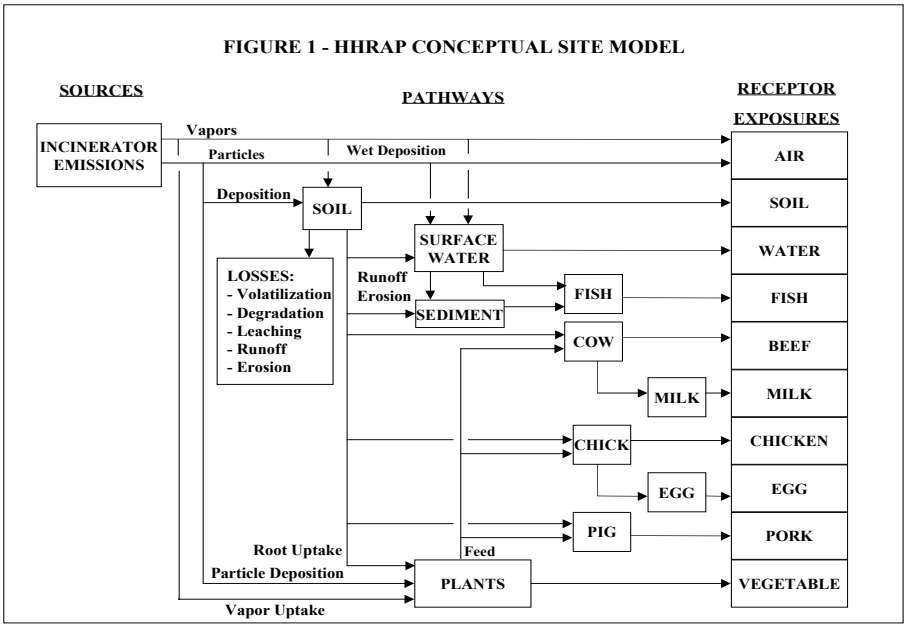
FIGURE 1 - HHRAP CONCEPTUAL SITE MODEL
SOURCES
PATHWAYS
RECEPTOR EXPOSURES
INCINERATOR EMISSIONS
Vapors
Particles
Wet Deposition
Deposition
SOIL
SURFACE WATER
LOSSES:
- Volatilization
- Degradation
- Leaching
- Runoff
- Erosion
Runoff
Erosion
SEDIMENT
FISH
COW
MILK
CHICK
EGG
PIG
Root Uptake
Particle Deposition
Vapor Uptake
Feed
PLANTS
AIR
SOIL
WATER
FISH
BEEF
MILK
CHICKEN
EGG
PORK
VEGETABLE

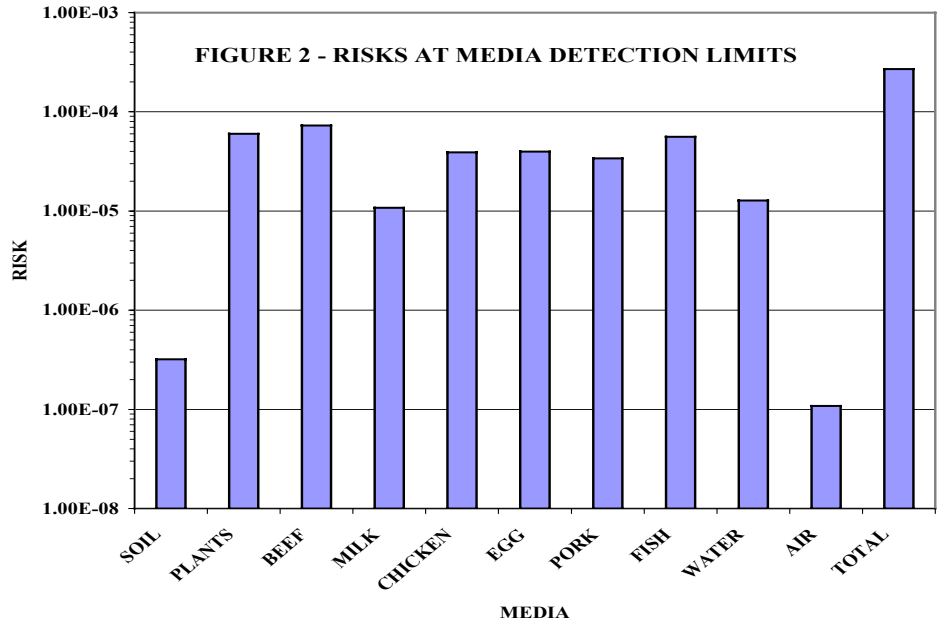
FIGURE 2 - RISKS AT MEDIA DETECTION LIMITS
RISK
1.00E-03
1.00E-04
1.00E-05
1.00E-06
1.00E-07
1.00E-08
SOIL
PLANTS
BEEF
MILK
CHICKEN
EGG
PORK
FISH
WATER
AIR
TOTAL
MEDIA