AN EVALUATION OF THE INHALATION HAZARD
POSED BY DIOXIN-CONTAMINATED SOIL

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ABSTRACT

Contaminant transport models were used to estimate the airborne concentrations of 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) vapor and particulates originating from soil containing 100 ppb TCDD. The upper-bound estimates of the airborne TCDD vapor concentration were 30 pg/m^3 on-site, and 12 pg/m^3 for TCDD vapor 100 meters downwind. The maximum plausible annual average concentration of TCDD bound to suspended soil was 0.7 pg/m^3 on-site and 0.07 pg/m^3 100 meters downwind. Assuming 70 years of continuous exposure to these concentrations, and a risk-specific dose (RSD) of 100 fg/kg/day (10^{-6} risk), the upper-bound cancer risks were estimated to be 8.1 x 10^{-5} and 3.2 x 10^{-5} for inhalation of vapors on and off the site. The upper bound risk for airborne dust on and off the site were 1.9 x 10^{-6} and 1.9 x 10^{-7} respectively. Due to the conservatisms inherent in the transport models, the actual airborne concentrations will almost certainly be 10-50 fold less than that predicted here. Since few sites have average soil concentrations as high as 100 ppb TCDD, this worst-case analysis indicates that inhalation will rarely, if ever, be an important route of exposure to TCDD-contaminated soil.

KEY WORDS

2,3,7,8-TCDD, risk assessment, dusts, vapor, contaminated soil

INTRODUCTION

Although 2,3,7,8-TCDD has been shown to have a negligible vapor pressure (1.5 x 10-9 mm Hg at 25 C; Schroy et al., 1985), numerous studies have claimed that airborne concentrations of TCDD from contaminated soil could be of a magnitude sufficient to pose a significant health risk (EPA, 1985). Other reports have suggested that inhalation of TCDD vapors and particulates are negligible routes of exposure that may be dismissed from consideration (Paustenbach et al., 1986). To resolve this issue, the highest plausible airborne concentrations of TCDD (vapor and particulate-bound) originating from a contaminated site were estimated and the attendant theoretical cancer risks were calculated.

METHODS

In this analysis, 10 acres of soil were assumed to be contaminated with 100 ppb TCDD to a depth of 10 cm. The potential TCDD vapor emissions from site soils were estimated using the model of Jury et al. (1983). This model estimates the time-dependent chemical vapor flux from contaminated soil, considering both the soil characteristics and the physical properties of the chemical (e.g., vapor pressure and soil binding coefficient). It accounts for the contaminant's volatility, absorption to soil particles, and upward leaching in the soil column due to evapotransporation. To ensure that the flux was not underestimated it was assumed that (a) the net water flux in the soil column was upward (b), the soil had a high porosity (45%), and (c) the soil contained a low level of organic matter (1%).

A box-model approach was used to predict the maximum plausible levels of on-site airborne TCDD vapor. The approach assumes that the air above the site is contained in a building having two open walls with wind blowing through the structure. An 8' tall ceiling was assumed (mixing height). The box-model assumes that the TCDD vapor travels only a very short distance prior to inhalation. It accounts for removal of TCDD vapor by the wind, but ignores dispersion effects. The approach produces an upper-bound airborne contaminant concentration. The SCREEN (EPA, 1988) Gaussian air dispersion model was used to estimate a worst-case concentration of TCDD at a distance of 100 meters off-site. As its name implies, SCREEN is also a screening model which produces upper-bound and often unrealistically high estimates of the actual airborne concentrations. SCREEN generates a maximum hourly air concentration estimate by accounting for horizontal and vertical dispersion during worst-case meteorological conditions (i.e. extremely slow, stable winds). For the purposes of this study, the annual average concentration was conservatively assumed to be 10% of the predicted maximum hourly concentration; an approach suggested in several regulatory air dispersion modeling guidelines (EPA, 1982; CAPCOA, 1987).

To estimate the maximum air concentration of TCDD bound to total suspended particulates (TSP) originating from the contaminated soil, it was assumed that the annual average ambient concentration of TSP is 0.07 mg/m3. This concentration is similar to the TSP levels measured in many rural areas (Trijonis et al., 1980; EPA, 1985a). Since there are many sources of airborne particulate matter (e.g. automobile exhaust, tire rubber), this factor was adjusted to account for the TSP fraction that conceivably originated from the TCDD-contaminated soil on the site (10%). Several studies have shown that only about 10-20% of airborne TSP is from soils (Lioy and Daisey, 1986). The airborne concentration of TCDD bound to particulates is simply the product of the ambient TSP concentration, the percentage of the TSP from the site soil, and the average TCDD concentration in the soil.

The EPA's unit risk value (URV)[1] of 4.4 x 10^{-5} (pg/m^3)$^{-1}$ and the URV of 2.7 x 10^{-6} (pg/m^3)$^{-1}$ suggested by Keenan et al., (1990a,b) (based on the recent re-analysis of Kociba et al. (1978)) were used to estimate the plausible excess lifetime cancer risk. The URV is defined as the theoretical cancer risk associated with breathing 1 pg/m^3 of the contaminant for a lifetime. The risk associated with exposure is based on the cancer potency factor for TCDD, and standard exposure assumptions (20 m^3/day breathing rate, 70 kg body weight). For the purposes of this report it was conservatively assumed that exposure occurs continuously for a 70-year lifetime.

RESULTS AND DISCUSSION

Using Jury's model, the TCDD vapor flux (emission rate per unit area of soil) was estimated at 1.1 x 10^{-13} mg TCDD/sec-cm^2. Given the conservative nature of the assumptions concerning soil conditions, this is an upper-bound estimate of the TCDD vapor flux. Using this figure and a low annual average wind speed of 3 meters/sec (about 7 miles/hour), the box-model predicted an on-site TCDD vapor concentration of 30 pg/m^3 above soil containing 100 ppb. This concentration could be associated with a lifetime cancer risk of 1.3 x 10^{-3} (URV of 4.4 x 10^{-5} pg/m^3) or 8.1 x 10^{-5} (URV of 2.7 x 10^{-6} based on a RSD of 100 fg/kg/day). Using a screening air dispersion model, the upper-bound off-site annual average air concentration (100 meters downwind of the source) was 12 pg/m^3. This concentration corresponds to an increased lifetime cancer risk of 5.3 x 10^{-4} (URV of 4.4 x 10^{-5}) or 3.2 x 10^{-5} (URV of 2.7 x 10^{-6}). The maximum airborne concentration of particulate-bound TCDD was estimated as 0.7 pg/m^3 from soil containing 100 ppb TCDD and this poses a plausible increased cancer risk of 1.9 x 10^{-6} (URV of 2.7 x 10^{-6}). Since the risk is directly proportional to the soil concentration, if the soil level was 1 ppb, then the combined hazard of inhaling both TCDD contaminated dust and vapor would be no greater than 8.2 x 10^{-7} (URV of 2.7 x 10^{-6} pg/m^3) or about 1.4 x 10^{-5} (URV of 4.4 x 10^{-5} pg/m^3).

Fate and transport models are commonly used to predict the movement of chemicals in the environment. Most models accepted by the regulatory community are designed to skew the uncertainty in their predictions such that the likelihood of overestimating the concentration at a receptor location is much greater than that of underestimating it. Nonetheless, screening models provide the risk assessor with a rapid, inexpensive method for separating contaminated sites into two categories: those that clearly do not warrant concern and those that require a more in-depth, realistic analysis.

Since numerous conservative modeling and exposure assumptions were employed in this analysis, it is expected that the risk estimates derived herein are upper-bound and may well over-predict the actual risk by at least 100-fold. It is unlikely that many of the worst-case assumptions used in this analysis would be applicable to any site for a period of 70 years.

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[1]The EPA (1989) actually reported a URV of 3.3 x 10^{-5} (pg/m^3)$^{-1}$, based on their estimate that only 75% of inhaled particulate-bound TCDD will actually be absorbed (EPA, 1985). Because we are estimating exposure to TCDD both in vapor form and bound to particulates, we have removed the absorption correction factors from the URVs used in this analysis.

The results of this analysis indicate that inhalation of TCDD vapor is not a significant exposure pathway and can be discounted in health risk assessments where the soil levels are less than 100 ppb. In addition, for most TCDD-contaminated sites, the inhalation of suspended soil will not pose a significant health risk.

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