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THE FOOD CHAIN AS A SOURCE OF HUMAN EXPOSURE TO ORGANIC CHEMICALS

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I. INTRODUCTION

Although human exposure to environmentally released pollutants can occur via several pathways including, inhalation, ingestion of contaminated food items, infant consumption of mother's milk, and dermal absorption, of particular concern are potential exposures from ingesting contaminated food items, since the food chain has been shown to be a primary source of human exposure to a large class of organics, including DDT, TCDD, pentachlorophenol, benzo(a)pyrene, and most pesticides (Hattemer-Frey and Travis, 1989; Lioy *et al.*, 1988; Travis and Arms, 1987; Travis and Hattemer-Frey, 1987).

For risk assessment purposes, an important objective in evaluating the environmental behavior and fate of various pollutants is predicting the major pathways and extent of human exposure. Many chemicals cycle in the environment with cross-media transfers occurring between air, water, soil, and biota. As a result of this cycling behavior and a chemical's presence in various environmental media, human exposure often results from multiple sources. Travis *et al.* (1987) found that 50-80% of all chemicals released into the environment result in human exposure through multiple media. The purpose of this presentation is to provide a perspective on the food chain as a source of human exposure to organic chemicals chronically released into the environment.

II. MEASURING ORGANICS IN HUMANS AND FOOD

There are two basic methods of determining the extent of human exposure to chemicals: measurement and prediction. One direct approach is to measure the concentration of a substance in various environmental media and then calculate the average daily intake. This procedure is often done in occupational and urban settings where chemicals are concentrated enough to allow direct measurement. In other situations, however, direct measurement is not feasible, because background concentrations are too low (below detection limits) or direct measurement would be prohibitively costly or impractical. Under these circumstances, indirect methods must be used to estimate human exposures. For example, past human exposure to some chemicals can be extrapolated from measured concentrations of organics in human tissue, hair, blood, and urine samples (Murphy *et al.*, 1983; Patterson *et al.*, 1986; and van den Berg *et al.*, 1986). In addition, biological markers can be used to identify and quantify past human exposure (Perera, 1986; Perera *et al.*, 1982; Perera and Weinstein, 1982; Shugart, 1986; Shugart *et al.*, 1983; Vainio *et al.*, 1983). These two techniques for indirectly estimating past human exposure to chemical carcinogens, however, are currently only applicable to a small number of chemicals. Hence, other predictive methods, such as the use of environmental transport models and predictive equations must also be used to evaluate the risks of human exposure to chemical carcinogens.

The extent of human exposure to organics in food can be estimated by: (1) determination of body burdens, and (2) estimation of dietary intakes from market basket studies. One of the best indicators of past and present exposure are residues that bioaccumulate in human tissues and fluids. Human adipose tissue is the primary repository for persistent, lipophilic compounds. The best quantitative data on the concentration of organics in human adipose tissues come from the National Human Monitoring Program (US EPA, 1986). Table 1 shows the occurrence of several selected organochlorine residues in human adipose tissue. These data show that 96% of the U.S. population has detectable levels of heptachlor epoxide stored in adipose tissue. Not surprisingly, DDT and PCB levels in human adipose tissue have declined since 1978, as the use of these chemicals was suspended in the early 1970's.

Direct determination of chemical concentrations in food is generally limited to a few studies. The best quantitative data on the concentration of various organics in food items comes from the USDA's Total Diet Studies (Gartrell *et al.*, 1985, 1986). Table 2 shows total daily intake of several selected organochlorine compounds. While market basket studies are helpful, they do not characterize the importance of the food chain as a source of human exposure. What is needed is a determination of the environmental fate of chemicals and their eventual partitioning into air, water, and soil.

III. QUANTIFYING THE CONCENTRATION OF ORGANICS IN ENVIRONMENTAL MEDIA

Various measurement and predictive techniques can be used to evaluate the movement and transfer of chemicals within and between environmental media, as well as the concentration of organics to which humans are exposed. The distribution, decomposition, and accumulation of any chemical in the environment depends largely upon its physical and chemical properties. Since organic chemicals tend to accumulate in the media in which they are most soluble, a few basic physicochemical properties can be used to predict the behavior and fate of chemicals released into the environment.

A. Octanol-Water Partition Coefficients

Organisms tend to accumulate chemicals in the lipid portions of their tissues. Thus, one way to determine the bioaccumulation potential of a chemical is to measure how lipophilic it is. Since it is difficult to directly measure a chemical's lipophilicity, researchers typically use the octanol-water partition coefficient (K_{ow}) to predict a chemical's tendency to partition between an octanol component (a good surrogate for fat) and water. Chemicals with large K_{ow} values (i.e., lipophilic compounds), such as dioxin (2,3,7,8-TCDD), DDT, and polychlorinated biphenyls (PCBs), are most likely to concentrate in organic matter. This class of chemicals tends to bioaccumulate in biota and vegetation, sorb strongly onto soil, sediment, and vegetation, and transfer to humans through the food chain. Conversely, chemicals with small K_{ow} values, such as benzene and trichloroethylene, tend to partition mostly into air or water and are expected to bioaccumulate in the food chain to a lesser extent than their more lipophilic counterparts.

Since octanol-water partition coefficients have been shown to be directly related to a chemical's tendency to bioconcentrate in biota (Chiou *et al.*, 1982; Chou and Griffin, 1986; Geyer *et al.*, 1982, 1987; Kenaga, 1980; Kenaga and Goring, 1980; Travis and Arms, 1988) and inversely correlated with water solubility (Chiou *et al.*, 1982; Kenaga and Goring, 1980), they are used extensively to estimate the bioconcentration potential of organics in biological systems. Correlations between a chemical's K_{ow} value and its potential to bioaccumulate in living organisms have been developed for several animal groups. Chiou *et al.* (1977), Kenaga (1980), Könemann and van Leeuwen (1980), Lu and Metcalf (1975), Mackay (1982), Neely *et al.* (1974), Travis and Arms (1988), and Travis *et al.* (1988) showed that a linear relationship exists between the bioconcentration of organics in fish, cows, swine, and humans and their K_{ow} values. These relationships can be useful for estimating human tissue concentrations that result from chemical exposure and retrospectively estimating past human exposure from known concentrations in animals.

B. Bioconcentration Factors and Biotransfer Factors

Assessing the environmental fate of chemicals depends largely on being able to predict the extent to which they will bioaccumulate in living organisms, including fish, cattle, and humans. Organisms can concentrate chemicals in their tissues at levels substantially higher than a chemical's concentration in water (in the case of aquatic organisms) or in food (in the case of terrestrial organisms). A traditional measure of a chemical's potential to accumulate in biota is the bioconcentration factor (BCF), which is defined as the equilibrium concentration of organic in an organism or tissue (mg/kg) divided by the equilibrium concentration of organic (mg/kg) in water (for aquatic organisms), soil (for vegetation), or food (for terrestrial organisms). This concept, however, is not readily applied to humans and other terrestrial organisms, because the amount of chemical in various food items (and, hence, total daily intake of pollutants) can vary markedly. Thus, for risk assessment purposes, it is more efficacious to examine the biotransfer factor (BTF), which is defined as the equilibrium concentration of organic in an organism or tissue (mg/kg) divided by the average daily intake of organic (mg/day) (Travis and Arms, 1988; Travis *et al.*, 1988).

IV. MULTIMEDIA TRANSPORT MODELS

The environmental fate of organic chemicals released into the environment can be studied using multimedia transport models. Steady-state compartmental models, such as Mackay *et al.*'s (1985a,b) Level III Fugacity model, estimate the steady-state concentration of a pollutant in various environmental media. Steady-state between phases (media) occurs when the fugacity for both phases are equal (Mackay *et al.*, 1985a). The Level III Fugacity model treats the environment as a "unit world" divided into six homogenous compartments: (1) air; (2) water; (3) soil; (4) bottom sediment; (5) suspended sediment (in water); and (6) aquatic biota (fish). The air, water, and soil compartments are interactively connected, while the sediment, suspended sediment, and biota compartments are connected with the water compartment only (Mackay *et al.*, 1985a,b). While multimedia transport models should not be viewed as exact replicas of the environment, they are considered acceptable for predicting the steady-state partitioning of non-particulate chemicals (Cohen and Ryan, 1985; Eberhardt *et al.*, 1976; Hushon *et al.*, 1983; Mackay *et al.*, 1985a,b; Mackay and Paterson, 1982). Preliminary attempts to validate fugacity models, however, show excellent agreement with measured environmental concentrations and estimates of human exposure (Hattemer-Frey and Travis, 1989; Mackay *et al.*, 1985a; Travis and Hattemer-Frey, 1987). Thus, multimedia transport models represent a useful tool for estimating the partitioning and fate of organics released into the environment, which is crucial if human exposures to organics from multiple media are to be accurately evaluated.

Mackay *et al.*'s Level III Fugacity model was modified to account for the uptake of organics through the food chain (Hattemer-Frey and Travis, 1989; Travis and Hattemer-Frey, 1987). Under conditions of continuous environmental release, the FFC model can be used to estimate unknown (or nondetectable) concentrations from (1) a chemical's physicochemical properties and (2) known (detectable) concentrations of a chemical in various environmental media, thus providing a coherent account of concentrations in all media (Travis and Hattemer-Frey, 1987). The FFC model estimates the concentration of a chemical in all six media and then uses those concentrations to predict the amount of chemical entering the food chain and the average daily intake of pollutant by the general population.

Input parameters required to predict the cross-media partitioning of a chemical include: (1) the physicochemical and biochemical properties of the compound; (2) estimates of emission rates for the compound into air, water, and soil; and (3) estimates of degradation rate coefficients for reactions that remove the compound from the system. The FFC model predicts the extent of human exposure to organic chemicals via inhalation and ingestion of contaminated produce, beef, milk, eggs, fish, and water.

V. UPTAKE OF ORGANICS THROUGH THE FOOD CHAIN

Evaluating the risks associated with exposure to chemicals through the food chain depends largely on the ability to predict their environmental fate and the extent to which they will bioaccumulate in the food chain.

A. Accumulation in Vegetation

Accumulation of organics in vegetation is a complex process that involves root uptake, foliar uptake (i.e., air-to-leaf transfer), and deposition onto outer plant surfaces (Figure 1).

1. *Root Uptake*

Root uptake of organics has been correlated with K_{ow} (Baes, 1982; Briggs *et al.*, 1982) and can be estimated from B_v, the soil-to-plant BCF. In the absence of measured data, B_v can be estimated from the geometric mean regression equation developed by Travis and Arms (1988). The concentration of organic in vegetation due to root uptake (CVR) on a dry weight (DW) basis can be estimated from the following equation:

$$\text{CVR (mol/kg)} = C_s * B_v \div D_s \, , \qquad (1)$$

where C_s equals the concentration of organic in soil (mol/m^3), and D_s is the bulk density of soil (1500 kg/m^3) (Hushon *et al.*, 1983; Mackay and Paterson, 1982).

2. *Foliar Uptake*

While some organics are taken up by plants directly from contaminated soil and translocated to upper plant parts, other compounds volatilize from the soil, and their vapors are absorbed by aerial plants parts (Bacci and Gaggi, 1985, 1986, 1987; Bacci *et al.*, 1989; Beall and Nash, 1971). Volatilization of organics from polluted soil may be as or more important than root uptake or direct deposition as a source of plant contamination. Buckley (1982), Nash and Beall (1970), and Mosbaek *et al.* (1988) reported that PCB, DDT, and mercury residues present in plant foliage were mainly due to vapor transport from the soil.

B_{va}, or the air-to-leaf BCF, is defined as equilibrium concentration of organic in upper plant parts (mg/g DW) divided by the concentration of organic in air as a vapor (mg/g). Bacci *et al.* (1989) determined the air-to-leaf BCF for ten organic compounds with a broad range of K_{ow} and H values. They found that B_{va} is best defined as K_{lw}/K_{aw}, where K_{lw} and K_{aw} represent the leaf-to-water and air-to-water partition coefficients, respectively. Intuitively, K_{lw} should be related to K_{ow}, while K_{aw} should be related to Henry's Law Constant (H) in atm-m^3/mol (Travis and Hattemer-Frey, 1988). Thus, B_{va} should be positively correlated with K_{ow}/H. Given this relationship, lipophilic chemicals must have a low H value to be accumulated to a large extent.

B_{va} can be estimated from the following geometric mean regression equation developed by Travis and Hattemer-Frey (1988) using the experimental data collected by Bacci *et al.* (1989) for ten chemicals with varying physicochemical properties:

$$\text{Log}\ (H * B_{va}) = -1.15 + 1.19\ \text{Log}\ K_{ow} \qquad (n = 10;\ r = 0.93)\ . \qquad (2)$$

Mackay (1982) and Halfon (1985) have argued that BCFs should be linearly related to K_{ow}, and Travis and Arms (1988) found that the relationship between BTFs for cow milk and beef were also linearly related to K_{ow}. Equation 2 indicates that this same relationship holds true for the bioconcentration of organics by aerial plant parts. We thus constrained the slope in equation 2 to be unity and refitted the data to yield:

$$B_{va} = 5.0\text{x}10^{-6}\ K_{ow} \div H\ . \qquad (3)$$

The concentration in vegetation due to the air-to-leaf transfer of organics (CVA) DW basis can be estimated from the following equation (Travis and Hattemer-Frey, 1988):

$$\text{CVA (mol/kg)} = C_a * F_v * B_{va} \div D_a \,, \qquad (4)$$

where C_a is the concentration of organic in air (mol/m^3), F_v represents the fraction of compound in air as a vapor, and D_a is the density of air (1.19 kg/m^3) (Mackay and Paterson, 1982).

3. Deposition

Deposition of organics onto outer plant surfaces can also contribute substantially to vegetative contamination. The concentration of pollutant on vegetation due to atmospheric deposition (CVD) DW basis can be estimated using the following equation derived from Travis *et al.* (1986):

$$\text{CVD (mol/kg)} = \frac{C_a * V_d * r/Y * F_p}{T½} \,, \qquad (5)$$

where: C_a = the concentration of organic in air (mol/m^3);

V_d = atmospheric deposition velocity (m/sec);

r/Y = vegetation-specific intercept fraction to productivity ratio (m^2/kg, DW basis) (Baes *et al.*, 1984);

F_p = fraction of chemical sorbed to particulates ($1 - F_v$);

$T½$ = weathering half-life (sec^{-1}) (Baes *et al.*, 1984).

In the absence of measured data, it is generally assumed that organics deposit onto plant surfaces at the rate of 0.0023 m/sec (Radian Corporation, 1987) and that organics deposited onto plant surfaces have a 14-day weathering half-life (Baes *et al.*, 1984).

Vegetation-specific differences in the intercept fraction for vegetation consumed by cows ($r/Y = 0.4\ m^2/kg$) and humans ($r/Y = 0.32\ m^2/kg$) (Baes *et al.*, 1984) require that different estimates of CVD be made for forage crops consumed by animals and exposed food crops consumed by humans. The total concentration of organic on vegetation consumed by animals and humans equals the sum of the individual contribution from all three pathways.

B. Accumulation in Beef and Milk

Kenaga (1980) and Travis and Arms (1988) showed that a positive correlation exists between K_{ow} and the bioconcentration of organics in cow whole tissue and milk. Biotransfer factors for organics in cow beef (B_b) and milk (B_m) can be estimated from the regression equation developed by Travis and Arms (1988) in the absence of measured data.

Ingestion of contaminated plants and soil can be a significant exposure pathway for terrestrial organisms, since lipophilic compounds, such as DDT and TCDD, and PCBs tend to accumulate in soil and plants. The predicted concentration of organic in cow beef (CB) and milk (CM) can be estimated from the following equation:

$$\text{CM or CB} = [(C_f * Q_f) + (C_g * Q_g) + (C_w * Q_w) + (C_s * Q_s) + (C_a * Q_a)] * B_m \text{ or } B_b, \quad (6)$$

where: CM or CB = the concentration of organic in milk or beef (mol/kg);

C_f = concentration of organic in forage (mol/kg);

Q_f = quantity of forage consumed by the animal (kg/day);

C_g = concentration of organic in grain (mol/kg);

Q_g = quantity of grain consumed by the animal (kg/day);

C_w = concentration of organic in water (mol/kg);

Q_w = quantity of water consumed by the animal (kg/day);

C_s = concentration of organic in soil (mol/kg);

Q_s = quantity of soil consumed by the animal (kg/day);

C_a = the concentration of organic in air (mol/m^3);

Q_a = quantity of air inhaled per day by the animal (m^3/day).

B_f = the chemical-specific BTF for cow adipose tissue (d/kg);

B_m = the chemical-specific BTF for cow milk (d/kg).

Since grains are protected by a husk, it was assumed that grains are not contaminated via the deposition or air-to-leaf transfer pathways (Stevens and Gerbec, 1988). Beef cattle destined for slaughter consume about 2.7 kg DW of forage and 5.0 kg DW grains per day, while dairy cattle consume about 7.1 kg DW of grain and 11 kg DW of forage per day (Shor *et al.*, 1982). Fries (1987) concluded that since lactating dairy cattle are rarely pastured, it is unlikely that soil ingestion would exceed 1% of dry matter intake. Similarly, although beef cattle do spend some time on pasture, they tend to pass most of their time confined to feed lots (Fries, 1987). Hence, we assumed that soil ingestion accounts for 1% of dry matter intake for beef and dairy cattle (0.08 and 0.18 kg DW/day) (Fries, 1987). Both feed lot and dairy cattle consume 1.5 kg of water and inhale 158 m^3 of air per day (Swenson, 1980).

C. Accumulation in Fish

Despite the low water solubility of many organic chemicals, fish can take up organics directly from contaminated water or from ingesting contaminated food items and concentrate them in their tissues at levels thousands of times greater than their concentration in water or food (Connolly and Pedersen, 1988; Isensee and Jones, 1975). Fish contamination levels are generally estimated by multiplying the concentration of chemical in water (C_w) times a chemical-specific BCF for fish. If measured BCFs for fish are not available, they can be estimated from the regression equation derived by Mackay (1982).

D. Estimating the Daily Intake of Pollutant

For risk assessment purposes, an important objective in evaluating the environmental fate of organic pollutants is predicting the major sources of human exposure. Table 3 lists the daily consumption values for various food groups of the U.S. diet (Yang and Nelson, 1986). Daily intake from food and water is estimated by multiplying the concentration of organic in beef, milk, vegetation, fish, and water (in mg/kg) times the average daily human consumption value (in kg/day). The concentration on vegetation consumed by humans was converted from a dry weight to a fresh weight basis using the conversion factors of 0.126 for above-ground vegetation and 0.222 for below-ground and protected vegetation (Baes *et al.*, 1984). The amount of organic inhaled is estimated by multiplying the concentration of organic in air (mg/m^3) times the average amount of air inhaled ($20\ m^3$) per day.

VI. APPLICATION OF THE FUGACITY-FOOD CHAIN MODEL: TCDD

Although there are actually more than 75 different molecular forms of dioxin, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) is often referred to as dioxin. TCDD is a highly lipophilic, extremely persistent compound that sorbs strongly to soil and sediment and bioaccumulates in the food chain. Because TCDD is the most potent chemical ever evaluated by the U.S. Environmental Protection Agency (US EPA, 1985), there is widespread fear that exposure to even small amounts of dioxin could lead to serious adverse human health effects.

A. Model Inputs

The physicochemical properties and degradation rates for TCDD are given in Travis and Hattemer-Frey (1987).

Measured concentrations of TCDD in air, water, and soil were used to estimate the distribution of release, assuming that measured environmental concentrations are in equilibrium with current environmental inputs. To calibrate the FFC model, emission rate estimates were adjusted so that environmental concentrations predicted by the model are consistent with the background concentration of TCDD in urban air (0.02 pg/m^3) measured by Rappe and Kjeller (1987) and the mean concentration in urban soil (1.0 to 1.4 ng/kg) reported by Nestrick *et al.* (1986). Total emissions of TCDD are estimated to be distributed between air, water, and soil in the ratio of 59:31:10, and the following emission rate estimates were used as inputs for the FFC model:

Air = 2.68×10^{-9} mol/hr ;

Water = 1.40×10^{-10} mol/hr ;

Soil = 4.41×10^{-10} mol/hr .

B. Environmental Fate of TCDD

The low solubility, low vapor pressure, and high octanol-water partition coefficient of TCDD result in its partitioning mainly between soil (86%) and sediment (13%) with less than 1% partitioning into air, water, suspended sediment, and biota. Table 4 shows that predicted environmental and food chain concentrations show reasonably good agreement with measured background values.

C. Uptake of TCDD in the Food Chain

Deposition of TCDD onto outer plant surfaces was expected to contribute substantially to vegetative contamination, since most of the TCDD deposited onto outer plant surfaces is likely to persist until the plant is harvested or consumed by animals (Isensee and Jones, 1971). Using the measured air concentration (C_a) of 0.02 pg/m^3 (Rappe and Kjeller, 1987), an F_p value of 0.6 (Bidleman, 1988), and a weathering half-life ($T_{1/2}$) of 14 days (5.7×10^{-7} sec^{-1}) (Baes *et al.*, 1984), the concentration of TCDD on forage due to deposition equals 19.3 pg/kg, and the concentration of TCDD on exposed food crops consumed by humans due to deposition equals 15.4 pg/kg.

Since TCDD is extremely lipophilic, and root uptake is inversely correlated with lipophilicity (Briggs *et al.*, 1982), root uptake was not expected to be a major source of vegetative contamination. Using the measured soil concentration of 0.96 ng/kg (Nestrick *et al.*, 1986) and a soil-to-root

BCF of 6.6×10^{-3} (Belcher and Travis, 1989), the concentration of TCDD in all plants due to root uptake is estimated to be 6.3 pg/kg.

Since TCDD is estimated to partition about 20% to 60% in the vapor phase in background urban air (Bidleman, 1988), air-to-leaf-transfer represented a potential major pathway of vegetative contamination. Using an air-to-leaf BCF of 9838 and assuming that the fraction of TCDD in the vapor phase is 40% (F_v = 0.4) (Bidleman, 1988), the concentration of TCDD on forage and exposed food crops due to uptake of TCDD vapors by aerial plant parts is 66.1 pg/kg.

Thus, the total concentration of TCDD on forage equals 91.7 pg/kg, 21% of which was due to foliar deposition, 72% of which was due to air-to-leaf transfer, and 7% of which was due to root uptake (Table 5). The total concentration of TCDD in exposed food crops equals 87.8 pg/kg. In this case, foliar deposition and air-to-leaf transfer accounted for 18% and 75%, respectively, of the TCDD contamination of exposed produce and vegetables consumed by humans, while root uptake accounted for only 7% (Table 5). The concentration of TCDD on grains and protected food crops (i.e., those plants contaminated via root uptake only) is 6.3 pg/kg (table 5).

Because of its high lipophilicity, TCDD readily accumulates in living organisms. Ingestion of contaminated plants and soil represents a major exposure pathway for terrestrial organisms. Using a steady-state daily intake-to-cow adipose tissue value of 0.8 d/kg and a daily intake-to-cow milk BTF of 0.03 d/kg (Washburn, 1989), the predicted daily intake of TCDD by beef and dairy cattle from inhalation and ingestion of contaminated water, soil, grains, and forage is given in Table 5. Ingestion of contaminated soil accounts for 21% and 14% of total daily intake of TCDD by beef and dairy cattle, respectively. These results confirm that ignoring soil ingestion as a pathway of exposure, especially when modeling lipophilic compounds, may lead to an underestimation of the concentration of organic in animal tissues (and subsequently in the amount humans take in as well). The vegetation ingestion pathway accounts for 82% of the daily TCDD intake by dairy cattle and 69% of the total daily intake by beef cattle.

The measured concentration of TCDD in whole eggs, 0.02 pg/g (Beck *et al.*, 1989), was entered into the FFC model.

TCDD levels measured in fish from lakes and rivers in the U.S. confirm that TCDD is bioaccumulating in fish and that low-level contamination of fish is widespread (Kaczmar *et al.*, 1985). Bioconcentration factors of 45 to 63,000 have been found in laboratory experiments that studied the accumulation of TCDD from water by fish (Adams *et al.*, 1986; Branson *et al.*, 1985; Isensee and Jones, 1975; Matsumura and Benezet, 1973; Yockim *et al.*, 1978). These reported BCFs, however, were determined through short-term studies in which the concentration of TCDD in fish may not have reached equilibrium. Travis and Hattemer-Frey (1989) estimated that the steady-state BCF for TCDD in fish is 100,000. Using this BCF, the predicted concentration of TCDD in fish (0.3 ng/kg, Table 4) agrees well with the measured value of 0.22 ng/kg (US EPA, 1987).

D. Extent of Human Exposure

Table 7 gives predicted average daily intake of TCDD by humans from air, water, and food. These data show that the food chain, especially meat and dairy products, accounts for 99% of human exposure to TCDD. Consumption of contaminated eggs, water, and fish are not major sources of human exposure to TCDD. The average, long-term intake be the general population of the U.S. is estimated to be 44 pg/day.

Ultimate proof of the widespread exposure to TCDD lies in the ability to detect TCDD in virtually all human adipose tissue samples. TCDD levels in human adipose tissue samples from non-occupationally exposed individuals in the U.S. and Canada ranged from 1.9 to 20.5 pg/g, with a geometric mean concentration of 7.4 pg/g (Graham *et al.*, 1985; Patterson *et al.*, 1986; Ryan *et al.*, 1985).

To verify the model estimate, the average daily intake of TCDD was also estimated using a linear, one compartment pharmacokinetic model (Geyer *et al.*, 1986):

$$I = \frac{(m_{ss})\ (\ln 2)}{T\frac{1}{2}}, \qquad (8)$$

where m_{ss} equals the total body burden of TCDD, and $T\frac{1}{2}$ is the half-life of TCDD in the human body, which was estimated by Poiger and Schlatter (1986) to be 2120 days. Assuming that the average human weighs 70 kg, has 20% body fat, and has 7.4 ng TCDD per kg fat, then the total body burden of TCDD is 104 ng, and the estimated long-term, average intake of TCDD is 34 pg per day.

In summary, two independent methods were used to estimate the long-term average daily intake of TCDD by the general population of U.S. An estimate based on the half-life of TCDD in the human body yields an intake estimate of 34 pg/day. Using measured and predicted concentrations of TCDD in various environmental media (the fugacity approach) yields an estimate of 44 pg/day. Both of these estimates agree well with Beck *et al.*'s (1989) market basket estimate of 25 pg/day.

VIII. APPLICATION OF THE FFC MODEL: PENTACHLOROPHENOL

The vast majority of the 23 million kg of pentachlorophenol (PCP) produced annually in the U.S. is used as a wood preservative (Cirreli, 1978). The fact that PCP has been measured in virtually all media, including human and animal tissues, indicates that humans are continuously being exposed to quantifiable levels of PCP (Hattemer-Frey and Travis, 1989).

The physicochemical and biochemical properties and emission and degradation rate estimates for PCP are given in Hattemer-Frey and Travis (1989). Results show that PCP partitions mainly into soil (97%) with 2% partitioning into water and less than 1% into air, sediment, suspended sediment, and biota. Table 8 shows that predicted environmental concentrations show relatively good agreement with measured background levels.

A. Uptake of PCP in the Food Chain

Since very little PCP (only about 10%) is sorbed to particulates, deposition was not expected to be a major pathway of vegetative contamination. Using the predicted concentration of PCP in air (0.17 ng/m^3), an F_p value of 0.9 (Bidleman, 1988), and a weathering half-life ($T\frac{1}{2}$) of 33 days (Hilton *et al.*, 1970), the concentration of PCP on forage due to deposition equals 0.08 μg/kg, and the concentration on exposed produce due to deposition equals 0.07 μg/kg (Table 5).

Although PCP readily adsorbs to roots, it is generally not translocated to upper plant parts (Casterline *et al.*, 1985). Using a soil-to-root BCF of 1.4 (Casterline *et al.*, 1985), the concentration of PCP in all plants due to root uptake is estimated to be 46.7 μg/kg.

Since PCP is estimated to partition primarily in the vapor phase (Bidleman, 1988), air-to-leaf-transfer was the major pathway of vegetative contamination. Using an air-to-leaf BCF of 8190 and assuming that the fraction of PCP in the vapor phase is 90% (Bidleman, 1988), the concentration of PCP on exposed produce and forage due to uptake of PCP vapors by aerial plant parts is 1.1 μg/kg.

Thus, the total concentration of PCP on forage and exposed produce equals 48 μg/kg, 97% of which was due to air-to-leaf transport, 2% of which was due to deposition, and 1% due to root uptake (Table 5). The total concentration of PCP on grains and protected food crops (i.e., those plants contaminated via root uptake only) is 47 μg/kg (Table 5).

Hattemer-Frey and Travis (1989) reported that predicted environmental concentrations showed good agreement with measured data with the exception of the concentration in cow milk. It was hypothesized that the BTF for cow milk was derived from a short-term study in which equilibrium tissue levels may not have been reached. Hence, we estimated more reasonable BTFs using the known concentration of PCP in cow milk. Rappe *et al.* (1987b) reported that the background concentration of PCP in whole cow milk was 15 μg/kg. Dividing this measured value by the total daily intake of PCP by dairy cattle predicted by the FFC model (865 μg/day, Table 6), a daily intake-to-cow BTF value of 0.017 d/kg. Since Travis and Arms (1987) showed that the BTF for cow beef is 3.2 times larger than the BTF for cow milk, a daily intake-to-cow beef BTF of 0.055 d/kg was assumed.

The predicted daily intake of PCP by beef and dairy cattle from inhalation and from ingestion of contaminated water, soil, grains, and forage is given in Table 6. These data show that ingestion of contaminated forage was the major pathway of exposure for dairy cattle accounting for 61% of total exposure, while ingestion of contaminated grains accounted for 64% of total daily intake of PCP by beef cattle. Unlike TCDD, ingestion of contaminated soil did not contribute substantially to PCP intake by cattle.

Again, the measured concentration of PCP in whole eggs, 0.95 μg/kg (OME, 1988), was entered into the FFC model.

Using a BCF of 5630 for PCP in fish, the predicted concentration in fish is 46 μg/kg (Table 8), which agrees well with the estimate of 50 μg/kg reported by Pierce *et al.* (1977).

B. Average Daily Intake of PCP by Humans

Table 9 show that the food chain, especially produce and dairy products, accounts for 99.9% of human exposure to PCP. Unlike TCDD, consumption of contaminated beef was not a major pathway of human exposure to PCP, accounting for only 15% of total daily intake. The average, long-term intake is estimated to be 12 μg/day, which agrees well with the estimate of 19 μg/day reported by Geyer *et al.* (1987) and an estimate of 11 μg/day based on urine excretion (Hattemer-Frey and Travis, 1989).

[Note that discrepancies in the results obtained for TCDD and PCP outlined in the previous sections versus results described in Travis and Hattemer-Frey (1987) and Hattemer-Frey and Travis (1989) reflect the modifications made to the FFC made since that paper was published and the use of more current chemical-specific data.]

X. GUIDELINES FOR ESTABLISHING MULTIMEDIA STANDARDS

An important objective in evaluating contaminant levels in environmental media is estimating the concentration that will not pose an unacceptable risk to humans. Traditionally, regulatory guidelines have been promulgated on a medium-by-medium basis and have focused on sources rather than exposures. The reliance on single-medium exposures to regulate toxic pollutants is misleading and not protective of human health, given that multimedia exposures are the rule and not the exception (Travis *et al.*, 1987). For example, the US EPA strives to limit inhalation exposure to TCDD to a lifetime excess cancer risk of $1x10^{-6}$ while neglecting a background ingestion risk to the U.S. population of $1x10^{-4}$ [background daily intake equals $7.1x10^{-10}$ mg/kg-day (Travis and Hattemer-Frey, 1987) times the cancer potency factor for TCDD ($1.56x10^5$ mg/kg-day^{-1})], which could result in more than 300 additional cancer deaths per year ($1x10^{-4}$ lifetime cancer risk x $2.2x10^8$ U.S. residents ÷ 70 years). If regulatory agencies are to fulfill their mandate of protecting human health, they must adopt a multimedia approach to setting environmental standards.

Adopting a multimedia approach enables regulators to establish guidelines that take into account the significance of each exposure pathway, since the principal pathway of human exposure is certain to differ for the broad spectrum of organics to which humans are routinely exposed. The initial apportionment of allowable daily intake between relevant exposure media should be made on the basis of the percentage of total exposure that each pathway is estimated to contribute to the exposure of the general population based on survey data, modeling techniques, or a combination of the two. Subsequent apportionments could be made to reflect intakes by subpopulations (e.g., children and infants) and limitations of existing analytical capabilities (e.g., some apportionments could result in a value below current limits of detection).

A. Setting Standards Based on Environmental Partitioning

The first step in determining maximum tolerable environmental concentrations is to evaluate, using survey data and modeling techniques, the environmental partitioning and subsequent average background daily intake of pollutants chronically released into the environment. Secondly, the relative contribution of each exposure pathway to total daily intake can be determined by dividing average daily intake via a specific pathway (i.e., air, water, or food) by total daily intake of pollutant. To apply this approach to TCDD, we multiplied the concentration of TCDD in air, water, soil, and food listed in Table 4 (as estimated from measured data and model predictions) by the average daily U.S. consumption rates for adults (Table 3) to quantify the total daily intake of TCDD by the general population of the U.S. and the contribution of each exposure pathway (Table 7). Thus, exposure to TCDD can be apportioned in the following manner:

Air - 1.0%
Food - 98.9%
Soil - 0.05%
Water - 0.01% .

These data show that direct ingestion of contaminated soil and water are negligible pathways of human exposure.

Since maximum environmental concentrations will partition in the same ratio as background environmental contamination, the ratio of the maximum allowable concentration of a compound to its background concentration will be equal to the ratio of the maximum acceptable daily intake divided by the background daily intake. Again, using TCDD as an example, the background daily intake of TCDD by the general population of the U.S. is estimated to be 44 pg/day (0.63 pg/kg-day). Assuming that the maximum allowable daily intake of TCDD is 10 pg/kg-day (Kociba *et al.*, 1978), the maximum tolerable daily intake for an individual weighing 70 kg (700 pg/day) is 15.9 times higher than the average background daily intake. Multiplying background environmental levels of TCDD by 15.9 yields the estimates of maximum tolerable environmental concentrations. These maximum tolerable environmental concentrations are equivalent to the following recommended guidelines for limiting human exposure:

Air = 0.32 pg/m^3
Water = 0.05 pg/L
Soil = 0.015 µg/kg .

These recommendations derived from the FFC model are based on an estimate of the maximum allowable daily intake (10 pg/kg-day), reflect the significance of each exposure pathway to total exposure, and could be readily adjusted should new data concerning the environmental partitioning or the acceptable daily intake of a chemical emerge.

The Federal-Ontario Ad Hoc Working Group (1988) considered the food chain as the primary pathway of human exposure to TCDD in setting maximum environmental standards. While we acknowledge that any attempt to set standards based on multimedia exposures is a complicated and controversial process, we do not support the Working Group's arbitrary increase in maximum tolerable soil and water concentrations to avoid setting standards that are below current detection limits. Our analysis shows that maximum levels proposed by the Working Group may not adequately and fully protect public health, since maximum tolerable levels of TCDD in air, water, and soil predicted by using the environmental partitioning approach are 15, 200, and 50 times lower, respectively, than the maximum tolerable levels suggested by the Working Group. This discrepancy is attributable to the Working Group's arbitrary assumption that an apportionment of less than 5% for water would result in a criterion that is below current detection limits, an assumption dictated by the fear that standards set below current detection limits would not be enforceable. We judge this preference unjustified since levels proposed by the Working Group would allow environmental concentrations to increase to the point where average daily intake of TCDD by humans would exceed threshold levels.

Figure 2 shows that the mean background concentration of TCDD in urban soil is about 1 pg/g (Nestrick *et al.*, 1986; Rappe and Kjeller, 1987). Using an environmental partitioning approach, we conclude that mean levels of TCDD in urban soil should not exceed 16 pg/g if the average individual's food chain exposure is to remain below threshold levels. We acknowledge, however, that soil concentrations will vary both temporally and spatially. Our approach allows regulators to account for such variability by presenting a distribution of maximum tolerable soil concentrations. Given that background environmental concentrations will vary, regulatory agencies may not be required to limit environmental concentrations immediately. For example, the Canadian government could adopt the limit proposed the Federal-Ontario Working Group as an initial standard. The F-O Working Group's limit of 100 pg/g is acceptable only if 10% of background sites tested do not exceed the proposed limit. Figure 2 shows that if 10% of the background urban soil samples have a TCDD content greater than 100 pg/g, then mean soil concentrations will exceed 16.2 pg/g and food chain intakes of TCDD will exceed maximum tolerable levels.

Using the total daily intake of 25 pg/g (0.36 pg/kg-day) reported by Beck *et al.* (1989), exposure to TCDD can be apportioned as:

Air - 1.6%
Food - 98.3%
Soil - 0.08%
Water - 0.02% .

Again, assuming that the maximum allowable daily is 700 pg/day, the maximum tolerable daily intake is 28 times higher than the average background daily intake reported by Beck *et al.* (1989). Multiplying background environmental levels by 28 yields the following apportionment:

Air - 0.56 pg/m^3
Water - 0.08 pg/L
Soil - 0.03 μg/kg .

These limits are 10, 215, and 50 times lower than the maximum tolerable concentrations proposed by the F-O Working Group. In this case, the F-O

Working Group's 100 pg/g limit is acceptable only if 20% of the background sites tested do not exceed the proposed limit. Figure 3 shows that if 20% of the background soil samples analyzed have a TCDD concentration greater than 100 pg/g, then mean soil concentrations and food chain intakes will exceed acceptable levels.

XI. CONCLUSIONS

Evaluating the risks associated with exposure to environmental pollutants involves identifying the sources and magnitude of environmental input, the transport and transformation of chemicals released into the environment, the pathways of human exposure, and the extent of exposure for populations at risk. Since organic chemicals tend to end up in the media in which they are most soluble, a few basic physicochemical properties can be used to predict the behavior and fate of chemicals released into the environment. Highly lipophilic compounds tend to sequester in soil, sediment, and biota, and the food chain is the primary pathway of human exposure. Volatile compounds tend to partition almost exclusively into air, and inhalation is the major source of human exposure.

Multimedia transport models, such as the FFC model, can be used to study the environmental fate of various pollutants released into the environment, their accumulation in the food chain (agricultural produce, beef, milk, and fish), and the extent and pathways of human exposure. Our results show that the food chain accounts for 99% of human exposure to TCDD and PCP.

Multimedia transport models represent useful tools for regulating human exposure to organic chemicals. Adopting a multimedia approach enables regulators to establish guidelines that take into account the significance of each exposure pathway, since the principal pathway of human exposure is certain to differ for the broad spectrum of organics to which humans are routinely exposed. These maximum tolerable environmental concentrations are equivalent to the following recommended guidelines for limiting human exposure:

Air = 0.32 pg/m^3
Water = 0.05 pg/L
Soil = 0.02 μg/kg .

The recommendations derived from the FFC model are based on an estimate of the maximum allowable daily intake (10 pg/kg-day), reflect the significance of each exposure pathway to total exposure, and could be readily adjusted should new data concerning the environmental partitioning or acceptable daily intake of a chemical surface.

APPENDIX A: ESTIMATING THE MEAN OF DATA SETS WITH NON-DETECTABLE VALUES

Interest in determining trace levels of contaminants in environmental media has increased with the recognition that even trace levels of pollutants can pose risks to human health and the environment. The analysis of trace level environmental data is frequently hampered, however, by chemical concentrations that are below detection limits established by analytical laboratories (Gilliom and Helsel, 1984; Porter et al., 1988). Traditionally, to estimate the mean concentration from such data, investigators assumed that all nondetectable values were (1) equal to zero (Arruda *et al.*, 1988; US EPA, 1980), (2) equal to half the detection limit (Staples *et al.*, 1985; Wallace *et al.*, 1987), or (3) equal to the detection limit (Birmingham *et al.*, 1988; Drury *et al.*, 1981). Such assumptions result in either an under- or an over-prediction of the true mean and standard deviation of the data (Porter *et al.*, 1988). We propose an alternative approach of estimating the mean that accounts for both detectable and nondetectable values.

Log-probit analyses have been shown to be a robust method for estimating the geometric mean and standard deviation of samples with values in the non-detectable range (Gilliom and Helsel, 1984). Although this method was introduced in the mid-1970s to analyze radiation data with nondetectable values (Denham and Waite, 1975; Gilbert and Kinnison, 1981; Miller *et al.*, 1977; Waite *et al.*, 1980) and has been used in connection with environmental pollution monitoring (Gilliom and Helsel, 1984; Porter *et al.*, 1988; US EPA, 1980), it has yet to receive the wide acceptance it deserves in risk assessments for chemical carcinogens.

In a log-probit analysis, all measurements (both detectable and nondetectable) are assumed to be taken from the same lognormal probability distribution. The assumption of lognormality for environmental data is fairly universal. The concentration of many compounds in air (Bencala and Seinfeld, 1976; Nero, 1985; US EPA, 1980; Wallace *et al.*, 1985), drinking water (Cothern *et al.*, 1986; Symons et al., 1975), and human biological samples (Grasmick and Huel, 1985; McLauglin *et al.*, 1973; Patterson *et al.*, 1986; US EPA, 1980; Wallace *et al.*, 1985) have been shown to be lognormally distributed. When samples from a lognormal distribution are plotted on a probit scale, they tend to lie on a straight line (Grosjean, 1983). In a probit analysis, nondetectable values are treated as unknowns, but their percentile values are accounted for. Thus, if there were 100 samples 30 of which were nondetectable, the first detectable data point would be plotted at the thirty-first percentile. If sufficient detectable values exist, they can be used, through linear regression, to define the line characterizing the entire data set. The geometric mean concentration of all data points (both detectable and nondetectable) is determined from the 50th percentile value or a probit value of five. The standard deviation can be estimated from the antilog of the slope of the regression line. Thus, a probit analysis allows the geometric mean to be extrapolated from detectable values even if it is below detection limits.

The following example illustrates the advantage of using a log probit analysis to estimate the geometric mean of data sets with nondetectable values. Because of its extreme toxicity, much concern and debate has arisen about human exposure to TCDD. Levels measured in fish taken from lakes and rivers in the U.S. confirm that TCDD is bioaccumulating in fish and that low-level contamination of fish is widespread (Kaczmar *et al.*, 1985). Many state and federal agencies have issued guidelines restricting or banning the consumption of TCDD-contaminated fish. The U.S. Food and Drug Administration (FDA), for example, suggests that a concentration of more than 25 picograms (pg) TCDD per gram (g) of fish fillet poses an unacceptable risk to humans (increased lifetime cancer risk equals $3x10^{-6}$) (FDA, 1981; Miller, 1983).

As part of their National Dioxin Study, the US EPA (1987) analyzed TCDD concentrations in fish sampled from 304 urban sites located near population centers or areas with known commercial fishing activity, including sites from the Great Lakes Region. None of the urban sites tested had a previous known source of TCDD contamination. Bottom feeders were analyzed first, because they were suspected to contain higher levels of TCDD than predator (or game) fish (US EPA, 1987).

Figure 3 presents the cumulative probability distribution of TCDD contamination (pg/g wet weight, WW) in bottom feeding fish fillets (n = 51) sampled from urban sites. TCDD concentrations in fish fillets ranged from nondetectable (71%) to 64 pg/g. The geometric mean (extrapolated from detectable concentrations) is estimated to be 0.4 pg/g (Figure 3). Assuming that all nondetectable values are equal zero and taking the average of all detectable measurements overestimates the true mean by a factor of 18 (average of detectable values equals 7.0 pg/g). Taking the geometric mean of all detectable values only (n = 18) overestimates the true mean by a factor of eight (geometric mean of detectable samples only equals 3.1 pg/g). Assuming that all nondetectable samples are equal to half the detection limit yields the estimate closest to the true mean (0.78 pg/g), while assuming that all nondetectable samples are equal to the detection limit overestimates the true mean by a factor of three (geometric mean equals 1.2).

Log-probit analysis provides a robust method of evaluating environmental data sets when a large percentage of values are below analytical detection limits. Applying the log-probit method is easy, and it provides a less biased and more accurate estimate of the mean than other, more frequently used methods (Gilliom and Helsel, 1984). We, therefore, advise that it become the method of choice for analyzing trace level environmental data.

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Table 1

Occurrence of selected organochlorine compounds in human adipose tissue

Compound	Frequency of detection	Concentration (ng/kg)
Benzene	96	ND - 97
Chlorobenzene	96	ND - 9
Toluene	91	ND - 250
PCBs	83	ND - 1700
Hexachlorobenzene	76	ND - 1300
Heptachlor epoxide	67	ND - 310
Tetrachloroethylene	61	ND - 93
DDT	55	ND - 540

Source: National Human Adipose Tissue Survey (US EPA, 1986)

Table 2

Daily dietary intake of selected organic chemicals (micrograms/day)

Chemical	FY 78	FY 81/82
ß-benzenehexachloride	0.06	0.03
DDT	0.56	0.07
Dieldrin	1.19	1.12
Heptachlor epoxide	0.56	0.14
Hexachlorobenzene	0.28	0.14
PCBs	1.89	0.21
Pentachlorophenol	ND	3.64
trans-nonachlor	0.07	0.07

Source: Gartrell *et al.*, 1986

Table 3

Average daily U.S. human consumption values

Food Group	Daily intake (g/day fresh weight)
Leafy and exposed vegetables	126
Protected produce	222
Grains	125
Milk and dairy products	309
Beef	88
Eggs	27
Fish	18
Water (tap water, water-based drinks, and soups)	1430

Source: Yang and Nelson, 1986

Table 4

Predicted and measured environmental concentrations for TCDD

(nanograms per kilogram except where noted)

Phase	Predicted concentration	Background concentration	Reference for measured value
Air	0.02[a]	0.02[a]	Rappe and Kjeller, 1987
Water	0.003[b]	<10	Birmingham *et al.*, 1988
Soil	0.96	0.96	Nestrick *et al.*, 1986
		1.42	Rappe and Kjeller, 1987
Sediment	0.30	1.2	Rappe *et al.*, 1987a
		2.7	Norwood *et al.*, 1989
Susp sediment	0.34	0.53[c]	Czuczwa *et al.*, 1984
Fish	0.28	0.40	Travis & Hattemer-Frey, 1989
		0.22	US EPA, 1987
Forage	0.09	NA[d]	
Grain	0.01	NA	
Exposed produce	0.09	0.005	Beck *et al.*, 1989
Beef	0.28	0.15	Beck *et al.*, 1989
Milk	0.04	0.008	Beck *et al.*, 1989
Eggs	0.02	0.02	Beck *et al.*, 1989

[a] Picograms per cubic meter. [b] Picograms per liter.

[c] Concentration of all tetrachlorinated dioxins (TCDDs).

[d] Measurement not available.

Table 5

Contribution of root uptake, deposition, and foliar absorption to total vegetative contamination by TCDD and PCP

	Exposed produce	Percent	Protected produce/ grains	Percent	Forage	Percent
TCDD	(ng/kg)		(ng/kg)		(ng/kg)	
Root uptake	6.4	7%	6.4	100%	6.4	7%
Deposition	18.8	21%	0	0%	23.5	24%
Air-to-leaf	66.2	72%	0	0%	66.2	69%
TOTAL	91.4	100	6.4	100	96.1	100
PCP	(μg/kg)		(μg/kg)		(μg/kg)	
Root uptake	46.7	97%	46.7	100%	46.7	97%
Deposition	0.07	1%	0	0%	0.08	1%
Air-to-leaf	1.1	2%	0	0%	1.1	2%
TOTAL	47.9	100	46.7	100	48.0	100

Table 6

Intake of TCDD and PCP by beef and dairy cattle from inhalation and ingestion of contaminated water, soil, forage, and grain

Source	Intake by beef cattle cattle	Percentage of total intake	Intake by dairy cattle cattle	Percentage of total intake
TCDD	(pg/day)		(pg/day)	
Inhalation	3.2	1%	32	0.3%
Water	0.004	<0.01%	0.004	<0.01%
Soil	74	21%	173	14%
Forage	244	69%	1010	82%
Grain	32	9%	45	4%
TOTAL	353	100	1228	100
PCP	(μg/day)		(μg/day)	
Inhalation	0.03	<1%	30	<1%
Water	0.01	<1%	10	<1%
Soil	2.6	1%	6000	1%
Forage	1283	5%	528	61%
Grain	234	64%	333	38%
TOTAL	364	100	866	100

Table 7

Predicted average daily intake of TCDD by the general population of the U.S.

Source	Daily Intake (pg/day)	Percent of total daily intake
Air	0.4	1%
Water	0.004	0.01%
Food (Total)	43.5	99%
Produce	2.0	4%
Milk	11.4	26%
Beef	24.8	57%
Fish	4.8	11%
Eggs	0.5	1%
TOTAL	44	100%

Table 8

Predicted and measured environmental concentrations for PCP

(micrograms per kilogram except where noted)

Phase	Predicted concentration	Measured background concentration	Reference for measured value
Air	0.17[a]	0.14[a]	Bevenue *et al.*, 1976
Water	8.2[b]	8.3[b]	Murray *et al.*, 1981
Soil	33.4	33.3	Arsenault, 1976
Sediment	0.7	0.8	Wegman & van den Broek, 1983
Susp sediment	0.7	0.5	Pierce *et al.*, 1977
Fish	46.2	50	Pierce *et al.*, 1977
Forage	47.9	50	Firestone *et al.*, 1979
Grain	46.7	50	Firestone *et al.*, 1979
		4.8	OME, 1988
		17.0	Manske and Johnson, 1975
Produce	47.9	4.8	OME, 1988
Root crops	46.7	0.5	OME, 1988
		10.0	Manske and Johnson, 1975
Beef	20.1	2.8	OME, 1988
Milk	15.0	15.0	Rappe *et al.*, 1987
		10.0	Manske and Johnson, 1975
Eggs	0.95	0.95	OME, 1988

[a] Micrograms per cubic meter. [b] Micrograms per liter.

Table 9

Total intake of PCP be the general population of the U.S.

Source	Daily intake (μg/day)	Percent of total intake
Inhalation	0.003	<1%
Water	0.01	<1%
Food	12.0	99.9%
Dairy	4.6	38%
Beef	1.8	15%
Produce	4.4	37%
Fish	0.8	7%
Eggs	0.4	3%
TOTAL	12.0	100