PCDD/Fs in the UK: Quantifying the Link Between Emissions and Human Exposure

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Abstract

We have made a quantitative estimation of how PCDD/Fs emitted to the UK environment effect human exposure by tracing their fate through the environment and into the human food-chain. We have reconstructed the mean annual air concentrations in the UK for the past 50 years. By incorporating these values into our model we have estimated a history of human exposure in the UK. From this we have calculated the expected serum concentrations of PCDD/Fs in the UK population according to age, and compared this to measured values. This is the first such integrated assessment of the link between sources and exposure for PCDD/Fs and, although many assumptions are made where information is lacking, the results have proved encouraging when tested against measured data.

Introduction

Polychlorodibenzo-p-dioxins and dibenzofurans (PCDD/Fs) are among the most toxic chemicals known to man. They are not produced intentionally but are formed as by-products of a number of industrial and combustion sources notably municipal waste incineration, metal smelting, pesticide manufacture and electricity generation. The major exposure pathway for humans is through consumption of contaminated food, principally dairy products, fish and meat. PCDD/F contamination of each of these food categories can be traced through a series of environmental pathways which are ultimately fed by the presence of PCDD/Fs in air. It is therefore difficult to exercise control over human exposure to PCDD/Fs other than by attempting to minimise their formation and emission to the atmosphere. In the UK, as in Japan, there has been a large investment over the past 10 – 20 years to identify the most important sources and apply improved technology to reduce PCDD/F emissions. This has been effective in bringing about a decline in PCDD/F levels measured in human serum, milk and adipose. However, sections of the UK population are still estimated to exceed the tolerable daily intake

(TDI) for PCDD/Fs, as recommended by the World Health Organisation (WHO). The UK Government is interested in knowing whether or not further emission control measures will be required to ensure a higher compliance in the future. To answer this we have assessed how much PCDD/F is released to the environment and how much of this the human population is exposed to. Our approach has been to form an understanding of the pathways which lead emitted PCDD/Fs to be incorporated into foodstuffs, and to study the individual sections of these pathways quantitatively. By recombining these sections we have attempted to quantify the link between emissions and human exposure to PCDD/Fs.

1 Identification and quantification of pathway from emissions to human exposure

1.1 *Diet*

PCDD/Fs are highly hydrophobic, semivolatile organic chemicals. Our main exposure to such chemicals is through dietary intake. A survey of the UK dietary habits coupled with studies of PCDD/Fs in major food types shows that the principal source of PCDD/Fs to the general population comes from milk and dairy products, with lesser inputs from fish and meat.

1.2 *Milk*

To investigate the factors controlling the concentrations of PCDD/Fs in cows' milk we conducted a three year study to look at how persistent organic contaminants behave in the lactating cow. Study animals were treated according to usual husbandry practice along with the rest of the herd. Their feed intake, weight and fat content were monitored and samples of their milk and faeces were collected over an 18 month period. From this study we obtained information on the efficiency of uptake of POPs across the gut wall, the extent of storage of POPs in the cow's adipose and other organs, the degree of metabolism of POPs by the cow and the transfer of POPs into its milk. For a set period of the study some cows were given relatively highly contaminated feedstock. The rate at which milk concentrations rose helped to develop a steady state model for the dynamics of POPs in the lactating cow. This enabled us to calculate the concentrations of PCDD/Fs in milk if we knew their concentration in the feedstock.

1.3 *Air/Grass*

In the UK dairy cows feed on fresh grass through the summer months and this is supplemented with silage during the winter. We studied the uptake of POPs by grass from the air in detail. Grass grows rapidly in the spring and early summer, some of which is cut and stored for use in the winter. We needed to understand how quickly the concentrations of PCDD/Fs in grass would respond to changes in air concentration, how much contamination the grass would accumulate over the winter months, whether fast new growth would have the effect of diluting the PCDD/F burden of the sward, how quickly newly grown grass would become contaminated and whether different species of grass would behave differently.

We found that the PCDD/Fs equilibrated between grass and air within two weeks. The grass would therefore even out any large fluctuations in air concentrations. Also the first spring cut would not contain an excess burden of PCDD/Fs accumulated over the winter, and new growth was sufficiently gradual to keep pace with contamination. Finally, we observed some small variation between species. From our results we could determine an effective relationship linking grass PCDD/F concentrations with air concentrations and temperature.

1.4 *Atmospheric fate*

A separate section of our research group have been studying the fate of POPs in the atmosphere. This has involved monitoring the concentrations of PCDD/Fs (and many other compounds) continuously at the Lancaster University field station for the past eight years. More recently we have intensified our monitoring campaign to study seasonal and diurnal changes in air concentrations of PCDD/Fs, the effect of temperature and air-mass origin. Whether the PCDD/Fs are in the free vapour phase of the air, or associated with airborne particles is critical to their fate in the atmosphere. Vapour phase PCDD/Fs are subject to degradation by reaction with OH radicals. They are available for absorption by plant surfaces and the organic fraction of soils. They will equilibrate between the air and water bodies and, to a minor extent, will be incorporated into rain. Particle bound PCDD/Fs are subject to both wet and dry deposition, and a large proportion of PCDD/Fs emitted to the atmosphere on particles will be deposited close to their source.

We used a McKay type fugacity model to describe atmospheric fate of PCDD/Fs from a general emission source. The model uses meteorological parameters such as particle deposition velocities and rainfall statistics, as well as physicochemical properties of the various PCDD/F compounds such as Henry's Law constants, partial pressures, octanol/water partition coefficients, octanol air/partition coefficients and rates of OH radical degradation, either taken from the literature or measured by our group. The model also took into account losses of PCDD/Fs due to long-range transport of air-masses away from the UK.

1.5 *Emission inventories*

Ruth Alcock recently published a congener specific inventory of PCDD/F emissions to the UK for 1996. This was based on a comprehensive set of measured emission data for many of the activities identified as major primary sources of atmospheric PCDD/F emissions. For some of the major known sources, where no direct data was available for the UK, emission factor ranges from the US EPA Inventory of Dioxin Sources Draft Review (1998) were used to estimate the output of such sources to the UK atmosphere. These emission data were entered into the atmospheric fate model to generate estimated air concentrations of several 'indicator' PCDD/F congeners. These were compared to air concentrations measured at the Lancaster Universtity field station. For many of the indicator congeners agreement between measured and predicted concentrations was found to be within a factor of two. For a few congeners, notably OCDD,

there was a large discrepancy. This could either mean that we have not identified all the OCDD emission sources or that our understanding of the fate of these congeners is less accurate.

1.6 *Estimation of PCDD/Fs in the UK diet*

Using the emission inventory data as input data to the subsequent set of calculations we found that the amount of OCDD in 1g milk fat was equivalent to that in $42m^3$ of air. Values obtained for a series of other indicator congeners ranged by a factor of four.

The dairy cow model was modified by eliminating lactation, altering the growth rate, fat accumulation and age of the animal to simulate the uptake of PCDD/Fs by cattle reared for beef production. For the purpose of our study we used beef to represent all 'meat' food products.

To derive estimates for human dietary exposure through consumption of fish, bioconcentration factors for individual PCDD/F congeners were taken from the European Union System for the Estimation of Substances (EUSES). These calculations could be related to air concentrations if it was assumed that these were in equilibrium with surface water concentrations and suspended particulate material, and that these, in turn, were at equilibrium with surface sediment.

1.7 *Human exposure*

These three food categories have been reported as the most significant dietary sources for PCDD/F exposure in the UK. By combining the estimated PCDD/F concentrations in these foods with information about the amounts of each food-type consumed by a typical person in the UK we calculated their exposure to PCDD/Fs.

2 Past exposure and its implications for human PCDD/F burdens

For contaminants that are as persistent as PCDD/Fs, human burdens can not be estimated from estimates of present day exposure. Past exposure has an important impact on both current and future body burdens. To investigate past exposure we reconstructed the air concentrations of PCDD/Fs in the UK for the past fifty years using three independent methods.

2.1.1 *Estimated past emission inventory*

We recalculated the UK PCDD/F emission inventory for 1990 based on measurements of PCDD/F emissions measured in 1990, and scaling each source activity appropriately to their relative size of operation in 1990.

2.1.2 *Historical sediment deposition record*

A record of atmospheric particle fallout is maintained in lake sediments. By analysing PCDD/Fs in sections of dated sediment cores we were able to reconstruct the atmospheric concentrations of PCDD/Fs over the past 150 years. This reconstruction also relied on our

knowledge of vapour/particle distribution of PCDD/Fs in the air, and on data for particle deposition velocities over the lake.

2.1.3 *Archived vegetation*

We measured PCDD/Fs in 16 grass samples that were collected at the same time each year and archived by the Institute of Arable Crop research in southern England. Reversing the direction of our air:grass model we again reconstructed the UK PCDD/F air concentrations over the period 1980 to 1995.

The reconstructed levels of PCDD/Fs in air from these three techniques are in agreement with each other and define a time trend that predicts concentrations in air for 1998 that match what we have measured. The sediment core data was therefore considered a reasonable record of PCDD/F air concentrations and these were used to estimate time trends of concentrations of PCDD/Fs in dairy, meat and fish.

2.2 *Historical exposure of the UK population to PCDD/Fs*

We also had access to trends in dietary habits of the UK population over these fifty years. From this we calculated the life-history of PCDD/F exposure and body burden for a typical person born in each year from 1920 to 1980. People's food consumption varies with their age. To take account of this we incorporated into our calculations published measured data for the variation of body weight and body fat with age. We have not been able to measure human uptake efficiencies for PCDD/Fs from food but, based on our measurements for the uptake of PCBs, we used a blanket value of 50% for all PCDD/F congeners. Half-lives for the metabolism of PCDD/Fs in occupationally exposed humans has been estimated tentatively to be between ten and twenty years for various congeners. We used these reported values, although a sensitivity test of our model found that variation of these values had minimal impact on predicted body burdens.

2.3 *Comparison of predicted human PCDD/F burden with measured values*

From these calculations we derived estimates for the PCDD/F serum concentrations for a hypothetical UK population in 1990 aged between twenty and seventy. The accuracy of our calculations was assessed by comparison of our hypothetical population to levels measured in people of all ages from the German population in 1990 (no data was available for the UK). For several congeners the agreement was very good considering the number of stages in the prediction and the number of assumptions made. For other congeners our prediction overestimated the measured serum concentrations by a factor of five to ten. These congeners were generally those for which very large ranges were given for emission factor estimates.

This model is in its infancy but we are greatly encouraged by the results obtained so far. We are currently attempting to identify the largest sources of uncertainty and will conduct further research in these areas to improve the accuracy and credibility of this first draft.