EXPOSURE COMMITMENT APPROACH TO THE DERIVATION OF ENVIRONMENTAL ASSESSMENT LEVELS: FEASIBILITY STUDY

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Abstract -

The results of this work will be used in the formulation of Government Policy, but views expressed in the report do not necessarily represent Government Policy.

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1. INTRODUCTION

1.1 EXPOSURE COMMITMENT – TERMS AND DEFINITIONS

Exposure Commitment is a time-independent measure of the total human exposure to a pollutant. It is assessed by considering the behaviour of the pollutant through a pathway, or number of pathways, comprising a series of environmental compartments from source to final human exposure. The basis of the method is a model incorporating the concentrations of a pollutant in the source, pathway and receptor compartments, and transfer factors between compartments.

Together these define the pathway to exposure, and allow calculation of the final "commitment" to exposure. Exposure or intake commitments to the receptor compartment are evaluated by multiplying the sequence of transfer factors in the pathway chain. Where there are a number of parallel pathways, each is considered separately, and the contributions to intake or exposure from each pathway are added to give the total human exposure to the substance.

Intake: The amount (of food, air, water, soil, contaminant) consumed per unit time. Intake via foods or inhalation refers to the amount passing through the Gastro-Intestinal tract (GI Tract) or lung, respectively. It is not the same as exposure since only a proportion of what is taken in is absorbed across the GI Tract or lung membranes.

Exposure: The exposure is defined as the intake multiplied by a number of transfer factors (absorption factors, partitioning factors).

Transfer Factors: These relate to the steady state concentrations in two compartments (e.g., soil and crop), and may be plant uptake factors, GI Tract, lung absorption factors, or partitioning factors.

Body Burden: The total amount (mass) of contaminant in the body (e.g., 150 mg per person).

Critical Concentration: The concentration of a contaminant in the target organ/tissue at the time any of its cells reaches a concentration at which adverse functional changes, reversible or irreversible, occur in the cell (adapted from Task Group on Metal Toxicity, 1976).

1.2 CAPABILITIES – STRENGTHS AND LIMITATIONS

Exposure Commitment determines the partitioning of the pollutant in a number of parallel pathways, and the amounts which ultimately reach the receptor. All assessments of exposure to a substance for the general population should identify the relative importance of different pathways and important components of the exposure pathways, in addition to calculating the total exposure from all sources. Exposure Commitment meets all the requirements of a general exposure assessment, and therefore the capabilities of the method can be summarised as follows.

1. Evaluation of combined human exposure to a pollutant from different media.

2. Comparative assessment of the importance of various pollutant pathways to human exposure.
3. Evaluation of exposure to a pollutant for the compartments within the pathways.
4. Estimation of equilibrium concentrations resulting from continuous releases.
5. Consequences of release to different media.

Strengths

It has become increasingly clear that multimedia transport models, such as Exposure Commitment assessment, represent useful tools for regulating human exposure to pollutants (Travis and Hattemeyer-Frey, 1991). Adopting a multimedia approach allows regulators to establish guidelines that take into account the significance of each exposure pathway, and this is important as the principal pathway to human exposure will vary for the wide range of contaminants in the environment.

Exposure Commitment is a time-independent assessment of total human exposure to a pollutant. Unlike time-dependent (dynamic) models, it does not require evaluation of actual transfer rate constants and compartment sizes, or a complete understanding of the complex physical and chemical mechanisms of transfer in the environment. In comparison to dynamic models it can proceed on a relatively limited database.

In addition, when using Exposure Commitment it is possible, in constant emission situations, simply to use equilibrium concentrations for the purpose of determining transfer factors, and therefore in calculating the environmental levels of a substance that result in a given level of exposure. Consequently, existing data on a particular substance and a general understanding of its behaviour in the environment and man can form a satisfactory basis for an Exposure Commitment.

Assumptions and Limitations

As with all models of environmental behaviour and exposure to a substance, the compartmental models produced using Exposure Commitment are a compromise between a sufficiently detailed and realistic representation, and a simplified, manageable version. The major compartments considered in exposure assessment are the atmosphere, oceans, soils, lakes and streams, ground water, diet and man. These compartments may be subdivided, however, depending upon the requirements of the application and nature of the exposure pathways.

The determination of transfer factors assumes steady-state conditions but this may not be applicable in all cases. For example, it may not be applicable for pollutants that do not occur in natural systems, or which have been introduced to the environment relatively recently as steady-state conditions may not be an appropriate assumption. However, for substances where the exposure commitment method is suitable, the steady-state assumption effectively allows the model to integrate the complex and variable processes that take place in the environment into a series of simple transfer factors.

When considering the level of a pollutant in an environmental compartment it is necessary to assume that the contaminant is reasonably well mixed. Exposure Commitment takes no

account of temporal or spatial variability but clearly, in reality, concentrations of the pollutant will vary through both time and space.

It is also assumed that the relationships between compartments are linear and that transfer factors between compartments remain constant. Therefore, a doubling of the concentration in the soil compartment would result in a doubling of the concentration in terrestrial plants, for example. However, experimental studies have identified many exceptions to this rule (e.g., for some metals as the total soil concentration increases, the proportion available to the plant, and therefore the rate of accumulation of the metal in the plant decreases; therefore doubling the soil concentration may result in only a slight increase in the levels in plants. It is important to highlight such cases when using Exposure Commitment.

Ideally exposure assessments should be based on measured data (Jones *et al.*, 1991). However, in some cases they may be based on data derived from validated mathematical models which predict environmental levels or concentrations in certain foodstuffs. Alternatively, in the absence of detailed data, assessments may focus on a few major food types that comprise a major portion of the human diet, and are considered as potentially important pathways of exposure for a particular substance. In certain cases when there are no analytical data available for a pollutant in a particular compartment, the transfer factor may be estimated from data on other chemicals with similar properties.

1.3 DEVELOPMENT OF THE EXPOSURE COMMITMENT APPROACH

Various government and regulatory agencies are now involved in assessing exposure to the general population (and population subgroups) for contaminants present in the environment (Jones *et al.*, 1991). The US EPA has issued guidelines which formalise exposure and risk assessment procedures (US EPA, 1986a,b) and other regulatory agencies. For example, the South Australian Health Commission, have attempted to formally refine exposure assessment for regulatory purposes (Langley, 1993). These examples illustrate the importance of setting up a consistent framework which can be applied to a wide range of substances, but one which can also be adapted according to the nature of the exposure pathways and the availability of data.

Summary Exposure Commitment assessments have been carried out by the Monitoring and Assessment Research Centre (MARC) for a number of substances, mainly metals, but also hexachlorobenzene (HCB), polychlorinated biphenyls (PCBs) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD), and these studies are summarised in Appendix 1.1. They provide an outline assessment in each case, with estimates of the contribution to total human exposure from two or more of the following pathways: inhalation of ambient air, ingestion of food from the terrestrial food chain, ingestion of food from the aquatic food chain, ingestion of drinking water.

Whilst being reasonable indications of human exposure, these studies can only be regarded as the basis for more up-to-date and precise assessments which examine exposure in more detail. In a number of cases there was an absence of data for pollutants in certain environmental media and data had to be derived. For example, in most cases the soil concentrations were derived using a standard deposition velocity for particulates in ambient air, a standard mixing depth for the soil and substance-specific half-lives in the soil; there is a great deal of

uncertainty associated with some of the assumptions made in these early studies. Since they were carried out a great deal more data have become available allowing more reliable assessments to be made.

Secondly, the contribution of the diet, which in most cases makes up at least 90 per cent of total exposure, was highly generalised in most cases. If Exposure Commitment is to be applied consistently, particularly as part of the process to derive Environmental Assessment Levels (EALs), then a more detailed measure of exposure from different foodstuffs is required. Within the last few years more reliable data on a wide range of substances in representative food groups have become available.

The development of a more detailed exposure assessment methodology in order to undertake more realistic measurements of total exposure to the general population is illustrated by a number of recent studies (Duarte Davidson and Jones, 1994; Beck *et al.,* 1994; Gilman and Newhook, 1991). Exposure to the general population from background environmental levels of contaminants have also been used recently to determine the impact of other routes of exposure, such as the application of sewage sludge to agricultural land (Wild *et al.,* 1994).

Duarte Davidson and Jones (1994) assessed human exposure in the UK to a number of polychlorinated biphenyl (PCB) congeners, and estimated body burdens and tissue concentrations for the general population using data on daily intakes of individual congeners. The importance of considering exposure at different ages was highlighted by deriving body burdens and tissue levels for individuals of different sex and age, as well as assessing the contribution from certain foodstuffs. The result is a more realistic estimate of lifetime exposure, that can identify critical periods in the average lifetime, but can also be used for comparison with other measures of exposure. For example the US EPA uses the Lifetime Average Daily Dose (LADD) (calculation of the LADD is outlined by Langley, 1993) as a single integrated measure of exposure. Exposure Commitment data can be used to calculate the current LADD for comparison with other LADD estimates and health-related Tolerable Daily Intakes (TDI).

A technique similar to that employed by Duarte Davidson and Jones (1994) for the assessment of exposure resulting from background environmental levels is utilised by the Canadian Government to assess risk to human health from priority substances (Meek *et al.,* 1994a). Multimedia exposure assessments are made for five age groups of the population to allow evaluation of the importance of breast-feeding, for example, to lifetime exposure. The assessment process incorporates standardised reference values for the human body, and consumption and inhalation rates, to derive an estimated daily intake by the general population at various ages. The estimates, together with the measured concentrations in specific environmental media, are compared with quantitative toxicity assessment data to characterise the extent of risk to human health, and allow classification of the substance in terms of toxicity.

The methods employed by Duarte Davidson and Jones (1994) and Meek *et al.* (1994a), and that developed at MARC, have been combined to form the basis of a formalised approach to Exposure Commitment using UK data. It is clear that using Exposure Commitment for deriving EALs on a consistent basis requires the clear definition of a process comprising a number of distinct stages.

1. Adoption of standard consumption rates for specific age groups in the general population for a wide range of representative foods, drinking water and soil, and inhalation rates for ambient air.
2. Selection of representative values for the substance in relevant environmental media and food. Where these data are unavailable a standardised method of estimating levels using qualified assumptions must be applied.
3. Definition of the pathways to final combined human exposure through examination of the literature and information on the environmental fate and behaviour of a substance.
4. Combining data on concentrations in drinking water, air, soil and food with standard consumption rates, to give Total Human Exposure (per unit time).
5. Derivation of body burdens and human tissue concentrations from the estimated intake and exposure, using standardised reference values for the final receptor (human body, and/or target organ/tissue) and pharmco-kinetic data where available.
6. Comparison of derived body burdens and levels in humans with measured data for the general population, to assess the accuracy of the model.
7. Estimation of acceptable levels of total exposure to the general population by comparison of either:
   (i) The estimated daily intake with an established TDI, or
   (ii) Estimated tissue levels in the target organ with a toxicologically-based critical concentration.
8. Use of the current estimates and acceptable levels of exposure to calculate EALs for air, soil, fresh water and coastal water from data on present background levels.

1.4 Refining Exposure Commitment – Application of the Method

General environmental exposure attempts to define the typical exposure for the general population rather than a particular target population. Consequently the exposure commitment model can be used and the sophistication of the assessments undertaken are dependent on the availability of suitable analytical data. Usually information from databases with typical concentrations of a contaminant in air, water, foodstuffs, human tissues, etc. must be relied upon. This type of information is generally available for selected chemicals through national monitoring programmes in developed countries, or the standard scientific literature.

The basic tasks in the application of the exposure commitment method are the selection of representative levels of the pollutant in the various compartments and the evaluation of transfer factors which relate exposure and intake commitments to the successive environmental compartments in the pathway(s).

The values assigned to the concentration of a pollutant in each of the environmental compartments, e.g., air, soil, water and selected foodstuffs, are the mean concentrations selected from national databases, or from the scientific literature.

Transfers of the pollutant may occur in various directions between compartments and at different rates. The key assumption when using the Exposure Commitment approach is that

the relationships between environmental compartments are at equilibrium. When this is the case, the transfer factor between compartments is simply the ratio of the average concentrations of the pollutant in the compartments. It is assumed that the relationships between compartments are linear, i.e., the transfer factor remains constant regardless of the concentration in the compartments, e.g., a doubling of the concentration in water results in a doubling of the concentration in fish.

A number of other transfer factors can be used to calculate the successive intakes and exposures to the compartments. These include values taken from the literature for plant uptake factors, livestock biotransfer factors, GI Tract and lung absorption factors, partitioning factors and consumption rates.

Standard Consumption Rates and Body Reference Values

Food

The food consumption rates for the general UK population have been obtained from the most up-to-date report from the Ministry of Agriculture, Fisheries and Food (MAFF), the 'National Food Survey 1993'. The data give average consumption of all the food groups in ounces per person per week for the years 1991, 1992 and 1993, and have been converted to annual consumption rates in kilograms. Where data are absent, e.g., offal, the consumption rate has been taken from previous MAFF reports. In the case of "other vegetables" for example, representative vegetable types have been selected from the data in MAFF Food Surveillance Paper no. 40 'The British Diet: Finding the Facts, 1989–1993', and their relative consumption rates calculated from the total consumption rate of the food group specified in the 'National Food Survey 1993'.

No attempt has been made to take account of the effects of preparation of food on levels of contaminants, although where literature is available mention of the possible implications on exposure has been made. With respect to vegetables, all the MAFF data are given for raw vegetables, as they generally represent the worst-case, e.g., in some cases contaminants may be concentrated on or in the skin of root vegetables for example, and therefore washing and peeling may reduce the total concentration of the contaminant in the food that is finally consumed. Therefore assuming that a particular food will always be prepared in the same manner is not considered good practice, and may result in underestimates of intake in certain instances. Conversely, some preparation of food may result in reduced levels of contaminants, e.g., levels of PCDD/Fs in meat may be reduced after cooking due to the loss of fat. The variations that may arise due to food preparation techniques have therefore resulted in most data being based on levels in raw food.

The food consumption rates at different ages are accounted for by using the estimated proportion of the adult diet consumed by each particular age group (Duarte Davidson and Jones, 1994).

Age	Proportion of adult dietary intake
3 months–2 years	0.125
2–7 years	0.25
7–14 years	0.5
14+ years	1.0

The consumption rates for the representative foods can be found in full in Appendix 1.2.

Drinking water consumption

The reasonable worst case drinking water consumption rate for adults is 2 litres per day (US EPA, 1989). Whilst acknowledging this to be an overestimate, there are also limited data on sensitive populations, e.g., people performing manual work who may consume considerably more than 2 litres per day (Langley, 1993). UK data indicate the mean consumption rate to be ~1.4 litres per day, but the higher estimate of 2 litres per day (730 litres per year) used by the US EPA is adopted in this study to provide a conservative estimate of drinking water consumption. The proportion of the adult intake for different age groups are assumed to be the same as for food consumption.

Breathing rate

The inhalation rates adopted are estimated using the data applied in US EPA risk assessments (originally taken from ICRP, 1975), and are shown below. The consumption rates for the age groups considered in this study have been estimated from these data and are summarised in Table 1.1.

	m^3/d	**m^3/yr**
Adult	22	8,030
Child (10 years old)	15	5,475
Infant (1 year old)	3.8	1,387
New-born	0.8	292

As breathing rates increase greatly with exercise, alternative exposure scenarios may be considered for members of the general population, e.g., for an adult who takes one hour of moderate exercise (jogging, tennis) per day, on average, the estimated inhalation rate is 24.6 m^3/d, or 8,979 m^3/yr, an increase of approximately 12 per cent.

Soil ingestion

The soil ingestion rates for different age groups vary as a result of the behaviour of individuals in each age group. Conservative estimates for four different age groups are presented below (Langley and El Saadi, 1991; ANZECC/NHMRC, 1992). The consumption rates for the age groups considered in this study have been estimated from these data and are summarised in Table 1.1.

Age (years)	Soil intake (mg/d)
0–1	negligible
1–5	100
5–15	50
Adult	25

Breast-feeding

The rate of ingestion of human milk during the breast-feeding period is calculated from the average lactation rate of 750 ml per day (ICRP, 1975) over an average breast-feeding period of three months.

Smoking

Smoking of Tobacco may contribute greatly to the intake and exposure to certain substances, e.g., arsenic and cadmium. However, in the application of Exposure Commitment the average person in the general population is used, and is assumed to be a non-smoker, and therefore estimated intakes from smoking have not been used in the calculation of EALs. Any exposure that may come about through smoking is in addition to the estimates of exposure from diet, air, soil and water, and therefore this additional exposure may represent a separate health risk. Where data on the estimated exposure from smoking are available they have been highlighted as additional information of interest.

Table 1.1 Summary of consumption rates and standard reference values for the human body used in this study

	0–3 mths	3 mths–2 yrs	2–7 yrs	7–14 yrs	Adult
Water (litres/yr)		91	183	365	730
Soil (kg/yr)		0	0.04	0.018	0.009
Air (m^3/yr)	300	1,500	4,000	6,000	8,030
Body weight (kg)	5	12	24	43	70

The method makes the assumption that the diet of children is a fractional proportion of the adult diet. However, the nature of the diet may also change with age, for example diet studies in Australia have found that younger children consume proportionally greater amounts of dairy products, and that adults consume proportionately greater quantities of shellfish. However, there are not sufficient data for individuals of different ages to be able to estimate the average consumption for the total diet for different age groups. This variation will influence not only the total amounts of a substance consumed, but also the relative proportions of various forms of the substance, e.g., in the cases of arsenic and mercury, the relative amounts of inorganic and organic forms.

The consumption rates for food, soil, air and water are given in full in Appendix 1.2.

Selection of Representative Values

The values assigned to the concentration of a pollutant in each of the environmental compartments, e.g., air, soil, water and selected foodstuffs, are the mean concentrations selected from national databases, or from the scientific literature.

As the Exposure Commitments are being used to derive EALs, the representative values used for foods, drinking water and urban air (i.e., the compartments in direct contact with humans) are 'conservative' in order to avoid underestimates of total exposure. Therefore, the representative values are generally the *highest recorded mean concentration* of a pollutant at background levels in the compartment.

However, for the source compartments (e.g., rural air and soil) the selected values are generally given as a range. The calculation of the EAL is based on the multiplication of background levels by an a factor derived by dividing the tolerable intake/exposure for the substance by the estimate for the current level of intake/exposure. Therefore, the lower value of the range for the source compartment would result in a lower EAL. Consequently, the lower value of the range represents a more conservative estimate with respect to a standard that is being set to limit the final human exposure to a pollutant, **and may underestimate the contribution to exposure of a particular pathway.**

Definition of Pathways to Exposure

In relevant cases, where a summary Exposure Commitment has already been carried out it can form the basis of a more detailed pathway analysis. Information on the environmental fate and behaviour of a substance can be obtained from the scientific literature, or predictions can be made using the physicochemical properties of the substance. The main pathways to exposure which must always be considered are inhalation, direct ingestion of soil, food consumption and drinking water.

Other sources of exposure must be considered on a pollutant-specific basis and, where possible, quantified. These may constitute direct sources of exposure, e.g., smoking, food contamination from packaging and processing, or indirect sources, e.g., application of phosphate fertilisers, agricultural application of sewage sludge.

Estimation of Total Human Exposure

Daily or annual intakes are calculated from the standard consumption rates and the representative levels of the substance in the various media. Calculating inhalation exposure involves combining the atmospheric concentration of the chemical and breathing rates to estimate the quantity of chemical taken into the lungs. Information on the rate at which the chemical is absorbed through the lung tissues can be used to calculate the dose or the quantity taken into the body via this route. Similarly, absorption via the GI tract influences the dose received via the ingestion route. However, such information for specific chemicals is not always available and is extrapolated or estimated from data for other chemicals usually from animal experiments.

Calculation of Body Burdens and Human Tissue Concentrations

Body burdens and tissue concentrations can be calculated using two methods. Method 1 is based on that used by Duarte Davidson and Jones (1994) and involves the calculation of body burden by using successive intakes over a number of time periods and accounting for metabolic losses by using the half-life of the contaminant. This method is particularly useful for substances with long half-lives in relation to the time periods used in the calculation (e.g., cadmium has a half-life of 30 years in the human body, and the calculations of body burden are done at approximately 10-year intervals).

The second method uses a steady-state assumption and is taken directly from Bennett (1981). It relates body concentrations to intake, body mass and retention time in the body, and assumes an equilibrium situation.

METHOD 1

Body Burden

The calculation of the body burden at different ages using the estimated daily intake is relatively straightforward. The following example demonstrates how the body burden at age 30 years can be calculated, before accounting for metabolic losses.

Body burden at age 20 years is 10 mg.
Daily intake of the substance is 1 µg/day.
Therefore during a 10-year period the total intake of the
substance is 1 x 365 x 10 = 3,650 ug, or 3.65 mg.

If the absorption factor across the GI Tract and lungs is 90 per cent, then the total exposure to the substance during the 10-year period is 0.9 x 3.65 = 3.29 mg.

The Body Burden at age 30 years, before accounting for metabolic losses, is simply the sum of the original body burden and the exposure during the 10-year period, i.e., 13.29 mg.

Accounting for metabolic losses

It is important to consider the elimination of a substance from the body due to metabolic losses in order to estimate the body burden more accurately. During a given period, a proportion of the original body burden will be lost, but during that same period there will also be a given intake of a substance resulting in additional exposure. All of the original body burden present at the beginning of a given period will be subject to a degree of elimination due to metabolic losses, and this loss may be calculated using the biological half-life and the length of the time period.

However, if it was assumed that the *additional exposure* (as a result of intake during the given period) is not subject to any metabolic losses during the exposure period, the body burden calculated would be an overestimate. Conversely if it was assumed that all of the *additional exposure* was subject to metabolic losses over the full period, the calculated body burden would be an underestimate. Therefore it is assumed that, on average, the *additional exposure* is subject to metabolic losses for half of the period.

The following example demonstrates how the body burden at age 30 years can be calculated, whilst accounting for metabolic losses.

The biological half-life in the body is used to account for metabolic losses in the body. If the half-time in the body is 30 years, then the retention time is 43.3 years (from the relationship: half-life = retention time x ln (2)).

It is assumed that metabolic losses are a linear function of time, therefore the proportion of the original body burden lost during the 10-year period is 10 ÷ 43.3.
Body burden at age 20 years is 10 mg.
The amount lost is (10 ÷ 43.3) x 10 mg = 2.31 mg.

This is subtracted from the original body burden, so the amount remaining at age 30 years is 7.69 mg.

The proportion of the total exposure lost during the 10-year period is 5 ÷ 43.3 (i.e., the total exposure amount is subject, on average, to metabolic losses for half of the exposure period). Therefore the amount lost is 3.29 x (5 ÷ 43.3) = 0.38 mg, and therefore the amount remaining at age 30 years is 2.91 mg.

Therefore the body burden at age 30 years, after accounting for metabolic losses, is simply the sum of the two remaining amounts, i.e., 7.69 + 2.91 = 10.6 mg.

Derivation of tissue concentrations

The tissue concentration is simply the total burden in the tissue divided by the mass of the particular tissue or target organ. Values for the mass of various tissues and organs are available from the ICRP (1975) report on "reference man" for both sexes at different ages.

In the case of lipophilic substances, for example, it is assumed that all the body burden is contained within the body's fat tissue. Some substances accumulate in specific organs (e.g., cadmium tends to accumulate in the kidney), in which case it is important to know what proportion of the total body burden is contained in the organ, and also the half-life of the substance in the target organ.

Comparison of estimated intakes, body burdens and tissue concentrations with measured values

Estimates of total exposure, daily intake, body burden and tissue concentrations can be compared with results of other relevant studies from developed nations to indicate whether the model is suitable for use in deriving EALs. There is obviously some variation between nations, often due to different dietary habits, but usually a range of reasonable estimates for the general population in other countries is available for comparison.

Comparison of estimated daily intake with Tolerable Daily Intake values and derived body concentrations with critical concentrations in target organs

Tolerable Daily Intakes

The estimated daily intake can be used to calculate acceptable levels in environmental compartments if the intake can be compared with a Tolerable Daily Intake for the substance. TDIs are normally expressed as a unit mass of substance per kilogram of body weight per day and should be well-established and internationally-recognised levels set with respect to the protection of human health. The World Health Organization (WHO) has set TDIs for a number of substances. They are based on experimental and epidemiological data and incorporate considerable margins of safety between the intakes at which effects have been observed and the levels that are considered tolerable. Tolerable daily intakes take into account long-term exposure, and it is generally believed that long-term chronic exposure to environmental carcinogens is of greater significance than short-term acute exposure (Harkov, 1982). **(This may or may not be the case for non-carcinogenic health effects of substances under consideration, e.g., long-term exposure to cadmium results in damage to the kidney, but high-dose acute exposure, such as the inhalation of high concentrations of cadmium compounds, may result in totally different health effects).**

As an example, the daily intake for a substance may be estimated at 35 ug per day for a 70 kg adult, the daily intake would be 0.5 µg/kg bw/day. Toxicological assessments have derived a TDI for the substance of 10 µg/kg bw/day. Therefore current exposure is approximately 20 times lower than what is considered tolerable. This factor can then be used, together with the transfer factors calculated by the Exposure Commitment, and the measured background levels in the environmental media to derive maximum acceptable concentrations in the environment that take into account the protection of human health.

The daily intake will vary with age, and therefore the age of highest daily intake may be considered the critical age. However, most TDI values refer to a lifetime intake assuming a mean body weight during the lifetime of 60 kg, and so the intakes for different age groups can be averaged to provide a lifetime average daily intake (equivalent to the US EPA Lifetime Average Daily Dose) which can be used for the purpose of comparison, or the daily intake at the "critical age" can be used.

Critical concentrations in target tissues

In some instances there are critical concentrations for certain substances in given tissues. For example, for cadmium there is a well-established critical concentration in the cortex of the kidney. Therefore, the estimated concentration in the target organ or tissue can be compared with the critical concentration and used to estimate an acceptable level of exposure and acceptable environmental concentrations. (Many pollutants accumulate in human tissues, so concentrations generally increase with age; therefore the critical age may be assessed according to the estimated levels at different stages in the human lifetime.) The measured levels in the background environment can then be used to derive EALs.

Comparisons of Environmental Assessment Levels calculated using Exposure Commitment with existing Environmental Standards

Whilst comparisons are made with existing Environmental Standards, it should be noted that these standards are derived using different principles, and with different regulatory objectives in mind. For example, freshwater EQS levels are set predominantly to protect the freshwater environment. This study aims only to introduce the concept of using Exposure Commitment for deriving Environmental Assessment Levels, and its applicability to certain substances, and before really meaningful comparisons can be made, more complete and robust UK datasets for all environmental media, food and human tissues will be needed. This will require co-ordination of the monitoring of all these environmental compartments.

1.5 REVIEW OF SIMILAR WORK ON ASSESSMENT OF EXPOSURE TO ENVIRONMENTAL CONTAMINANTS FOR THE GENERAL POPULATION

A detailed review of work on exposure assessment has been carried out, and the findings discussed with particular reference to factors which must be considered when quantifying background environmental exposure. These are highlighted by work on particular substances, as the factors to be considered are often dependent upon the nature of the substance, or group of substances. These include inhalation of indoor air (rather than outdoor, ambient air), dermal absorption and the consideration of all congeners when assessing exposure to a simple mixture of chemicals.

Particular attention has been paid to cases where exposure assessment already forms part of the regulatory process for limiting the risk to public health from environmental exposure. The way in which the problem of insufficient analytical data can be tackled is discussed (e.g., mathematical models for predicting environmental concentrations), as are studies on the use of tolerable daily intakes, critical concentrations in human tissues and the derivation of environmental standards.

Organic chemicals

General exposure assessments are often hampered by a lack of data, as routine monitoring data are limited to only a few organic chemicals. This is largely because the government agencies responsible for undertaking routine monitoring have limited resources, and therefore select a limited number of priority contaminants for inclusion in their monitoring programmes. Consequently exposure assessments have been published for only a relatively small number of organic compounds.

Ideally, various criteria should be used to select the priority organics of particular concern to government agencies. These could include such properties as chemical production volume, likelihood of environmental release (or patterns of use), persistence in the environment, tendency to accumulate in the food chain and oral ingestion/mammalian toxicity. Potential for accumulation in the food chain can be assessed quickly by referring to established bioconcentration databases, or can be calculated from equations relating the bioconcentration factor to the physicochemical properties of the chemical.

Estimates of total exposure to the UK general population from food, water and air have been carried out for PCBs, pentachlorophenol (PCP) and polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDD/Fs), all of which are considered priority contaminants.

A detailed assessment of background exposure to PCP in the UK, and the relative contributions of the main exposure pathways, found that diet was the major contributor (92 per cent), followed by drinking water (7 per cent) and air (1 per cent) (Wild and Jones, 1992). The estimated total daily intake and values for the half-life of PCP in the body, were used to calculate the average UK body burden. By comparing the UK estimates with measured body burdens for other countries it was concluded that either the half-life for PCP in the human body had previously been underestimated, or that the estimated UK body burden is lower than in other countries.

Mixtures of organic chemicals

In the case of PCBs, dietary intake has again been shown to be the main source of exposure to the general UK population (Duarte Davidson and Jones, 1994). A detailed congener-specific estimate of daily intake was used to derive estimated theoretical body burdens and body fat concentrations for individuals of different ages and sex in the UK. The relative contributions to $\sum$PCB exposure were estimated for diet (96.56 per cent), air (3.4 per cent) and drinking water (0.04 per cent) and the contributions of specific food types were estimated. However, the absence of data on certain food groups means that the study may have underestimated actual PCB exposure. It was found that vegetables played a major part in the intake of lower chlorinated compounds, whilst fatty foods were of greater importance for the intake of higher chlorinated compounds. It was also predicted that the lower chlorinated congeners, which are more readily removed from the human body, would reach an equilibrium concentration in humans. In contrast, the higher chlorinated congeners appear to accumulate in the body throughout life. The study is an excellent example of the need to consider all congeners when undertaking an exposure assessment for a simple mixture or group of chemicals, due to the variability of their behaviour in the environment and the human body.

Mixtures of chemicals, such as PCDD/Fs may also be considered by using the Toxic Equivalents (TEQs) system. (The toxic potency of each compound in the mixture is related to a reference compound, usually the most toxic. The concentration of each compound is multiplied by the relative toxic potency, or Toxic Equivalency Factor, and the results summed to give the total TEQ concentration). Background exposure in the UK from air, food and water has been estimated for all seventeen 2,3,7,8-substituted PCDD/Fs found in human tissues for which TEQs are available (Wild *et al.*, 1994), and used to assess the impact on background human exposure (in terms of elevated concentrations of PCDD/Fs in food) of the application of sewage sludge to agricultural land. This included an estimated dietary intake based on data on PCDD/F levels in a few selected food types; some data, e.g., root vegetables and various dairy products, were derived rather than measured values. However, it does illustrate that where data may be absent, good estimates for certain food groups can be made by applying some simple assumptions.

Previous exposure assessments had concentrated on 2,3,7,8-TCDD alone (Jones and Bennett, 1989), and whilst providing useful information, they failed to take account of the threat posed by the other 16 congeners. As with PCBs, it is important to recognise that the environmental behaviour of PCDD/Fs is congener-specific, therefore a risk assessment model centred on one

congener cannot easily be extrapolated to others. Wild *et al.* (1994) clearly showed that by using TEQs a more complete estimate of exposure can be made. In addition, while general exposure assessment may be concerned with diffuse sources, Wild *et al.* (1994) showed that it can be used to investigate a particular source of exposure.

Due to their apparent ubiquity in the environment, and the growing public debate over their possible health effects at low concentrations, PCDD/Fs have been the subject of assessments of background exposure in a number of countries.

In Germany, food has been identified as the main source of exposure to 2,3,7,8-substituted PCDD/Fs (Beck *et al.*, 1994) by using a protocol similar to that used by Wild *et al.* (1994). Other sources and pathways were found to be of minor importance. Food of animal origin contributes most, although human exposure begins with atmospheric emissions depositing these compounds on plant surfaces or soil. The assessment examined lifetime exposure and concluded that the relatively high levels of exposure associated with breastfeeding (albeit for a short period of time), due to the accumulation of PCDD/Fs in human milk, are not tolerable with respect to prospective health care, therefore efforts should be made to minimise or avoid PCDD/F emissions into the environment. In particular, air concentrations of these substances must be reduced to decrease the food chain accumulation leading at present to levels in human milk. Similarly, Gilman and Newhook (1991) used the same technique to assess exposure to PCDD/Fs in Canada, and concluded that PCDD/Fs presently enter the environment in quantities that constitute a danger to public health.

Workers in the Netherlands, Italy, Norway, Germany, Canada and USA have attempted to quantify human exposure to PCDD/Fs by using data on a few of the major food groups (meat, fish, and dairy products) which constitute the major pathways to exposure (Theelen *et al.*, 1993; Birmingham *et al.*, 1989a,b; Beck *et al.*, 1989; Furst *et al.*, 1990; DiDomenico, 1990; Faeden, 1991; Theelen, 1991). Other studies have examined lifetime exposure from key food groups, including human breast milk, in order to highlight the periods of maximum exposure (Schecter *et al.*, 1994), and also examined the additional risks posed to certain sub-groups of the population through abnormally high consumption of certain food types (Theelen and Liem, 1994). Although such studies provide reasonably good measures of exposure, estimation of total exposure from all sources is more appropriate for the consideration of EALs, given the variability of routes of human exposure for the wide range of organic substances present in the environment.

Metals

Compounds of the same metal may differ greatly in toxico-kinetics and toxicological effects and this, together with the environmental fate and behaviour of the metal, should be taken into account when assessing exposure and setting standards for metals (Zielhuis, 1984). Therefore, in terms of human health, while some metals may present only a limited risk, certain compounds of the same metal may have significant human health effects. Despite this fact, nearly all assessments of exposure to metals deal only with total concentrations of the metal itself due to a lack of data on the environmental levels of individual compounds.

As part of the survey of cadmium in food, MAFF carried out a very basic multimedia exposure assessment for the general population using selected food types, air and drinking water. However, many of the recorded levels for food were below the limit of detection, and

in such cases assumed to be equal to the limit of detection. Therefore total daily intake from the diet was overestimated. Daily intakes from the diet (<20 μg), drinking water (1–2 μg) and air (urban air, <0.4 μg) were estimated. A total exposure of 140 μg/week was suggested as representative of the adult UK population, and compared with the World Health Organization/Food and Agriculture Organization of the United Nations (WHO/FAO) provisional tolerable weekly intake (PTWI) of 500 μg/week. It was concluded that the safety margin between this and actual intakes in the UK was not great, and recommendations were made to continue surveillance of certain key food types for cadmium. It was noted that there was insufficient information to give a tolerable intake of cadmium for children (MAFF, 1983). The Ministry of Agriculture, Fisheries and Food has also carried out surveys of the following metals in the UK diet: copper, zinc, arsenic, lead, aluminium, antimony, chromium, cobalt, indium, nickel, thallium, tin and mercury.

Average intakes have been calculated for cadmium for a number of different countries, although perhaps the best for comparative purposes used data for cadmium collected by the Joint FAO/UNEP/WHO Food Contamination Monitoring Programme (GEMS/Food). Daily intakes of cadmium were calculated for the average population in each country, together with daily intakes for lead, mercury, and PCBs. It was concluded that the majority of the population in the reporting countries are not exposed to levels above the provisional tolerable levels recommended by national authorities. In local areas and for certain groups of consumers, however, the dietary intakes may approach or even exceed tolerable levels (Moy *et al.*, 1993). Clearly, as with all assessments of environmental exposure, it is important to recognise that individual exposures within the general population may vary considerably depending upon the personal activities, lifestyle and diet of the individual.

A similar multimedia assessment for cadmium has been carried out in Canada, limited to total cadmium due to insufficient information on the forms of cadmium in environmental media. The main route of exposure was found to be food, with exposure from drinking water, soil and air negligible. Smoking could contribute substantially to the total exposure. The available data indicated that some members of the general population in Canada are exposed to cadmium in amounts that are at or near those that have been associated with mild effects on the kidney in cross-sectional epidemiological studies (Newhook *et al.*, 1994).

Background exposure to several other metals in Canada has been assessed including arsenic, which focused on inorganic arsenic as the form of primary toxicological concern. It was found that the greatest exposure was to young children, and the principal sources of exposure at all ages are drinking water and food (Hughes *et al.*, 1994). In the case of nickel, although it is well recognised that certain groups of nickel compounds (sulphidic, oxidic and soluble) are carcinogenic, there were insufficient data on individual compounds in environmental media so the estimate of exposure is related to total nickel only. The principal route of nickel intake for all ages was food, followed by drinking water and breathing air. Smoking could contribute up to 3 per cent of the total daily intake in an adult (Hughes *et al.*, 1994).

Consideration of exposure sources

The importance of considering all potential routes of exposure is clearly illustrated by total exposure estimates which have identified sources other than food as the main source. In Japan, human exposure to dichloromethane, carbon tetrachloride, 1,1,1-trichloroediane, trichloroethylene and tetrachloroethylene from inhalation of ambient air, drinking water,

milk, meat, fish, vegetables and fruit have been quantified (Yoshida, 1993). Estimated exposure doses suggested that the main route of exposure is inhalation. A physiologically-based pharmaco-kinetic model was used to evaluate distribution of the substances in the human body giving a more complete picture of exposure. Data used were derived from an environmental fate model, and were in good agreement with measured levels in Japanese air, water, soil and sediment.

However, the available data may not always be adequate to carry out a complete assessment of exposure, as in the case of the assessment of PAH exposure in Canada (Meek *et al.,* 1994). Five PAHs were considered; benzo(a)pyrene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene and indeno(1,2,3-cd)pyrene. The exposure to these PAHs from air was quantified, but there were not sufficient data to estimate exposure from food. However, a US "Total Human Environmental Exposure Study" (THEES) which investigated multimedia exposure to one of these PAHs, benzo(a)pyrene (BaP), has been carried out using measured environmental levels to quantify the potential risk to health from a number of sources (Butler *et al.,* 1993). Environmental exposure was found to be predominantly through food, again highlighting that all routes of exposure should be accounted for to avoid underestimation.

Indoor Air

Many exposure assessments routinely use concentrations in outdoor air, even though an average person spends nearly 23 hours a day indoors. Concentrations of many organics (particularly volatile organic compounds) may be at least an order of magnitude higher in indoor air (Anderson and Hites, 1988; Petreas and Lia, 1989; Wallace, 1987) so assessments based on outdoor concentrations may routinely underestimate exposure. Therefore in certain cases it is important to use data for concentrations in indoor air, if available, in order to produce an accurate measure of exposure. For example, assessment of exposure to dichloromethane in Canada showed that indoor air appears to be the most important route of exposure, with the estimated intake being more than an order of magnitude greater than those from ambient air, food (reliable data on levels in Canadian foods were not available, so measured levels from the US Food and Drug Administration's Total Diet Program were used) or drinking water (Long *et al.,* 1994).

Similarly, measured and predicted concentrations of benzene in the environment were used to estimate its accumulation in the food chain, and the subsequent extent of human exposure from inhalation, drinking water, vegetables, dairy products, beef and fish (Hattemeyer-Frey *et al.,* 1990). Results showed that inhalation contributes nearly all of the total daily intake of benzene (99.96 per cent). Ingestion of contaminated food (0.03 per cent) and drinking water (0.01 per cent) represent minor pathways of human exposure. Although inhalation is the primary route of human exposure to benzene from background levels in the environment (Hattemeyer-Frey *et al.,* 1990), smoking was found to be the largest anthropogenic source of background human exposure. An average smoker is exposed to three times more benzene from smoking than from all other sources. The Total Exposure Assessment Methodology had previously shown that benzene levels in indoor air are approximately double those in outdoor air (Wallace, 1986).

Dermal Absorption

Another area where there is considerable uncertainty with respect to human exposure to organic chemicals is the direct exposure of pollutants to the skin, and subsequent absorption into the body. Between August 1989 and July 1990 the State of California carried out a programme to eradicate Mediterranean fruit fly in urban areas of Southern California by aerial applications of the insecticide Malathion. Chronic dose rates to the population in the area were calculated for dermal exposure, inhalation and ingestion of contaminated unwashed vegetables. Exposure from direct contamination of the skin and via ingestion of vegetables were both approximately 2,000 times higher than from inhalation. No estimates of secondary pathways (bioaccumulation in livestock) were made, but this study still highlights the potential exposure that may arise from direct dermal exposure to some atmospheric pollutants (Marty *et al.*, 1994). Despite this fact, the majority of general exposure assessments do not examine dermal absorption, and there is a paucity of information on both skin absorption rates and assessment methodology for the majority of environmental contaminants under consideration.

Specific subgroups of the general population

The investigation of human exposure to malathion in specific areas of California demonstrates the flexibility of the exposure assessment approach, in its application not just to general population exposure, but also to specific cases. Another example of an attempt to quantify exposure for a particular population is the multimedia assessment of exposure to the general population in Los Angeles County to the biodegradable volatile organic chemicals perchloroethylene (PCE), trichloroethylene (TCE), 1,1-, cis-1,2-, trans-1,2-dichloroethylene (DCE) and vinyl chloride (VC) (Yeh and Kastenberg, 1991). The risk to human health from the estimated exposure levels was calculated using dose-response models, and allowing for biodegradation.

Availability of analytical data and the use of derived values

Information about human exposures to environmental agents is a crucial component of informed decisions about protection of public health, and exposure data are useful for monitoring status and trends in environmental health (Sexton *et al.*, 1992; Goldman *et al.*, 1992). The availability of data on contaminant concentrations in environmental media is fundamental, and varies greatly according to the substance and the media in question. For example there are a large number of studies which have investigated the background levels of organic chemicals such as PCDD/Fs in cows' milk, but very few studies which have measured levels in background air.

Over the past few years MAFF have carried out a number of studies surveying the levels of various environmental contaminants in food consumed in the UK. In Canada a food analysis programme, comprising a total diet study, has been carried out for a number of years, and is conducted every five years; the data are used in assessing risk to human health from environmental contaminants (Conacher and Mes, 1993).

Data required for exposure assessment have been discussed with respect to the availability, suitability and quality of data for incorporation in risk assessments in the US (Graham *et al.*, 1992). Exposure-related databases must include measurements of actual exposures and doses for relevant human populations. Therefore, the principal requirements for a reliable exposure assessment are good quality field data, a good understanding of the pathways, transport

mechanisms and fate of chemicals under consideration. In the absence of detailed data, assessment models may focus on a few basic food types which comprise a major portion of the human diet and are considered major pathways of exposure (Jones *et al.,* 1991), or may use data derived from validated mathematical models.

To account for this simplification and/or aggregation in environmental pathways, assessment models are often given a conservative bias to reduce the probability of underestimation. This bias is most important if an evaluation is to have implications with respect to regulatory standards, or effluent discharge objectives (Jones *et al.,* 1991).

As food is often the major source of exposure, predicting concentrations in food in the absence of reliable analytical data may provide a reasonable estimate of exposure in many cases. The use of mathematical models for predicting the chemical concentration in the food chain for health risk assessment has been reviewed (Giordano, 1994). There are basically two levels of analysis; Level 1 concentrates on five key food groups recommended by the US EPA as representing the most common sources of chemical exposure through the food chain; fruit and vegetables, beef, fish, dairy milk, and human breast milk. Level 2 analysis may take into account features of the local environment and other important food groups, and is site-specific. Models comprising Level 1 analysis were reviewed and recommended as being suitable for screening and refined risk assessments. Therefore, where data for a substance in food are not available there are validated mathematical models which may be used for prediction of concentrations in food, but it must be recognised that any exposure assessments based on such predictive models must account for the uncertainty and shortcomings associated with the models used for generating data.

Multimedia modelling of the complex physical, chemical and biological processes that govern the fate and transport of substances in the environment can be used to simulate the concentration of a substance in various environmental compartments. The capabilities and limitations of the major types of models have been reviewed (Hsieh and Ouimette, 1994), stressing that the knowledge of concentrations in environmental compartments is essential for realistic assessments of human exposure.

Compartmental models, linking several single-medium models to mimic a multimedia system, are the most traditional approach. However, spatial models (e.g., fugacity model) which describe the equilibrium and steady-state distribution of a substance are also used, and require much less data than compartmental models. A more recent approach has been a combination of the two – Spatial Multimedia Compartment Model (SMCM) – which allows screening level calculations whilst retaining as many physical realities as possible. The SMCM model has been used in several studies and the predicted concentrations were found to be in reasonable agreement with the available field data (Cohen *et al.,* 1990).

Whilst these models provide useful estimates of concentrations, their application to prediction of environmental levels for exposure assessments, and subsequent EAL derivation, is recommended to provide provisional estimates of exposure, which can be verified by the collection of analytical data in the environment.

The role of Ecotoxicology in the assessment of human exposure to chemical substances has been examined by Calamari (1992). With respect to the assessment of risk to the general

population, and any subsequent steps to limit exposure, sound scientific judgement must be applied to the knowledge of expected environmental concentrations and toxicological properties of the substance. While ecotoxicology will play an important role in predicting environmental concentrations for many exposure assessments, the ideal is to use measured levels where data are available.

Toxicological assessment – tolerable daily intakes and critical concentrations in human tissues

Environmental regulation on behalf of public health is becoming more prominent, and quantitative risk assessments are becoming increasingly sought after for exposure standard-setting in relation to public health risks posed by low-level environmental exposures. Part of the process requires the examination of current estimates for background exposure in relation to levels of exposure that are considered "acceptable", and these are often expressed as Tolerable Daily Intakes (TDIs) or as critical concentrations in a target organ or tissue.

Reliable values for TDIs and critical concentrations are crucial in assessing the risk to the general population of background exposure to a substance, and therefore for setting exposure standards to limit the risk. These values may be derived from toxicological assessments using toxicity tests on certain animal species, or by using empirical epidemiological data. The use of empirical epidemiological data in the complex social process of assessing environmental risks to health and setting exposure limits has been examined by McMichael (1989).

In order to formulate exposure standards, quantitative assessment of the risks associated with low levels of exposure in humans is required. However, much of the scientific evidence relating exposures, particularly low dose exposures, to health risks is beset with uncertainty. It may be either statistically imprecise, or the animal experimental evidence may be of uncertain relevance to human populations. For example, there can be clear-cut evidence of toxic or carcinogenic effects at high exposure levels, or in animals, but little evidence of effects at the lower exposure levels that apply widely within the community.

This is an area of great uncertainty, and the evidence of subtle, sub-chronic health effects in the general population due to long-term, low-level exposure is very difficult to identify. In addition to their intrinsic uncertainty at low exposure levels they refer only to average risks at specified exposures. They do not, and cannot, allow for either inter-individual variability in response, or greater than additive interactions of the specified exposure with other coexistent exposures. For these reasons, estimates of acceptable environmental levels based on quantitative exposure and toxicological assessments can only form guidelines to judgement.

1.6 EXPOSURE COMMITMENT IN THE REGULATORY PROCESS

Evaluation of potential health risks, using exposure assessments, are required by Government agencies who seek to balance these risks with other social and economic factors (Hawkins *et al.*, 1992). Quantitative exposure assessments for the general population, together with dose-response relationships from toxicological investigations, are used to estimate risk to human health from harmful substances in the environment in the US (Naugle and Pierson, 1991) and Canada (Meek *et al.*, 1994). Once current exposure and risk are determined then controls on exposure levels may be back-calculated according to risk levels desired (Jones *et al.*, 1991).

These assessments form a fundamental part of the regulatory process, and are essentially the same tools being considered in this study for use in formulating EALs.

In Canada the multimedia approach to the assessment of total human exposure has for some time been an integral part of the assessment of risk to human health. It allows the development of measures to be taken to effectively protect human health by identifying the relative contributions of each pathway to exposure (Meek *et al.*, 1994).

The Canadian Ministry of Environment and Health have developed a formalised approach for the assessment of exposure to Priority Substances for the general population. Where possible these assessments are based on data acquired in national surveys of ambient air, indoor air, drinking water, soil, foodstuffs and consumer products in Canada. Mean concentrations in various environmental media are used in estimating exposure (where data include results less than the limit of detection (LOD), the result is assumed to be equal to the LOD although it is recognised that this will lead to an overestimate of exposure). Extensive and detailed reference values are set out for assessing the total daily intake of priority substances by five different age groups of the general population in Canada (Meek *et al.*, 1994).

Where data are absent, predicted levels (e.g., from the Fugacity Model) are used, but owing to the uncertainty associated with predicted values, they are only relied upon for assessments when the resulting exposure is several orders of magnitude lower than the considered tolerable exposure level. Assessment of simple mixtures of chemicals, such as PCDD/Fs by expressing the daily intake in toxic equivalents has been used in a number of cases.

Background exposure to the Canadian population has been assessed for a large number of organic chemicals and metals using this procedure, and the results published in a single volume (Journal Environmental Science & Health 1994: Environ. Carcino & Ecotox Revs C12(2)). These assessments demonstrate the usefulness of a consistent and well-defined protocol which can be applied to the wide range of contaminants in the environment. The approach is very similar in principal to the method proposed in this study, and is already a key part of the regulatory process in Canada.

1.7 DERIVATION OF ENVIRONMENTAL QUALITY STANDARDS USING EXPOSURE COMMITMENT

In a number of countries, different regulatory agencies too often propose and enforce standards which on closer scrutiny are incompatible. Often there is little or no co-operation between agencies, and the standards do not account for the fact that during a lifetime humans are exposed to the same substances through various pathways. The concept of using Total Human Exposure as the guiding scientific principle in risk assessment, to formulate risk reduction/management programs and drive environmental policy is now widely accepted (Wesolowski, 1992).

For some time it has been recognised that standard setting for the chemical quality of air, drinking water and food in order to protect human health requires a step by step approach that takes account of total exposure through various routes of entry (Zielhuis, 1984). Traditionally, regulatory guidelines have been established on a medium-specific basis, and have focused on sources rather than exposures.

Increasingly, health and environmental regulatory bodies are being asked to collect and explain exposure data, and relate them to public health concerns. Determination of human exposure to environmental contaminants from multiple routes is fundamental to the process, and should form an integral part of environmental regulation (Johnson, 1992).

An excellent example of the suitability and associated advantages of Exposure Commitment for the derivation of maximum tolerable environmental levels has been presented by Travis and Hattemeyer-Frey (1991). They used the multi-media approach to determine the proportion of the allowable daily intake of 2,3,7,8-TCDD contributed by air, food, soil and water. By using a maximum allowable daily dose for 2,3,7,8-TCDD compatible with human health it was estimated that the tolerable daily intake was 20 times greater than the current intake. Multiplication of background environmental levels by a factor of 20 resulted in estimates of the maximum tolerable environmental concentrations for limiting human exposure to acceptable levels. These were then compared with examples of environmental standards in force at the time, and found to be several times lower than the existing standards.

The advantage of adopting a multi-media approach is clearly demonstrated, as it enables regulators to establish guidelines that take into account the significance of each exposure pathway. This is of particular importance as the principal pathway to human exposure will differ for the wide range of substances to which humans in the general population are exposed. The levels derived in this study were based on an estimate of the maximum allowable daily intake, and could be readily adjusted if new data on environmental concentrations or acceptable daily intake became available, demonstrating the flexibility of the multi-media approach to the derivation of EALs.

The approach used by Travis and Hattemeyer-Frey (1991) is almost exactly the same as the method used to determine EALs for cadmium, PCDD/Fs, nickel, mercury and arsenic in this study.

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2. EXPOSURE COMMITMENT ASSESSMENT FOR POLYCHLORINATED DIBENZO-P-DIOXINS AND DIBENZOFURANS

2.1 INTRODUCTION

Detailed estimates of intake and exposure to polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) have been made using UK data (where these have not been available data from studies in other industrialised nations have been used) and internationally-accepted reference values for the human body. These estimates compare well with a number of other exposure assessments carried out by other workers. The estimated body burden and tissue levels are in good agreement with measured levels in the general population. The review of toxicological information has found that there is still a great deal of uncertainty regarding an "acceptable level of exposure" to dioxins and furans. The World Health Organization (WHO) recommended Tolerable Daily Intake (TDI) is 10 pg TEQ/kg bw/day. The study estimates lifetime intake to be 3.1 pg TEQ/kg bw/day for the average individual in the UK. Environmental Assessment Levels (EALs) for air, fresh water and soil have been calculated.

Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans are two groups of tricyclic aromatic compounds with similar chemical and physical properties. Ubiquitous in the environment, PCDD/Fs do not occur naturally, nor are they produced intentionally. The bioavailability of PCDD/Fs depends on the matrix they are in and the route of exposure.

The main pathway to human exposure from the environment is the ingestion of herbage and soil by livestock, accumulation in the animal tissues and the subsequent consumption of dairy and meat products. Transfer from the soil to plants is very inefficient due to the physical properties of PCDD/Fs, and most PCDD/Fs in plants are of atmospheric origin.

Data on retention of PCDD/Fs in tissues of various species show a high variability between congeners. Limited data on humans indicate half-lives for 2,3,7,8-substituted PCDDs and PCDFs of 2–6 years. Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans are predominantly stored in fat, excreted in milk and pass through the placenta; they appear in blood and the vital organs at lower concentrations. Concentrations in the fat of human adipose tissue of up to 55 ng/kg fat have been recorded in the general population with no known specific exposure (Beck *et al.,* 1994). Higher levels have been reported in cases without evidence of disease. Average levels of PCDD/Fs in the body increase with age.

There are difficulties in drawing conclusions as to the relative resistance of humans to the toxic effects of PCDD/Fs. This is due firstly to the uncertainty of the real dose received by humans, and secondly the difficulty in assessing toxic effects other than chloracne, incidents of which are well documented following accidental and occupational exposure. Studies on animals have found 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) to be one of the most powerful carcinogens known to man, and yet there is still a great deal of uncertainty regarding its carcinogenic effects on humans.

2.2 ENVIRONMENTAL TRANSPORT AND BEHAVIOUR

Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans are transported in the atmosphere and may be in the vapour phase or adsorbed onto particles. Analysis of ambient air has shown that tetra- and penta-chlorinated PCDD/Fs are present at relatively high concentrations in the vapour phase (the ratio between vapour and gas phases depends on the temperature and the amount of particles present in the air) but the higher chlorinated, more lipophilic PCDD/Fs are preferentially found on particles rather than in the gas phase (Hutzinger and Fiedler, 1993). Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans can travel considerable distances in the atmosphere and are removed from the atmosphere by dry and/or wet deposition; in general, dry deposition is the dominating process (Hutzinger and Fiedler, 1993).

Their low volatility and water solubility in ambient conditions mean that movement of PCDD/Fs through the water and soil compartments is inhibited. They adsorb strongly to organic materials in soils and are not readily leached out by rain or ground water, but may be moved along with the particles to which they have absorbed. Most PCDD/Fs found in soil are present in the top 15 cm of top soil (Young, 1983); they are persistent in the soil and have half-lives in the order of years, e.g., 2,3,7,8-TCDD half-life in soil is estimated as 10–12 years (Young, 1983). The more highly chlorinated congeners are the least mobile (Helling *et al.*, 1973).

In water PCDD/Fs are preferentially adsorbed onto sediments, with about 90 per cent present in the aquatic medium in the adsorbed state, and it has been recognised that aquatic transfer mechanisms will play a minor role in exposure (Hutzinger and Fiedler, 1993).

When contamination of soil particles is excluded, PCDD/Fs in plant shoots and leaves are almost exclusively of atmospheric origin due to the very inefficient transfer from soil to plant, as PCDD/Fs adsorb strongly to soil particles and are relatively insoluble in water. For example the maximum translocation of 2,3,7,8-TCDD from soil to the aerial parts of oats and soya bean plants was estimated as 0.15 per cent (Isensee and Jones, 1971) greatly limiting transport from contaminated soil to the shoot. Although not taken up systematically, PCDD/Fs volatilizing from the soil have been shown to adsorb on to the aerial parts of plants (Sacci *et al.*, 1986) but more recent evidence suggests that evaporation of PCDD/Fs from soils to plants is not an important process (Hutzinger and Fiedler, 1993).

Similarly, high PCDD/F levels in soil do not lead to increased PCDD/F concentrations in fruits, and even when grown on highly contaminated soil, airborne PCDD/Fs are still the major source of contamination for tree fruits. After the Seveso industrial accident levels of up to 50 mg TCDD/kg were found in vegetation (Firestone, 1978). In the years following the accident when there was no direct contact with the aerosol cloud, the concentration of dioxins decreased by several orders of magnitude (Wipf and Schmid, 1983). In the fruit bodies of apples, pears, peaches and corn the levels of PCDDs were below the limit of detection (1 ng/kg) but about 100 ng/kg was detected in fruit peels, suggesting contamination due to dust settling, and not from plant uptake. It has been suggested therefore, that for consumption, peeling of fruit is a feasible precaution to reduce PCDD/F intake whereas washing has no effect (Muller *et al.*, 1993).

It has been suggested that the movement of PCDD/Fs from soil into root crops could be a major potential source of human exposure (Prinz *et al.*, 1990), but it is generally considered that concentrations in roots and tubers are unaffected by soil concentrations (Wild *et al.*, 1994). Some studies have found increased levels in the roots of plants following dosing of the soil with PCDD/Fs, but no significant increases in aerial parts of the plant (Faccheti *et al.*, 1986). A more recent study which investigated transfer of PCDD/Fs from soil to a range of crops found that only unpeeled potato tubers showed increased levels with increasing soil concentrations (Hulster and Marschner, 1993). In contrast to unpeeled potato tubers PCDD/F levels in peeled potato tubers were not related to the level of soil contamination. Peeling may therefore be a practical means of minimising PCDD/F levels in potato products for human consumption (Hulster and Marschner, 1993), and it should still be considered that under certain circumstances contamination of crops by soil particles may become an important pathway for PCDD/F accumulation.

Wild *et al.* (1994) carried out an extensive assessment of the potential influence of sewage sludge applications to agricultural land on human exposure in the UK. They concluded that the application of sludge to crop land will not significantly influence human exposure, due to the inefficiency of transfers from soil to plant tissues. However, transfers of PCDD/Fs into livestock via ingestion of soil and sludge adhered to vegetation are critical with respect to human exposure. Since the application of sludge to grazing land may result in elevated levels of PCDD/Fs in meat tissue and cows' milk (McLachlan *et al.*, 1994), it has been suggested that its use for this purpose should be banned (Kello and Yrjanheikki, 1992). Clearly the amount of sewage sludge applied and the amount of soil in harvested feeds used for livestock are important factors in determining the extent of the increase in PCDD/F concentrations in food (McLachlan *et al.*, 1994).

The main route of human exposure is from the consumption of meat and dairy products, with the main pathway from the environment being the ingestion of herbage and soil by livestock. Sources other than food (i.e., paper products, soil, air and cigarettes) are of minor importance compared with the daily exposure from the diet.

There are little data on the absorption of PCDD/Fs across the Gastro-Intestinal Tract (GI Tract) and the lung, but some analyses of faecal levels estimate absorption to be >87 per cent. Given the lack of adequate data it is assumed that, given the highly lipophilic nature of PCDD/Fs, 100 per cent of the ingested and inhaled amounts is absorbed to the blood.

2.3 ENVIRONMENTAL LEVELS

Air

The atmosphere is the principle means of transport of PCDD/Fs in global cycling. Although the loading of these compounds in air is small in proportion to the total loading in other environmental compartments (Harrad and Jones, 1992), the atmosphere plays an important role in the long-range transport and redistribution of PCDD/Fs.

Urban air

As part of the Toxic Organic Micropollutants (TOMPs) project, sponsored by the UK Department of the Environment, PCDD/Fs have been monitored at four urban sites since

January 1991. The results for the first two years of the project are summarised by Duarte Davidson *et al.* (1994) and the median levels for the individual congeners for each site are shown in Appendix 2.1. The highest median value for each congener at the four sites was used as the representative value for each congener in urban air. Naf *et al.* (1990) also produced a congener-specific analysis of air samples for Sweden, which has been used by other workers (Wild *et al.*, 1994) as representative of background levels in urban and rural air.

Rural air

At present there are little data available on levels of individual PCDD/F congeners in background rural air. Due to the importance of atmospheric inputs to agroecosystems as a route of human exposure to PCDD/Fs via the food chain, rural air concentration data are needed in order to carry out an Exposure Commitment. As more data become available it will be possible to use measured levels of PCDD/Fs. For the purpose of this assessment rural air concentrations have been derived from results for urban air in the UK.

Lorber *et al.* (1994) assessed the data available in the US, and found that although congener-specific profiles for rural settings are generally not available, studies that have measured rural air concentrations have found concentrations approximately 4–6 times lower than in urban settings. Other studies have found that PCDD/F levels in air differ from industrialised to rural regions by a factor of up to 15 (McLachlan and Hutzinger, 1990). On a local level the congener-profile (concentration of individual congeners relative to 2,3,7,8-TCDD) may vary as a result of point sources. It has been assumed that the profiles in urban and rural air are the same.

The rural background levels for individual congeners in the UK (see Appendix 2.2) were estimated by dividing the mean urban concentration (derived by taking the mean for each congener from the four sites) by 4 and 15 to give a range of values. The derived concentrations for rural air from the UK data are similar to those derived by Lorber *et al.* (1994) for the US.

Soil

Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans concentrations are significantly higher in urban areas than in rural areas, probably as a result of higher rates of atmospheric deposition of PCDD/Fs from urban air (Rappe and Kjeller, 1987; Creaser *et al.*, 1990). However, there are very few data on congener-specific background concentrations in either urban or rural soil for the UK. Kjeller *et al.* (1991) described changes in the PCDD/F content of soil and herbage due to atmospheric deposition in samples collected at the same 'semi-rural' plot from the mid-1800s to 1986, and provides a congener specific profile for tetra to octa PCDD/Fs for samples collected in 1986. These data were taken as being representative of agricultural soils in the UK, and data on urban soil levels are taken from Creaser *et al.* (1990) (Appendix 2.3).

Water

There are very few data on levels of PCDD/Fs in water, and none has been identified for sea water. A study investigating levels in unfiltered river water in two Swedish rivers has been identified as the only reference (Rappe *et al.*, 1989). However, it is known that the rivers sampled had been subject to some contamination by polychlorinated biphenyls (PCBs) in the

past, therefore the levels could not be considered to be representative of background concentrations due to the association between PCDD/F and PCB contamination. However, Travis and Hattemeyer-Frey (1991) estimated the level of 2,3,7,8-TCDD in fresh water, and this estimate together with the congener profile from the work by Rappe *et al.*, has been used to calculate predicted background levels of the 2,3,7,8-substituted congeners, by taking the estimated 2,3,7,8-TCDD concentration and using the congener profile from Rappe *et al.* (1989) to calculate levels of the remaining sixteen 2,3,7,8-substituted PCDD/Fs (Appendix 2.4).

2.4 PCDD/Fs IN FOOD

The key reference is the survey of dioxins in British foods carried out by the Ministry of Agriculture, Fisheries and Food (MAFF, 1992), but where data were absent measured levels from other sources have been used. In most cases the congener profile for specific foodstuffs is remarkably consistent between data from different studies. In addition the levels of PCDD/Fs in foods is similar in a number of industrialised nations, i.e., Western Europe and North America, although exposure may vary between average individuals in different countries as a result of differences in dietary habits.

Several papers report congener-specific levels for 2,3,7,8-substituted PCDD/Fs in a range of foods comprising national diets (Beck *et al.*, 1994; Schecter *et al.*, 1994; Schecter *et al.*, 1992; Furst *et al.*, 1990). In cases where the data are presented on a fat weight basis, conversion to fresh weight levels can be carried out using the mean per cent fat composition of the foodstuff, e.g., meat is approximately 22 per cent fat by mass, therefore a concentration of 5 ng/kg (fat weight) is equivalent to a fresh weight concentration of 1.1 ng/kg.

All data used in this assessment are taken from studies of foods from areas of background contamination, or from control samples in studies of specific contamination. Results less than the Limit of Detection (LOD) were assumed to be equal to the LOD. For each foodstuff a representative mean ΣTEQ level has been selected, and compared with other levels reported in the literature, and the congener-specific concentrations are presented in Appendices 2.5 to 2.8.

Meat
MAFF carried out analyses on a pooled sample of beef, pork and lamb (mean ΣTEQ 0.63 ng TEQ/kg), and these data are used as representative values for the combined fresh meat group. In comparison, a summary of the levels reported from Germany (Beck *et al.*, 1994; Furst *et al.*, 1990), USA (Schecter *et al.*, 1994) and the Netherlands (Theelen *et al.*, 1993) indicates that the levels measured in meat in the UK are similar to those reported in meat from areas exposed to background PCDD/Fs in other industrialised countries. Where data are reported on a fat weight basis, conversion to fresh weight has been carried out assuming that meat is 22 per cent fat by mass (MAFF, 1994).

Table 2.1 Levels of PCDD/F TEQs (ng TEQ/kg fresh weight) in meat

Source	Beef	Pork	Lamb
Beck *et al.*, 1994	0.57	0.06	0.36
Furst *et al.*, 1990	0.82	0.27	0.53
Schecter *et al.*, 1994	1.51	0.26	0.41
Theelen *et al.*, 1993	0.39	0.10	0.41
Lorber *et al.*, 1994	0.41		

Offal

There are few data on levels of PCDD/Fs in offal. The only congener-specific data (MAFF, 1992) (mean ΣTEQ 0.64 ng TEQ/kg). This level includes derived values for congeners where data are missing, when the assumptions made were that the congener profile is the same in offal and meat, and that PCDD/Fs are present exclusively in the fat of meat and offal. Given that offal (liver and kidney) are approximately 7 per cent fat by mass (ICRP, 1975), the fat concentration is approximately 9 ng TEQ/kg fat. The only other data reported in the literature are 5.7 ng TEQ/kg fat (cows liver) and 15.3 ng TEQ/kg fat (pigs liver), respectively (Theelen *et al.*, 1993). Therefore the MAFF data appear reasonably consistent with other studies.

Poultry

The reported mean ΣTEQ for poultry in the UK is 0.34 ng TEQ/kg (MAFF, 1992). Levels reported in chicken drumsticks in the USA are an order of magnitude lower, 0.03 ng/kg (Schecter *et al.*, 1994). Although it is not clear why there is such a significant difference in the levels reported in the two studies, it may be a result of variation in the fat composition of the samples. If it is assumed that poultry has approximately the same fat content as meat, then the UK data would equate to 1.6 ng TEQ/kg fat. Reported data in German poultry give levels of 2.25 ng TEQ/kg fat (Beck *et al.*, 1994) and 1.41 ng TEQ/kg fat (Furst *et al.*, 1990), so again the UK data appear to be comparable with those from other nations.

Meat products

The data reported by MAFF (mean ΣTEQ 0.2 ng TEQ/kg) represent levels in a number of pooled samples comprising a selection of all meat products consumed. Meat products (e.g., ham, bacon, sausages) may have quite variable fat composition, and the effect of preparation on PCDD/F levels is unquantified at present. Charcoal grilling or smoking may increase levels, whereas loss of fat during cooking may decrease the overall PCDD/F level. Therefore it is not appropriate to convert this result to a fat weight basis level for the purposes of comparison. However, Schecter *et al.* (1994) measured PCDD/Fs in cooked ham in the USA, on a fresh weight basis (0.03 ng TEQ/kg) and there are also reported levels in Russian meat products including sausages (0.15–0.6 ng TEQ/kg), cooked hamburger and meatball (both 0.02 ng TEQ/kg) by Schecter *et al.* (1992).

Milk

A number of studies have reported PCDD/F levels in cows' milk. The mean of 0.09 ng TEQ/kg reported by MAFF (1992) for whole milk represents milk in glass bottles as delivered to the doorstep. MAFF reports data for winter and summer, with the winter levels generally slightly higher than the summer levels. Therefore for the purpose of this report the

data used are the winter data. Schecter *et al.* (1994) reported a mean level of 0.04 ng TEQ/kg in milk in the US (based on only one sample, and not specified whether whole milk or semi-skimmed). A further study in the UK (Startin *et al.*, 1990) reports congener-specific levels for whole milk from farms in rural locations remote from likely sources of contamination, and a mean 0.045 ng TEQ/kg.

Assuming that whole milk is approximately 4 per cent fat, (DeJong *et al.*, 1993; Wild *et al.*, 1994) then the level reported by MAFF equates to 2.25 ng TEQ/kg fat. Furst *et al.* (1990) reports a background level for cow's milk in Germany of 0.6–1.6 ng TEQ/kg fat, whilst a further study of 43 dairies in North Rhine-Westphalia, Germany (Furst *et al.*, 1992) reports a mean level of 1.35 ng TEQ/kg fat (range 0.76 to 2.63 ng TEQ/kg fat). Lassek *et al.* (1993) recorded a mean level of approximately 1.0 ng TEQ/kg fat, based on samples from rural areas of Bavaria. A study of nine industrial dairies in Switzerland measured a mean level of 1.31 ng TEQ/kg fat (Schmid and Schlatter, 1992). These ranges seem to reflect the background levels of PCDD/Fs in milk from industrialised countries, and are similar to those reported by MAFF for milk in the UK.

There is only one example of congener-specific data for semi-skimmed milk (MAFF, 1992) which gives a mean level of 0.04 ng TEQ/kg. This is approximately half the level in full milk, and as semi-skimmed milk contains approximately half the fat of whole milk (~2 per cent fat) this result appears to support the assumption that, where data for semi-skimmed milk or other milk-based products are unavailable, levels can be estimated from the concentration in whole milk and the relative fat content. This method has been used by Wild *et al.* (1994) to estimated PCDD/F levels in cream, yoghurt, ice cream and butter and assumes that all PCDD/Fs are present in fat.

Cheese

MAFF reported congener-specific data for cheddar cheese (0.161 ng TEQ/kg) and reduced fat medium cheese (0.12 ng TEQ/kg). Schecter *et al.* (1994) measured individual congeners in a number of cheeses in the US; mean ΣTEQ levels of 0.04 (cottage cheese), 0.7 (soft blue cheese), 0.3 (soft cream cheese) and 0.3 ng/kg (American cheese slices) (Schecter *et al.*, 1994). The variation in PCDD/F concentrations in these results is most likely a result of the variation in fat content, and data reported on a fat weight basis from a number of studies are reasonably consistent; 1.83 ng TEQ/kg fat (Furst *et al.*, 1992), 0.6–1.3 ng TEQ/kg fat (Schecter *et al.*, 1992) and 0.98 ng TEQ/kg fat (Furst *et al.*, 1990). The representative data chosen were the MAFF data for cheddar cheese.

Butter

The congener-specific data reported by MAFF give a ΣTEQ level of 1.07 ng/kg and, assuming butter is 80 per cent fat (Wild *et al.*, 1994), this equates to a concentration of 1.34 ng TEQ/kg fat. Further reported data include 0.81 ng TEQ/kg fat (Beck *et al.*, 1994); 0.66 ng TEQ/kg fat (Furst *et al.*, 1990); 1.4 ng TEQ/kg fat (Schecter *et al.*, 1992); 1.1 ng TEQ/kg fat (Furst *et al.*, 1992).

Milk products

The data on miscellaneous milk products reported by MAFF give a mean ΣTEQ level of 0.218 ng/kg. There are no comparable data in the literature.

Ice cream and yoghurt
Schecter *et al.* (1992) reports congener-specific data for ice cream with a level of approximately 0.006 ng TEQ/kg. It is assumed that ice cream and yoghurt have approximately the same fat content (Wild *et al.*, 1994) and that this level is representative of the combined intake of these foods.

Cream
There are no UK data for levels of PCDD/Fs in cream. Congener-specific levels are reported in American heavy cream, 0.37 ng TEQ/kg (Schecter *et al.*, 1994), and in Russian cows' cream, 0.95 ng TEQ/kg (Schecter *et al.*, 1992). A derived value of 0.56 TEQ ng/kg for cream in the UK can be calculated using the whole milk level of 0.09 TEQ ng/kg, if it is assumed that cream is 25 per cent fat (Wild *et al.*, 1994). For the purpose of this assessment the higher of the two reported levels (MAFF, 1992) will be used (0.95 ng TEQ/kg).

Eggs
Congener-specific data on a fresh weight basis are reported by MAFF (mean 0.195 ng TEQ/kg). These compare with a recorded level of 0.59 ng TEQ/kg in US eggs (Birmingham *et al.*, 1989). The only other data are recorded on a fat weight basis (Beck *et al.*, 1994) with a mean level of 1.52 ng TEQ/kg fat.

Fats and oils
Two mixed samples of fats and oils based on the Total Diet Survey was analysed by MAFF, although data for 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD and 2,3,4,7,8-PeCDF were not available for one of the samples. As the samples analysed were a mixture of animal and vegetable oils it is not appropriate to calculate derived values from congener profiles. Therefore data from one set only are used (i.e., with complete congener-specific data, and highest ΣTEQ level).

Studies which have measured PCDD/Fs in animal oil and vegetable oils indicate that nearly all of the PCDD/F intake from consumption of oils is of animal origin. Values of 0.015 ng TEQ/kg fat for vegetable oil (Beck *et al.*, 1989) and 0.006 ng TEQ/kg fat for sunflower oil (Theelen *et al.*, 1993) have been reported, whereas data from Germany for animal lard indicate a mean of 0.8 ng TEQ/kg fat (Furst *et al.*, 1990). Levels in animal oils therefore appear to be approximately 2–3 orders of magnitude higher than those in vegetable oil.

Green vegetables
The only congener-specific data available are that of MAFF (mean 0.032 ng TEQ/kg, fresh weight). This value includes derived levels for congeners where data are absent, using the congener profile for PCDD/Fs in general herbage (Kjeller *et al.*, 1991). A mean level for vegetables in Germany of 0.015 ng TEQ/kg (fresh weight) has been measured by Beck *et al.*, 1989.

Potatoes
Congener-specific data are reported for two sets of samples (MAFF, 1992) although there are no data available for 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD and 2,3,4,7,8-PCDF for one of the sample sets. Therefore individual congener concentrations are taken from the more complete data set (mean 0.04 ng TEQ/kg, fresh weight).

Other vegetables
MAFF reported data for a pooled sample of "other vegetables", including carrots, onions, parsnips, tomatoes (mean 0.104 ng TEQ /kg). There are no other reports of PCDD/Fs on a congener-specific basis in the literature, but levels of between 0.1 and 0.4 ng TEQ/kg (dry mass) have been measured (Hulster and Marschner, 1993). Assuming that vegetables are 10–15 per cent dry mass this equates to a range of 0.01–0.06 TEQ ng/kg on a fresh weight basis. It appears therefore that the MAFF estimate (including derived data) is an overestimate, and therefore the data from the more complete sample result from the MAFF study, with a ΣTEQ level of 0.079 ng/kg (fresh weight) is used.

Fruit and fruit products
MAFF report data for fresh fruit (mean 0.056 ng TEQ/kg, fresh weight). German studies report similar concentrations; 0.015 ng TEQ/kg (Beck *et al.*, 1989) and 0.015–0.05 ng TEQ/kg for apples and pears (Muller *et al.*, 1993). There are no data for fruit products (mainly fruit juices) from MAFF, but levels of 0.01 to 0.079 ng TEQ/kg are reported in fruit juices packaged in bleached paperboard containers (Ryan *et al.*, 1991). The ΣTEQ of 0.056 ng/kg appears to be representative of the levels in fruits and fruit products.

Sugar and preserves
MAFF did not report data for sugars and preserves, and there are no other data on PCDD/Fs in sugar. For the purpose of this assessment the levels in sugar and preserves are assumed to be the same as those in fruit.

Bread and cereals
Most cereals contain little fat, however cereal products such as biscuits and cakes usually contain significant levels of added fat, which may have appreciable levels of PCDD/Fs (MAFF, 1992). MAFF estimated a level of 0.05 ng TEQ/kg for the cereals food group based on the fat content of cereal products and the levels of PCDD/Fs in the oils and fats group. This has been converted into congener-specific levels by using the congener profile for the fats and oils group.

Beverages
There are no data for PCDD/Fs in tea and coffee in the UK. The intake of PCDD/Fs by the consumption of beverages should not be high due to the hydrophobic nature of these substances. The levels in beverages were estimated by assuming that, as the beverages are plant-based, the PCDD/F content in coffee/tea is equal to that of green vegetation and, using the dilution factor from MAFF (1983), 118 g tea/coffee to 800 g final consumption, equivalent to a dilution factor of approximately 7 if it is assumed that levels in water are negligible. Approximately 60 per cent of TCDF present in a tea bag is extracted to the final beverage (MAFF, 1992). For example, the level of TCDD in green vegetables is <0.01 ng/kg. Therefore the estimated level of TCDD in a beverage is 0.01 multiplied by 0.6 divided by 7, giving an estimated level of 0.0008 ng/kg. If this is applied to all congeners the derived ΣTEQ level is 0.003 ng/kg.

Fish
A number of studies have investigated PCDD/F levels in fish. UK data include congener-specific levels for plaice (0.54–1.42 ng TEQ/kg), mackerel (0.61 ng TEQ/kg), herring (1.84 ng TEQ/kg), cod (0.18 ng TEQ/kg), skate (0.29 ng TEQ/kg) and coley (0.15 ng

TEQ/kg). Congener-specific mean values have been calculated, giving a mean ΣTEQ level of 0.74 ng TEQ/kg (Startin *et al.*, 1990). These data are also reported in MAFF (1992). The catch area was unknown but it is assumed that it was varied and generally distant from UK coastal waters. These data seem to be the most representative of fish consumed in the UK. If it is assumed that fish is approximately 9 per cent fat by mass (Schecter *et al.*, 1992) then the UK data equate to a concentration of 8.2 ng TEQ/kg fat. Several other studies have reported PCDD/Fs in fish on a fat weight basis (ng TEQ/kg fat) and are summarised below (Table 2.2)

Table 2.2 Levels of PCDD/F TEQs (ng/kg fat weight basis) in fish

Fish type	Mean ΣTEQ (ng TEQ/kg fat)	Source
Herring	34, 50	Beck *et al.*, 1994; Ende, 1990
Cod	43	Beck *et al.*, 1994
Fatty sea fish	7	Theelen *et al.*, 1993
Lean fish	49	Theelen *et al.*, 1993
Freshwater fish	13, 17, 18	Furst *et al.*, 1990; Ende, 1990; Frommberger, 1991
Sea fish	17, 29	Furst *et al.*, 1990; Ende, 1990

The data for fish relate to raw, unprocessed samples of fish muscle tissue, but it has been shown that the practice of smoking fish before consumption can increase PCDD/F levels significantly. The fresh weight concentration in uncooked fish was measured as 0.07 ng TEQ/kg, and is almost one order of magnitude lower than that of smoked fish (Schecter *et al.*, 1992).

2.5 CONGENER PROFILES FOR ENVIRONMENTAL COMPARTMENTS AND SELECTED FOODS

Dioxins

The profiles for meat, milk and air are similar, with a trend of increasing levels with increased chlorination, with the OCDD level approximately 50 times higher than TCDD levels (Figures 2.1 and 2.2).

Figure 2.1 Congener profile for PCDDs in milk and meat

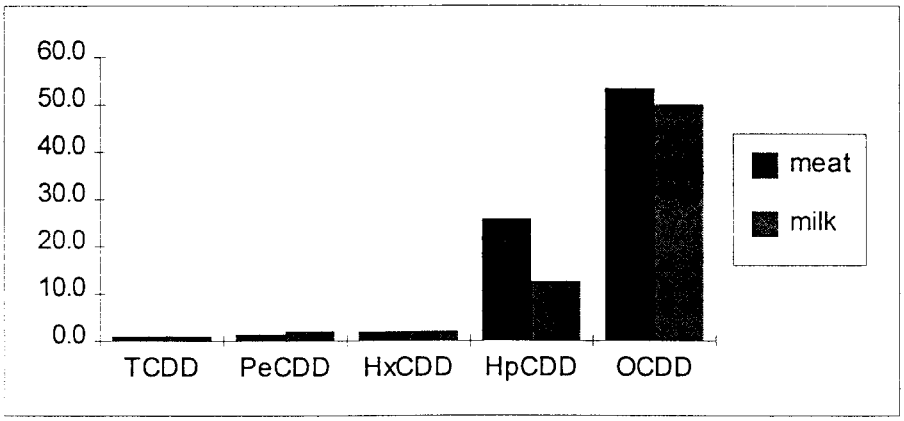
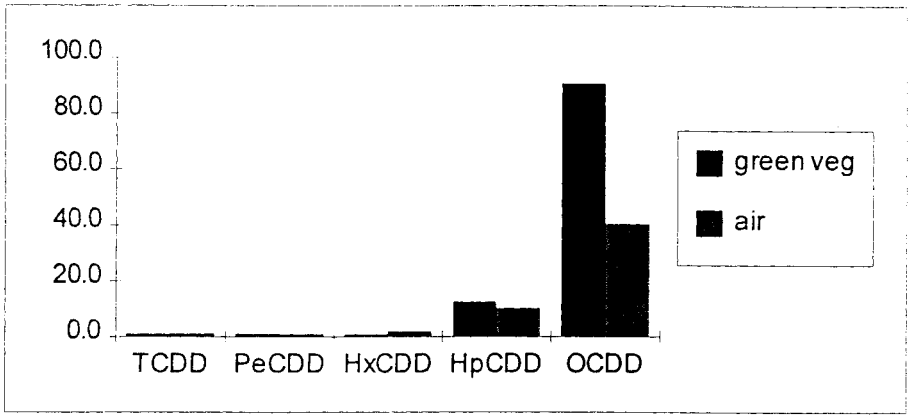
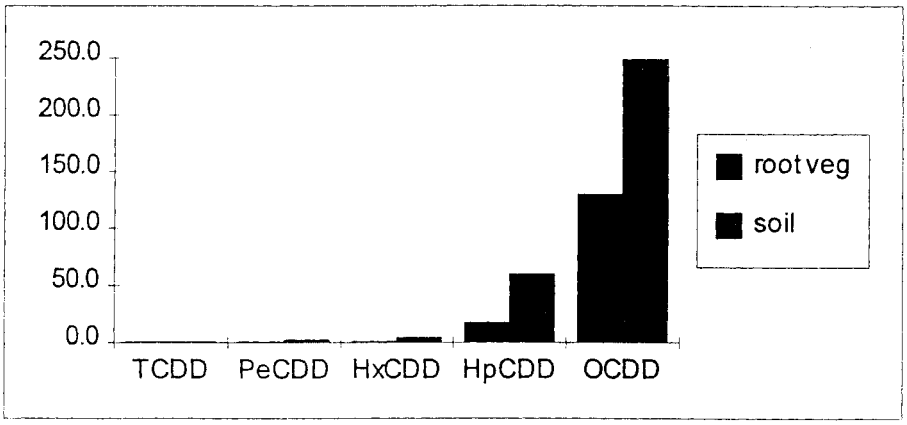


Figure 2.2 Congener profile for PCDDs in green vegetation and air



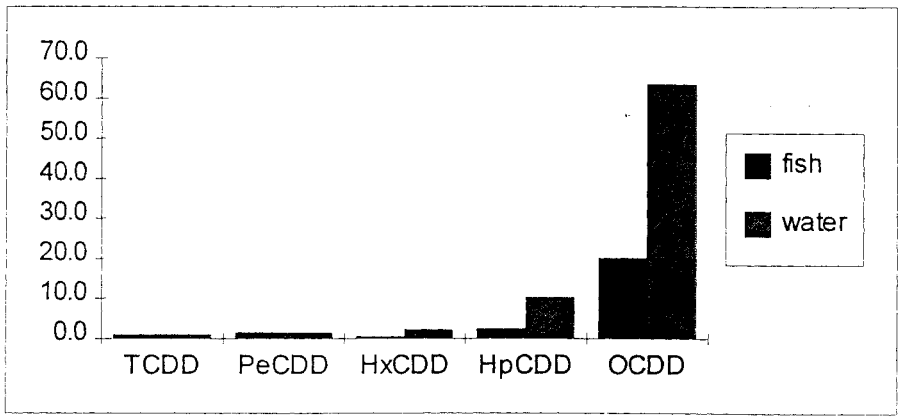
A similar trend is seen in green vegetables, but the difference between TCDD and OCDD levels is greater (approximately 130 times). Soil levels of OCDD are significantly higher than HpCDD levels (Figure 2.3).

Figure 2.3 Congener profile for PCDDs in soil and root vegetables



In fish there is a less marked increase from TCDD to OCDD (approximately 20 times greater), but in water the levels of OCDD are approximately 60 times higher than TCDD levels (Figure 2.4).

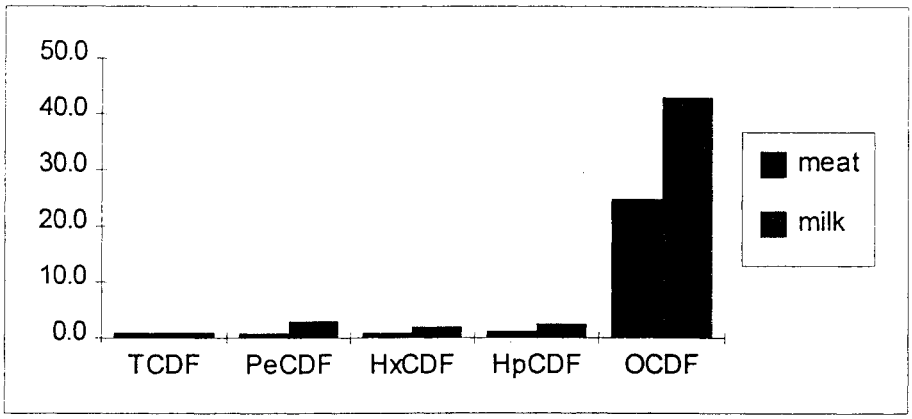
Figure 2.4 Congener profile for PCDDs in fish and water



Furans

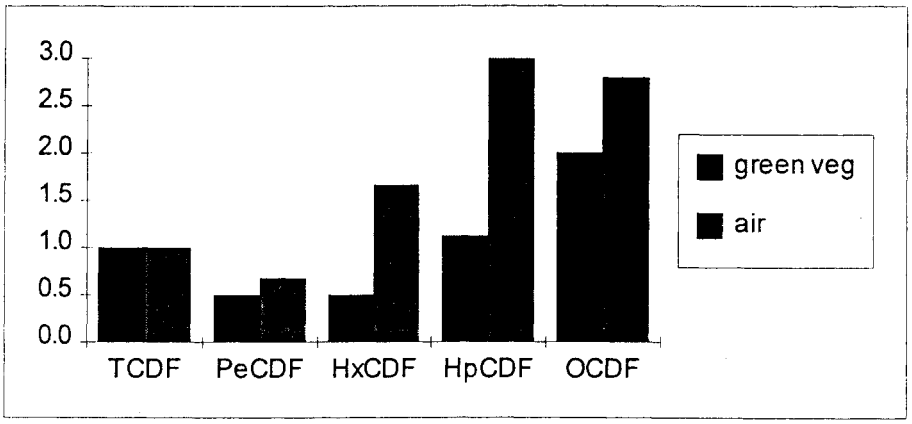
The profiles for meat and milk are similar to dioxins with OCDF levels 40 times higher than TCDF levels (Figure 2.5).

Figure 2.5 Congener profile for PCDFs in meat and milk



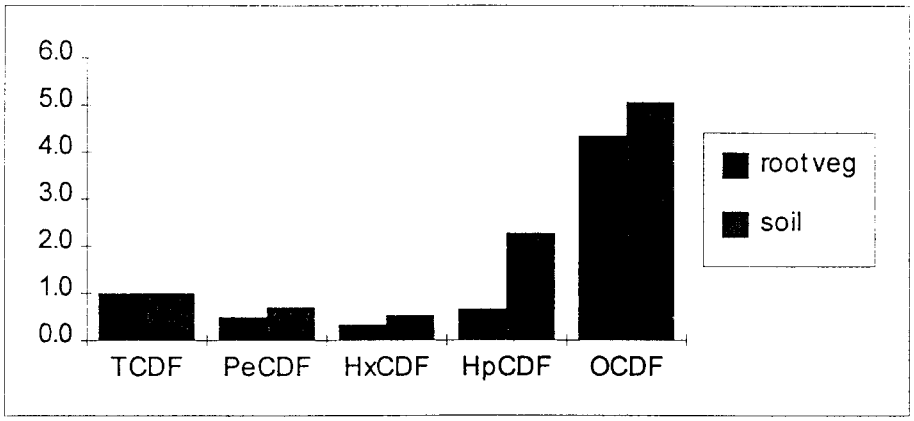
The trend for air and green vegetation concentrations is less distinct than that for meat and milk (Figure 2.6).

Figure 2.6 Congener profile for PCDFs in green vegetation and air



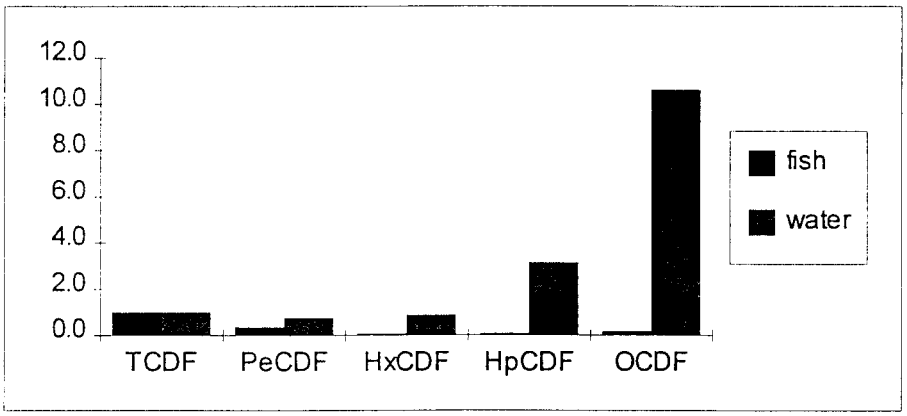
Soil shows a trend of slight increase with increasing chlorination, OCDF levels being 15 times higher than TCDF levels, and a similar but less marked trend is seen in root vegetables (Figure 2.7).

Figure 2.7 Congener profile for PCDFs in soil and root vegetables



In water there is a slight increase in concentrations with increasing chlorination, but in contrast levels of OCDF in fish are lower than levels of TCDF by an order of magnitude (Figure 2.8).

Figure 2.8 Congener profile for PCDFs in fish and water



The general trend in air, animal and vegetable matter is increasing levels with higher chlorination. In fish the levels of the more toxic TCDD relative to the less toxic congeners is higher than in animal matter, and in fish the trend for furans appears to be decreasing levels with increased chlorination. The profiles of the food types do seem to reflect the relative abundance of the different congeners in the relevant environmental media, and may also be dependent upon the congener-specific transfer into the food chain. (It should be noted, however, that due to the difficulties in analysing for PCDD/Fs, many of the reported levels are near or below the limit of detection. Therefore, the trends may also be influenced by the difference in limits of detection for the various congeners in the various studies).

2.6 INTAKE AND EXPOSURE – ESTIMATES FOR AVERAGE INDIVIDUALS IN DIFFERENT AGE GROUPS

Estimated intake through breast-feeding
(See also Appendix 2.9)

The intake for breast-fed infants can be estimated from the measured levels of PCDD/Fs in human milk in women from the general population and the average lactation rate over a given period of breast-feeding. A number of studies have measured PCDD/Fs in human milk, but results for women in the UK (Birmingham) are used in this assessment (Rappe, 1992), being 44.5 ng TEQ/kg fat.

Polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans have also been measured in Germany (35.4 ng TEQ/kg fat), Belgium (33.7 ng TEQ/kg fat), and the Netherlands (39.7 ng TEQ/kg fat) (Rappe, 1992). Further studies in Germany (Schecter *et al.*, 1991) and the Netherlands (Koopman-Esseboom *et al.*, 1994) have measured levels of 27.0 ng TEQ/kg fat and 30.2 ng TEQ/kg fat, respectively. As concentrations of PCDD/F TEQs in mothers milk and adipose tissue for west European countries are comparable (Jensen, 1989), intake of PCDD/Fs seems to be similar for the different countries.

The fat concentration was converted to a fresh weight concentration, assuming that human milk is 3 per cent fat and the specific gravity is 1.03 (ICRP, 1975).

Example:
6.5 ng TCDD/kg fat is equivalent to 0.189 pg TCDD/ml.

The consumption rate of milk by the infant is assumed to be the lactation rate of 750 ml/day (ICRP, 1975) over a three-month breast-feeding period (Duarte Davidson and Jones, 1994). The intake of TCDD for example is estimated to be 12.8 ng over the 90-day period, equivalent to a daily intake of 142 pg, or approximately 28 pg TCDD/kg bw/day (see Appendix 2.10).

The estimated daily intake of $\sum$TEQ is 1.1 ng (194 pg TEQ/kg bw/day) and is similar to the average daily intake for breast fed German infants of 142 pg TEQ/kg bw/day (Beck *et al.*, 1994).

Child and adult intake
(See also Appendices 2.5 to 2.8).

The estimated total intake is 173 pg TEQ/day for an adult consuming the average diet, with the diet contributing 96 per cent (165 pg TEQ/day) of the total. This is equivalent to a dietary intake of 2.5 pg TEQ/kg bw/day for an average 70 kg adult, with major contributions from meat (31 per cent), dairy products (22 per cent) and fats/oils (15 per cent).

Daily exposure for the general population to PCDD/Fs from food in industrialised countries is in the order of 70–260 pg TEQ (Schecter *et al.*, 1991). MAFF (1992) reported the daily intake to be ~125 pg TEQ. Estimated daily intakes have been reported for the Netherlands (Theelen *et al.*, 1991) and Canada (Birmingham *et al.*, 1989a,b), in each case the main

contributors were meat and dairy products. Reported German daily intakes from food (Beck *et al.*, 1989; Furst *et al.*, 1990) estimate that meat, dairy and fish products each contribute approximately 30 per cent of the total intake. In Italy, a significant intake from vegetables is reported (Di Domenico, 1990). Further estimates of intakes from food for non-occupationally exposed persons have been carried out in Germany (Beck *et al.*, 1994) and the US (Schecter *et al.*, 1994). The results are summarised in Table 2.3.

Table 2.3 Dietary intakes of PCDD/Fs (pg TEQ/day) from various studies

Country	Intake (pg TEQ/day)	Source
	165	This study
UK	125	MAFF, 1992
Netherlands	115	Theelen *et al.*, 1991
Canada	92	Birmingham *et al.*, 1989a
Canada	140	Birmingham *et al.*, 1989b
Germany	94	Beck *et al.*, 1989
Germany	85	Furst *et al.*, 1990
Italy	260–480	Di Domenico, 1990
Germany	161	Beck *et al.*, 1994
US	21–189	Schecter *et al.*, 1994

If the intake at different ages is considered it can be seen that, during the breast-feeding period, the intake is much greater in relation to body weight than during adult life (Table 2.4).

Table 2.4 Current intake (pg TEQ/kg bw/day) at various ages

	0–3 mths	3 mths–2 yrs	2–7 yrs	7–14 yrs	Adult	Lifetime
Intake (pg TEQ/kg bw/day)	194	1.8	1.9	2.1	2.5	2.6

It is generally agreed that diet represents the main route of exposure to PCDD/Fs and, because of the lipophilic nature of these contaminants, foodstuffs of animal origin are of special importance. On the basis of lifetime exposure, the diet is by far the main source of PCDD/F exposure (95.2 per cent), with breathing air the only other contribution of any significance (4.6 per cent). Ingestion of soil (0.1 per cent) and water (0.1 per cent) make negligible contributions to total lifetime intake (Table 2.5).

Table 2.5 Percentage contribution of different sources to lifetime exposure and for various age groups

	0–3 mths	3 mths–2 yrs	2–7 yrs	7–14 yrs	Adult	Lifetime
Diet	99.97	93.9	91.6	93.8	95.8	95.2
Air	0.03	6.0	7.8	6.0	4.1	4.6
Water	0	0.1	0.1	0.1	0.1	0.1
Soil	0	0	0.5	0.1	0	0.1

The contribution of the inhalation of air to total exposure to PCDD/Fs is relatively high in this assessment compared with some previous studies, probably due to the conservative selection of values for each congener. The contribution of inhalation will be lower for suburban and rural populations.

Jones *et al.* (1993) estimated that the average human uptake resulting from all sources was 2–10 pg TEQ/kg bw/day. The data from this assessment indicate that when the intake over a 70-year life span is considered, the daily intake is equivalent to 2.6 pg TEQ/kg bw/day (average lifetime body weight 60 kg).

Contribution of individual congeners to ΣTEQ intake

From Appendix 2.5 it can be seen that the main contributors to the ΣTEQ intake are 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD and 2,3,4,7,8-PeCDF, as these are the most toxic congeners with the highest Toxicity Equivalent Factors (TEFs). However, some of the less toxic congeners also make a significant contribution due to the higher levels present in the environment and diet, e.g., OCDD contributes 2.6 per cent of the ΣTEQ intake, despite being the least toxic of the PCDDs (1,000 times less toxic than 2,3,7,8-TCDD with a TEF of 0.001), due to the fact that it is present in foods at levels approximately two orders of magnitude higher than 2,3,7,8-TCDD. Therefore, although many of the PCDD/Fs are significantly less toxic than 2,3,7,8-TCDD, they may be of toxicological importance due to their greater environmental abundance. In addition, the environmental behaviour of PCDD/Fs is congener-specific, and therefore a risk assessment based on one congener cannot be extrapolated to other congeners (Wild *et al.*, 1994).

PCDD/Fs in food packaging materials – potential exposure

PCDD/Fs associated with chlorine-based bleaching processes have been found at significant concentrations in paper products, and migration into food in contact with these products could be an additional source of exposure. Analyses of milk products and their containers for PCDD/Fs has indicated the migration of trace amounts of these toxic compounds from the bleached container into the food. It has been suggested that continuous consumption of milk from bleached cartons could contribute significantly to human exposure (Ryan *et al.*, 1991), and a maximum level of 1 ng TEQ/kg in such food packaging materials has been recommended (Kello and Yrjanheikki, 1992).

Based on calculations of the carry-over rates, the average daily intake per person for PCDD/Fs from paper containers is estimated to be 1 pg TEQ/day (Beck *et al.*, 1991), less than 1 per cent of the total daily intake. It appears that packaging materials do not make a significant contribution to the body burden of PCDD/Fs.

Smoking – potential exposure

Based on the determination of PCDD/F in cigarette smoke (Ball *et al.*, 1990), an intake of 2 pg TEQ/day from smoking 20 cigarettes per day has been derived. Compared with the daily intake of approximately 165 pg TEQ/day from the diet it can be seen that even for long-term heavy smokers, the intake from cigarettes will not contribute significantly to the body burden.

ESTIMATION OF THE BODY BURDEN AND FAT CONCENTRATION AT VARIOUS AGES

New-born baby

There are no measurements of PCDD/F levels in new-born babies. Beck *et al.* (1994) measured levels of PCDD/Fs in infants, and reports congener-specific data for a number of infants aged 4 to 9 months. The representative level is that of a 5-month-old infant who has not been breast-fed (it is assumed that there is negligible exposure to PCDD/Fs from consumption of infant milk formula). The levels are converted to body burden values by multiplication by the mass of fat in the new-born baby of 400 g (ICRP, 1975), e.g., fat concentration of 0.2 pg TCDD/g gives a body burden of 0.08 ng. This was repeated for all congeners, and the ∑TEQ body burden at birth is estimated as 0.89 ng.

Importance of exposure to PCDD/Fs through breast-feeding

Clearly the infants intake of 194 pg TEQ/kg bw/day greatly exceeds the upper limit of the TDI of 10 pg/kg bw/day. Other studies have also concluded that the estimated PCDD/F levels ingested by infants could exceed the lifetime allowable daily intake levels set by some regulatory authorities (Beck *et al.*, 1994; Schecter *et al.*, 1991). From this assessment it is estimated that breast-feeding contributes 99 ng TEQ to the total lifetime intake of 3313 ng (70-year lifetime), and therefore despite the high intake with respect to body weight during the breast-feeding period, the overall contribution to lifetime exposure is approximately 3 per cent.

Due to the high dose/body weight ratio during breast-feeding, elevated childhood exposure may be cause for concern despite the fact that the TDI is a guideline to lifetime exposure (Law and Gudaitis, 1994). Although for risk assessment the daily intake over the entire lifetime has to be considered, the short-term intake of levels exceeding the toxicological limits during breast-feeding is of concern both because of the long half-lives of some of these compounds, and also in the light of prospective health care.

According to the German regulatory authority, Bundesgesundheitsant (BGA), no adverse health effects have been demonstrated in breast-fed infants, and it is generally agreed that the advantages of breast-feeding outweigh the theoretical risks (Beck *et al.*, 1994) and therefore is generally recommended by WHO. The concentrations of PCDD/Fs in target organs are not expected to be dramatically elevated as a consequence of breast-feeding because of the rapid increase in the amount of fatty tissue during infancy (Gilman and Newhook, 1991).

Estimated exposure to PCDD/Fs and calculated body burden

The estimated intake from all sources can be used to calculate the exposure to the body, by multiplying the intake by the absorption factor. There are virtually no data on absorption factors for the various congeners, and the most useful information available is from a study on a human volunteer who ingested radio-labelled TCDD in corn oil (Poiger and Schlatter, 1986). Faecal analysis was used to estimate absorption at >87 per cent. For the purpose of this assessment it is assumed that PCDD/Fs ingested and inhaled are absorbed completely, i.e., exposure = intake.

Body burden is estimated by using biological half-time values for PCDD/Fs in the body to account for metabolic losses. Again there are very little data on the behaviour of PCDD/Fs in the body with respect to derivation of biological half-lives. Poiger and Schlatter (1986) derived a half-life for TCDD in the body of 5.8 years, and a study by Geyer *et al.* (1986) calculated a half-life for TCDD of 3.5 to 6.9 years. A study which derived and validated a mathematical model to evaluate the dose received during breast-feeding (Sullivan *et al.*, 1991) used a half-life for PCDD/Fs of 8 years. For the purpose of the general assessment the half-life was assumed to be 8 years.

Example:
The half-life is 8 years, therefore the retention time in the body is 11.54 years
(from the relationship: Retention time = half-life ÷ l n (2)).
The estimated body burden of TCDD at 14 years is 53 ng.
The estimated exposure between 14 and 20 years is 85 ng.
The body burden at age 15, before accounting for metabolic losses, is 53 + 85 = 138 ng.
If the retention time is 11.54 years, and it is assumed that metabolic losses are a linear function of time, the proportion of the original body burden lost during the 6 year period is 6 ÷ 11.54. Therefore the amount remaining is 25.4 ng.
The proportion of the TCDD intake lost during the 6-year period is assumed to be 3 ÷ 16 (i.e., the intake amount is subject to metabolic losses for the mean period of time, 3 years). Therefore the amount of the intake remaining after the 6-year period is estimated as 62.9 ng.
Therefore the estimated body burden at age 15 years is 25.4 + 62.9 = 88.3 ng.

(If it was assumed that the intake is not subject to any metabolic losses during the 6-year period the body burden calculated would be an overestimate; conversely if it was assumed that 100 per cent of the intake was subject to metabolic losses over the full 6-year period, the calculated body burden would be an underestimate).

The concentration in fat is calculated by dividing the body burden by the mass of fat at the given age (from ICRP, 1975), and was carried out for both males and females. There are significant differences between males and females in the amount of fat tissue in the body at various ages, and these will affect the predicted concentrations of PCDD/Fs in fat tissue (Appendix 2.11)

An alternative method of calculating the body or tissue concentrations can be carried out using the equation from Bennett (1981), which relates the time-independent steady-state compartment concentration to intake, compartment size and retention time in that compartment. This involves the calculation of the concentration from the following equation:

$C = T/M \times F$ Where C = Concentration in compartment
T = Retention time
M = Mass of compartment
F = Flux or intake to the compartment

In the case of this calculation, the retention time is 11.54 years (as mentioned previously), and the mass refers to the mass of fat tissue in average individuals in each age group (kg). The concentration is the level in the fat tissue (ng TEQ/kg) and the flux is the annual total intake

of PCDD/Fs (ng TEQ/year). The figures used in the calculation are summarised below in Table 2.6, and the proportions of fat in the body, the average body mass and annual intakes for each age group are the same as those used to calculate the fat concentration using the half life, as previously detailed.

Table 2.6 Data used for the calculation of fat tissue concentrations of TEQs at various ages

	0–3 mths	3 mths–2 yrs	2–7 yrs	7–14 yrs	Adult	Lifetime
Intake (ng/yr)	355	8	17	32	63	56
Body mass (kg)	5	12	24	43	64	58
Fraction of fat	0.19	0.2	0.16	0.17	0.23	0.22
Mass of fat (kg)	1.0	2.4	3.8	7.3	16.1	13.2
Fat concentration (ng/kg)	**4,311**	**39**	**50**	**51**	**47**	**48**

As can be seen from Table 2.6, this approach provides a reasonable estimate for the equilibrium concentration in adults, but does not detect the trend of increasing fat levels of PCDD/Fs with age, which is well documented in the literature and demonstrated using the alternative approach. Secondly, this method overestimates the levels in infants and young children, particularly in the case of the infant where the estimate is several orders of magnitude higher than measured levels.

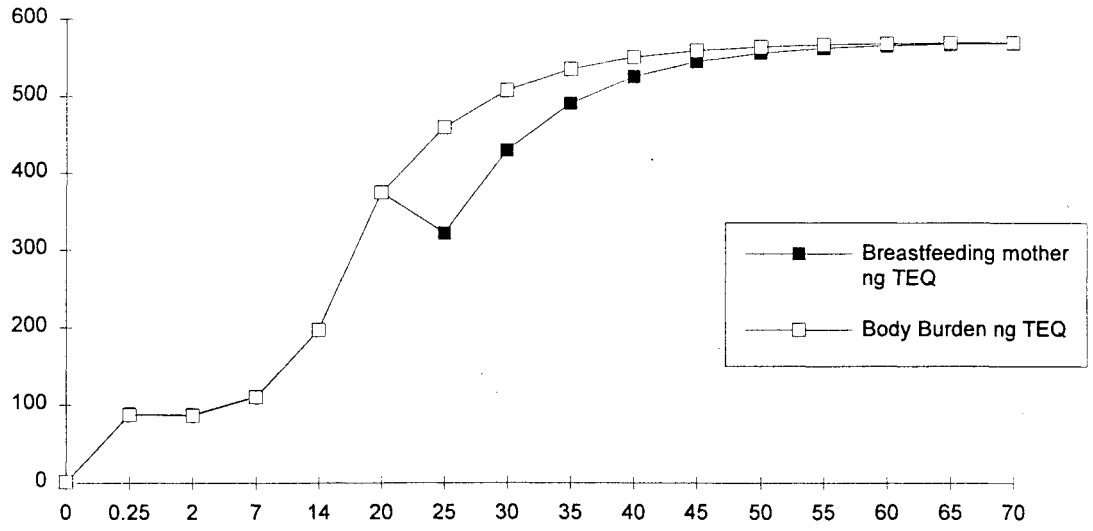
This is a result of the long retention time in the body in comparison with the relatively short periods of time being considered, and therefore it is not possible for steady-state concentrations to be reached in the body. This demonstrates the limitation of the use of the above relationship to calculations of steady-state body and tissue concentrations when the retention time is substantially longer than the period of time for which the calculation is being made. Therefore the time-dependent calculation using the half-life and adjusting for metabolic losses appears to produce more realistic estimates of the tissue concentrations. It is suggested, therefore, that the steady-state relationship is only valid for calculations when the half-life is relatively short in comparison to the period of time in question, or the period of time for which the steady-state concentration is relatively long, i.e., in the case of substances with half-life of several years, the calculation of the steady-state concentrations is acceptable only for the intake, which for adults occurs over a period of 56 years, and gives sufficient time for concentrations to approach equilibrium.

Effect of breast-feeding on the mother's body burden

It has been suggested that maternal losses of PCDD/Fs through breast milk may be significant (Sullivan *et al.*, 1991). To estimate the loss of PCDD/F body burden it was assumed that the woman undertakes two three-month breast-feeding periods between the ages of 20 and 25. The daily loss of PCDD/Fs through breast-feeding was estimated from the lactation rate and the mean levels of the PCDD/F congeners present in human milk, e.g., TCDD, concentration is 0.189 pg TCDD/ml, daily loss is 161 pg. Total loss over the two breast-feeding periods was 28.9 ng.

The estimated loss of PCDD/Fs was then subtracted from the intake between 20 and 25 years before calculating the body burden. The results are shown graphically in Figure 2.9.

Figure 2.9 Body burden (mg) vs. age (years) for the average individual and a breast-feeding mother



Comparison of estimated body burden with measured body burdens

Polychlorinated dibenzo-p-dioxin and polychlorinated dibenzofuran levels measured in human tissues in industrialised countries are in general greater than levels in less industrialised countries (Schecter *et al.*, 1991). A number of studies have measured the levels of PCDD/Fs in human milk fat, adipose tissue and blood fat in adults, and are summarised below. The concentrations have been converted to body burdens by assuming that the average adult contains 16.1 kg of fat tissue (ICRP, 1975) (see also Appendix 2.12).

Table 2.7 PCDD/F concentrations in human tissues (ng TEQ/kg fat weight basis) and estimated body burdens from various studies

COUNTRY	CONCENTRATION (ng TEQ/kg fat)	BODY BURDEN (ng)	SOURCE
Blood			
USA	41	656	Schecter *et al.*, 1991
Germany	40	640	Schecter *et al.*, 1991
Adipose Tissue			
Germany	55	880	Beck *et al.*, 1994
Canada	38	608	Teschke *et al.*, 1992
Human milk			
UK	45	720	Rappe, 1992
Germany	35	560	Rappe, 1992
Belgium	38	608	Rappe, 1992
Netherlands	40	640	Rappe, 1992

In this assessment the estimated body burden for 20 to 70-year-old adults has a mean value of 530 ng (Appendix 2.11). The mean concentration can be calculated by dividing by the mean mass of fat in the body, giving a fat concentration of 33 ng/kg. These data appear to agree

with the argument that fat concentrations in western European countries are comparable, and therefore the intake of PCDD/Fs in these industrialised nations is similar.

In the US an extensive monitoring programme, the National Human Adipose Tissue Survey (NHATS), operated by the US EPA, has assessed human exposure to potentially toxic compounds. It has produced a whole population estimate (by using samples from subjects in age, sex and racial categories, representative of the entire nation) of fat concentrations of 29 ng/kg fat (430 ng body burden). This is slightly lower than the other estimates, as the studies by Beck *et al.* (1994) and Teschke *et al.* (1992) used samples only from adults. The NHATS has detected statistically significant differences between men and women, and reports that the average PCDD/F fat concentrations tend to increase with age. If the average lifetime body burden is considered from this study the body burden is approximately 421 ng, very close to that measured by the NHATS survey in the whole population.

This assessment has assumed that PCDD/Fs are equally distributed in fat tissue, i.e., the concentrations on a fat weight basis are the same in adipose tissue, blood and human milk. However in general, PCDD/F levels are higher in blood fat and lower in milk fat, so human tissue levels on a fat weight basis can most readily be compared using data for the same tissue type (Schecter *et al.*, 1991).

Comparison of measured and estimated congener-specific body burdens and estimation of congener-specific half-lives

The measured levels of PCDD/Fs in adult fat tissue are compared with the levels derived using the estimated daily intake and an overall half-life in the body for all congeners of 8 years. Using the measured levels of PCDD/Fs in adult subjects (Schecter *et al.*, 1991), and the estimated intake of specific PCDD/Fs from this assessment, the half-lives of individual congeners can be calculated as follows.

The retention time is calculated from the following relationship (Bennett, 1981):

$C = T/M \times F$ Where C = Congener concentration in body (ng/kg fat)
T = Retention time in body in years
M = Mass of fat in the body (16.1 kg)
F = Intake rate of congener (ng/yr)

The half life can then be calculated from the following relationship (Bennett, 1981):

Half-life = Retention Time x ln (2).

The derived half-lives vary considerably, but it is interesting to note that the mean of all PCDD/Fs is 5.7 years, and therefore similar to the overall half-life of 8 years used in this assessment.

The half-life of 2,3,7,8-TCDD has been measured at about 5.8 years (Poiger and Schlatter, 1986), but when possible excretion in the urine was taken into account the estimated half life was 4.5 years. Geyer *et al.* (1986) calculated the 2,3,7,8-TCDD half-life to be between 3.5

and 6.9 years. A shorter half-life for the furan 2,3,7,8-TCDF of 2 years has been calculated from information on incidents of rice oil poisonings (Kunita *et al.*, 1984).

Gorski *et al.* (1984) reported half-lives of 3.5, 3.2 and 5.7 years for HxCDD, HpCDD and OCDD respectively in a young girl exposed to pentachlorophenol. The same study reported half-lives of less than 1.8 years for HpCDF and OCDF.

Table 2.8 Estimated half-lives for PCDD/F congeners (from predicted body fat concentrations), measured and predicted equilibrium concentrations (ng TEQ/kg) and half-lives reported in the literature

	Measured concentration	Predicted concentration	Half-life (years)	Reported half-lives
2,3,7,8-TCDD	5	9	4.0	3.5–6.9
1,2,3,7,8-PCDD	21	13	8.5	
1,2,3,4,7,8-HxCDD	13	10	9.5	3.5
1,2,3,6,7,8-HxCDD	84	25	28.0	3.5
1,2,3,7,8,9-HxCDD	15	11	9.5	3.5
1,2,3,4,6,7,8-HpCDD	187	200	5.5	3.2
OCDD	1,174	1,070	6.0	5.7
2,3,7,8-TCDF	3	24	0.5	2
2,3,4,7,8-PCDF	13	20	4.0	
1,2,3,7,8-PCDF	3	9	1.5	
1,2,3,4,7,8-HxCDF	15	14	5.5	
1,2,3,7,8,9-HxCDF	1	6	2.0	
1,2,3,6,7,8-HxCDF	14	9	5.0	
2,3,4,6,7,8-HxCDF	4	9	1.5	
1,2,3,4,6,7,8-HpCDF	36	42	4.5	<1.8
1,2,3,4,7,8,9-HpCDF	2	8	1.0	<1.8
OCDF	4	159	<0.5	<1.8
Mean half-life			**5.7**	

2.8 TOXICOLOGICAL ASSESSMENT

Humans are exposed to mixtures of PCDD/Fs, and the TDI for all PCDD/F congeners is related to the toxicological data (predominantly on 2,3,7,8-TCDD) by TEFs to produce toxic equivalents. Therefore the TDI of 10 pg TCDD/kg bw/day should refer to a TDI for 2,3,7,8-TCDD and its equivalents. The TEQs calculated by multiplying the concentration of the congener by its TEF are summed, assuming that the toxic effect of the congeners in a mixture is additive. It is important to calculate exposure to all 2,3,7,8-substituted PCDD/Fs rather than 2,3,7,8-TCDD alone, for the simple reason that 2,3,7,8-TCDD makes up only about 22 per cent of the ΣTEQ intake; a risk assessment based on 2,3,7,8-TCDD alone would seriously underestimate total exposure to ΣPCDD/Fs by a factor of 4.55.

Based on a combination of experimental toxicology and epidemiological data, and applying a safety factor of 100 to account for inter-species and individual variation, the generally accepted supposition is that man would not be affected by a lifetime exposure to PCDD/Fs of

1–10 pg TCDD/kg bw/day. Consequently, a number of regulatory agencies have adopted "Tolerable Daily Intake" values for ΣPCDD/Fs which range from 1 to 10 pg TEQ/kg bw/day.

An expert group convened by the WHO Regional Office for Europe recommended a TDI for 2,3,7,8-TCDD of 10 pg/kg bw/day, derived by extrapolation from laboratory animal studies, but also taking into account differences in the pharmokinetics of 2,3,7,8-TCDD in laboratory animals and man (WHO, 1991). For the derivation of this value, 2,3,7,8-TCDD was considered to be non-genotoxic, acting as a promoter-carcinogen, and consequently the TDI was established on general toxicological effects (Kello and Yrjanheikki, 1992). The majority of European countries have accepted this value.

The UK Department of Health Committee on the Toxicity of Chemicals in Food, Consumer Products and the Environment, recommends that an intake of 1 pg TEQ/kg bw/day be considered as a guideline value (i.e., a level which exceeded should trigger investigation and measures to reduce environmental levels generally). It is not intended that this level is interpreted as a "safety level" or as an "acceptable daily intake". MAFF recommends that when considering mixtures of PCDD/Fs, the TDI can be regarded as 10 pg TEQ/kg bw/day, but that the TEFs used to calculate TEQ from the levels of individual congeners should come under review as new data become available. In Germany, the BGA fixed a TDI of 1–10 pg TEQ/kg bw/day.

The Canadian Government have also adopted a TDI of 10 pg TEQ/kg bw/day, based on a threshold assumption for TCDD and related congeners, calculated by dividing the no-observed-adverse-effect level (NOAEL) by a multiple of several safety factors (McColl, 1989). However, they also recommend a "value of precaution" of 1 pg/kg bw/day, and emphasise that efforts should be made to limit daily uptake to less than 1 pg/kg bw/day. It is apparent that current intakes in most industrialised nations, including the UK, are greater than the UK's guideline and the Canadian "value of precaution".

Other workers have suggested TDIs in the range of 1–10 pg TEQ/kg bw/day, including Liem *et al.* (1991), who quote a tolerable intake of 240 pg TEQ/day, equivalent to a TDI of 3.4 pg TEQ/kg bw/day for a 70 kg adult. The margin between this value and the current estimates of intake in the UK (2.6 pg TEQ/kg bw/day) is small.

In contrast, US EPA treats 2,3,7,8-TCDD and related congeners as complete carcinogens and the estimates of tolerable exposure are considerably lower than the ones accepted in Europe. The estimated dose, over a 70-year lifetime, which would lead to one excess cancer per one million people is 0.0064 pg/kg bw/day (Safe, 1991). However, it has been suggested that 2,3,7,8-TCDD may act as a tumour promoter rather than an initiator, and therefore have a toxicity threshold (Greenlee *et al.*, 1991). According to Law and Gudaitis (1994) the US EPA estimate of the toxic potency is very conservative. Less conservative estimates of the cancer potency factor (CPF) used to calculate the acceptable daily intake value, based on a re-evaluation of the original data, result in a higher acceptable value of 0.1 pg/kg bw/day (Keenan *et al.*, 1991). Despite this, the value is considerably lower than the WHO TDI based on general toxic effects, and is at least one order of magnitude lower than the current intakes.

It has been suggested that body or tissue concentrations accumulated over time may better predict health effects. The body burden or fat levels as a result of the dose remain elevated

over time and may be continually available to body tissues, particularly in the case of compounds with long biological half lives such as PCDD/Fs (Sullivan *et al.*, 1991).

2,3,7,8-TCDD is toxic in animals at very low concentrations and adverse effects occur in rats at concentrations of 3 ng/kg (Kueger *et al.*, 1990) but the evidence in humans is inconclusive. Results from the Seveso population, in 1976, with 2,3,7,8-TCDD concentrations in blood fat of up to 56,000 ng/kg revealed no TCDD-induced clinical, immunological or other biochemical effects, except cases of chloracne immediately after the exposure. It has been suggested that, in light of these findings, levels found in fat tissue of the non-occupationally exposed population in Europe (30–50 pg/g) are not a cause for concern (Schmid and Schlatter, 1992).

Kello and Yrjanheikki (1992) have suggested that reproductive effects in humans are unlikely to occur at doses substantially different to those causing other effects. Therefore the TDI may be appropriate for these human health effects as well as general toxicity. However, by using a reproductive end-point, rather than cancer, Rao and Brown (1990) estimated 125 pg TEQ/day (equivalent to 1.8 pg TEQ/kg bw/day) to be the highest daily dose compatible with human health. This figure is less than the current estimates of daily intake in many industrialised nations, and therefore the current levels of exposure may be cause for concern with respect to human reproductive effects.

In summary, the current levels of exposure appear to be higher than the 1 pg TEQ/kg bw/day "value of precaution" above which action to reduce environmental levels generally should be taken. Many workers have suggested that PCDD/F emissions should be reduced and where possible prevented altogether. The level of 10 pg TEQ/kg bw/day can be considered as a "preventative value" (Prinz *et al.*, 1993), and in the absence of reliable epidemiological evidence linking environmental exposure to current background levels of PCDD/Fs with human health effects, it is this value which has been used in the calculation of Environmental Assessment Levels (EALs). However, given the considerable debate surrounding the human health effects of PCDD/Fs and the wide range of suggested tolerable levels of exposure/intake, the EALs derived from this TDI should be treated with considerable caution, and the overriding aim should be to reduce current emissions and prevent new emissions of PCDD/Fs into the environment.

2.9 DERIVATION OF EALS – FROM TDI VALUES AND ESTIMATED INTAKE AND BACKGROUND ENVIRONMENTAL LEVELS

As mentioned previously, the current lifetime daily intake of 2.6 pg TEQ/kg bw/day is higher than the "guideline value" of 1 pg TEQ/kg bw/day, and therefore steps to reduce environmental levels generally should be appropriate. Secondly, the estimated intake is significantly higher than both the US EPA estimate (0.0064 pg TEQ/kg bw/day) and the less conservative estimate of 0.1 pg/kg bw/day (Keenan *et al.*, 1991) of the dose which results in one excess cancer per million people. However, evidence of carcinogenicity of PCDD/Fs in humans is inconclusive.

The TDI, as recommended by the WHO Regional Office for Europe, is 10 pg TEQ/kg bw/day, and MAFF consider the TDI for mixtures of PCDD/Fs as 10 pg TEQ/kg bw/day

using the international TEFs. Environmental assessment levels for 2,3,7,8-substituted PCDD/Fs have been estimated using this value.

Air

As the levels in rural air lead to the main exposure through the food chain, the EALs are related to the current intake of 2.6 pg TEQ/kg bw/day and the concentrations of the congeners in rural air. At present there are no data available for rural air, and so the levels are derived from measurements of urban air (Duarte Davidson *et al.*, 1994) and factors from the literature that estimate rural air concentrations to be 4–15 times lower than urban air, and therefore the upper and lower ranges of the estimated EALs are given below (Appendix 2.2).

Table 2.9 EALs for PCDD/F congeners in UK air

Congener	EAL (pg/m^3)	EAL (pg/m^3)
2,3,7,8-TCDD	0.01	0.04
1,2,3,7,8-PCDD	0.01	0.03
1,2,3,4,7,8-HxCDD	0.02	0.07
1,2,3,6,7,8-HxCDD	0.02	0.08
1,2,3,7,8,9-HxCDD	0.02	0.07
1,2,3,4,6,7,8-HpCDD	0.10	0.37
OCDD	0.39	1.47
2,3,7,8-TCDF	0.02	0.08
2,3,4,7,8-PCDF	0.01	0.04
1,2,3,7,8-PCDF	0.02	0.07
1,2,3,4,7,8-HxCDF	0.04	0.16
1,2,3,7,8,9-HxCDF	0.01	0.04
1,2,3,6,7,8-HxCDF	0.07	0.28
2,3,4,6,7,8-HxCDF	0.02	0.08
1,2,3,4,6,7,8-HpCDF	0.08	0.29
1,2,3,4,7,8,9-HpCDF	0.05	0.20
OCDF	0.06	0.23
Total PCDD/Fs	**0.96**	**3.60**
TEQ	**0.05**	**0.17**

Soil

Despite the fact that PCDD/Fs in soil are not effectively transferred to plant material, soil is an important source of exposure via the terrestrial food chain due to the ingestion of soil by livestock. The EALs for soil are calculated from the current intake of 2.6 pg TEQ/kg bw/day and the concentrations of the congeners in semi-rural soil recorded by Kjeller *et al.* (1991) (see Appendix 2.3). These data may be revised if levels of PCDD/Fs in rural soil in the UK are available, at present the data are presented as a mean value for all UK soils by Creaser *et al.* (1989).

Table 2.10 EALs for PCDD/F congeners in UK soil

Congener	EAL (ng/kg)
2,3,7,8-TCDD	0.4
1,2,3,7,8-PCDD	1.1
1,2,3,4,7,8-HxCDD	1.4
1,2,3,6,7,8-HxCDD	2.4
1,2,3,7,8,9-HxCDD	1.8
1,2,3,4,6,7,8-HpCDD	22.0
OCDD	91.3
2,3,7,8-TCDF	4.0
2,3,4,7,8-PCDF	4.6
1,2,3,7,8-PCDF	2.1
1,2,3,4,7,8-HxCDF	2.7
1,2,3,7,8,9-HxCDF	0.04
1,2,3,6,7,8-HxCDF	3.3
2,3,4,6,7,8-HxCDF	2.6
1,2,3,4,6,7,8-HpCDF	16.6
1,2,3,4,7,8,9-HpCDF	1.6
OCDF	20.3
Total PCDD/Fs	**177.0**
TEQ	**5.1**

Fresh water

Despite the low levels of PCDD/Fs in water, the ingestion of fish may be a significant pathway of exposure. The EALs for fresh water are calculated from the current intake of 2.6 pg TEQ/kg bw/day and derived concentrations for PCDD/Fs in fresh water. The only data available are from two rivers which had been subject to some contamination by PCBs. Therefore, these concentrations are probably higher than general background freshwater levels. Background levels in uncontaminated river water have been estimated from the congener profile for PCDD/Fs in the Swedish rivers sampled by Rappe *et al.* (1989) and the level of 2,3,7,8-TCDD in fresh water estimated by Travis and Hattemeyer-Frey (1991); the measured level of 2,3,7,8-TCDD was 0.022 pg/l, whereas the estimated level of 2,3,7,8-TCDD in fresh water, not contaminated from point sources, is considerably lower, 0.003 pg/l. The predicted levels in uncontaminated rivers are shown in Appendix 2.4, and the resulting EALs in Table 2.11.

Table 2.11 Calculated EALs for PCDD/F congeners in UK fresh waters

Congener	EAL (pg/l)
2,3,7,8-TCDD	0.01
1,2,3,7,8-PCDD	0.02
1,2,3,4,7,8-HxCDD	0.02
1,2,3,6,7,8-HxCDD	0.04
1,2,3,7,8,9-HxCDD	0.02
1,2,3,4,6,7,8-HpCDD	0.12
OCDD	0.74
2,3,7,8-TCDF	0.01
2,3,4,7,8-PCDF	0.01
1,2,3,7,8-PCDF	0.01
1,2,3,4,7,8-HxCDF	0.01
1,2,3,7,8,9-HxCDF	0.01
1,2,3,6,7,8-HxCDF	0.01
2,3,4,6,7,8-HxCDF	0.01
1,2,3,4,6,7,8-HpCDF	0.06
1,2,3,4,7,8,9-HpCDF	0.02
OCDF	0.14
Total PCDD/Fs	**1.26**
TEQ	**0.04**

2.10 ENVIRONMENTAL STANDARDS FOR PCDD/FS

Two recent studies have examined the use of an integrated approach consistent with the exposure commitment methodology to develop standards and guidelines for PCDD/Fs in environmental media.

Prinz *et al.* (1993) conducted a study in Germany that used an integrated approach to produce guidelines for PCDD/Fs in ambient air. It was stressed that although the existence of PCDD/Fs in a number of environmental media means that various standards and criteria are needed, an integrated approach is not only desirable, but absolutely necessary. Calculations were based on the precautionary principle employed by the Federal German Government, and the standards were established using the TDI for PCDD/Fs, pathways to exposure and transfer factors between different compartments.

Air quality standards were derived by back-calculation from the current and tolerable intakes. In terms of human exposure, the lower limit of the TDI (1 pg TEQ/kg bw/day) was identified as a precautionary level, and the upper limit of the TDI (10 pg TEQ/kg bw/day) as a preventative level. It was also noted that the current intake is estimated at around 2 pg TEQ/kg bw/day in Germany, greater than the defined precautionary level. The transfer factors used were defined as "quantitative relationships between continuous compartments or media", the same definition as that used for transfer factors in exposure commitment.

Standards were calculated for both deposition rates of PCDD/Fs, and for PCDD/F air concentrations in non-rural areas expressed in TEQs. Although in the current study the concentrations of individual congeners is preferred, the TEQ values calculated for the range of air EALs is in the range 0.05–0.17 pg TEQ/m^3 (see Table 2.9). The quality standard calculated by Prinz *et al.* (1993) **using a similar approach to the one used in this study,** was 0.12–0.15 pg TEQ/m^3, indicating that the results from the two studies are comparable.

The second study used a similar multi-media approach (Travis and Hattmer-Frey, 1991) to estimate environmental standards for 2,3,7,8-TCDD. Pathways and the extent of human exposure to 2,3,7,8-TCDD in the adult general population of the US were quantified. The results are very similar to those from this assessment: diet 98.8 per cent, air 1.1 per cent, soil 0.05 per cent and water 0.01 per cent (see Table 2.5 for comparison of these results with the current study). Based on a TDI of 10 pg TCDD/kg bw/day for a 70 kg individual (i.e., a daily intake of 700 pg TCDD per person), and a background daily intake of 35 pg TCDD per person (c.f. 29 pg TCDD per person in this assessment) it was suggested that the TDI was 20 times higher than the average daily intake. Background environmental levels were derived from the Fugacity Food Chain (FFC) model (Travis and Hattemer-Frey, 1987) and multiplied by 20 to give estimates of the maximum tolerable environmental concentrations for limiting human exposure to 2,3,7,8-TCDD to tolerable levels.

Air	0.4 pg TCDD/m^3
Water	0.06 pg TCDD/l
Soil	20 ng TCDD/kg

The major disadvantage of assessing exposure to 2,3,7,8-TCDD alone is that it does not account for the exposure from the other sixteen 2,3,7,8-substituted congeners thus demonstrating the problem of using a single congener. The daily intake of 10 pg/kg bw/day is applied to TCDD equivalents, i.e., TEQs (for example by MAFF) for mixtures of PCDD/Fs. From the current assessment it has been calculated that the contribution of 2,3,7,8-TCDD to ΣTEQs is approximately 22 per cent. Therefore the TDI for 2,3,7,8-TCDD is 700 multiplied by 0.22, giving a TDI for 2,3,7,8-TCDD of 154 pg TCDD/per person/day. Therefore the TDI for 2,3,7,8-TCDD is 4.4 times higher than the average daily intake of 35 pg TCDD/per person/day. Therefore the maximum tolerable concentrations calculated from the revised values are:

Air	0.09 pg TCDD/m^3
Water	0.01 pg TCDD/l
Soil	4.4 ng TCDD/kg

These compare with the EALs calculated in this assessment using the same principles, but using a TDI of 10 pg TEQ/kg bw/day, an average lifetime intake of 2.6 pg TEQ/kg bw/day and measured environmental levels rather than levels derived from the FFC model; the environmental levels are elevated by a factor of 4.6.

Air	0.01–0.04 pg TCDD/m^3
Water	0.01 pg TCDD/l
Soil	0.44 ng TCDD/kg

A number of different organisations have proposed ambient air quality standards for PCDD/Fs, and again the range of values proposed varies greatly. The State of Connecticut have suggested an Air Quality Standard for 2,3,7,8-TCDD of 1 pg/m^3 (Rao and Brown, 1990) and the Federal Government of Canada a standard of 5 pg/m^3 (Federal-Ontario, 1988). The values derived by Travis and Hattemeyer-Frey (1991) and using exposure commitment are approximately 25 to 500 times lower than those suggested by these two organisations. It is unclear whether these standards apply to urban air alone or general background air quality. Given that urban air levels of PCDD/Fs are probably 4–6 times higher than background levels, and that the margin between current and tolerable exposure is a factor of 4.6, it is clear that the current urban levels are approaching the EALs proposed using exposure commitment. This appears to demonstrate the importance of using a multimedia approach to assess total exposure, and therefore derive environmental quality standards that relate to human health aspects.

In contrast in the US, New York authorities stipulate a limit of 0.09 pg TCDD/m^3 as an annual arithmetic mean, which is far more compatible with the proposed EALs, and the standards suggested by Travis and Hattemeyer-Frey (1991), and Prinz *et al.* (1993).

In Canada the guideline for ∑PCDD/Fs is 30 pg/m^3, which is higher than the total concentration of 2,3,7,8-substituted PCDD/Fs suggested by this study of 0.96 to 3.6 pg/m^3.

The US EPA has set a maximum allowable level for 2,3,7,8-TCDD in drinking water as low as 0.013 pg/l based on an estimated cancer risk of one excess cancer per million. If it is assumed that this standard applies also to surface waters, then it can be compared with suggested EAL for 2,3,7,8-TCDD for fresh waters of 0.01 pg/l. The two standards, derived from different data and using different methods are nevertheless, comparable.

The various Environmental Standards for TCDD, ∑PCDD/Fs and TEQs are summarised in Table 2.12 below.

	RANGE	SOURCE
Air	0.05-0.17 pg TEQ/m^3	This study
	0.12-0.15 pg TEQ/m^3	Prinz *et al.*, 1993
	0.4 pg TCDD/m^3	Travis and Hattemeyer Frey, 1987
	0.01-0.04 pg TCDD/m^3	This study
	1 pg TCDD/m^3 *	Rao and Brown, 1990
	5 pg TCDD/m^3	Federal-Ontario, 1988
	0.09 TCDD/m^3	Travis and Hattemeyer Frey, 1991
	30 pg ∑PCDD/F /m^3 **	Travis and Hattemeyer Frey, 1991
	0.96-3.6 pg ∑PCDD/F /m^3	This study
Water	0.013 pg TCDD/l	US EPA
	0.01 pg TCDD/l	This study
	0.06 pg TCDD/l	Travis and Hattemeyer Frey, 1991

2.11 SUMMARY

1. Data on PCDD/Fs in environmental media are limited; there are reasonable data for urban air, but virtually none for background air, soil or fresh water. No data on levels in sea water have been found.
2. There have been a number of studies investigating PCDD/Fs in food, data are consistent and estimates of intake, exposure and body concentrations appear to be reasonably accurate.
3. The main sources of exposure to PCDD/Fs are meat (30 per cent), dairy products (22 per cent) and fish (8 per cent).
4. Limited transfer from the soil to above ground plant tissue makes this route of exposure less important than atmospheric deposition onto grazing land, and the ingestion of contaminated pasture and soil and subsequent accumulation of PCDD/Fs by livestock.
5. The total daily intake of 2.6 pg/kg bw/day appears representative of the lifetime of an average UK individual.
6. The Tolerable Daily Intake of 1–10 pg/kg bw/day is the most widely-used measure of acceptable exposure to PCDD/Fs.
7. Using the TDI and current estimates of intake, maximum allowable environmental concentrations compatible with human health (accounting for lifetime exposure) are 3.85 times greater than current background levels. Environmental Assessment Levels have been calculated on this basis.
8. Comparison with results from other attempts to set air quality standards for PCDD/Fs indicate the proposed EALs are similar to those proposed by workers in other countries.

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3. EXPOSURE COMMITMENT ASSESSMENT FOR CADMIUM

3.1 INTRODUCTION

Detailed estimates of intake and exposure to cadmium have been made using UK data (where these have been unavailable, data from other industrialised nations have been used) and internationally accepted reference values for the human body. These estimates and those of body burden and tissue levels compare well with a number of other exposure assessments carried out by other workers, and measured levels in the body tissues of the general population. The review of toxicological information has identified the critical concentration of 200 μg Cd/g (wet weight) in the renal cortex, defined by the WHO Task Group on Metal Toxicity, and the Provisional Tolerable Weekly Intake (PTWI) for adults of 500 μg defined by the World Health Organization/Food and Agriculture Organization of the United Nations (WHO/FAO), as the key measures of acceptable levels of exposure. Environmental Assessment Levels (EALs) for air, fresh water, soil and sea water have been calculated.

Cadmium is present in the earth's crust and may be released to the atmosphere by volcanic activity, vegetation and in the form of wind-blown dusts. The atmosphere plays a major role in the long-range transport of cadmium. The availability of soil cadmium to plants is dependent on a number of factors, and while generally cadmium is readily taken up by plants, a straightforward relationship between plant and soil levels is not always possible. While atmospheric deposition onto aquatic environments is important, for fresh waters the major input is from soil erosion.

Due to the input of cadmium to the human food chain from the use of phosphate fertilisers, the exposure commitment has been used to quantify the contribution to total human exposure, and account for this source when calculating tolerable environmental concentrations.

Animals and man acquire cadmium mainly through ingestion, and in the body it accumulates mainly in the liver and kidneys, the levels increase with age. The major routes of exposure for the non-smoking general population is via food, and the contribution to exposure from other pathways is relatively small. However, tobacco may make a considerable contribution, and heavy smoking may give rise to exposure equal to that due to food consumption.

Occupational exposure to cadmium is mainly through inhalation, and there is evidence that long-term occupational exposure to high cadmium levels may contribute to the development of lung cancer, and accumulation in the renal cortex of the kidney can cause renal tubular dysfunction. The kidney is considered the target organ for both the general and occupationally exposed populations.

In humans one third of the total body burden is in the kidney and the renal cortex concentration is approximately 1.5 times that of the whole kidney. Movement of cadmium through the placenta is limited (WHO, 1992), and the new-born baby is practically free of cadmium.

In chronically exposed human populations the critical concentration appears to be between 180–220 μg/g. The WHO Task Group on Metal Toxicity defined the critical concentration in the renal cortex as 200 μg Cd/g (wet weight). However, cadmium accumulated under these

conditions is distributed between more than one compartment, a major fraction for example is bound to metallothionein and may be relatively inert (Foulkes, 1986). Therefore this value may be an underestimate in terms of the concentration at which real adverse health effects take place.

3.2 ENVIRONMENTAL TRANSPORT AND BEHAVIOUR

Cadmium occurs naturally in the earth's crust and may be released to the atmosphere by volcanic activity, vegetation and in the form of wind-blown dusts. The atmosphere represents an important means of long-range transport. In the environment it is found with zinc, and the two metals have chemical and physical similarities.

Atmospheric cadmium is deposited on soils by dry and wet deposition and is efficiently bound in clay and basic soils. Cadmium is more mobile in sandy and acidic soils. From the soil it is readily taken up by plants, and some leafy species (e.g., spinach, lettuce) can accumulate very high levels of cadmium in their edible parts; levels of up to 1,070 µg/kg (fresh weight) have been found in spinach grown in the UK in agricultural areas treated with sewage sludge (MAFF, 1983). Generally, the fruit and seeds of plants tend to contain less cadmium than the leaves (Bingham, 1979). For other plant species, however, cadmium may have toxic effects (interfering with photosynthesis) at much lower concentrations.

The uptake of cadmium into plants generally depends on the availability of the metal in the soil solution. It is not solely dependent on soil concentration, but also the soil pH and composition, the nature of the soil clays and the organic matter content. The relationship between soil cadmium levels and plant uptake is not a simple one, because of the wide variety of soil characteristics that affect the extent of cadmium uptake. Therefore, a straightforward correlation between plant and soil levels is not always possible (Boudene, 1979).

Phosphate fertilisers, which have been widely applied to agricultural land, may contain high levels of cadmium, so long-term use leads to elevated cadmium levels in soil resulting in higher than background levels in plants (Baker *et al.*, 1979). Crop plants grown near atmospheric point sources of cadmium may contain elevated levels (WHO, 1992b), but it is not always possible to distinguish whether the cadmium is derived directly from surface deposition, or originates from root uptake, since the soil concentrations in the affected area are generally higher than background levels.

Atmospheric cadmium may also be deposited on aquatic environments, although a major input to fresh waters is from soil erosion. A number of cadmium salts are practically insoluble in water (e.g., cadmium oxide) while others are water-soluble (e.g., cadmium nitrate). The speculation of cadmium in the aquatic environment therefore is of great importance to the extent of exposure through the aquatic food chain and drinking water.

Much of the cadmium entering fresh waters is rapidly adsorbed by particulate matter, which may remain suspended or settle out; therefore it is possible to detect low dissolved cadmium concentrations in fresh waters that receive considerable inputs of from contaminated discharges. Rivers contaminated with cadmium can contaminate the surrounding land either through irrigation, or dumping of dredged sediments and flooding.

Once in the aquatic environment cadmium is readily accumulated by aquatic plants and shellfish, and may also accumulate to high concentrations in the liver and kidneys of fish. Freshwater organisms are affected by cadmium at lower concentrations at increasing salinity and water hardness, both of which reduce the uptake by organisms and therefore the toxic impact. The organic content of water can also greatly affect the environmental toxicity of cadmium by binding it and reducing its availability to organisms. Calcium competes with the free cadmium ion in aquatic organisms, reducing its toxicity, but in contrast zinc increases toxicity to aquatic invertebrates.

The main pathways to human exposure from environmental sources are via the consumption of vegetables and cereals, although individuals who consume large quantities of seafood (especially shellfish) and offal (liver and kidney) may receive considerable exposures via these routes. Smoking of tobacco is also a substantial source of cadmium, which for heavy smokers may be equal to exposure from the diet.

Reported values for the absorption across the Gastro-Intestinal Tract (GI Tract) vary, with the reported transfer factor ranging from 0.01 to 0.12 (Bennett, 1981). However, several studies have found the average absorption factor to be around 0.05 (WHO, 1992a) and 0.045 (Friberg *et al.*, 1974). For example, a figure of 0.06 is widely accepted and used for risk assessment purposes by the US Environmental Protection Agency (US EPA) (Owen, 1990) and is the value used in this assessment. Similarly, values for absorption across the lung vary from 0.15 to 0.5 (Bennett, 1981), with an estimated mean of 0.25 (Friberg *et al.*, 1974). More recently a value of 0.4 has been used regularly for risk assessment purposes (Owen, 1990).

Cadmium tends to accumulate in the liver and kidneys of both animals and humans. Data on tissue concentrations of cadmium in humans and animals have been reported and all show the highest concentrations to be found in the kidney, followed by the liver. The concentration in the cortex of the kidney is approximately 1.5 times higher than in the kidney as a whole. Approximately one third of the total body burden is in the kidneys (Bennett, 1981; Friberg *et al.*, 1974; WHO, 1992a), and therefore the partitioning factor for cadmium exposure to the kidney in the body is 0.33. The kidney is considered the target organ for both the general and occupationally exposed populations.

Phosphate fertiliser application and its contribution to soil levels relative to atmospheric deposition

In Europe the amount of cadmium entering agricultural soils from the application of phosphate fertiliser is similar to, if not greater than, the total input from all atmospheric sources (Hutton, 1982). Cadmium is removed from the atmosphere by dry deposition and by precipitation (WHO, 1992a). A representative value for agricultural areas in the EC is suggested by Hutton (1982) as 3 g/ha/yr. A measured rate of 2.6 g/ha/yr is recorded for Norfolk (Horler and Barbour, 1979).

The rate of application of cadmium to arable land due to phosphate fertiliser application in the UK is estimated at 6.5 g/ha/yr (Hutton, 1982).

Sewage sludge application may give rise to markedly higher soil cadmium levels on a local level contributing up to 90 per cent of total soil cadmium, but as it is applied to less than

5 per cent of agricultural land in the UK, on a national and regional level it is thought to account for less than 2 per cent of the total input (Hutton, 1982). Long-term use of phosphate fertilisers will give rise to elevated soil cadmium levels, but there is also evidence that its addition may actually reduce the bioavailability of soil cadmium.

It is assumed that the concentration in agricultural soils is a result of atmospheric input and fertiliser input. Given the estimated application rate for fertiliser in the UK (6.5 g/ha/yr) and the estimated atmospheric deposition rate in the UK (2.6 g/ha/yr) it is possible to calculate the relative contributions of the two sources, assuming that the input rates and soil concentrations are at equilibrium, as being 30 per cent from the atmosphere and 70 per cent from fertiliser application. On a national basis, the proportion of cadmium reaching UK crop land from air is estimated to be 41 per cent, from fertilisers 54 per cent and **5 per cent from other sources, such as sewage sludge** (Yost and Miles, 1979). A more conservative estimate, used in this assessment, is that approximately 50 per cent of the cadmium present in vegetable and cereal crops originates from phosphate fertiliser use, to attempt to ensure that the atmospheric contribution to final exposure is not underestimated, and therefore that the EALs calculated are not overestimates (and therefore not suitable for the protection of human health).

3.3 ENVIRONMENTAL LEVELS

(See Appendix 3.3)

Air

Rural air

Concentrations range from 0.1–6 ng/m^3 (Bennett, 1981 – who used 1 ng/m^3 as the representative value in a summary Exposure Commitment for cadmium). The range of annual means for two sites in Wales has been recorded as 0.12–0.83 ng/m^3, with an overall average for both sites over the four year period of 0.42 ng/m^3 (Bertorelli, 1994). Cawse (1977) also gives a range of annual averages for seven rural sites in the UK of 1–2.7 ng/m^3. The levels in rural air are given as a range, and the EALs calculated are therefore also expressed as a range of possible values. For the purpose of the calculation of EALs, background rural air was assumed to have cadmium levels in the range **of 0.12 to 0.83 ng/m^3, based on the most recent data collected by Bertorelli (1994).**

Urban air

Cadmium concentrations in urban areas are higher than in rural localities, corresponding in general to an increase of approximately one order of magnitude (Hutton, 1981). Levels vary between 1–50 ng/m^3 according to location and degree of industrialisation (Bennett, 1981). The range of annual means for five urban sites in England and Scotland was 0.9–10 ng/m^3, with an overall average over the five-year period of 3.8 ng/m^3 (Bertorelli, 1994). The range of annual means for four urban sites in Wales was 0.43–3.5 ng/m^3, with an overall average over the four-year period of 1.5 ng/m^3. The range of annual means for five sites in London was 3.6–8.9 ng/m^3 (Branson and Pattendon, 1979).

The level in urban air is used for the calculation of intake by the general population, as the vast majority of the UK population live in urban areas, and also this gives the assessment of the inhalation pathway an appropriately conservative bias. The representative value chosen

for background cadmium levels in urban air was 9 ng/m^3, corresponding to the highest annual mean recorded in an urban UK setting.

Soil

Background soil concentrations will vary to some extent depending on the parent rock from which they are derived (Hutton, 1982). Levels in soil are generally less than 1,000 μg Cd/kg in non-polluted areas, and are usually between 200–400 μg Cd/kg (Bennett, 1981). In the major US agricultural areas the mean soil level ranges from 150–370 μg Cd/kg, with an overall mean of 270 μg Cd/kg (Holmgren *et al.*, 1985).

It was assumed that urban soil concentrations are an order of magnitude higher than the mean background concentration. (It was assumed that the increase of one order of magnitude in urban air concentrations relative to background concentrations would result in a corresponding increase in the rate of atmospheric deposition, and therefore in the soil concentration).

Taking a representative mean value of 300 μg/kg for background soil (not subject to the addition of phosphate fertiliser) an urban soil level of 3,000 μg/kg was used for the calculation of total exposure. The representative values for background soil concentrations have been expressed as a range of 200–400 μg Cd/kg for the purposes of calculating the EAL.

Water

Sea water contains between 0.04 and 0.3 μg/l (Friberg *et al.*, 1979), whilst levels in fresh water are generally less than 1.0 μg/l (Bennett, 1981). The representative values used in the calculation of EALs were 0.5 μg Cd/l for fresh water (half the limit of detection) and 0.04–0.3 μg Cd/l for sea water.

3.4 CADMIUM IN FOOD

(See Appendix 3.3)

The Ministry of Agriculture, Fisheries and Food (MAFF) carried out a comprehensive survey (MAFF, 1983) but many of the data were below the limit of detection (LOD). Therefore where data from other sources are available, and are given as actual measured levels rather than <LOD, these data have been used.

Meat and offal

A number of key references have been identified which provide data for countries in Europe, including the UK. In animals, as in humans, cadmium accumulates in the liver and kidneys, with the highest reported levels in the kidneys. In comparison, levels in meat muscle tissue are low. The representative values are summarised below (the literature sources and ranges of recorded levels can be found in Appendix 3.1).

Table 3.1 Cadmium levels in meat (μg Cd/kg fresh weight)

MEAT TISSUE	LEVEL (μg Cd/kg fresh weight)	SOURCE
Beef	6	Falandysz, 1993
Pork	1.5	Niemi *et al.*, 1991
Lamb	2	Schulzschroeder, 1991
Liver	271	Schulzschroeder, 1991
Kidney	830	Schmidt *et al.*, 1988

Meat products

Levels are assumed to be approximately the same as meat muscle tissues; the representative value has been calculated by averaging the levels in beef, pork and lamb. The representative level is therefore approximately 3 μg Cd/kg (assuming that the three meats constitute equal proportions of meat products).

Poultry

High levels of cadmium are found in phosphate poultry feeds (1–67 mg/kg) resulting in relatively high exposure to poultry from dietary cadmium in these feed supplements, in comparison to environmental sources (Sullivan *et al.*, 1994). In the US there are already restrictions on the use of kidneys from mature poultry as human food due to concern over cadmium levels (Coleman *et al.*, 1992). Therefore the transfer of cadmium to humans from environment due to consumption of poultry products can be regarded as negligible in comparison with direct input to the human food chain through the use of phosphate feeds. The data can be found in Appendix 3.1, and the representative value is 12 μg Cd/kg (Piekacz, 1976).

Whole milk and milk products

Levels in milk and milk products are generally low due to the low biotransfer factor for cadmium from cattle feed to milk (Stevens, 1991). Even milk from cattle in cadmium-polluted areas has been found to have a low cadmium content (Schwarz *et al.*, 1991). In Denmark it has been estimated that milk and milk products contribute less than 4 per cent of the total Danish dietary intake of cadmium, and can be considered as toxicologically insignificant (Larsen and Rasmussen, 1991). Some representative mean values for whole milk from cattle in uncontaminated areas include <2 μg Cd/kg (MAFF, 1983), 0.044 μg Cd/kg (Jeng *et al.*, 1994), and <1.0 μg Cd/kg (Larsen and Rasmussen, 1991). The selected representative value is 1 μg Cd/kg. As there are no data for other milk products, it has been assumed that the levels are the same as in whole milk.

Eggs

Despite the importance of phosphate feeds, which can contain relatively high concentrations of cadmium, transfer from the bird's diet to eggs is relatively low (Sullivan *et al.*, 1994). MAFF (1983) have detected cadmium in eggs, the mean value being <30 μg Cd/kg (range <30–40 μg Cd/kg) and the mean level in eggs from birds not fed phosphate supplement is estimated at 3.8 μg Cd/kg (Jeng and Yang, 1995). Levels in eggs may increase fivefold when fed with a supplement containing 75 Cd mg/kg (Bokori *et al.*, 1995), and generally the levels of cadmium in commercially used phosphate feed supplements range from 1 to 67 mg Cd/kg (Sullivan *et al.*, 1994). Assuming the phosphate feeds contain approximately 60 mg Cd/kg,

and using the levels recorded in eggs from birds raised without feed supplements, it is estimated that the use of phosphate feeds will increase the background levels in eggs by a factor of 4. Therefore, the representative value is estimated to be 16 μg Cd/kg (i.e., 3.8 μg Cd/kg multiplied by 4).

Vegetable oil

The range of cadmium levels in vegetable oils (e.g., corn oil and olive oil) is surprisingly narrow (23–33 μg Cd/kg) (Sattar *et al.*, 1993). The assigned value is 33 μg Cd/kg.

Animal oil

The only recorded data found were from MAFF, 1983, with a range of <30–40 μg Cd/kg and a mean value of <30 μg Cd/kg. The assumption made was that levels in animal oils are similar to those in other meat products, and the assigned value is 3 μg Cd/kg.

Vegetables

There are a number of sources of data for levels in cadmium in vegetable crops, and there is a great deal of scientific literature documenting the factors affecting uptake of cadmium and the distribution of cadmium within plants. A number of key references have been identified which provide data for vegetable crops from non-contaminated areas in the UK, other European countries and the US. These values have been compared, and in each case the highest recorded mean value from the literature sources has been used as the representative value; these values are summarised in Table 3.2 (literature sources and ranges can be found in Appendix 3.2).

Table 3.2 Cadmium levels in vegetables (μg Cd/kg fresh weight)

VEGETABLE	LEVEL (μg Cd/kg fresh weight)	SOURCE
Cabbage	44	Kaferstein *et al.*, 1979
Lettuce	93	Page *et al.*, 1981
Peas	27	Dowdy and Larson, 1975
Potato	50	Kaferstein *et al.*, 1979
Onion	80	Page *et al.*, 1981
Carrot	89	Giordano *et al.*, 1979
Tomato	30	Page *et al.*, 1981

This may well result in an overestimate of the total dietary intake of cadmium for the majority of the population. It is well documented that elevated cadmium levels in soil give rise to significantly higher concentrations in vegetables, so the estimated intake in this report may be an underestimate for populations consuming vegetables exclusively from contaminated areas, treated with sewage sludge, or with naturally elevated soil cadmium levels. However, the aim of the assessment is to estimate the intake and exposure for the general UK population as a whole.

Fruit

In comparison to vegetables there are relatively little data on cadmium levels in fresh fruit. However, MAFF 1983 measured mean levels of <100 μg Cd/kg in raspberries (range <10–390 μg Cd/kg). Levels in different fruit species also vary, e.g., mean values for strawberry 24 μg Cd/kg, blackcurrant 2 μg Cd/kg, redcurrant 7 μg Cd/kg and apple <1 μg Cd/kg

(Tahvonen and Kumpulainen, 1991). It is possible that the lower concentrations found in tree fruits are due to the fact that they are less contaminated with soil particles than strawberries, for example, which fruit close to the ground.

The level in fresh fruit has been calculated using the relative proportions of strawberries, blackcurrants, redcurrants and apples consumed by the general UK and the data from Tahvonen and Kumpulainen (1991). The ratios are 1:1:1:8 for strawberries, blackcurrants, redcurrants and apples, respectively. The weighted mean from these data is 3.7 μg Cd/kg, and was used as the representative value.

MAFF (1983) recorded a mean value for fruit products (mainly fruit juices) of <10 μg Cd/kg (range <10–20 μg Cd/kg). The level in fruit products was assumed to be the same as for fresh fruit.

Sugar and preserves

Levels of cadmium in sugar are generally low, although samples of sugar beet usually contain higher levels than cane-sugar (Sattar *et al.*, 1993). Data include a mean of <20 μg Cd/kg, range <10–20 μg Cd/kg for sugar (MAFF, 1983), and 1 μg Cd/kg for sugar and 3.5 μg Cd/kg for syrup (Khoulif *et al.*, 1993). The representative value for sugar and preserves used is 3.5 μg Cd/kg.

Breads and cereals

Levels of cadmium recorded in bread and cereals are reasonably consistent. Tahvonen and Kumpulainen (1994) recorded mean levels of 30 μg Cd/kg and 27 μg Cd/kg in wholemeal bread and white bread, respectively. These compare well with the concentrations measured by MAFF (1983) of 30 μg Cd/kg for wholemeal bread, 20 μg Cd/kg for white bread, and 30 μg Cd/kg for flour. The selected value for all bread and cereals was 30 μg Cd/kg.

Fish

MAFF, 1983 has extensive records on cadmium in fish caught in UK coastal waters. The selected representative values are freshwater fish (trout) 29 μg Cd/kg, marine fish (Cod) 170 μg Cd/kg and shellfish (mussels) 940 μg Cd/kg. The value for canned fish is 20 μg Cd/kg (MAFF, 1983).

Drinking water

Levels in drinking water are generally low, <1 μg/l (Bennett, 1981); 95 per cent of samples contained less than 2 μg/l, and only one sample contained more than 4 μg/l (MAFF, 1983). The representative value for drinking water is 1 μg/l.

3.5 INTAKE AND EXPOSURE – ESTIMATES FOR AVERAGE INDIVIDUALS IN DIFFERENT AGE GROUPS

(See Appendices 3.4–3.7)

The data on cadmium levels in selected representative food types, drinking water, urban soil and urban air have been used together with information on the average consumption rates of different foods in the UK, the consumption rate for drinking water and soil, and the representative breathing rate to calculate the annual intake of cadmium for the average

individual in the general population in five age groups. The annual intake is obtained by multiplying the amount consumed, or breathed, by the average cadmium concentration in the media.

Intake from breast-feeding
(See Appendix 3.8)

Cadmium intake and exposure through breast-feeding was estimated using a mean of 0.08 ng/ml in human milk (calculated from 210 samples, Dabeka *et al.*, 1986) and assuming an average of 750 ml of milk excreted per day by the mother during lactation (IRCP, 1975) over a three-month period of breast-feeding. The estimated intake due to breast-feeding is 5.4 µg over the three-month period, and is negligible in comparison to lifetime exposure due to dietary intake.

The daily intakes (µg Cd/kg bw/day) are summarised in Table 3.3, and the relative contributions to intake from different sources are summarised in Table 3.4.

Table 3.3 Daily intake of cadmium (µg Cd/kg bw/day) in various age groups

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Intake	0.01	0.34	0.35	0.38	0.46	0.44

Table 3.4 Percentage contribution to intake from different sources

SOURCE	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Diet	0.89	92.9	89.3	92.1	93.0	92.3
Soil		0.0	3.6	0.9	0.2	0.5
Water		6.2	5.9	6.1	6.2	6.1
Air	0.11	0.9	1.2	0.9	0.6	0.7

Sources of exposure to the general population
The exposure to the body (the blood) is calculated by multiplication of the intakes by the absorption factors for the GI tract (food, soil, water) and the lung (air). As mentioned previously, these transfer factors are 0.06 (GI tract) and 0.4 (lung). The exposure to the body was then divided by 3 to give the exposure to the kidney, as approximately one third of the body burden is found in the kidneys. The relative contributions to the final exposure for the five age groups is summarised in Table 3.6.

Table 3.5 Exposure to the body (µg Cd/kg bw/day) and kidney (µg Cd/g kidney/day)

EXPOSURE	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Exposure to the body (µg/yr)	2.39	93	197	376	734	641
Exposure (µg/kg bw/day)	0.001	0.02	0.02	0.02	0.03	0.03
Exposure to kidney (µg/yr)	0.8	31.1	65.6	125.5	244.6	214
Kidney mass (g)	26	50	105	170	290	
Exposure (µg Cd/g kidney/day)	0.03	0.62	0.62	0.74	0.84	0.81

Table 3.6 Percentage contribution to lifetime cadmium exposure to the renal cortex for various age groups

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	14–70 YRS	LIFETIME
Diet	55	88	84	87	90	90
Air	45	6	7	6	4	4
Water	–	6	6	6	6	6
Soil	–	–	3	1	0	0

Ingested cadmium is the major source of cadmium intake for the general population, with the main route of exposure being via the ingestion of food (90 per cent).

The main route of exposure for the general population is air-soil-plant-diet-blood-kidney. The ingestion of plant matter (vegetables and cereals) makes up approximately 78 per cent of the total dietary intake, and contributes 70 per cent of the lifetime exposure to cadmium for the average individual. Given the relationship between plant and soil cadmium concentrations, and due to elevated soil levels due to the application of phosphate fertilisers, this pathway represents a major source of exposure.

The air-soil-plant-livestock-diet pathway represents a much smaller source of exposure (9.4 per cent from meat and all dairy products), whilst consumption of fish (9.2 per cent) is also a relatively small, but potentially significant, source of exposure. For the general population inhalation is a minor pathway of exposure (0.6 per cent), and negligible contributions are made by the ingestion of soil (0.02 per cent) and drinking water (0.01 per cent).

Smoking

Tobacco plants naturally accumulate high levels of cadmium in their leaves. It has been reported that an average cigarette contains 1–2 μg, and that about 10 per cent of this amount is inhaled, although this may be reduced by filters (WHO, 1992a). Therefore a person smoking 20 cigarettes per day would take in an additional 4 μg of cadmium (equivalent to 0.06 μg/kg bw/day), and in terms of exposure this would mean an extra 0.02 μg/kg bw/day absorbed to the blood assuming that 40 per cent of the cadmium is adsorbed. This would mean that smoking would contribute an additional 17 per cent in terms of intake, but an additional 70 per cent in terms of exposure. Several studies have found that heavy smoking may contribute an equal amount to daily exposure as that derived from the diet. Therefore, smoking is an important source of cadmium exposure in certain individuals.

3.6 BODY BURDENS AND CONCENTRATION IN THE RENAL CORTEX – ESTIMATES FOR INDIVIDUALS AT VARIOUS AGES

The new-born baby

The movement of cadmium through the placenta is limited (WHO, 1992a), and is minimised in the normal, healthy placenta by the binding of cadmium to metallothionein. Levels of cadmium in the organs of new-born babies are lower by 2 to 3 orders of magnitude than the levels in organs of adult females (Henke *et al.*, 1970), and the new-born baby is practically free of cadmium. The renal cortex concentration was estimated as being 0.25 μg/g at birth (from the literature it was estimated that the concentration in the renal cortex of a 30-year-old

individual is approximately 25 μg/g). The whole kidney concentration was calculated to be ~0.17 μg Cd/g, and using the mass of the kidneys at birth (23 g) from ICRP (1975) the body burden at birth was estimated as 12 μg.

The annual exposure to the kidney is used to calculate the changes in body burden and renal cortex concentrations with time. One third of the total body burden is estimated to reside in the kidney, and the renal cortex concentration is approximately 1.5 times that of the whole kidney (these values are widely used in the literature, and are considered as standard assumptions).

The estimated exposure to the kidney can be used to calculate the estimated kidney burden by using biological half-time values for cadmium in the kidney to account for estimated metabolic losses. Experimental and epidemiological evidence indicates strongly that the biological half-time in the whole body is extremely long (many years), and the average biological half-time in the kidneys is 10–40 years (Friberg *et al.*, 1974), 17 years (Tsuchiya *et al.*, 1976) with a best estimate of 30 years according to Elinder *et al.* (1974).

Example
Kidney burden at 7 years is 356 μg.
Estimated exposure to the kidney between 7 and 14 years is 870 μg.
The kidney burden at age 14, before accounting for metabolic losses, is
356 + 870 = 1,226 μg.

If the half-time is 30 years then the retention time in the kidney is 43.3 years (from the relationship: half-life = retention time x ln (2)).

It is assumed that metabolic losses are a linear function of time, the proportion of the original body burden lost during the 7-year period is $7 \div 43.3 = 0.16$.
The amount remaining of the original body burden is $0.84 \times 356 = 299$ μg.

The proportion of the intake lost during the 7-year period is assumed to be $3.5 \div 43.3 = 0.08$ (i.e., the intake amount is subject to metabolic losses for the mean period of time, 3.5 years).

The amount of the intake remaining after the 7-year period is therefore $0.92 \times 870 = 800$ μg.

The estimated kidney burden at age 14 years, when metabolic losses are accounted for is
800 + 299 = 1,099 μg.

(If it was assumed that the intake is not subject to any metabolic losses during the 6-year period the body burden calculated would be an overestimate; conversely if it was assumed that 100 per cent of the intake was subject to metabolic losses over the full 6-year period, the calculated body burden would be an underestimate).

The concentration in the kidney as a whole is calculated by dividing the kidney burden by the mass of the kidney at the given age (from ICRP,1975). This concentration is then multiplied by 1.5 to give the concentration in the renal cortex. The total body burden is calculated by multiplying the kidney burden by 3.

The concentrations and body burdens calculated are shown in Appendix 3.9.

An alternative method of calculating the body or tissue concentrations can be carried out using the equation from Bennett (1981), which relates the time-independent steady-state compartment concentration to intake, compartment size and retention time in that compartment. This involves the calculation of the concentration from the following equation:

$C = T/M \times F$ Where C = Concentration in compartment
T = Retention time
M = Mass of compartment
F = Flux or intake to the compartment

In the case of this calculation, the retention time is 43.3 years (as mentioned previously), and the mass refers to the average mass of the whole body in each age group (kg). The concentration is the level in the body (µg/kg) and the flux is the annual total intake of cadmium (µg/year). The figures used in the calculation are summarised below in Table 4, and the average kidney mass, the average body mass and annual intakes for each age group are the same as those used to calculate the renal cortex concentration using the half-life, as previously detailed.

Table 3.7 Data used for the calculation of renal cortex concentrations of cadmium at various ages

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Intake (µg/yr)	2.4	93	197	376	734	641
Body mass	5	12	24	43	70	60
Body concentration (µg/kg)	21	337	355	379	454	463
Body Burden (mg)	0.10	4.04	8.52	16.30	31.77	27.76
Kidney burden (µg)	35	1,347	2,841	5,433	10,590	9,252
Kidney mass (g)	26	50	105	170	290	258
Kidney concentration (µg/g)	2.0	40.4	40.7	48.0	54.8	53.9

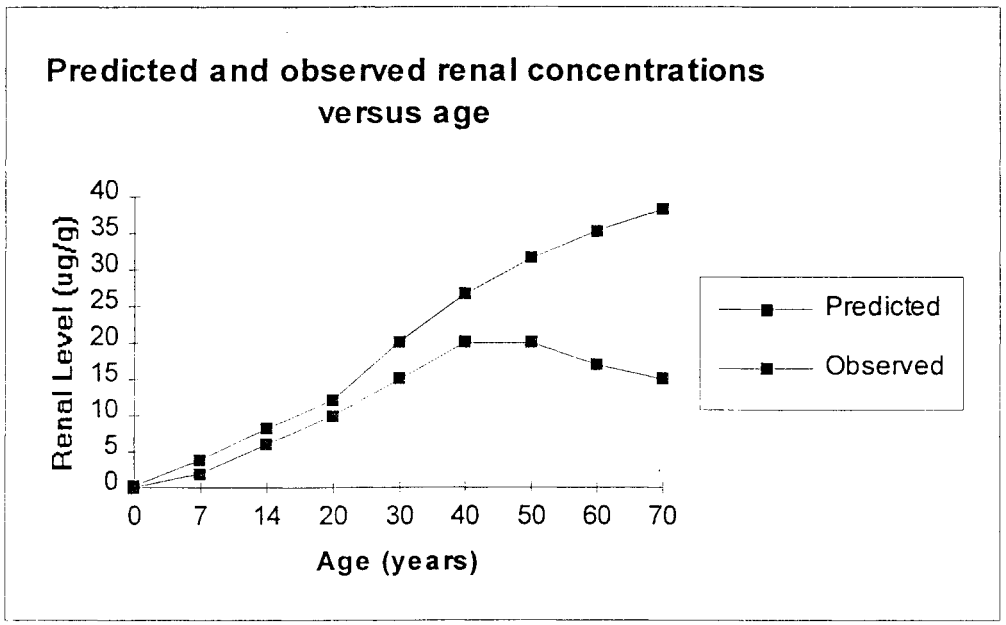
As can be seen from Table 3.7, this approach provides an estimate for the equilibrium concentration in the adult renal cortex that is significantly higher than the one estimated using the time-independent approach (54.8 µg/g compared with 38.2 µg/g using the method using half-life and accounting for metabolic losses). In addition this method overestimates the concentration in the renal cortex of infants by an order of magnitude, and the levels in the cortex of children by a significant factor (see Appendix 3.9). This demonstrates that the relationship is not particularly appropriate for a substance such as cadmium, which has a very long half life in the body. Therefore the time dependent calculation detailed earlier is more appropriate to substances with very long retention times.

Comparison of estimates of the renal cortex concentration and body burden with values from the literature
(See Appendix 3.9)

After birth the body burden gradually increases to the age of about 40–50 years, and after the age of about 50–60 years the concentration in the kidney cortex gradually decreases (WHO, 1992a). It is not clear whether the documented decrease in renal cortex levels is a result of physiological changes, or whether the individuals in the older age groups of the studies have been exposed to lower levels of cadmium during their lifetimes.

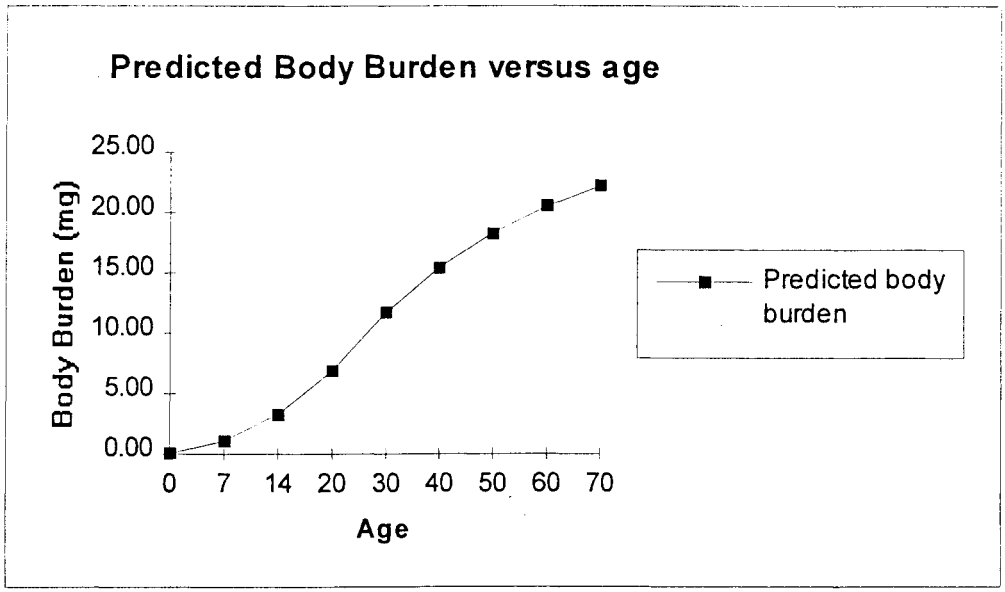
The predicted renal cortex concentration from this study for 40 to 59-year-old individuals is estimated as 26–35 μg/g, and it can clearly be seen that the predicted renal concentration increases with age (Figure 3.1). Measured renal cortex concentrations in individuals aged 40–59 years varies between 14–27 μg/g (Ante and Schneider, 1971), 23–29 μg/g (Piscator *et al.*, 1972) and in the general adult population from a number of studies between 11–46 μg/g (WHO, 1992a). However, the model does not predict the well documented decrease in cadmium levels in the renal cortex, which occurs after the age of about 50 years, therefore the critical age for the purpose of calculation of EALs was taken to be 50 years. The observed renal cortex concentrations shown in Figure 3.1, are taken from Kjellstrom (1979), and are values for non-smoking US males. Several studies have established a highly significant correlation between smoking and increased renal cortex concentrations (Vahter, 1982).

Figure 3.1 Predicted and observed levels of cadmium in the renal cortex (μg/g kidney, fresh weight) against age (years)



The average body burden in non-smoking adults not exposed to excessively high cadmium varies between 5–20 mg (WHO, 1992a). This range compares well with the estimated body burden at various ages from this study (shown graphically in Figure 3.2), and it is suggested that the estimates of intake, exposure and accumulation of cadmium presented in this study are reasonably representative of those of the general population in the UK.

Figure 3.2 Predicted Body Burden (mg) versus Age (years)



3.7 TOXICOLOGICAL ASSESSMENT

The renal cortex is the primary target organ for cadmium, and the seriousness of human exposure to cadmium is commonly assessed in terms of the extent to which levels are permitted to approach a critical value. In chronically exposed human populations this appears to lie between 180–220 μg/g. However, cadmium accumulated under these conditions is distributed between more than one compartment, a major fraction, for example, is bound to metallothionein and may be relatively inert (Foulkes, 1986). In addition, when a critical concentration in the cortex is reached, nephrotoxicity results in a considerable fall in kidney concentration due to excretion. Therefore, renal concentrations after nephrotoxicity has developed may not provide reliable data from which to judge body burden or critical concentration. These factors were not considered fully when the WHO Task Group assessed the probable renal cortical concentration at 200 μg/g, as the data considered included some derived from occupationally exposed persons with nephrotoxicity (MAFF, 1983). Therefore the actual critical concentration in the renal cortex which gives rise to kidney damage may be higher than the WHO critical concentration.

Whilst acknowledging that this value may be a conservative underestimate, the critical concentration in this study is taken to be 200 μg/g, and used to calculate EALs.

Alternatively, it may be possible to calculate the EALs from the current daily intake by comparison with the tolerable daily intake (TDI) as proposed by the FAO together with the WHO. The FAO/WHO PTWI for cadmium is 400–500 μg, and MAFF (1983) concluded that the margin of safety between this and actual intakes in the UK is not great. This PTWI is equivalent to a TDI of 0.82–1.02 μg Cd/kg bw/day for an average adult individual (mean body weight, 70kg). These data are also used to calculate EALs from the current estimates of cadmium intake.

3.8 DERIVATION OF ENVIRONMENTAL ASSESSMENT LEVELS FROM DATA ON THE ESTIMATED INTAKE AND RENAL CORTICAL CONCENTRATIONS

The model has been used to estimate the renal cortical concentration at various ages, associated with the current intake and background environmental levels. This concentration, at the critical age (i.e., the maximum concentration, in this instance in individuals of approximately 50 years of age), is compared with the critical concentration from the toxicological assessment. This critical concentration is equivalent to a maximum allowable lifetime exposure and is used to derive a maximum allowable intake for cadmium, which may then be equated to maximum allowable environmental levels.

Current Background Levels

	ASSUMED VALUE	RANGE
Air	0.5 ng Cd/m^3	0.12–0.83 ng Cd/m^3
Soil	0.3 mg Cd/kg	0.2–0.4 mg Cd/kg
Fresh water	**0.5 µg Cd/l**	**<1.0 µg Cd/l**
Sea water	0.1 µg Cd/l	0.04–0.3 µg Cd/l

Using the predicted levels in the renal cortex and the critical concentration
(See Appendix 3.9)

Critical concentration	200 µg Cd/g
Current levels at critical age	31.5 µg Cd/g

Therefore the maximum acceptable environmental levels (i.e., those associated with a renal cortex concentration of 200 µg Cd/g) are 6.35 times (200 ÷ 31.5) higher than current background levels. Using this calculation, the EALs would be:

	EAL RANGE
Air	**0.76–5.3 ng Cd/m^3**
Soil	1.3–2.5 mg Cd/kg
Fresh water	**<6.4 µg Cd/l (3.2 µg Cd/l)**
Sea water	0.3–1.9 µg Cd/l

As mentioned previously, the application of phosphate fertiliser to agricultural soil represents a substantial source of exposure to cadmium for the general population. Given that the contribution to lifetime exposure from breast-feeding is negligible, approximately 70 per cent of the total lifetime exposure comes from consumption of vegetables and cereals.

From the data on the relative inputs to agricultural soil from atmospheric deposition and fertiliser application, it is estimated that approximately 50 per cent of cadmium in these foodstuffs comes from phosphate fertilisers. Therefore, approximately 35 per cent of total lifetime exposure to cadmium comes not from background environmental sources, but from the direct input of phosphate fertilisers to soils and crops. For a renal cortex concentration of 31.5 µg Cd/g, it follows that 35 per cent (11.02 µg Cd/g) is due to fertiliser application, and the remaining 20.48 µg Cd/g from background environmental sources.

Assuming that the contribution from phosphate fertilisers will remain constant, it can be calculated that for a critical concentration of 200 μg Cd/g, up to 189 μg Cd/g may be of environmental origin.

Given a current exposure (renal cortex concentration) of 20.48 μg Cd/g, and applying the same assumptions as previously, the maximum acceptable environmental levels are 9.2 times (189 ÷ 20.48) higher than current background levels. Using this calculation, the EALs would be:

	EAL RANGE
Air	**1.1–7.6 ng Cd/m^3**
Soil	1.8–3.7 mg Cd/kg
Fresh water	**<9.2 ng Cd/ (4.6 ng Cd/l)**
Sea water	0.4–2.8 μg Cd/l

Using current intake estimates and the TDI

Alternatively EALs can be calculated using the TDI of 1.02 μg/kg bw/day, and the current estimate of the daily intake of cadmium over the lifetime. The current estimate of lifetime intake is 0.44 μg/kg bw/day, and the intakes during various stages of life are summarised in Table 3.3.

These data would mean that the TDI is approximately 2.3 times higher than the current intake, and therefore the following EALs have been calculated from the background environmental levels in the UK.

	EAL RANGE
Air	0.3–1.9 ng Cd/m^3
Soil	0.5–0.9 mg Cd/kg
Fresh water	<2.3 ng Cd/l (1.1 ng Cd/l)
Sea water	0.1–0.7 μg Cd/l

As with the calculation from the critical concentration in the renal cortex, it may be possible to calculate the EALs taking into account the contribution from fertiliser application. The current contribution to lifetime exposure from fertiliser application is estimated to be approximately 35 per cent, therefore given a current intake of 0.44 μg/kg bw/day, 0.15 μg/kg bw/day would be from fertiliser application. Given a TDI of 1.02 μg/kg bw/day, this would mean that up to 0.87 μg/kg bw/day could come from background environmental input. Given that the current intake from this source is approximately 0.29 μg/kg bw/day, this would result in a factor of 3 between current intake levels and maximum tolerable intake levels, **assuming that exposure as a result of fertiliser application remains unchanged**. The resulting EALs calculated from this figure are shown below.

	EAL RANGE	MID RANGE
Air	0.36 – 2.5 ng Cd/m^3	1.5 ng Cd/m^3
Soil	0.6 – 1.2 mg Cd/kg	0.9 mg Cd/kg
Fresh water	<3 μg Cd/l	1.5 μg Cd/l
Sea water	0.12 – 0.9 μg Cd/l	0.3 μg Cd/l

In summary, it is possible to take the upper and lower estimates of the EALs calculated to give a range which is largely governed by the conservatism of the estimates and the critical concentration and TDI. The range and mid-range values are given below:

	EAL RANGE	MID RANGE
Air	0.3 - 7.6 ng Cd/m^3	4.0 ng Cd/m^3
Soil	0.5 - 3.7 mg Cd/kg	2.1 mg Cd/kg
Fresh.water	<2.3 - <9.2 µg Cd/l	<5.8 µg Cd/l
Sea water	0.1–2.8 µg Cd/l	1.5 µg Cd/l

3.9 ENVIRONMENTAL STANDARDS FOR CADMIUM

The current EALs and EQSs for cadmium are:

Air	5 ng/m^3
Fresh water	5 µg/l (soluble and insoluble)
Coastal water	2.5 µg/l (dissolved)

The existing standards for cadmium are very similar to the mid-range values c lated in this assessment, and are well below the upper limit of the EALs proposed using ex re commitment. Therefore it appears that the current standards are set at values t ffectively limit human exposure to levels that are compatible with the long-term protect f human health.

3.10 SUMMARY

1. The data on background levels of cadmium in soil and air are consiste n a number of studies, and the ranges given can be considered to be representativ on ambient levels of cadmium in the aquatic environment, particularly in fresh w e poor and whilst a tentative value has been used it should be recognised that th re not sufficient to set an EAL with confidence.

2. There have been many studies investigating cadmium in food, and are reasonably consistent. The estimates of intake, exposure and body should be representative of the general UK population. The long half-life and e data on the behaviour of cadmium in the body mean the estimates of body bur l tissue concentration can be considered to be representative of the general ation.

3. The main sources of lifetime exposure to cadmium are vegetables uit (53 per cent), cereals (18 per cent) and seafood (8 per cent). Drink ater (6 per cent) and air (4 per cent) are relatively minor contributions.

4. Despite the fact that direct soil ingestion constitutes only a small rtion of lifetime exposure, the contribution of the terrestrial food pathway and th y of some edible plants to accumulate high levels of cadmium, means that soil ca l levels are very important. The use of phosphate fertilisers makes a major contri l to total human exposure.

5. The total daily intake of 0.44 μg Cd/kg bw/day appears to be representative for the lifetime of an average UK individual.

6. The most widely-accepted measures of the maximum allowable exposure are the critical concentration in the renal cortex of 200 μg Cd/g (fresh weight), and the PTWI of 500 μg (for an adult).

7. Using the critical concentration the maximum allowable environmental levels compatible with human health when consideration is given to lifetime exposure to cadmium are estimated to be 6.35 times greater than current background environmental levels. Environmental Assessment Levels have been calculated on this basis. A second calculation has been made to take into account the input of phosphate fertilisers, and used to estimate EALs for air and soil.

8. The margin is smaller when the PTWI is used as the measure of acceptable exposure, tolerable intake being a factor of only 2.3 times higher than current intake.

9. The current long-term EAL for cadmium in air, and the EQS values for cadmium in fresh water and coastal water, appear to be compatible with those calculated using exposure commitment, and therefore may be considered as effective with respect to the long-term protection of human health.

3.11 REFERENCES

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4. EXPOSURE COMMITMENT ASSESSMENT FOR NICKEL

4.1 INTRODUCTION

Detailed estimates of intake and exposure to both total and inorganic nickel have been made using UK data (where these have not been available, data from other industrialised countries have been used) and internationally accepted reference values for the human body. Estimates of daily intake compare well with previous studies. There is no tolerable daily intake (TDI) or critical concentration in a target organ which can be used as acceptable levels of exposure. A surrogate TDI of 800 μg is used for the calculation of Environmental Assessment Levels (EALs), **this value being the highest daily intake which did not appear to result in adverse health effects being recorded in a human population.**

Nickel is a ubiquitous element that has been detected in different media in all parts of the biosphere. It is introduced into the environment from both natural and man-made sources and is circulated throughout all environmental compartments by means of chemical and physical processes, as well as through the biological transport mechanisms of living organisms. A major source of nickel in the environment is the combustion of fossil fuels, particularly coal. Atmospheric nickel is considered to exist mainly in the form of particulate aerosols.

Nickel from soil and water is absorbed and metabolised by plants and micro-organisms and these small quantities of nickel are widely present in all foods and water. Nickel may be accumulated by some plants, and growth retardation has been reported in some species at high nickel concentrations. Some foods contain relatively high concentrations of nickel, but these quantities have not been correlated with adverse health effects. The chemical and physical forms of nickel and its salts strongly influence bioavailability and toxicity.

Inhalation is an important route of exposure to nickel and its salts with respect to health risks, compared with the exposure from the gastrointestinal route which, despite being considerably larger in terms of quantity of intake, is of lesser importance. There is evidence of a carcinogenic risk through the inhalation of nickel metal dusts and some nickel compounds. Very high tissue concentrations of nickel are required to produce teratogenic or genotoxic effects. The consequences of the effects of nickel on the immune system are not clear. There is no evidence that nickel may undergo biotransformation, though it does undergo complexation. Nickel has been shown to be essential for the nutrition of many micro-organisms, a variety of plants, and for some vertebrates.

4.2 TRANSPORT AND BEHAVIOUR IN THE ENVIRONMENT AND MAN

Atmospheric nickel is considered to exist mainly in the form of aerosols; the different nickel concentrations in particles depend on the type and source of nickel. A major source of nickel in the environment is from the combustion of fossil fuels.

A comprehensive overview of nickel in the terrestrial Canadian environment (McIlveen and Negusanti, 1994) revealed that the available information dealing with nickel is substantially less than for the other metals such as copper, lead, zinc and cadmium, but that a reasonable body of knowledge has been accumulated both in Canada and other developed nations.

Human activities that introduce nickel into the natural environment also frequently cause other contaminants to be co-deposited, and it is a rare situation when only one material of concern is emitted to the environment at a time, causing considerable difficulty in assessing the impact of metals such as nickel.

Nickel in the soil occurs in a large number of mineral forms and chemical compounds, and it is the availability of these forms to plant roots and other organisms, and the subsequent accumulation of the forms which is important in terms of human exposure. Nickel can exist as inorganic crystalline minerals, or complexed with organic matter or clay particles (Hutchinson *et al.,* 1981). Uptake of nickel from soil to plants will depend not only on the total soil concentration, but also on the proportions of the various forms present, and their bioavailability.

The major source of nickel accumulation in terrestrial plants is the increased occurrence of nickel in soils. The concentration of nickel in plants is in the range of 5 to 500 μg/kg fresh weight assuming that plants are 10 per cent dry matter (NAS, 1977). Nickel is relatively more toxic to plants than most other heavy metals, and levels in excess of 5,000 μg/kg are toxic to most plant species. Levels of nickel in various plant species grown in the same soils vary widely, as may the concentrations in various parts of a single plant and levels in seeds may be three times higher than in stems and leaves (NAS, 1977) and, for example, higher levels have been found in oat grains than in oat stems (Halstead *et al.,* 1969).

Contamination of food crops can occur through deposition of nickel from the atmosphere or through uptake of nickel from soils contaminated by nickel. Uptake of nickel from soils is important as it is phytotoxic, but in many plant species nickel is not taken up readily. However, some edible plants (lettuce and carrots) do take up significant amounts of nickel.

Nickel is introduced into the hydrosphere by atmospheric deposition, surface run-off, discharge of industrial and municipal wastes, and also following the natural erosion of soils and rocks. In rivers, nickel is mainly transported in the form of a precipitated coating on particles in association with organic matter, but in lakes it is transported in the ionic form, also in association with organic matter. There is no evidence of biomagnification of nickel in aquatic food chains (WHO, 1991).

Absorption is related to the solubility of the compound; nickel carbonyl, due to its lipophilicity, the most rapidly and completely absorbed of the nickel compounds in the lungs. It has been shown that forms which are removed only slowly from the lungs may accumulate in this tissue following chronic exposure to low levels of atmospheric nickel (Williams *et al.,* 1980). Ingestion is the major form of intake in humans, but much is eliminated unabsorbed, mainly in the faeces. It is known that nickel crosses the human placenta; and it has been found in both the foetal tissue and the umbilical cord. Appreciable amounts of nickel have also been found in breast milk.

Gastrointestinal absorption of nickel is variable and depends on the composition of the diet. In a recent study on human volunteers, absorption of nickel from water was 27 per cent compared with less than 1 per cent from food (WHO, 1991). A number of food components are known to affect nickel absorption in the gastro-intestinal tract (GI tract) (Solomons *et al.,* 1982), but generally absorption of ingested nickel is assumed to be less than 10 per cent.

Nickel is rapidly cleared from blood plasma and excreted predominantly in urine. Animal studies indicate that 80 to 90 per cent of the injected amounts of nickel are excreted within the first three days following exposure (Bennett, 1982). The International Commission on Radiological Protection (ICRP) has recommended for assessment that 70 per cent of the amount absorbed into blood is rapidly excreted through the kidneys. The remainder is assumed to be uniformly distributed throughout all organs and tissues of the body and retained with a biological half-time of 1,200 days (ICRP, 1981). Transplacental transfer has been demonstrated in rodents.

The absorption factors for nickel across the GI tract used were 0.05 (Owen, 1990) and for absorption across the lungs the factor used was 0.06. The absorption from water is far more efficient than from food, and the factor used was 0.27 (WHO, 1991).

4.3 ENVIRONMENTAL LEVELS

Air

Owing to the large number of sources releasing nickel into the atmosphere and their uneven distribution over the globe, ambient nickel concentrations may vary over several orders of magnitude. Atmospheric nickel concentrations in remote areas range from <0.1 to 3 ng/m^3, urban and rural areas usually exhibit air nickel levels ranging from 5 to 35 ng/m^3 (Bennett, 1984). Ambient levels of nickel in air have been reported to be 6 ng/m^3 in non-urban areas of the USA, and 17 and 25 ng/m^3 in urban areas in summer and winter respectively (NAS, 1975). At a semi-rural site in England, the average concentration during 1957–74 was 19 ng/m^3 with a standard deviation of ±50 per cent (Salmon *et al.*, 1978). There were wide variations over the period (<10–50 ng/m^3), but no overall trend, and little difference between average levels in summer and winter. The annual mean concentrations in air from three Belgian cities between 1972–1977 ranged from 9 to 34 ng/m^3 (Kretzschmar, 1980), and the relatively small variation in these levels in urban areas appears to indicate that diffuse sources, such as traffic fumes, predominate.

Rural air

Good UK data exist for ambient atmospheric nickel concentrations. The mean concentration for three rural sites (Oxfordshire, Nottinghamshire and Cumbria) between 1972–1981 was 6.9 ng/m^3, with a range of means of 4.6–9.4 ng/m^3 for the three sites. A trend of decreasing nickel levels was detected at each of the sites, and the mean level between 1982–1991 was 3.8 ng/m^3, with a range of 2.6–5.6 ng/m^3 (Bertorelli, 1994). For the purposes of the calculation of the EALs the most recent UK data from Bertorelli (1994) was used, and EALs were derived from background levels of 2.6–5.6 ng/m^3.

Urban air

Nickel has also been monitored in UK urban air at five sites (Bertorelli, 1994), and the mean concentrations for the period 1985–1990 were 8.7 ng/m^3 (central London), 10.7 ng/m^3 (outer London), 19.4 ng/m^3 (Motherwell), 8.3 ng/m^3 (Glasgow) and 11.7 ng/m^3 (Leeds). The range of annual means for all five sites was 4.1–23.0 ng/m^3, with an overall mean of 11.8 ng/m^3. For the purpose of estimating the exposure to nickel via inhalation, the representative value used was 12 ng/m^3.

Water

Levels in natural waters have been found to range from 2–10 µg/l in fresh water (WHO, 1991), and a similar range of 2–13 µg/l with a mean of 6 µg/l is reported for fresh waters by Amavis *et al.* (1975). Levels in sea water are lower, with a range of 0.2 to 0.7 µg/l (WHO, 1991). The background levels used for the calculation of EALs were a range of 2–10 µg/l for fresh waters, and 0.2–0.7 µg/l for sea water.

Soil

Farm soils contain between 3 and 1,000 mg Ni/kg **(Bennett, 1982).** Berrow and Burridge (1980) suggest a normal range of nickel in cultivated soils of 5–500 mg/kg, with a typical value of 50 mg/kg. The distribution of values for 750 farm soils sampled in the UK showed that most values are in the range 4–80 mg/kg, with a median value of 26 mg/kg. There are no data for levels of nickel in urban soils, but it is assumed that they are generally higher than those in rural areas. The EALs have been derived based on a background range of 4–80 mg/kg. **The level in urban soil was assumed to be 1,000 mg/kg, the highest recorded data for agricultural soils (no data on urban UK soils were available). This may be unreasonably conservative, resulting in an overestimate of the contribution from this pathway but, in the absence of reliable data on urban levels, it represents the reasonable worst-case scenario.**

4.4 NICKEL IN FOOD

(See Appendix 4.2)

Data on nickel in foodstuffs are relatively scarce in comparison to some of the other heavy metals. It is fairly evenly distributed in the diet, with the highest concentrations occurring in food groups which contain processed foods, e.g., canned vegetables, sugars and preserves, bread and cereal food groups, suggesting a contribution from food processing equipment and possibly from food cans (Smart and Sherlock, 1987).

Certain food items (meat, some fish, particularly marine fish, dairy products and some fruits) are unlikely to have a high nickel content, and generally contain less than 100 µg/kg. Nickel concentrations in European and USA foods are usually below 500 µg/kg fresh weight (Ellen *et al.,* 1978) although certain vegetables (peas, beans, cabbage, lettuce), nuts and oatmeal have been found to contain higher levels of nickel in the range 1,000–3,000 µg/kg.

Meat

Generally meat and meat products contain less than 100 µg/kg, but higher concentrations have been measured by US surveys, with a range of 60–400 µg/kg (Stoeppler, 1980; NAS, 1975). Other studies have found lower levels in meat, between 2–20 µg/kg (Boudene, 1979). The mean value for all meat products from the Ministry of Agriculture, Fisheries and Food (MAFF) survey was <280 µg/kg. More recent UK data for beef (<50 µg/kg), lamb (<90 µg/kg) and pork (<60 µg/kg) are generally lower than those measured during the MAFF survey, although this may be a result of more sensitive analytical methods (Smart and Sherlock, 1987). **The representative values used in this assessment are the limits of detection for the most recent UK data, i.e., beef – 50 µg/kg, lamb – 90 µg/kg and pork – 60 µg/kg. Similarly the only value for poultry is from the same study – <70 µg/kg, and 70 µg/kg is the representative value used in this assessment.**

Offal
The only data available for liver are those from Smart and Sherlock (1987), which found a level of <100 μg/kg, and from Evans et al (1978) which found 50 μg/kg. The representative value used in this assessment is 50 μg/kg. There are even fewer data available for the levels of nickel in kidney, the only available results quote a concentration of <250 μg/kg (Smart and Sherlock, 1987).

Meat products
There are no data for meat products, although it has been suggested that levels in processed meats may be higher than in fresh meat. The representative value used is the average for the meat groups beef, pork and lamb, **i.e., <70 μg/kg, and the representative value used is 70 μg/kg (assuming that meat products are comprised of equal amounts of the three fresh meats).**

Whole milk, semi-skimmed milk and milk products
For foods such as milk the levels are usually around 20 μg/kg (Veien and Andersen, 1986). and 4–25 μg/l (Boudene, 1979). Other studies that have measured levels in cows' milk have found the usual range of concentrations to be between 10–30 μg/l, with the most comprehensive investigation finding a range of 28–38 μg/l, with a mean concentration of 32 μg/l (Lavi and Alfassi, 1990). The results from the MAFF survey between 1976 and 1981 found levels in milk to be <20 μg/l on average. For milk and all dairy products, as there are no data on levels in specific products other than whole milk, the representative value selected was 20 μg/l.

Eggs
There are no data available for eggs and so the representative value was assumed to be approximately the same as for the dairy product group (20 μg/l).

Oils
The only data available for the fats/oils group are from MAFF, and indicate a mean level of <90 μg/kg.

Vegetables
Levels in potatoes are usually between 100 and 500 μg/kg (Ellen *et al.*, 1978) and in vegetables in general the range suggested is 20 to 2,700 μg/kg (Stoeppler, 1980; NAS, 1975). Levels of 110 μg/kg have been measured in spinach (Evans *et al.*, 1978), but measured levels in lettuce in Denmark are significantly higher, with a mean value of 1,000 μg/kg (Veien and Andersen, 1986). A UK study found that concentrations in canned vegetables were higher than those in frozen vegetables (Smart and Sherlock, 1987); the highest level recorded in canned vegetables was 400 μg/kg, compared with 100 μg/kg in frozen carrots and spinach. The levels of nickel in various vegetable crops are given in Appendix 4.1 and the representative values summarised in Table 4.1.

Table 4.1 Representative nickel concentrations in selected vegetables

VEGETABLE	CONCENTRATION (µg/kg)	SOURCE
Cabbage	270	Smart and Sherlock, 1987
Lettuce	1,400	McIlveen and Negusanti, 1994
Peas	300	Veien and Andersen, 1986
Potato	170	McIlveen and Negusanti, 1994
Onion	40	MAFF, 1985
Carrots	10	MAFF, 1985
Tomato	110	Wolnik *et al.*, 1985

Fruit

Levels in fruits are usually in the range 100–500 µg/kg (Ellen *et al.*, 1978) whilst other reports suggest a range of 20–2,700 µg/kg (Stoeppler, 1980; NAS, 1975). The levels in fresh fruit in the UK have been measured, with a mean value of 70 µg/kg for apples, and 210 µg/kg for pears, and 170 µg/kg for soft fruits. Levels in fruit juice were <110 µg/kg (Smart and Sherlock, 1987); concentrations of 60 µg/kg and 120 µg/kg have been measured in apricots (Evans *et al.*, 1978; Flint and Packirisamy, 1995). Other results include values for apples (10 µg/kg), pears and apricots (100 µg/kg) and raspberries (900 µg/kg). The representative value selected for this assessment is 200 µg/kg, as it is approximately the highest mean value measured in UK fruit.

Levels in fruit products, for example, fruit juices, are generally low with concentrations for apple and orange juices in the range of 20–30 µg/kg (Veien and Andersen, 1986), and the representative value selected for fruit products is 30 µg/kg.

Sugar

Levels in white sugar were <130 µg/kg (Smart and Sherlock, 1987) and nickel concentrations of 200 µg/kg have been measured in Danish honey (Veien and Andersen, 1986). The representative level used was 130 µg/kg.

Cereals

Ellen *et al.* (1978) report a range of 100–500 µg/kg, whereas Stoeppler (1980) and NAS (1975) cite a range of 20–2,700 µg/kg in cereals. Wholemeal products can have significantly higher concentrations than more refined products, due to the high nickel content of wheatgerm, with UK data recording levels of <60 µg/kg in white bread, and 190 µg/kg in wholemeal bread (Smart and Sherlock, 1987). A Danish survey found mean concentrations in white bread of around 200 µg/kg, and levels in wholegrain bread of 80 µg/kg (Veien and Andersen, 1986), which appear to contradict the evidence from the UK studies. The selected representative value for bread is the mean of the two UK results, 125 µg/kg.

Other cereal products, such as biscuits (150 µg/kg) and rice (420 µg/kg) also have relatively high levels of nickel. In various cereals analysed in a Danish survey the mean levels ranged from 100 to 400 µg/kg (Veien and Andersen, 1986). The selected representative mean for other cereal products was 300 µg/kg.

Beverages
The nickel content of coffee when made up is around 8 μg/kg (Veien and Andersen, 1986) and this is the only datum available.

Fish
Levels in seafood vary considerably, with a wide range of 20 to 20,000 μg/kg (fresh weight) reported. For example, data found for various fish and shellfish species include 400–600 μg/kg in mussels, 60–100 μg/kg in mackerel/herring and 30 μg/kg in prawns. Molluscs may accumulate high levels of nickel if there are high concentrations in the water (Friedricks and Filice, 1976), and some other aquatic organisms may contain relatively large amounts of nickel, e.g., oysters 1,500 μg/kg, salmon 1,700 μg/kg (Boudene, 1979).

In uncontaminated waters the range of concentrations reported are 20 to 2,000 μg Ni/kg (whole fish, wet weight) with a mean of 700 μg/kg in fish from the UK total diet survey (Smart and Sherlock, 1987). Levels in tuna have been measured as 40 μg/kg (Evans *et al.*, 1978). The representative values for the various fish types are 100 μg/kg (marine fish), 1,700 μg/kg (freshwater fish), 40 μg/kg (tinned fish) and 1,500 μg/kg (shellfish).

Drinking water
The average concentration in drinking water samples measured at the tap during a comprehensive US survey was 4.8 μg/l (NAS, 1975). In Italy a similar survey found nickel levels in drinking water were mostly below 10 μg/l (Clemente *et al.*, 1980) and in Germany household drinking water contains 6.8–10.9 μg/l (Schumann 1980). An important additional source of exposure may come from pipework containing nickel, as leaching of nickel from taps and fittings may introduce significant amounts of nickel to the drinking water supply, and raise levels in water, which has been allowed to stand in the pipework, to as high as 200 μg/l. However, the representative value used for the calculation of exposure was 10 μg/l.

The representative values for nickel in all selected food types and environmental media are summarised in Appendix 4.2.

4.5 INTAKE AND EXPOSURE – ESTIMATES FOR AVERAGE INDIVIDUALS IN DIFFERENT AGE GROUPS
(See Appendices 4.3 to 4.6)

The data on nickel levels in selected representative food types, drinking water, urban soil and urban air have been used together with information on the average consumption rates of different foods in the UK, the consumption rate for drinking water and soil, and the representative breathing rate to calculate the annual intake of cadmium for the average individual in the general population in five age groups. The annual intake is obtained by multiplying the amount consumed, or breathed, by the average nickel concentration in the media.

Intake from breast-feeding
(See Appendix 4.7)

The level of zinc in human milk has been measured by a number of workers, but there are fewer available data on nickel levels. The available data are reviewed by Feeley *et al.* (1983)

who found that levels of nickel in human milk did not change during lactation, and that the concentration is usually between 14 and 17 ng/l. The main intake for infants is from breast-feeding, with a negligible intake from inhalation.

Table 4.2 Total intake of nickel at various ages and over the lifetime (μg/kg bw/day)

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Intake	2.6	1.9	6.1	3.2	2.9	3.1

Table 4.3 Percentage contribution of various sources to nickel intake at various ages

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Diet	99.9	89	27	57	78	73
Soil		0	69	36	12	18
Water		11	3	7	10	9
Air	0.1	0	0	0	0	0.1

As can be seen from Table 4.2 the highest intake (relative to body size) occurs in the 2 to 7-year-old age group, and this is a result of the high contribution of soil ingestion (possibly an overestimate due to the extremely conservative value used for urban soil) relative to the other routes for other age groups (Table 4.3). It can also be seen that in terms of lifetime intake of nickel that the diet is the main contributor, although significant contributions also come from the ingestion of soil and drinking water. The intake of nickel from the breathing of air is negligible.

Food has been found to be the main source of nickel intake by man by a number of previous studies, and nickel intake via the GI tract can be high compared with that of other trace elements. The intake from air and water is lower than from food, but nickel in these media may be more biologically available than nickel in food. The amount of nickel entering the human respiratory tract is in the range of 0.1–0.7 μg/day (WHO, 1991), and from this assessment it is estimated that the average UK individual living in an urban area inhales 0.26 μg/day. This is low in comparison with the estimated daily intake from food which is approximately 160 μg/day. The mean daily intake from water for adults is estimated to be between 7.5 and 15 μg/day (WHO, 1991), which is comparable with the intake estimated for adults in this study of 20 μg/day. In one study on infant diets, where estimates were made from nickel concentrations in milk and infant foods, the daily intake was estimated to be 30–300 μg/day (Clemente *et al.*, 1980), but the estimate from this assessment is lower, being approximately 13 μg/day.

Estimates of the daily intake of nickel in the diet compiled by other studies from a number of developed countries are summarised in Table 4.4. Daily intake from food varies from 100–800 μg/day, but the mean dietary intake in most countries is usually 100–300 μg/day (WHO, 1991). Mean dietary nickel intakes in the UK (1981–1984) were between 140 and 150 μg/day (Smart and Sherlock, 1987). This assessment calculated an average intake of approximately 160 μg/day from the diet, which compares well with the previous assessments for the UK and other countries. The essentiality of nickel to humans is still unclear, but estimates of the daily nickel requirements vary between 35 and 50 μg.

Table 4.4 Reported daily intakes from diet (µg/day)

DAILY INTAKE	COUNTRY	SOURCE
140–150	UK	Smart and Sherlock, 1987
300–500		Schroeder *et al.*, 1962
290		Nodiya, 1972
260		Horak and Sunderman, 1973
165		Myron *et al.*, 1978
100–300		Clemente *et al.*, 1980
200		Flent and Packirisamy, 1995
<300		Hamilton and Minski, 1973
111–256	Germany	Anke *et al.*, 1991
63–300	Worldwide	Anke *et al.*, 1991
360	New Zealand	Dick *et al.*, 1978
460	Canada	Kirkpatrick and Coffin, 1974
170	USA	Myron *et al.*, 1978

The exposure to the body (the blood) is calculated by multiplication of the intakes by the absorption factors for the GI tract (food, soil, water) and the lung (air). As mentioned previously, these transfer factors are 0.05 for food (GI tract), 0.27 for water (GI tract) and 0.06 (lung). The exposure to nickel (µg/kg bw/day) and the relative contributions of different media are summarised in Tables 4.5 and 4.6.

Table 4.5 Daily exposure to nickel at various ages (µg/kg bw day)

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Exposure	0.13	0.14	0.35	0.21	0.21	0.22

Table 4.6 Percentage contribution from various sources to nickel exposure at various ages

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Diet	99.9	59	24	43	54	51
Soil		0	60	27	8	14
Water		40	16	30	37	35
Air	0.1	0	0	0	0	0.1

It can be seen that although food contributes 73 per cent of the total intake (Table 4.3), the lower availability of nickel in food relative to some other media means that over a lifetime it contributes only about one half of the total exposure. Soil and water also make substantial contributions, with drinking water being over one third of the total lifetime exposure, mainly as a result of the higher bioavailability of nickel in water compared with other media. The contribution to lifetime exposure from breathing air is negligible.

Food processing and cooking utensils

It is primarily the use of nickel in utensils, food processing equipment, from the grinding of flour and in catalysts which can lead to the contamination of food by nickel (Smart and Sherlock, 1987). Nickel based catalysts are employed in the hydrogenation of edible oils and fats (Berman, 1980), and may lead to some contamination of products, such as margarine.

Some studies that have investigated the contribution made by cooking utensils have concluded that the contribution to total exposure from this source is negligible, being in the region of 0–8 μg/person/day (Flint and Packirisamy, 1995). Others have concluded that cooking utensils can make a significant contribution to total intake, in the region of 100 μg/person/day (Smart and Sherlock, 1987). It is still unclear whether the release of nickel from kitchen utensils contributes significantly to oral intake.

Smoking

A potentially important route of nickel exposure is tobacco smoking. Smoking 20 cigarettes a day may result in the inhalation of 1–12 μg Ni/day, and importantly it is believed that nickel carbonyl is formed in the mainstream smoke. Nickel carbonyl is one of the most toxic forms of nickel. Nickel intake from all sources is approximately 200 μg/day for an adult, but as nickel carbonyl in air is more bioavailable than other forms, smoking may make a significant contribution to nickel exposure.

4.6 BODY BURDEN AND WHOLE BODY CONCENTRATIONS – ESTIMATES FOR INDIVIDUALS AT VARIOUS AGES

The body burden has been estimated using the relationship between retention time and intake, represented by the following equation:

$C = T/M \times F$ Where C = Body concentration (μg/kg)
T = Retention time (days)
M = Body weight (kg)
F = Exposure to body tissues

A number of assumptions are made in order to estimate body burden from our estimates of intake. As 70 per cent of the nickel absorbed to the blood is excreted almost immediately via the kidneys (ICRP, 1975), the actual exposure to the body tissues is considered to be the daily exposure to the blood (μg/day) multiplied by a factor of 0.3. The half-life of the remaining nickel in the tissues is estimated to be 1,200 days, so the retention time is approximately 1,730 days (from the relationship; retention time = half-life ÷ ln (2)). The data used in the calculations are summarised in Table 4.7 and, as can be seen, the body burden of the average adult is estimated to be 7.6 mg.

Table 4.7 Data used for the calculation of the body burden at various ages

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT
Blood exposure (μg/day)	0.64	1.67	8.41	9.14	14.58
Tissue exposure to body	0.19	0.50	2.52	2.74	4.37
Body mass (kg)	5	12	24	43	70
Body concentration (μg/kg)	66	72	182	110	108
Body burden (mg)	0.3	0.9	4.4	4.7	7.6

Comparison of estimates of the body burden with values from the literature

There are no available data on the body burden or levels in the new-born infant, and no data on human tissue concentrations of nickel in adults. Inconsistencies in the correlation between

exposure levels and biological measurements of nickel are mainly due to the fact that exposure is not to a single chemical species, but to a variety of nickel compounds of very different solubility, absorption, transportation and elimination rates.

In humans, wide variations have been reported in body levels. This makes it difficult to compare the results obtained by other investigations with those from this assessment. In addition to variations in the geographical origin of data and individual dietary and smoking habits, major differences can be attributed to the analytical methods employed. However, the normal ranges of nickel concentrations in tissues are not significantly influenced by age, sex or pregnancy (WHO, 1991), and as can be seen from Table 4.7 there does not appear to be a relationship between body concentrations of nickel and age of individuals.

A previous assessment of nickel metabolism in human beings indicated that the body burden of nickel in normal adults averages 0.5 mg per person, approximately 7 μg/kg for a 70 kg adult (Bennett, 1984). It was concluded that the oral intake of nickel averaged 170 μg/day, of which about 5 per cent would be absorbed. Inhalation of nickel averaged 0.4 μg/day for urban dwellers, of which 35 per cent was retained (0.07–0.14 μg/day). This involved the assumption that 70 per cent of the nickel absorbed into the blood is promptly excreted by the kidneys and that the remaining 30 per cent is deposited in the tissues, with a mean retention time of 200 days. This retention time appears to be an underestimate as the ICRP (1975) figure is a half-life of 1,200 days, equivalent to a retention time of 1,730 days.

As mentioned previously, this assessment has estimated the average body burden in UK adults to be 7.6 mg. This figure is comparable with that quoted in a Japanese study of about 5.7 mg for a body weight of 55 kg (Sumino *et al.*, 1975), equivalent to 7.3 mg for a 70 kg adult. Other studies have indicated a body burden of about 10 mg for an adult person (Schroeder *et al.*, 1962).

Measured levels of nickel in the blood vary greatly (Lavi and Alfassi, 1990). One recent study has found the mean level to be approximately 37 ng/ml (range 15–38 ng/ml), but other studies have measured ranges of 1–580 ng/ml.

4.7 TOXICOLOGICAL ASSESSMENT

There is no available TDI in the literature, although a number of authors have cited a recommended daily intake value of 50 μg/day. This value is therefore not a maximum permissible level of intake, but a guideline for the optimal intake level of nickel. No data are available on the reproductive and developmental effects of nickel in human beings.

In general, nickel is relatively non-toxic through the oral route due to limited intestinal absorption. However, a number of investigators have shown that ingested nickel can cause exacerbation of hand eczema in patients who are already sensitised to nickel (Flint and Packirisamy, 1995). Nickel metal and compounds can have strong sensitising effects on skin leading to dermatitis. This is particularly the case for direct contact from nickel-containing products, but ingested nickel can aggravate chronic dermatitis.

Excessive exposure to nickel compounds may cause a variety of local effects but only nickel carbonyl is associated with systemic effects in man. Exposure to nickel-containing mists and

dusts may cause asthma, pneumoconiosis and irritation of nasal membranes. Inhalation exposure of animals to nickel sulphide, nickel oxide and nickel carbonyl have been found to cause cancer in the lung. From increased incidences of cancer of the nasal cavity and the lungs in workers in nickel refineries it is concluded that it is likely that nickel in some forms is also carcinogenic to man.

In terms of human health, nickel carbonyl is the most acutely toxic nickel compound and although some, and perhaps all, forms of nickel may be carcinogenic, there is little or no detectable risk in most sectors of the nickel industry at current exposure levels. This includes some processes which in the past were associated with very high lung and nasal cancer risks. It seems unlikely therefore from the evidence presented in the literature that long-term ingestion of nickel poses a serious threat to human health.

Recently the WHO has proposed a tentative TDI of 5 µg/kg bw/day in their drinking water guidelines, equivalent to a daily intake of 300 µg/day over the lifetime of an average 60 kg individual. EALs have been calculated using this value in the absence of an established TDI for all sources, but should be considered as only provisional until a well-established TDI becomes available. This value appears conservative as there has been no established link between environmental exposure to the general population and human health effects, even at the highest recorded mean daily intakes (800 µg/day from the diet for an average adult).

4.8 DERIVATION OF ENVIRONMENTAL ASSESSMENT LEVELS FROM DATA ON THE ESTIMATED INTAKE

There are well established links between nickel exposure and certain skin disorders, but these are predominantly due to dermal contact with nickel-containing consumer products, such as jewellery, although ingested nickel can exacerbate this type of reaction. It is very difficult, therefore, to establish a dose-response relationship for nickel and its effects on human health. The lack of a well established TDI value means that the results should be treated with some caution.

The WHO TDI is equivalent to 4.29 µg/kg bw/day for a 70 kg adult. Given that the current UK lifetime intake of nickel can be estimated with some confidence to be approximately 3.1 µg/kg bw/day, EALs can be calculated on the basis that they are 1.38 times higher than current background environmental concentrations. Therefore these can be considered as provisional EALs, until a TDI is established.

Current Background Levels

	RANGE	MID-RANGE
Air	2.6–5.6 ng Ni/m^3	4.1
Soil	4–80 mg Ni/kg	42
Fresh water	2–10 µg Ni/l	6
Sea water	0.2–0.7 µg Ni/l	0.5

EALs

	EAL Range	Mid-range
Air	3.6–7.7 ng Ni/m^3	5.7
Soil	5.5–110 mg Ni/kg	58
Fresh water	2.8–13.8 µg Ni/l	8.3
Sea water	0.7–2.6 µg Ni/l	0.7

4.9 Environmental standards for nickel

The most recent EAL proposed for nickel and its compounds in air is 200 ng/m^3. The EQS for nickel in fresh water varies according to the hardness of the water between 50 and 200 µg/l, and for coastal and estuary waters is 30 µg/l.

In the guidelines for drinking water quality (WHO, 1984) no guideline value was set for nickel in drinking water, as the toxicological data available at the time indicated that a guideline value was not necessary. In the air quality guidelines for Europe (WHO, 1988) no safe level was recommended for nickel because of its potential carcinogenic properties.

At the present time there are no established standards for nickel in any terrestrial medium in Canada. However, a number of guidelines are in use, including those for surface soil, foliage in general and forage. The most recent standards are for soil clean-up guidelines, which are 200 mg/kg (McIlveen and Negusanti, 1994).

The freshwater standard depends on the hardness of the water, with harder waters (i.e., those with a higher calcium carbonate concentration) having the higher EQS as the toxicity of nickel to aquatic organisms is reduced by competitor ions. The EAL range proposed by this study is approximately 15 times lower than the current EQS, but it should be noted that it has been calculated using a tentative TDI for nickel, and the true health-related TDI may be higher than the value used. **Due to the difficulty in finding a reliable TDI of nickel, no meaningful conclusions can be attached to the difference between the EAL calculated in this study and current regulatory standards.**

4.10 Summary

1. Data on background concentrations of nickel in the environment are good for all media, and the results from a number of different studies are consistent. The representative values can be assumed to be close to actual mean background concentrations.
2. There are good and consistent data on nickel levels in foodstuffs and the estimates of intake, exposure and body burden appear consistent with previous studies.
3. The main sources of lifetime exposure to nickel are the diet (mainly from vegetables, fruit and cereals) and drinking water. Direct soil ingestion may also contribute to significant exposure in younger children, **although this may be as a result of the extremely conservative value used for urban soil in the absence of reliable data on UK urban soils.**

4. The total lifetime exposure to nickel of 3.1 µg Ni/kg bw/day appears to be representative of the average UK individual.

5. There is no TDI for nickel, as it is generally considered to be non-toxic through the oral route due to limited absorption. **A lifetime TDI of 300 µg for an average 60 kg individual has been used.** It is recognised that consequently the EALs produced may be overly conservative, and should only be considered as provisional suggestions until such time as a TDI, or another measure of acceptable exposure to nickel, is established.

6. The maximum allowable environmental levels compatible with human health when consideration is given to lifetime exposure to nickel are estimated to be approximately 1.38 times greater than current background environmental levels. Environmental Assessment Levels have been calculated on this basis.

7. The EALs proposed are considerably lower than the current standards in force. However, the current standards have been derived for different regulatory purposes and **it is probable that the use of the tentative TDI and in some cases extremely conservative environmental levels,** has resulted in EALs that are more stringent than limits necessary to protect human health.

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5. EXPOSURE COMMITMENT ASSESSMENT FOR ARSENIC

5.1 INTRODUCTION

Detailed estimates of intake and exposure to both total and inorganic Arsenic (As) have been made using UK data (where these have not been available, data from other industrialised nations have been used) and internationally accepted reference values for the human body. These estimates compare well with a number of exposure assessments carried out by other workers. The estimated body burden and tissue levels are in good agreement with measured levels reported in the literature. The review of toxicological information has identified the World Health Organization/Food and Agriculture Organization of the United Nations (WHO/FAO) Tolerable Daily Intake (TDI) of inorganic arsenic of 2 μg As/kg bw/day as the key measure of acceptable levels of exposure. The study estimates lifetime intake to be 0.45 μg As/kg bw/day for the average individual in the UK. Environmental Assessment Levels (EALs) for air, fresh water, soil and coastal waters have been calculated.

Arsenic is a naturally occurring element which may be present as inorganic or organic compounds in environmental media. The toxicity of arsenic depends not only on the concentration, but also on the chemical form of the element. Inorganic arsenical compounds, especially compounds, such as trivalent and pentavalent arsenic, are toxic; the trivalent form being more toxic than the pentavalent form. Organoarsenic compounds, like arsenobetaine which occurs in fishery products (Luten *et al.,* 1983) are generally less toxic.

Traditionally concentrations of arsenic compounds found in environmental samples have been reported as total arsenic following digestion. Limited data indicate that the proportion of inorganic arsenic in food ranges from 0 per cent in saltwater fish to 75 per cent in dairy and meat products. Arsenic is toxic to plants, and it has been suggested that this toxicity to plants effectively protects humans to harmful levels of exposure from that route.

Arsenic is methylated in the body, and this may act as a detoxifying mechanism but even at low exposures this process is not complete, and at higher exposures it can become saturated leading to a sudden increase in body levels of inorganic arsenic. In humans with relatively low exposures, the highest arsenic concentrations are found in the skin, hair and nails.

From the results of epidemiological studies there is good evidence that inorganic arsenic is a cause of human cancer at several sites. Arsenic is unusual as there is no good animal model for arsenic as a carcinogen, but in humans it is generally accepted that arsenic exposure through inhalation can cause lung cancer, and ingested arsenic may cause skin cancer. The risks associated with chronic exposure to low doses of arsenic is a problem that has been recognised with respect to specific instances, e.g., mining communities, where human activities have given rise to elevated soil arsenic concentrations. Calculation of lifetime risk of lung cancer from inhaled arsenic, and skin cancer from ingested arsenic, has been carried out for a number of specific instances. A recent assessment of cancer risk from arsenic in drinking water concluded that measures to reduce arsenic levels in water supplies should be considered (Smith *et al.,* 1992).

5.2 ENVIRONMENTAL TRANSPORT AND BEHAVIOUR

The link between anthropogenic releases of arsenic to the atmosphere and elevated environmental levels is well established. Increasing levels of arsenic in surface waters in the US between 1974–1981 have been attributed to increasing coal combustion (Smith *et al.*, 1987); vegetables grown near to industrial emissions have been found to contain elevated levels of arsenic (MAFF, 1982). The dominating pathway of arsenic from a factory to leafy vegetables grown nearby is direct atmospheric deposition, and the arsenic found on leafy crops is predominantly inorganic (Larsen *et al.*, 1992). Therefore the atmosphere is an important means of transport of arsenic in the environment.

Atmospheric deposition of arsenic fluctuates between years due to variations in emissions or meteorological conditions, but the uptake of arsenic in the vegetables from the soil varies less. Therefore a reduced emission of arsenic from a point source will cause the contamination of the aerial portions of plants by arsenic to drop (however, uptake from soil to root crops, e.g., carrots and potatoes, will persist).

Arsenic has low to moderate mobility in the soil (Sheppard, 1992) and is rarely found as the free element in soil but is present as a component of sulphidic minerals (Pyles and Woolson, 1982). Arsenic uptake by plants is greater (and its toxicity more severe) in light-textured soils than heavy-textured soils. Agricultural inputs, such as fertilisers and pesticides, are the major sources of arsenic in soils, although the contribution to the total human exposure is difficult to quantify. Arsenic accumulation in soils due to repeated application of arsenic-containing fertilisers may raise levels in crops grown on such soils.

Few edible plants are able to translocate arsenic effectively from the lateral roots to the edible portion. The concentrations of arsenic in pasture herbage examined in a UK study were generally low even on the highly contaminated soils. Unwashed samples, however, showed markedly higher As contents due to contamination on the grass (Abrahams and Thornton, 1994). It is possible that some arsenic accumulates on root surfaces during uptake of water while plants are growing (Weaver *et al.*, 1984).

The highest levels of arsenic in plants occur in the roots, followed by the aerial parts – seeds and fruit (NRCC, 1978). A UK study investigated the relationships between the arsenic contents of beetroot, lettuce, onion and pea and found that levels increase in the edible tissues with increasing soil concentration. Arsenic in carrots and beans was not significantly related to soil content (Xu and Thornton, 1985), and despite high soil concentrations, all vegetable samples examined were well below the permitted level for arsenic in the UK. In studies of fruits grown on arsenate-contaminated soils, it has been found that although the levels were elevated compared with levels in fruits grown on uncontaminated soil, they were still well below levels that would constitute a risk to the health due to human consumption (Creger and Peryea, 1992).

There is a very narrow margin between background concentrations and concentrations that are toxic to plants (Sheppard, 1992), and phytotoxicity has been observed in plants with relatively low levels of arsenic (MAFF, 1982), and it appears that this phytotoxicity limits the upper levels of arsenic in vegetables. Both arsenite and arsenate have been found to decrease crop yields and increase As concentration in crops (Jiang and Singh, 1994).

It appears, therefore, that due to the limited uptake of arsenic from the soil and its phytotoxicity at relatively low concentrations, plants may act as barriers in the environment, and may effectively protect humans from harmful levels of exposure from this route. The same argument has been used to suggest that high dietary intake of arsenic by livestock is prevented, therefore protecting humans from harmful levels from this route also (Sheppard, 1992). These contributions, however, must be assessed in relation to other pathways of exposure, i.e., water supplies, inhalation of air, atmospheric deposition onto crop surfaces, and direct ingestion of contaminated soils and dusts.

Soil contamination of pasture is thought to be an important pathway of dietary intake of arsenic for livestock grazing on contaminated land (in extreme circumstances contributing up to 97 per cent of the total arsenic intake by the animals). However, as very little arsenic in the soil is actually absorbed by grazing animals, health problems in grazing livestock are uncommon. Drinking water is an important source of exposure to arsenic, therefore direct releases of arsenic to the freshwater environment may have particular significance with respect to human exposure.

Most environmental transformations of arsenic appear to occur in the soil, sediments, plants and animals, and in zones of biological activity in the oceans. Biomethylation and bioreduction are probably the most important environmental transformations of the element, since they can produce organometallic species that are sufficiently stable to be mobile in air and water. Arsenates occur naturally in oxygenated environments, while arsenite is probably the dominant form under moderately reducing conditions such as flooded soils. Inorganic arsenic in the environment may be converted into organic arsenic compounds by micro-organisms.

The composition of the diet can influence absorption of arsenic in both its dissolved and adsorbed states through physical and chemical interactions with various components, such as other trace elements, proteins and fatty materials (Pershagen, 1978). The uptake of inorganic arsenic from the gastro-intestinal tract (GI tract) is high for dissolved compounds, whereas insoluble compounds show less absorption. In humans more than 90 per cent of inorganic arsenic given in a water solution is absorbed from the GI tract (Vahter and Norin, 1980). The organic arsenic compounds present in seafoods are almost completely absorbed when ingested. Over 80 per cent of the ingested dissolved inorganic arsenic is absorbed from the GI tract (Fowler *et al.,* 1979), and the US Environmental Protection Agency (US EPA) risk assessment procedure uses an absorption factor of 0.98 (Owen, 1990) and this is the factor used in this assessment.

Absorption of arsenic following inhalation is uncertain. Retention of ambient aerosols is approximately 35 per cent and a large fraction of this is transferred from the lungs to the blood (50–80 per cent) giving an absorption factor of 0.28 (NRCC, 1978). The lung absorption factors used by the US EPA for risk assessment is 0.34 (Owen, 1990) and is the value used in this assessment. Skin is a possible route of absorption as indicated by some occupational accidents.

Inorganic arsenic absorbed from the GI tract is methylated into less toxic, organic forms at various sites in the body, in particular the liver and kidney, and is transported by the blood to

different organs in the body mainly in the form of dimethylarsenic acid. Even at low background levels of arsenic exposure, methylation is far from complete (Foa *et al.*, 1983), and at high exposures the methylation process may become saturated resulting in a sudden increase in levels of inorganic arsenic in the blood. Inorganic trivalent arsenic is systematically more poisonous than the pentavalent form and it is possible that pentavalent arsenic is reduced to the trivalent form before exerting any toxic effects (Jonnalagadda and Rao, 1993). In humans with exposures to relatively low concentrations of arsenic, the highest arsenic concentrations are found in the skin, hair and nails.

The arsenic concentrations in animal tissues are usually related to arsenic levels in the diet (Vreman *et al.*, 1986). Mammals, including man, have been shown to methylate arsenic (Tam *et al.*, 1979). Since methylated arsenic compounds are generally less toxic than inorganic arsenic compounds, methylation may act as a detoxifying mechanism **and it is possible that a limited number of plant species may also be able to detoxify arsenic to an extent using a similar mechanism (Tam *et al.*, 1979).**

Arsenic is eliminated from the body at a rapid rate, and the mean retention time for inorganic arsenic in the body is approximately 8 days (Bennett, 1981), whilst organic forms have shorter retention times (approximately 4 days). At least 50 per cent of ingested arsenic is excreted in the urine within one week, thus the relatively short half-time (WHO, 1994). More than 90 per cent of arsenic in the blood is rapidly cleared, with the remaining arsenic cleared 10–100 times more slowly in two additional phases.

Most absorbed inorganic arsenic is excreted in urine as organic acids, although a small portion of absorbed arsenic is excreted in the urine without methylation. The relative fraction of non-methylated arsenic compounds increases with the intensity of the exposure. The urinary concentration of arsenic may be a useful indicator of the exposure to inorganic arsenic since the main route of excretion is via the kidneys. Levels of arsenic in the urine of people who often ate higher than average quantities of flatfish and shellfish were 1.5 times higher than those of people who ate less flatfish, and those people who ate different types of seafood (Vahter, 1986).

5.3 ENVIRONMENTAL LEVELS

Traditionally concentrations of arsenic compounds found in environmental samples have been reported as total arsenic following digestion, but the toxicological properties of arsenic compounds are dependent on the chemical nature of the compound as well as the quantity present. Limited data indicate that the proportion of inorganic arsenic in food ranges from 0 per cent in saltwater fish to 75 per cent in dairy and meat products.

Air

Airborne arsenic particulates in ambient air may contain both inorganic and organic arsenic compounds, and in rain from an urbanised area only 35 per cent was present in the inorganic trivalent form (generally regarded to be the toxicologically important form of arsenic). In the UK a declining trend has been observed during the period 1957–1974 at one site which was attributed to legislation restricting industrial emissions (Salmon *et al.*, 1978).

Rural air

Concentrations in ambient air in non-urbanised areas are generally less than 10 ng As/m^3 (Pershagen, 1978). Arsenic has been measured in UK air in rural areas, and a range of annual mean levels <0.5–12.3 ng As/m^3 recorded (Cawse, 1977). Levels in rural areas of the USA, UK and Canada range from 0.3–6.0 ng As/m^3 (NAS, 1977; NRCC, 1978). The mean ambient range for the US (1977–1981) was 5–10 ng As/m^3 (US EPA, 1984). The value selected for the calculation of the EALs was the range of values recorded by Cawse for UK air, i.e., <0.5–12.1 ng/m^3.

Urban air

Measurement of arsenic levels in London and Manchester found levels of 94 ng As/m^3 and 52 ng As/m^3, respectively (Goulden, 1952). Hughes *et al.* (1994) measured a mean concentration of 1 ng As/m^3 in 11 Canadian cities, but levels in urban air in the US are around 20 ng As/m^3 (IARC, 1980). Levels in the industrialised areas of the Great Lakes have been measured at 60 ng As/m^3 (Landsberger *et al.*, 1993).

Soil

The typical background concentration for soil is 1–10 mg As/kg (Sheppard, 1992). The mean level in soil in the earth's crust is 6 mg As/kg with a range of 0.1–40 mg As/kg according to Bowen (1979), and the average background level is approximately 7 mg As/kg (NRCC). Mean levels in various uncontaminated Canadian soil types lie in the range 5–14 mg As/kg (Hughes *et al.*, 1994), US background soil levels have been estimated to range from 0.2–40 mg As/kg, but rarely exceed 10 mg As/kg (Smith *et al.*, 1992) and 5.2–13.3 mg As/kg (Cherian *et al.*, 1990).

Mean levels in UK farming areas range from 5.7–8.3 mg As/kg, with a general trend of decreasing arsenic levels with increasing depth of soil (Li and Thornton, 1993). The trend of decreasing arsenic levels with increasing soil depth has also been noted by Ori *et al.* (1993) during a comprehensive survey of soil arsenic in an agricultural area. The mean background levels in Whitburn (a small town in Scotland used as a comparison with a contaminated area) was recorded as 8.1 mg As/kg (Smith *et al.*, 1986). Danish arable soil levels are 2–4 mg As/kg (Andersen, 1980).

The representative value for urban soil used in the calculation of exposure was 40 mg As/kg (there are no specific data for urban soil, so the highest **mean** level measured in background soil has been adopted) **and for the calculation of the EAL the range of values used for rural soil are those quoted for arable soils (Li and Thornton, 1993), i.e., 5.7–8.1 mg As/kg.**

Water

Organic arsenic is mainly found in shallow coastal water, and no organic arsenic is found in river water (Bennett *et al.*, 1981). A total arsenic concentration in the North Sea of 0.9 µg As/l has been measured by Haring *et al.* (1982). Mean levels of inorganic arsenic in sea water have been measured, with levels in open sea water around 1.54 µg As/l, and in coastal waters 1.08 µg As/l (Sturgeon *et al.*, 1986). Concentrations in sea water generally increase with increasing depth, and in the open sea inorganic arsenic levels are in the range 1.4–2.0 µg As/l (mean 1.5 µg As/l) and in coastal waters the levels are 0.6–1.2 µg As/l (Fujiwara *et al.*, 1990).

Narasaki (1988) measured the levels in six unpolluted Japanese rivers, and found a range of 1–4 μg As/l (mean 2.2 μg As/l). Further studies of the unpolluted Arakawa River in Japan has given levels of 0.5–2.7 μg As/l (Narasaki *et al.*, 1992). Ho *et al.* (1984) investigated background levels in river water (0.4 μg As/l), while the mean concentration in unpolluted German rivers is estimated to be 2.2 μg As/l (Haring *et al.*, 1982).

The representative levels selected for fresh water is a range 0.4 to 2.2 μg/l and for coastal water is 0.6 to 1.2 μg/l.

5.4 ARSENIC IN FOOD

(See Appendix 5.2).

The Ministry of Agriculture, Fisheries and Food (MAFF) completed a comprehensive study in arsenic in the UK diet (MAFF, 1982), but data from other studies have been used to supplement the UK data where unavailable and replace it when more recent data from studies using more sensitive analytical techniques is available, and the MAFF data are recorded as values lower than the limit of detection (LOD).

Meat

The MAFF survey found that the mean levels in meat were <30 μg As/kg. Studies investigating levels in beef in West Germany have found a mean level of 9 μg As/kg (Holm, 1978), and more recently in the Netherlands levels of 4 μg As/kg (Vos *et al.*, 1987) and 4–5 μg As/kg (Vreman *et al.*, 1986) have been found.

In sheep a mean level of 1 μg As/kg has been recorded (Vos *et al.*, 1988). The levels found in the tissues of Dutch sheep were comparable to those found in Dutch swine, and in general slightly higher levels have been found in cattle slaughtered in the Netherlands (Vos *et al.*, 1988). No relationship is observed between age and the arsenic concentration in the meat and organs. Elevated residues in pig and chicken products are almost certainly due to the use of organo-arsenical feed additives used for growth promotion or medicinal purposes (MAFF, 1982). Jorhem *et al.* (1991) detected a decrease in levels of arsenic in pig tissues between 1984 and 1988, and this was attributed to the decrease in the use of fish meal (high in organic arsenic) in pig feed.

The selected representative values were 9 μg As/kg (beef), 1 μg As/kg (pork) and 1 μg As/kg (lamb).

Liver

The data from MAFF have mean levels of <400 μg As/kg (pig), <100 μg As/kg (cattle) and <100 μg As/kg (lamb).

The levels recorded in the livers of cattle from various countries lie within a relatively small range, and the levels found in sheep and pigs livers are lower than those found in cattle. The levels in the livers of cattle are not greatly elevated in comparison with those in the muscle tissue. The representative value selected is 17 μg As/kg.

Table 5.1 Arsenic levels in livers from various livestock studies

COUNTRY	ANIMAL	LEVEL	SOURCE
Netherlands	Cattle	11	Vaessen and Ellen, 1985
Netherlands	Cattle	13	Vos *et al.*, 1987
Netherlands	Sheep and pigs	5	Vos *et al.*, 1988
Sweden	Pigs	23	Jorhem *et al.*, 1991
West Germany	Cattle	13	Holm, 1978
US	Cattle	17	National Bureau, 1976

Kidney

The levels found in kidneys by the MAFF survey all had mean values lower than the LOD (110 μg As/kg).

The selected value is 48 μg As/kg.

Table 5.2 Arsenic levels in kidneys from various livestock studies

COUNTRY	ANIMAL	LEVEL	SOURCE
Netherlands	Cattle	47	Vaessen and Ellen, 1985
Netherlands	Cattle	48	Vos *et al.*, 1987
Sweden	Pigs	19	Jorhem *et al.*, 1991
Netherlands	Sheep and pigs	11	Vos *et al.*, 1988
West Germany	Cattle	34	Holm, 1978

Poultry

There are no available data on levels in poultry, so it is assumed that the levels are approximately the same as those in sheep and pigs, and therefore the representative value selected is 1 μg As/kg.

Meat products

No data are available for meat products and so it has been assumed that the level is equal to the weighted mean of the other three meat types, and therefore a selected value of 4 μg As/kg is used.

Whole milk, semi-skimmed milk and milk products

It is thought there is a blood-mammary barrier to arsenic, demonstrated by several separate papers in which cattle were fed arsenic-supplemented diets without a significant increase of the arsenic content of their milk (Jonnalagadda and Rao, 1993) and this may be the reason for the consistently low levels found in milk and other dairy products.

Cervera *et al.* (1994) carried out an extensive analysis of levels in various milk products in Spain; results for whole milk and semi-skimmed milk were 0.32 μg As/kg and 0.48 μg As/kg, respectively. They concluded that the low levels of arsenic present in cows' milk do not pose a toxicological problem to humans. In the Netherlands an investigation found the average arsenic content to be 0.3 μg As/kg (Koops *et al.*, 1989). Other studies have found similarly low levels of <0.5 μg As/kg (Ihnat and Miller, 1977), <1.0 μg As/kg (Vreman *et al.*, 1986) and <0.4 μg As/kg (Dabeka and Lacroix, 1987). The selected representative values for whole

and semi-skimmed milk were those reported by Cervera *et al.* (1994), i.e., 0.32 and 0.48 μg As/kg, respectively.

No data are available on other milk products – cream, butter, cheese and ice cream, although MAFF (1982) did carry out analyses and found all samples to have less than 250 μg As/kg. Given that the LOD for the MAFF data is high, it is assumed that the levels in these products are approximately the same as in whole milk. Therefore a representative value of 0.4 μg As/kg has been used.

Yoghurt

Canadian data (Dabeka and Lacroix, 1985) report levels of 1–2 μg As/kg, which are higher than those levels reported for whole milk. This may be as a result of the other ingredients in the yoghurts, e.g., fruit, which contain higher levels of arsenic than whole milk, and therefore will elevate the concentrations found in yoghurts.

Eggs

Results indicate a substantial increase in arsenic residues in eggs following the use of organic arsenical feeds (Donoghue *et al.*, 1994), and this may cause concern with respect to human health. Slightly greater levels have been found in eggs than in other animal products (Boudene, 1978), about 60 μg As/kg. The MAFF data found levels to be <50 μg As/kg. The selected representative value for eggs in the UK was assumed to be that from the MAFF data, and consequently a selected representative level of 50 μg As/kg was used.

Oils

Although arsenic is seldom determined in plant oils, 200 μg As/kg has been reported for crude rapeseed oils (Elson *et al.*, 1983). A total diet study in Canada found levels of approximately 19 μg As/kg in fats and oils, and in a study investigating levels in olive oil, all samples were found to have levels <20 μg As/kg (Miliadis and Liapis, 1993). The representative value used was 20 μg As/kg.

Vegetables

Levels in vegetables are generally less than 10 μg As/kg even in areas previously exposed to moderate arsenic contamination (Miliadis and Liapis, 1993). In most vegetables consumed in the UK, levels of arsenic are <10 μg/kg (MAFF, 1982), the exception in the selected types being lettuce, for which a mean level of 20 μg/kg has been reported. All data for tomatoes were <10 μg As/kg, and the highest recorded value is 8 μg/kg (Pyles and Woolson, 1982). The representative values used in the assessment are summarised in Table 5.3, the literature sources and ranges of the recorded data can be found in Appendix 5.1.

Table 5.3 Levels of arsenic in certain vegetable crops

VEGETABLE	LEVEL (μg As/kg fresh weight)
Cabbage	10
Lettuce	20
Peas	10
Potatoes	10
Onions	10
Carrots	10
Tomatoes	8

Fruit

Lead arsenate is approved for use as a pesticide for the control of moths and sawfly in apples, pears and cherries, consequently its use for this purpose may represent a direct input of arsenic to the food chain via the consumption of fruit. Data for apples and apricots grown in soil of normal background levels are <15 μg As/kg and <10 μg As/kg respectively (Creger and Peryea, 1992). Other studies have found similar concentrations in apples of 4–14 μg As/kg (Wiersma *et al.*, 1986). A study of a number of fruits found all had a mean level of <10 μg As/kg (Miliadis and Liapis, 1993). The selected representative value was 14 μg As/kg, as this was the highest mean value recorded not expressed as <LOD.

Sugar

A survey of levels in different sugar products has illustrated the difference between levels in refined products and raw sugar. Dried raw pulp and molasses had levels of 320 and 270 μg As/kg, respectively, whilst levels in white sugar were less than 50 μg As/kg (Hujibregts *et al.*, 1985). The selected representative value was 50 μg As/kg, as in the UK the main constituent of this food group is refined white sugar (MAFF, 1994).

Cereals

The arsenic levels in cereals are high compared with those of other crops, probably as a result of the high dry matter contents of cereal grains. Wiersma *et al.* (1986) found levels of 45 μg As/kg and 67 μg As/kg in wheat and barley, respectively. Dabeka *et al.* (1993) measured levels of approximately 25 μg As/kg in bakery goods and cereals. MAFF found mean levels of between <20 and <50 μg As/kg in bread and other cereals, and all measurements were <50 μg As/kg. The selected representative mean in bread and cereals was 50 μg As/kg.

Beverages

An extensive study of beverages in Denmark found that for all beverages investigated the mean arsenic contents were at a constantly low level of 3–11 μg As/kg. The average total daily intake from beverages for Danes was estimated to be 6 μg As/day, constituting approximately 5 per cent of the total from food and beverages (Pedersen *et al.*, 1994). Levels for specific beverages were recorded (11 μg As/kg for wine; 7 μg As/kg for beer; 3 μg As/kg for soft drinks; 7 μg As/kg for juice; 4 μg As/kg for coffee). The selected representative value used was 11 μg As/kg.

Fish

The mean levels in freshwater fish in the US for the period 1976–1984 range from 140–270 μg As/kg (Schmitt and Brumbaugh, 1990). Levels in freshwater fish in Germany have also been measured by Fecher and Nagengast (1994) who found a range of 66–892 μg As/kg. The levels measured in farmed Rainbow Trout in Scotland were between 400–1,900 μg As/kg, with an overall mean level of 1,200 μg As/kg (MAFF, 1982).

Most arsenic present in marine organisms is present as organic arsenic compounds of high chemical stability (Pershagen, 1978). MAFF (1982) found consistently higher arsenic levels in fish species which live on or near the sea bed. The mean concentration in fish consumed in the UK, from the MAFF total diet study, was 1,550–3,600 μg As/kg. Levels in shellfish are high in comparison to other foodstuffs (MAFF, 1982) with levels in crab (10,900 μg As/kg), mussels (2,500 μg As/kg), prawns (8,800 μg As/kg) and whelks (9,000 μg As/kg) often three orders of magnitude higher than in meat and vegetables, and approximately four orders of magnitude greater than levels in dairy products. Canned fish generally have lower concentrations than fresh fish caught in coastal waters, most samples containing less than 600 μg As/kg (MAFF, 1982).

The representative values used for the various fish types in this assessment are 1,200 μg/kg (freshwater fish), 3,600 μg/kg (marine fish), 600 μg/kg (canned fish) and 10,900 μg/kg (shellfish).

Drinking water

Standards for drinking water are an upper limit of 50 μg As/l, but for long-term safe consumption a level of less than 10 μg As/l is recommended (WHO, 1971). The EC directive relating to the quality of water intended for human consumption (80/778/EEC) lists a maximum allowable concentration of 50 μg/l. A survey of tap water in Bradford and Reading (MAFF, 1982) found levels in all samples to be less than 5 μg As/l. Most major US drinking water supplies contain levels lower than 5 μg/l (Smith *et al.,* 1992). A mean level of 5 μg As/l was used for the assessment by Hughes *et al.* (1994) for water in Canada, although it was recognised that levels in most Canadian surface drinking water supplies were considerably less than this value. In Germany a mean concentration of 1.3 μg As/l has been measured by Haring *et al.* (1982).

Concentrations in ground water depend on the arsenic content of the bed rock, and often exceed 5 μg/l (Hughes *et al.,* 1994), and therefore represent an important source of exposure in cases where ground waters are used for human consumption. The representative level for drinking water used in the calculation of the exposure is 5 μg As/l.

All the representative concentrations for the various foods and environmental media can be found in Appendix 5.2.

5.5 INTAKE AND EXPOSURE – ESTIMATES FOR AVERAGE INDIVIDUALS IN DIFFERENT AGE GROUPS

(See Appendices 5.3 to 5.10)

The data on arsenic levels in selected representative foods, drinking water, urban soil and urban air have been used together with information on the average consumption rates of

different foods in the UK, the consumption rate for drinking water and soil, and the breathing rate, to calculate the annual intake of total and inorganic arsenic for the average individual in the general population in five age groups. The annual intake is obtained by multiplying the amount consumed, or breathed, by the average (either total or inorganic arsenic) concentration in the media. The inorganic arsenic concentration in the media is calculated by multiplying the total arsenic concentration by the fraction of arsenic present in the inorganic form in those food or media. The fractions are obtained from the literature and are as follows for the various media:

i. 100 per cent of total arsenic in air, water and soil is in the inorganic form.

ii. 75 per cent of total arsenic in terrestrial foods and freshwater fish is in the inorganic form.

iii. 0 per cent of total arsenic in seafood is in the inorganic form.

The figure of 75 per cent for foods from the terrestrial food chain is probably a conservative estimate, as arsenic in the foodstuffs is generally between 0 and 75 per cent inorganic arsenic, and it is known that a wide range of organisms methylate arsenic as a detoxifying mechanism, and so much of the arsenic in plants and animals is in the organic form (Hughes *et al.*, 1994). Other studies have found 5–10 per cent of arsenic in fish to be present in the inorganic form, but given the extremely conservative estimate for terrestrial foods, the assumption of 0 per cent inorganic arsenic in seafood, as used in the Canadian Government's assessment of risk to health from environmental exposure to arsenic, is considered reasonable.

Intake from breast-feeding

Arsenic in human milk has been measured as 0.7 μg As/kg (range 0.2–1.1 μg As/kg) by Dang *et al.* (1983). Ninety-five per cent of arsenic exposure to the breast-feeding infant comes from milk, and as can be seen from Table 5.7, the exposure relative to body weight in infants is low in comparison with that of adults. Breast-feeding contributes a negligible proportion of the total lifetime exposure to arsenic.

Intake

It can be seen from Table 5.4 that adults have the highest daily intake of total arsenic per unit body weight, but that only approximately 35 per cent of this is in the inorganic form. The adult daily intake is equivalent to 91 μg per day total arsenic and 32 μg per day inorganic arsenic (for a 70 kg adult).

Table 5.4 Intake (μg/kg bw/day) of total and inorganic arsenic over the lifetime and at various ages

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Total	0.12	0.95	1.13	1.10	1.30	1.25
Inorganic	0.12	0.34	0.48	0.38	0.46	0.45

Table 5.5 clearly shows that food is the main contributor to total arsenic intakes (84 per cent), but for inorganic arsenic intake the dietary contribution is less (57 per cent), and drinking water is a major source of intake (30 per cent). This is because much of the total arsenic from dietary sources comes from fish, and is predominantly in the methylated (organic) form, whereas in other media, such as drinking water it is present in the inorganic form.

Table 5.5 Percentage contribution to total arsenic intake from various sources

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Diet	95	86	72	82	86	84
Soil		0	15	4	1	2
Water		11	9	11	11	11
Air	5	3	4	3	2	3

Table 5.6 Percentage contribution to inorganic arsenic intake from various sources

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Diet	95	60	34	47	60	57
Soil		0	36	12	3	6
Water		31	22	31	31	30
Air	5	10	9	10	6	7

Exposure

The exposure to the body (i.e., the amount absorbed by the blood) is calculated by multiplication of the intakes by the absorption factors for the gastro-intestinal tract (GI tract) (food, soil, water) and the lung (air). As mentioned previously, these transfer factors are 0.98 (GI tract) and 0.34 (lung). The exposure to arsenic (μg/kg bw/day) and the relative contributions from different sources to both total arsenic and inorganic arsenic exposure for the five age groups are summarised in Tables 5.7, 5.8 and 5.9.

Table 5.7 Exposure (μg/kg bw/day) to total and inorganic arsenic over the lifetime and at various ages

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Total	0.11	0.91	1.08	1.06	1.25	1.21
Inorganic	0.11	0.31	0.44	0.35	0.44	0.42

The total lifetime exposure to total arsenic is equivalent to approximately 72 μg/day, and 25 μg/day for inorganic arsenic. The exposure to total arsenic at various ages is used in the calculation of the body burden (see Table 5.10). The percentage contributions from various sources are similar to those for intake for both total and inorganic arsenic.

Table 5.8 Percentage contribution to lifetime total arsenic exposure for various age groups

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Diet	95	88	74	84	87	86
Soil		0	15	4	1	2
Water		11	9	11	11	11
Air	5	1	1	1	1	1

Table 5.9 Percentage contribution to lifetime inorganic arsenic exposure for various age groups

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Diet	95	58	36	51	62	59
Soil		0	38	13	3	7
Water		38	23	31	32	32
Air	5	4	3	4	2	3

The main sources of lifetime exposure to inorganic arsenic are the diet (59 per cent) and drinking water (32 per cent). Soil (7 per cent) and air (3 per cent) make smaller contributions, although the direct ingestion of soil by young children may be an important source of exposure. Seafoods make up a large proportion of the exposure to total arsenic, but as it is present in an organic form which is not considered toxic in humans, virtually none of the exposure to toxicologically significant arsenic comes from marine foods.

Comparison with daily intakes from other studies

Total diet studies carried out by MAFF in the 1970s found the average daily intake of total arsenic from food for adults to be 89 μg As/day (MAFF, 1982), of which fish contributed ~75 per cent. In this study the total intake of arsenic for adults from all sources is estimated to be 91 μg As/day, and from dietary sources alone is estimated to be 78 μg As/day, slightly less than the MAFF estimate. MAFF concluded that for UK adults, significant intakes from either air or water would be unlikely. However, the contribution of fish and shellfish consumption to dietary intake is similar (74 per cent) and it is estimated that fish consumption represents 63 per cent of the total lifetime exposure to total arsenic.

Surveys in Canada, France, and USA indicated a range of 7 to 60 μg As/day with an average of about 30 μg As/day (WHO, 1973). In Canada it has been estimated that the average daily intake from food would not exceed 36 μg As/day (Smith *et al.*, 1975) and the estimated daily intake from food for an 18-year-old male in the US was estimated to fall from 71 μg As/day in 1967 to 14 μg As/day in 1974 (Jelinek and Corneliussen, 1977). The average daily dietary ingestion of total arsenic by the average Canadian is 38 μg, and varies from 15 μg for the 1 to 4-year-old group to 59 μg for the 20 to 39-year-old group (Dabeka *et al.*, 1993).

The mean daily intake of arsenic from food sources for adults in the US has been estimated to be 46 μg As/day (Gartrell *et al.*, 1986a). In a separate report, the mean daily intakes for infants and two-year-olds were estimated to be 1.26 μg As/day and 15.5 μg As/day, respectively (Gartrell *et al.*, 1986b). For the majority of people not living in areas with elevated atmospheric or water concentrations, it was concluded that the major source of exposure to arsenic is the diet. However, in food much of the arsenic is in organic forms (IPCS, 1981) that are rapidly excreted unchanged (Foa *et al.*, 1984).

On average, 114 μg arsenic is obtained from a Danish diet per day (National Food Agency, 1990). The main exposure to inorganic arsenic is through ingestion (Smith *et al.*, 1992). Current dietary intake of total arsenic in US adults, excluding tap water, has been estimated to be around 45–50 μg As/day (US EPA, 1988; Gunderson, 1988). Most of the arsenic derives from seafood, meat and poultry (80 per cent) and grains and cereals (17 per cent). Although seafood has a high arsenic content, only 5–10 per cent is in the inorganic form and the

organic forms (mainly arsenobetaine) are excreted unchanged (Vahter, 1986). Similar to this study in which 74 per cent of dietary arsenic came from fish, and 13 per cent from cereals.

The total daily intake of arsenic from environmental exposure may range from 10 μg As/day to 1,000 μg As/day **(Bennett *et al.*, 1981)** and is greatly influenced by the amount of seafood in the diet (Bennett et al, 1981). Several other workers have found that dietary arsenic intake depends on the seafood content. Assessments of exposure to populations in the vicinity of anthropogenic inputs of arsenic to the environment, have found that the immediate residential environment is only a minor source of exposure, and that food (particularly seafood) from outside the impact area is the major source of measured arsenic **intake, although exposure is limited from this source due to the rapid excretion of organic forms of arsenic** (Polissar *et al.*, 1990).

An assessment of risk to health in Canada from environmental exposure to arsenic focused principally on inorganic arsenic, the form of primary concern (Hughes *et al.*, 1994). Most of the arsenic in drinking water, air and soil was assumed to be present in the inorganic form, while limited data indicate that the proportion of inorganic arsenic in food ranges from 0 per cent in saltwater fish to 75 per cent in dairy and meat products. The estimated total amount of inorganic arsenic ingested daily ranged from 0.2–0.8 μg As/kg bw/day, with the greatest exposure being in young children. We have found the greatest exposure to be in adults and the 2- to 7-year-old age group, around 0.44 μg/kg bw/day (see Table 5.7). However, the principal sources of exposure at all ages are the same, i.e., drinking water and food. According to Hughes *et al.* (1994) only relatively minor amounts of inorganic arsenic are inhaled daily (0.0003 to 0.0004 μg As/kg bw/day), **and whilst this study found the exposure from inhalation sources to vary between 0.010 and 0.015 μg As/kg bw/day (greatest exposure being in young children aged 2–7 years), both studies have found the contribution of inhalation to the intake of inorganic arsenic to be low.**

Investigation of the deposition of atmospheric arsenic surrounding a wood preservation factory, its uptake by vegetables and the assessment of risk to the population, it was found that although consumption of vegetables grown near the source would result in an increased intake of inorganic arsenic, the intake from the total diet would still be below the tolerable daily intake established by FAO/WHO (Larsen *et al.*, 1992). An attempt to establish the extent and degree of contamination in an area of elevated environmental levels in the UK (Cornwall), and to establish how much arsenic is taken into the edible tissues crops, found that all vegetables sampled were below the UK statutory limit of 1,000 μg As/kg (fw). However, it was stressed that implications for health should be assessed in relation to other exposure routes via water, air and directly ingested dust and soil (Xu and Thornton, 1985).

Dermal uptake of arsenic is of minor importance except in some cases of occupational accidents (Pershagen, 1978). The dermal exposure of arsenic, although not well characterised, appears to be low and exposure through skin contact with soil would seem to be minor (US EPA, 1984).

Arsenic concentrations in water supplies can vary considerably, and given the contribution to exposure to inorganic arsenic from this source (about 30 per cent) from background levels, the contamination of drinking water supplies may have significant influence on the lifetime

exposure of individuals using such supplies. Therefore, levels of arsenic in drinking water are of major importance when seeking to limit human exposure to arsenic.

The US EPA estimated the average total intake of inorganic arsenic from food, water and other beverages to be about 17 μg As/day, of which 5 μg As/day comes from drinking water (US EPA, 1988). For the majority of the US population about 30 per cent of ingested inorganic arsenic comes from drinking water. However, if inorganic arsenic were present at the current US standard (50 μg As/l), drinking water could contribute almost 100 μg more to the daily intake of inorganic arsenic, representing 90 per cent of the daily intake. In this study the intake of inorganic arsenic in adults from all sources was estimated as 29 μg As/day, of which 10 μg As/day comes from drinking water (i.e., 34 per cent of the total intake).

Smoking

Cigarette smoking is estimated to contribute an additional 0.01–0.04 μg As/kg bw/day (Hughes *et al.*, 1994). Given that in adults the intake of total arsenic is approximately 91 μg As/day, this will lead to an additional 1–3 per cent exposure to total arsenic. For inorganic arsenic the total daily intake is estimated as approximately 32 μg As/day, and therefore smoking 20 cigarettes a day will lead to an additional 2–8 per cent exposure to inorganic arsenic. Therefore heavy smoking may contribute significantly to lifetime exposure to arsenic.

5.6 BODY BURDEN – ESTIMATES FOR INDIVIDUALS AT VARIOUS AGES

The body burden has been estimated using the relationship between retention time and intake, represented by the following equation:

$C = T/M \times F$ Where: C = Body concentration (μg/kg)
T = Retention time (days)
M = Body weight (kg)
F = Exposure to body tissues

Arsenic is rapidly eliminated from the body and the mean retention time is estimated to be approximately 8 days (Bennett, 1981). Therefore, with a constant intake the steady-state equilibrium concentration in the body will be reached in a relatively short time. In studies of occupational exposure a steady state of exposure and elevated urine levels of arsenic was reached after only one day of exposure (Vahter, 1986). The calculation of body concentration and body burden is carried out using the exposure to total arsenic for each age group. It has been assumed that the retention time of arsenic in the body is the same for both organic and inorganic arsenic, as the calculations and estimates of retention times both relate to total arsenic, although the retention time of organic forms of arsenic is generally shorter than that of inorganic forms.

Table 5.10 Data used for the calculation of the body burden at various ages

	BIRTH–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT
Exposure (µg/day)	0.54	10.9	25.8	45.5	87.8
Body mass (kg)	5	12	24	43	70
Body concentration (µg/kg)	0.9	7.3	8.6	8.5	10.0
Body burden (µg)	4	87	207	364	702

Comparison of estimates of the body burden with values from the literature

The new-born baby

There are no available data on the levels of arsenic in the body of the new-born, or the transport of arsenic across the placenta. Therefore, it has been assumed that the blood concentration in the foetus is approximately equal to the blood concentration of an adult female, therefore a new-born baby would be expected to have a body burden of approximately 35 µg (adult body burden is 702 ug; adult body weight is 70 kg; new-born body weight is 3.5 kg). If this is the case then it would be expected that the body burden would fall during breast-feeding due to relatively low levels of arsenic exposure from ingestion of human milk.

Previous studies have estimated the total body content of arsenic in an adult to be 984 µg (Liebscher and Smith, 1968). This compares reasonably well with the estimate of body burden in adults in this study of 702 µg (Table 5.10).

Table 5.11 Tissue levels and burdens in the human body

TISSUE	As LEVEL (µg/kg fresh weight)	TISSUE MASS (kg)	BURDEN (µg)
Muscle	13	28.0	370
Bone	47	5.0	240
Skin	34	2.6	89
Blood	7	5.5	40
Liver	8	1.8	14
Lungs	21	0.57	12
Hair	41	0.02	8
Nails	28	0.003	1
Other Tissue	8	26.5	210
Total			**984**

Source: From Liebscher and Smith, 1968

From Table 5.11 it can be seen that approximately 4 per cent of the total body burden is found in the blood. Therefore, given an adult body burden of 702 µg the mean concentration in the blood would be approximately 5.1 µg As/kg (blood mass in an adult is 5.5 kg, ICRP, 1975). This estimate compares favourably with measured data from studies by Dang *et al.* (1983) and Cross *et al.* (1979), summarised in Table 5.12. The hair has the highest concentration of arsenic, and concentrations in particular organs may vary by an order of magnitude between individuals (Dang *et al.*, 1983).

Table 5.12 Measured levels of arsenic in various body organs

Organ	Dang et al. (1983)	Cross et al. (1979)	Larsen et al. (1972)	Leibscher and Smith (1968)	Brune et al. (1978)
Liver	14.5	7.8	11.0	8.0	6.0
Kidney	12.4	7.5	7.0		5.0
Brain	3.9	2.8			
Lungs	19.9	16.2	10.0	21.0	11.0
Spleen	15.2	5.0	3.0		
Hair	457.0	510.0		410.0	
Blood	5.8	5.7		7.0	

5.7 TOXICOLOGICAL ASSESSMENT

Human indices of exposure to arsenic are difficult to establish due to the differing toxicity of organic and inorganic compounds, and the need for identifying and quantifying chemical species, not just total concentration of arsenic, is well recognised (Vanhoe, 1993). There is also difficulty in relating tissue concentrations to effects, because there is not a generally accepted critical organ for arsenic (Lafontaine, 1978).

Detoxification of arsenic through methylation is believed to be important, but the mechanisms and the quantitative relationships are not yet understood. It is suspected that inorganic arsenic is the toxic agent and that the metabolic processes to detoxify inorganic arsenic by methylation become saturated. Above this level further increases in the ingestion of inorganic arsenic may lead to large increases in the dose of inorganic arsenic delivered to target tissues. The dose level at which saturation occurs may depend on genetic and dietary factors that determine the availability of enzymes necessary for the detoxification processes (Warner North, 1992).

Skin lesions are the most common expression of arsenic toxicity following long-term exposure. Elevated levels of arsenic in drinking water have also been reported to have neurological effects (Abernathy and Ohanian, 1992). Literature suggests that the nervous and cardiovascular systems are targets for arsenic toxicity in children (ATSDR, 1988). Skin absorption may occur after spillage of soluble arsenic salts on to the skin of workers and may lead to death. In cases of occupational exposure to inhaled arsenic, respiratory diseases including cancer have been clearly observed (Tsuda *et al.*, 1992).

The Joint Expert Committee of the FAO/WHO (1983) set the maximum tolerable daily intake of inorganic arsenic at 2 μg As/kg bw/day assuming a considerably lower toxicity of organo-arsenic compounds. The US EPA has adopted a reference dose of 1 μg As/kg bw/day (Diaz-Berriga *et al.*, 1993).

Arsenic is one of the few chemicals recognised as a human carcinogen (classified by the US EPA as a known carcinogen when exposure occurs by inhalation and oral routes), but it has not clearly been shown to produce cancer in animals (Marcus and Rispin, 1988). In humans arsenic exposure through inhalation is judged to cause lung cancer and ingested arsenic is judged to cause skin cancer. It has been suggested that both skin cancer and other

toxic effects are demonstrated only above intakes of 200 µg As/day (inorganic) and that this may be considered as a practical threshold (Warner North, 1992). The TDI of 2 µg As/kg bw/day is based on these data, and derived by applying a safety factor of 100.

There appears to be a strong correlation between the effects of arsenic and the age at which individuals are exposed, with an increase in cancer rates for older persons in groups of mixed ages who have been exposed to arsenic for the same length of time (Murphy and Toole, 1989). It has been hypothesised that arsenic neither initiates nor promotes cancer, but acts as a gene amplifier. As older people have a higher probability of completion of the initiation and promotion stages of cancer, they would also have a higher probability of displaying an effect from arsenic exposure (Brown and Chu, 1983).

A recent assessment of cancer risk from arsenic in drinking water concluded that measures to reduce arsenic levels in water supplies should be considered (Smith *et al.*, 1992). Studies by Fishbein (1972) and Perrose (1974) have shown arsenic to be carcinogenic to humans and arsenosis may result from low intake levels. Further studies have presented evidence that at chronic low doses, arsenic exposure may lead to lung and skin cancer (Doull *et al.*, 1980).

5.8 Derivation of Environmental Assessment Levels from Data on the Estimated Intake

It has been suggested that the risks associated with the current US drinking water standard place arsenic at the forefront of cancer risks associated with environmental exposures, comparable even with cancer risks associated with inhalation of environmental tobacco smoke. Serious environmental cancer risks are posed by arsenic in drinking water (Smith *et al.*, 1992).

The critical value is the TDI of the toxicologically significant inorganic arsenic of 2 µg As/kg bw/day. The current estimated intakes at various ages are shown in Table 5.4, and from these a lifetime daily intake of 0.45 µg As/kg bw/day for inorganic arsenic can be calculated.

Current Background Levels

	Range	Mid-Range
Air	0.5–12.3 ng As/m^3	6.4
Soil	5.7–8.3 mg As/kg	7.0
Fresh water	0.4–2.2 µg As/l	1.3
Coastal sea water	0.6–1.2 µg As/l	0.9
TDI	2 µg As/kg bw/day	
Current intake	0.45 µg As/kg bw/day	

Therefore the maximum acceptable environmental levels (i.e., those associated with a TDI of 2 μg As/kg bw/day) are 4.4 times (2 ÷ 0.45) higher than current background levels. Using this calculation the EALs would be:

	EAL	MID-RANGE
Air	2–54 ng As/m^3	28
Soil	25–37 mg As/kg	31
Fresh water	2.2–11.9 μg As/l	5.7
Coastal sea water	2.6–3 μg As/l	4.0

5.9 ENVIRONMENTAL STANDARDS FOR ARSENIC

The current EAL of 200 ng/m^3 for arsenic in air is considerably higher than the one proposed in this study, the difference being a factor of about 7. Given that the estimates of intake are consistent with a number of other studies (and can therefore be considered representative of the general UK population) and that the TDI for inorganic arsenic is a well-established and widely accepted value, **this difference between the current EAL and the level proposed by this study may merit further discussion and investigation.**

However, it may also be argued that due to the limited uptake of arsenic by plants and the relatively low levels accumulated in livestock as a result, the potential exposure from the atmospheric deposition of arsenic to soil is limited. Certainly, the current difference between the EAL and proposed EAL merits further investigation.

The EQS for arsenic in fresh waters is 50 μg/l, and the proposed EAL is considerably lower (5.7 μg/l). Given that the drinking water route has been identified as highly important in terms of human exposure to arsenic, **close scrutiny and further investigation of the exposure to the general population from this route is necessary.**

The proposed EAL for coastal waters is 4 μg/l, again considerably lower than the current EQS of 25 μg/l. However, as the exposure route from the marine environment involves the consumption of fish, in which arsenic is present in the toxicologically insignificant organic form, this difference may not have a significant bearing on the long-term protection of human health.

5.10 SUMMARY

1. There is good agreement on the background levels of arsenic in environmental media from a number of sources. Therefore the current background environmental levels can be taken as being representative.
2. There are a number of consistent data from different sources on arsenic levels in foods and the estimates of intake, exposure and body burden appear consistent with previous studies.

3. The main sources of lifetime exposure to inorganic arsenic are drinking water (32 per cent), cereals (25 per cent) and vegetables and fruit (16 per cent). Despite high levels of total arsenic in fish, the majority is present in organic forms which are rapidly excreted from the body, and considered to be far less toxicologically important than inorganic forms.

4. It has been suggested that arsenic toxicity to plants will prevent levels reaching those which could pose a threat to human health.

5. Arsenic levels in drinking water are of primary concern when consideration is given to limiting human exposure for the general population to arsenic.

6. The total intake of inorganic arsenic of 0.45 μg As/kg bw/day appears to be representative for the lifetime of an average UK individual.

7. The most widely-accepted measure of the maximum allowable exposure is the FAO/WHO Tolerable Daily Intake for inorganic arsenic of 2 μg As/kg bw/day.

8. The maximum allowable environmental levels compatible with human health when consideration is given to lifetime exposure to inorganic arsenic are estimated to be approximately 4.4 times greater than current background environmental levels. Environmental Assessment Levels have been calculated on this basis.

9. The proposed EALs are considerably lower than those standards currently in force. In the case of the EAL for arsenic in air, and the EQS in fresh water, the current standards may require further examination to ensure that they are sufficiently stringent to protect human health. Arsenic exposure from the marine environment is mainly in the organic form which is not considered to be of toxicological significance, therefore the EQS for coastal waters may be sufficient to protect human health, despite being considerably higher than the proposed EAL.

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6. EXPOSURE COMMITMENT ASSESSMENT FOR MERCURY

6.1 INTRODUCTION

Detailed estimates of intake and exposure to mercury have been made using UK data (where these have not been available, data from other industrialised nations have been used) and internationally-accepted reference values for the human body. These estimates compare well with a number of other exposure assessments carried out by other workers. The estimated body burden and tissue levels are in good agreement with measured levels. The review of the toxicological information has identified the World Health Organization/Food and Agriculture Organization of the United Nations (WHO/FAO) Provisional Tolerable Weekly Intake (PTWI) for total mercury (300 μg) and organic methylmercury (200 μg) as the key measures of acceptable levels of exposure. The study estimates lifetime intake to be 0.08 μg/kg bw/day for the average individual in the UK. Environmental Assessment Levels (EALs) have been calculated for air, fresh water, soil, and coastal waters.

Mercury occurs naturally in three forms: inorganic (divalent), elemental and organic (methyl). It is released in to the atmosphere from volcanic activity and due to soil degassing; most anthropogenic mercury originates from waste incineration plants, coal combustion, chlor-alkali, metal smelter, electrical and paint industries. Man-made sources currently form about 25 per cent of the total emissions to the atmosphere and the anthropogenic contribution is greater in the Northern Hemisphere (WHO, 1990).

The most important of the three forms in terms of human exposure, is methylmercury (ATSDR, 1992). Environmental methylmercury arises mainly from the methylation of inorganic mercury (Gerhardsson *et al.,* 1994), and accumulates in aquatic organisms.

Uptake of the three forms by the human body varies considerably, and consequently influences the extent to which the routes of exposure contribute to total exposure. The proportions of inorganic, organic and elemental mercury present in environmental media have been studied and, from this information for the purposes of this assessment, it is assumed that all atmospheric mercury is in the elemental form; all mercury present in seafood is in the organic form and, in all other media, mercury is in the inorganic form.

Behaviour in the human body is complicated, but generally accumulation can occur in the brain and kidneys. The retention times of the different forms in the body also vary quite significantly. The key site of toxic effects is the brain, with neurological disturbances affecting the central nervous system. Mercury crosses the placental barrier, and the infant may receive appreciable exposure during breast-feeding. The greater susceptibility of the foetus and infant to the toxic effects of mercury make this period critical in terms of human health effects.

6.2 ENVIRONMENTAL TRANSPORT AND BEHAVIOUR

The global cycle of mercury almost exclusively involves inorganic and elemental mercury (WHO, 1990). Atmospheric transport and air/sea exchange are the main pathways of cycling (Fitzgerald and Clarkson, 1991), and in the atmosphere elemental mercury is the principal

species (Fitzgerald and Clarkson, 1991). It has been reported that more than 95 per cent of mercury in air is elemental mercury vapour, with the rest present as methylmercury and bivalent inorganic mercury (Johnson and Braman, 1974). However, a Canadian study of atmospheric mercury composition in the vicinity of Toronto (Schroeder and Jackson, 1987) found that approximately 75 per cent of the total concentration was present as elemental mercury vapour, with the remainder being present as methylmercury (20 per cent) and inorganic mercury (5 per cent).

Mercury enters the soil by atmospheric deposition and is very effectively fixed in the uppermost layer of soil (Andersson, 1979; Aastrup *et al.,* 1991), probably as a result of a tendency to associate strongly with humic substances, both in aquatic and terrestrial environments (Strohaland Huljev, 1971; Hakanson, 1974; Benes *et al.,* 1976; Andersson, 1979; Schnidzer and Kerndorf, 1981; Rae and Aston , 1982; Lodenius *et al.,* 1983; Zvonarev and Zyrin, 1983). However atmospheric mercury concentrations do not necessarily reflect those in soil (Barghigiani and Ristori, 1994) because the turnover of mercury in soil is extremely slow (Johansson *et al.,* 1991). Therefore short-term atmospheric fluctuations may not result in detectable changes in soil levels.

Despite accumulation in some soil systems almost no soil mercury is translocated from the root to the shoots of plants due to a barrier against movement of mercury into the above ground parts (Rauter, 1976; Beauford *et al.,* 1977; Huckabee *et al.,* 1983; Bargagli and Baldi, 1984; Fukuzaki *et al.* 1986; MAFF, 1987; Godbold and Huttermann, 1988; Lenka *et al.,* 1992).

Atmospheric deposition is the main source of mercury to remote aquatic systems (Rada *et al.,* 1989; Fitzgerald and Clarkson, 1991; Lindqvist *et al.,* 1991; Vandal *et al.,* 1991) as the mercury emitted to the atmosphere is converted to soluble forms, and deposited by rain water on to land and surface water bodies where the elemental mercury is oxidised to the divalent mercuric ion (Jonnalagadda and Rao, 1993). In aquatic systems subject to direct human influence, industrial and municipal discharges can introduce considerable amounts of mercury, and runoff may also be an important means of transport for mercury into freshwater systems (Iverfeldt and Johansson, 1988). In the sea and large lakes most mercury associated with humic matter is deposited to the sediments. Once in the sediments, the reactive inorganic mercury species derived from atmospheric precipitation are methylated mainly through microbiological processes (Jensen and Jernelov, 1969; WHO, 1972; Mason and Fitzgerald, 1990). This may also occur to a lesser extent in the soil (Kitamura *et al.,* 1969; Yamada and Tonamura, 1972) and the water column (ATSDR, 1992).

Mercury salts and organic mercury are readily taken up by aquatic organisms and accumulated in the tissues as methylmercury (Jonnalagadda and Rao, 1993). Due to the slow biological turnover in fish, with half-lives of several years (Boudou and Ribeyere, 1990; Meili, 1990), concentrations increase with age and size. In addition, species at the top of the food chain contain higher levels of mercury than those occurring at the bottom (Wood *et al.,* 1968; Akielaszek and Haines, 1981; Evans, 1986; MAFF, 1987). In a study carried out by the Ministry of Agriculture, Fisheries and Food (MAFF, 1971), 90 per cent of the total mercury in fish occurred as methylmercury, and in shellfish the proportion was 40 to 90 per cent. More recent studies have found that the proportion of methylmercury in fish varies from 50 to 80 per cent (MAFF, 1987), and 70 to 90 per cent (ATSDR, 1992).

The bioconcentration factor of methylmercury in fish tissue is usually between 10,000 and 100,000 (US EPA, 1990) and accumulation may be enhanced in fresh waters by low pH, alkalinity and conductivity (Bouzes *et al.*, 1977; Suns *et al.*, 1980; Wren and MacCrimmon, 1983; Bjorklund *et al.*, 1984; Lindqvist *et al.*, 1984; Hakanson *et al.*, 1988; Fitzgerald and Clarkson, 1991; Johansson *et al.*, 1991; Grandjean *et al.*, 1994).

Uptake and behaviour of the different forms of mercury in the human body varies considerably. In the case of the gastro-intestinal tract (GI tract) uptake of metallic mercury is low – around 0.01 per cent (HMSO, 1976) – but is higher for inorganic salts (15 per cent) and for organic species (>80 per cent) (Clarkson, 1972; Piotrowski and Coleman, 1980). Other workers have concluded that the absorption of inorganic mercury salts from the GI tract is lower, less than 10 per cent (Sollmann and Schreiber, 1936; Clarkson and Shapiro, 1971; Miettinen, 1973; Rahola *et al.*, 1973; ATSDR, 1992; Jonnalagadda and Prasada Rao, 1993). A more recent study reported GI tract absorption for methylmercury of 90 per cent, and for inorganic mercury a value of 10 per cent (Halbach, 1994). The absorption factors for methylmercury and inorganic forms in the GI tract, according to the US Environmental Protection Agency (US EPA) risk assessment purposes, are 95 per cent and 15 per cent, respectively and have been used in this assessment.

There is also variation in the absorption of the different forms across the lungs; about 80 per cent of elemental mercury vapour and methylmercury is absorbed while only 50 per cent of the bivalent inorganic mercury is absorbed (Kudsk, 1965, 1969; WHO 1976; Berlin, 1986; ATSDR, 1992; Jonnalagadda and Rao, 1993; Elinder *et al.*, 1994). The US EPA risk assessment absorption factor for elemental mercury is 75 per cent. The value used in this assessment is 75 per cent for elemental mercury.

Elemental mercury and inorganic and organic mercury compounds may be absorbed through the skin, although no quantitative data are available (WHO, 1980; Berlin, 1986; ATSDR, 1992).

Distribution in the body varies with the chemical form but, in general, between 3–10 per cent of the body burden of total mercury is found in the brain, 5–10 per cent in the blood, up to 44 per cent in muscle tissue and up to 30 per cent in the liver and kidneys (MAFF, 1971; OECD, 1974; Kitamura *et al.*, 1975, 1976; WHO, 1976; Ontario Ministry of Labour, 1977; Kershaw *et al.*, 1980). Some of the mercury accumulated in the brain is slowly eliminated with a biological half-life that may exceed several years (Kosta *et al.*, 1975; Rossi *et al.*, 1976; Elinder *et al.*, 1994). The shortest elimination half-life of both total mercury and methylmercury in the human brain reported so far is about 240 days (Takeuchi *et al.*, 1974; Takeuchi and Eto, 1975).

Organomercury species, especially methylmercury, are the most toxic to humans because they are lipophilic and bioaccumulate in the body (Piotrowski and Coleman, 1980; Zillioux *et al.*, 1993). Once in the body, methylmercury is distributed to all tissues in about four days (Kershaw *et al.*, 1980; Gerhardsson *et al.*, 1994). It is quite resistant to biodegradation and tends to be more evenly distributed in the body than other mercury compounds (MAFF, 1971; Elinder *et al.*, 1994), but concentrations in the brain can reach quite high levels. The brain is the ultimate site of toxic effects of methylmercury (Elinder *et al.*, 1994) and it contains

between 3 and 7 per cent (Inskip and Piotrowski, 1984), or up to 10 per cent (Aberg *et al.*, 1969) of the methylmercury body burden. Miettinen (1973) found methylmercury concentrations in the brain to be about six times higher than in the blood. Methylmercury is converted to the divalent inorganic form in the tissues where it is likely to bind to sulfhydryl groups on proteins, restricting its mobility in the human body (Berlin, 1986).

The behaviour of methylmercury in the brain tissue depends on the nature of exposure. Following typical exposure to methylmercury, demethylation is unlikely to occur in the brain, with about 95 per cent of mercury in the brain in the organic form (Berlin, 1986). In a study of an unexposed population, it was found that over 80 per cent of the mercury present in the brain was in the inorganic form (Kitamura *et al.*, 1975, 1976). Takizawa (1986) obtained a similar figure of 88 per cent in a study carried out in an unpolluted area.

Inorganic and elemental mercury are mainly found in the kidneys (Piotrowski and Coleman, 1980; Berlin, 1986; Elinder *et al.*, 1994) and, to a lesser extent, in the liver (Berlin, 1986). The oxidation of elemental mercury to its divalent inorganic form in the blood does not occur rapidly enough to prevent the passage of elemental mercury through the blood-brain barrier, the placenta and other tissues (Elinder *et al.*, 1994). After oxidation has taken place in these tissues, the inorganic mercury is trapped and held, leading to accumulation in brain (Piotrowski and Coleman, 1980) and foetal tissues (WHO, 1976). Inorganic mercury does not readily traverse the blood-brain or the placental barrier but it is capable of accumulation in the brain (Piotrowski and Coleman, 1980; Elinder *et al.*, 1994), placenta (Mitani *et al.*, 1978), foetal membranes and amniotic fluid (Wannag and Skjaerasen, 1975; Suzuki *et al.*, 1977). Most inorganic mercury salts disassociate into ions in the body (ATSDR, 1992). The tissue distribution and excretion pathways of inorganic mercury salts are similar to those of elemental mercury vapour (ATSDR, 1992).

Mercury accumulates to a much greater extent in the foetal brain than in adult tissue, reaching levels 2–4 times higher in the foetal cerebellum and cerebrum than the corresponding maternal tissues (Yang *et al.*, 1972; Null *et al.*, 1973). Under normal conditions of chronic exposure the placenta does not prevent foetal blood and tissue mercury accumulation (Pink and Reynolds, 1975; Reynolds and Pitkin, 1975; King *et al.*, 1976). This is probably due to higher foetal blood concentrations (Suzuki *et al.*, 1971), greater binding of mercury, and a longer half-life in the foetal brain (Yang *et al.*, 1973). Methylmercury (Suzuki *et al.*, 1967; Childs, 1973) crosses the placental barrier (Clarkson *et al.*, 1972; Greenwood *et al.*, 1972) and is also distributed to the breast milk (Smart, 1961; Bland and Egan, 1963; Friberg and Vostal, 1972; HMSO, 1976; WHO, 1976; Skerfving, 1988; Lindquist *et al.*, 1991; ATSDR, 1992). The elimination half-time of methylmercury in the mother's decreases to almost one third during lactation, possibly due to the excretion of mercury in the milk (Grandjean *et al.*, 1994).

As a result of the variations in behaviour of the different forms of mercury, there are differences in the half-lives reported in the literature. In general, for mercury in the whole body, it has been indicated that approximately 80 per cent is excreted with a half-life of around 60 days (Rahola *et al.*, 1971; Hursh *et al.*, 1976), and overall the half-life in the body has been estimated to be between 70 and 90 days (Harries *et al.*, 1969).

Of the different forms, methylmercury has the longest half-life for the whole body of about 70 days (Swedish Working Group, 1971; Piotrowski and Coleman,.1980; ATSDR, 1992; Elinder *et al.*, 1994) because of its higher affinity for proteins with sulphur-containing groups (HMSO, 1976). The half-life of inhaled elemental mercury is about 60 days (Hursh *et al.*, 1976; ATSDR, 1992) or 40–70 days (Piotrowski and Coleman, 1980). The elimination half-life of inorganic mercury is the shortest, about 40 days (ATSDR, 1992).

The capacity of urinary excretion of mercury decreases with age (Gundersen and Lie, 1980; Lie *et al.*, 1982; Skare *et al.*, 1990; Akesson *et al.*, 1991). In the most heavily exposed population groups (exceptionally high consumption of fish), a significant correlation between blood mercury and age has been demonstrated (Hansen, 1983; Grandjean *et al.*, 1992).

6.3 ENVIRONMENTAL LEVELS

Air

Measurements of mercury in air may quantify either total mercury (i.e., elemental mercury vapour plus particulate mercury) or particulate mercury. Unless otherwise stated the values in this assessment refer to total mercury concentrations. As stated previously, the relative proportions of elemental and particulate mercury in background air are approximately 95 per cent and 5 per cent, respectively. The proportion of particulate mercury increases in urban air to approximately 25 per cent (Schroeder and Jackson, 1987).

It has been suggested that concentrations of mercury vapour in the atmosphere are so low that inhalation of ambient air does not contribute significantly to human exposure (Palusova *et al.*, 1991). Mercury concentrations in air close to the ground are typically higher than those occurring a short distance (1–2 m) above the ground, indicating a flux of the metal from the soil. This is probably from soil degassing of elemental mercury formed during bacterial action on inorganic mercury (Matheson, 1979).

Rural air

There are good data from a number of rural UK sites, which indicate a mean background concentration of between 0.086 and 0.26 ng/m^3 for particulate mercury (Bertorelli, 1994) and earlier studies have found similar levels of between 0.04 and 0.2 ng/m^3 (Peirson *et al.*, 1973). Not surprisingly, these concentrations are slightly higher than the particulate mercury levels measured in air in some remote areas of Europe, for example, 0.015 ng/m^3 in northern Norway (Heindreyckx *et al.*, 1973) and 0.025 ng/m^3 in the Swiss Alps (Heindreyckx *et al.*, 1973).

Urban air

The total mercury concentration in the general atmosphere in urbanised areas is usually between 10 and 20 ng/m^3 (Williston, 1968; Lindqvist *et al.*, 1984). Particulate levels in urban areas in the UK appear to vary between <0.01 and 14 ng/m^3 (Welsh Office, 1975). Data for Paris indicate a mean concentration of 11 ng/m^3 (Bogen, 1973), whilst the average level in US urban areas is around 7 ng/m^3 (NAS, 1978). The representative value for ambient urban air used in this assessment is 20 ng/m^3. [Assuming that particulate mercury constitutes 25 per cent of the total atmospheric mercury concentration in urban areas, exposure to the total mercury concentration in urban air in the UK is between <0.04 and 56 ng/m^3].

It should be noted that for mercury, as with some organic chemicals, there may be a significant difference between indoor air levels and ambient air levels. For example, mercury-containing paint can raise the total indoor air mercury concentration by a factor of 1,000 (ATSDR, 1992). Therefore any assessment that uses the concentrations of mercury in ambient air may underestimate the actual exposure to inhaled mercury in some instances. However, there are insufficient data to determine the proportion of the population exposed to indoor air containing elevated levels of mercury as a result of the use of mercury-containing paints, or the extent of exposure on an individual basis. In this study it is assumed that the entire mercury is in the form of elemental vapour and 80 per cent of the mercury inhaled is absorbed.

Water

Levels in uncontaminated surface waters have been measured in the UK (HMSO, 1976) and the mean concentration is <0.05 μg/l. Levels measured in the River Thames were found to range from 0.045 μg/l–0.4 μg/l (Smith *et al.*, 1973). Other studies have found mean levels as low as 0.001–0.012 μg/l in surface waters (Aastrup *et al.*, 1991, Lindqvist *et al.*, 1991). However, the mercury content of surface water is not necessarily a good indicator of mercury contamination in the aquatic environment, since the solubility of many mercury compounds is low, and in polluted surface waters mercury is predominantly particle-bound (Fonds, 1971; Hasselrot, 1971; Smith *et al.*, 1971) and associated with humic matter, which means that most mercury is found in sediments (OECD, 1974). For the purpose of the calculation of the EALs, the background concentration of mercury in rivers is 0.045 μg/l.

The principal exchange of mercury between terrestrial sources and the marine environment occurs through the air-sea interface (Fitzgerald and Clarkson, 1991). However, coastal and middle distance waters may be affected by anthropogenic pollution through contaminated rivers or waste disposal to the sea (MAFF,1987). Levels in the open ocean have been measured and range between 0.0005 and 0.003 μg/l (Lindqvist *et al.*, 1984), but other studies have estimated a mean oceanic concentration of 0.05 μg/l (MARC, 1977). Levels in coastal waters may be nearly one order of magnitude greater than oceanic levels, ranging from 0.002 to 0.015 μg/l (Lindqvist *et al.*, 1984). The EALs were calculated from a background range of 0.002 to 0.015 μg/l for coastal waters.

Soil

The divalent inorganic form of mercury dominates in soil, and the mean concentration of mercury in European soils is generally <0.1 mg/kg, (Frissell *et al.*, 1973), whilst a similar level (0.08 mg/kg) has been estimated as the average abundance of mercury in the earth's crust (Taylor, 1964). Background levels in UK soils are in the range of 0.01–0.06 mg/kg (Martin, 1963) and studies in other European countries have found similar levels, for example, mean levels in Swedish soils lie in the range 0.018–0.07 mg/kg (Stock and Cucuel, 1934; Andersson, 1967; Andersson and Nilsson, 1972). Levels in Finnish soils are generally 0.02–0.2 mg/kg (Hasanen, 1973); further estimates include mean values of 0.1 mg/kg (Kloke and Schenke, 1973) and 0.095 (Wimmer and Haunold, 1973).

A study which examined mercury levels in Scottish agricultural soils found a mean level of <0.2 mg/kg, with a range of 0.01–0.89 mg/kg (DAFS, 1976). The same study found levels of 0.06–1.96 mg/kg in non-agricultural soils, with the mean being <0.4 mg/kg. Other investigations of levels in non-rural settings have found mean levels of 0.06 mg/kg

(Kick *et al.*, 1980), 0.11 mg/kg (Frank *et al.*, 1976; Brune and Ellinghaus, 1982), 0.12 mg/kg (Hoffmann *et al.*, 1982), 0.2 mg/kg (Driel and Smilde, 1982), and 0.06–0.36 mg/kg (Wiersma *et al.*, 1986).

For the purposes of this assessment it is assumed that the background soil concentration of mercury in the UK is between 0.01 and 0.06 mg/kg, and that the concentration in urban soils is 0.4 mg/kg.

6.4 MERCURY IN FOOD

(See Appendix 6.1)

The mercury present in food may result from its natural occurrence in the environment and/or from pollution arising from industrial and agricultural human activities. The estimation of mercury levels in the different foodstuffs constituting the British diet was based on the MAFF surveillance of mercury in food. However, results from other studies have been used where there are insufficient data and also for the purpose of comparison with the UK data. A number of the MAFF data recorded were below the limit of detection (LOD) and thus other data, where available, were considered.

Meat

(See Appendices 6.2 and 6.3)

A comparative study of different European countries (Falandysz, 1993) found that levels of mercury in meat were generally very low (1.2–1.9 μg/kg). Other studies have reported that meat generally shows low or very low mercury contents (Barghigiani and Ristori, 1994) and that the proportion of methylmercury in meat is negligible (HMSO, 1976). Mercury is stored mainly in the inorganic form (Grandjean *et al.*, 1992) in the liver and, to a greater extent, in the kidney (Bombosch, 1983; Vreman *et al.*, 1986; Vos *et al.*, 1987; Stevens, 1992). Therefore concentrations in tissues generally follow a decreasing trend from kidney to liver to muscle (Wright *et al.*, 1973).

In the Netherlands, the provisional legal limits for mercury content in cattle are 50 μg/kg in meat, 50 μg/kg in liver and 100 μg/kg in kidney (Vos *et al.*, 1988). The same levels are applied in USA for imported meat, except for muscle meat (40 μg/kg). These concentrations are extremely conservative compared with the tolerated concentrations in fish.

Beef

The MAFF survey found mean levels in beef to be <7 μg/kg, a result which is comparable to data from other sources. Data from a large number of studies fall within a relatively small range (1–11 μg/kg), with most results being in the range 1–7 μg/kg. The representative value selected for beef in this assessment is 7 μg/kg.

Pork

Mean levels in British pork are <7 μg/kg (MAFF, 1984). A number of other studies have measured similarly low levels in pork, 9 μg/kg (Jorhem *et al.*, 1991), 11 μg/kg (Niemi *et al.*, 1991) and 2 μg/kg (Falandysz, 1993). The representative value selected for pork was 7 μg/kg.

Lamb
A mean concentration of <6 μg/kg was recorded by MAFF, and other studies have found levels of between 1 and 5 μg/kg in lamb meat. The representative value used in this assessment for lamb is 5 μg/kg.

Meat products
There are few data on mercury in meat products, but concentrations measured in bacon, ham and salami ranged from 2 to 2.5 μg/kg (Barghigiani and Ristori, 1994). These concentrations are similar to those found in fresh meat. The representative value used in this assessment is 2.5 μg/kg.

Liver and kidneys
(see Appendix 6.3)

There are numerous studies which provide data on levels of mercury in liver and kidneys of livestock. The representative values used were from Jorhem *et al.* (1991) and were 16 μg/kg and 19 μg/kg for liver and kidney, respectively. These values were selected as this particular study probably represents the most comprehensive and precise indication of the current levels of mercury in these foodstuffs.

Poultry
Levels in chicken consumed in the UK have been measured by MAFF, which found a mean level of <5 μg/kg, which is consistent with the concentrations measured by other studies of <2 μg/kg (Barghigiani and Ristori, 1994). A study which investigated mercury levels in a number of poultry products (Falandysz, 1991) found slightly higher concentrations in the muscle and offal of turkey and ducks than in chicken and geese. Some additional input to the food chain comes about from the use of fish flour, which may contain quite substantial quantities of mercury, in poultry feeds (MAFF, 1987) and from grains which may have been treated with the fungicide alkylmercury (Smart and Lloyd, 1963; WHO, 1972; Soares *et al.*, 1973; March *et al.*, 1983; MAFF, 1987; Cuadrado *et al.*, 1995). Indeed, levels of mercury in some game birds have risen to toxic levels due to the agricultural use of alkylmercury, and relatively high concentrations have been measured in pigeons in the UK, 10 μg/kg (MAFF, 1987). For the purpose of this assessment the representative value for mercury in poultry is 5 μg/kg.

Eggs
The levels of mercury in eggs do not appear to be significantly different from those of poultry meat (Palusova *et al.*, 1991; Barghigiani and Ristori, 1994). The mean mercury concentration found in eggs by MAFF (1985) was <4 μg/kg, and other studies have found mean levels of 8 μg/kg (Palusova *et al.*, 1991) and <2 μg/kg (Barghigiani and Ristori, 1994). The Netherlands have introduced legislation which limits the concentration of mercury in eggs for human consumption to less than 30 μg/kg. The egg albumen appears to contain higher total, and organic, mercury concentrations than the yolk (MAFF, 1987; Jeng and Yang, 1995), almost certainly due to the binding of mercury to the ovalbumin protein (Sell *et al.*, 1974). The representative value used in this assessment was 4 μg/kg.

Milk

Data on the mercury content in milk and milk products are fairly comprehensive, but the values reported are subject to considerable variations (Koops and Westerbeek, 1984). MAFF (1982) recorded a mean concentration in whole milk of <1 μg/kg, and other studies have produced similar results of 1.2 μg/kg (De Ruig, 1977), 0.1–0.5 μg/kg (Fotdzinska and Zmyewska, 1979), 0.5–2.3 μg/kg (Vreman *et al.*, 1986) and <2 μg/kg (Barghigiani and Ristori, 1994). Levels as low as 0.07 μg/kg have been measured (Koops and Westerbeek, 1984), whereas one survey has found higher mean values of approximately 5 μg/kg (Heeschen, 1982). The Netherlands' authorities have suggested an allowable mercury level in milk of 10 μg/kg (Landbouwadviescammissie, 1980). The levels determined by MAFF and most other authors were well below this provisional level. The representative value for whole milk used in this assessment was 1 μg/kg.

Skimmed milk

The levels in skimmed milk have been measured by two investigations, and are 0.1 μg/kg (Kaiser *et al.*, 1978) and 0.5–1.0 μg/kg (Deharis *et al.*, 1982). The representative value for skimmed milk used in this assessment was 1 μg/kg.

Other dairy products

Data for milk products are available only for cheese, so it has been assumed that the remaining products (butter, ice cream, yoghurt and cream) have the same mercury concentrations as whole milk. The level of mercury in cheese might be estimated based on the actual content of this element in milk and assuming a uniform distribution of this metal over the proteins, which are converted from milk to cheese by a factor of 10 (Koops and Westerbeek, 1984). Therefore, from the MAFF data it would appear that the mean level of mercury in cheese is probably <10 μg/kg. Actual measurements of mercury in cheese suggest lower levels, with mean concentrations of 1.0–1.6 μg/kg (Koops and Westerbeek, 1984) and <2.0 μg/kg (Barghigiani and Ristori, 1994). The suggested allowable mercury concentration in cheese in the Netherlands is 30 μg/kg (Landbouwadviescammissie, 1980), relatively high compared with current measured levels. The representative value used for cheese in this assessment is 2 μg/kg.

Oils

The only available data were from MAFF for oils and dairy products as a food group (1982) and fats (1979). The mean mercury concentration was the same for both groups (<1 μg/kg) and therefore the representative value used was 1 μg/kg.

Vegetables

(See Appendix 6.4)

Very little mercury is translocated from the soil into plants. However, mercury may accumulate to a certain degree in the aerial parts of plants following atmospheric deposition, and can, to a small extent, be transported to the rest of the plant (Rauter, 1976). The amount of methylmercury found in plant produce is very small or nil (WHO, 1972). Some species of mushrooms are good accumulators of mercury present in soil (Schelenz and Diehl, 1973; Stijve and Besson, 1976; MAFF, 1987). Among leafy vegetables the highest concentrations have been found in herbs, such as sage (De Temmerman *et al.*, 1986; Barghigiani and Ristori, 1994), ranging from 4–30 μg/kg (partly due to its perennial life cycle). The level reported by

Barghigiani and Ristori (1994) for sage was much lower (<0.002 mg/kg) and the mean mercury concentration in rosemary was 0.0468 mg/kg wet weight. However, the consumption of herbs is very low, and so represents a small exposure.

Cabbage and lettuce

Both cabbage and lettuce are capable of accumulating mercury because of the large leafy area exposed to deposition of mercury from the atmosphere (Barghigiani and Ristori, 1994). Levels in cabbage are around 2 μg/kg, with mean values of around 1.9 μg/kg (De Temmerman *et al.*, 1986) and < 2 μg/kg (Lenka *et al.*, 1992). In lettuce the measured levels are 1.4 μg/kg (Aichberger, 1976; Barudi and Bielig, 1980), 1 μg/kg (Varo *et al.*, 1980), 3 μg/kg (De Temmerman *et al.*, 1986), 2 μg/kg (Wiersma *et al.*, 1986) and 1.8 μg/kg (Barghigiani and Ristori, 1994). The maximum acceptable level of mercury in lettuce in the Netherlands is 30 μg/kg (Klitsie, 1983; Staatscourant, 1985), and all measurements of current levels in lettuce are considerably lower than this concentration. For the purpose of this assessment the representative values for cabbage and lettuce are 2 μg/kg and 3 μg/kg, respectively.

Carrots

The mean concentration of mercury in carrots varies between <1.0 and 2.8 μg/kg. A study of plant storage organs, such as carrots, found that they are unlikely to accumulate mercury (De Temmerman *et al.*, 1986). Recorded concentrations include means of 1 μg/kg (Aichberger, 1976), 2 μg/kg (Wiersma *et al.*, 1986), 2.6 μg/kg (Barghigiani and Ristori, 1994) and 2.8 μg/kg (De Temmerman *et al.*, 1986). The maximum acceptable level of mercury in carrots in the Netherlands is 30 μg/kg (Klitsie, 1983; Ned Staatscourant, 1985). The representative mean concentration in carrots was considered to be 2.8 μg/kg for the purposes of this assessment.

Potatoes

Potatoes and onions contain less mercury than other root plants, such as carrots, despite the fact that they are all storage organs (De Temmerman *et al.*, 1986). MAFF measured levels of 1–4 μg/kg in potatoes, with a mean concentration of 1 μg/kg, and other studies include measurements of 4 μg/kg (Bundesgesundheitsanet, 1975), 2 μg/kg (Varo *et al.*, 1980), 0.9 μg/kg (De Temmerman *et al.*, 1986), 3 μg/kg (Wiersma *et al.*, 1986) and 2.2 μg/kg (Barghigiani and Ristori, 1994). The maximum acceptable level in potatoes in the Netherlands of 20 μg/kg (Klitsie, 1983; Ned Staatscourant, 1985) is well above the levels found in the literature. As reliable UK data indicate levels in potatoes to be <1 μg/kg, the representative value for potatoes was 1 μg/kg.

Onions

Low levels of mercury have been detected in onions by two studies, with concentrations of <2 μg/kg (Barghigiani and Ristori, 1994) and 0.5 μg/kg (De Temmerman *et al.*, 1986). The representative value used in this assessment was 0.5 μg/kg.

Tomatoes

Among vegetables, tomatoes accumulate the lowest levels of mercury, (Barghigiani and Ristori, 1994). Lenka *et al.* (1992) reported that mercury bioconcentrates in the leafy branches of tomatoes but not in the fruit. Unfortunately, the only data available for UK (Cross *et al.*, 1978) are from a study with a relatively insensitive analytical technique, resulting in a

probable overestimate of the true concentration due to a higher limit of detection than many of the subsequent studies. Other studies have found levels of 0.6 μg/kg (Aichberger, 1976), 2 μg/kg (Barudi and Bielig, 1980), 1.7 μg/kg (De Temmerman *et al.*, 1986), 1.3 μg/kg (Wiersma *et al.*, 1986) and 1.3 μg/kg (Barghigiani and Ristori, 1994). The Dutch government have proposed a maximum acceptable level of mercury in tomatoes of 30 μg/kg (Klitsie, 1983; Ned Staatscourant, 1985). The representative concentration selected for this assessment is 2 μg/kg.

Peas

The only available data for peas are from De Temmerman *et al.* (1994) who measured low levels of 0.75 μg/kg. This was the representative value used in the assessment.

Cereals

Mercury levels in cereal grains are generally lower than the rest of the plant (Johnels *et al.*, 1979; Skortsova *et al.*, 1984). Concentrations in British bread are in the range <0.5 to 1 μg/kg with a mean of <0.6 μg/kg (MAFF, 1987). Mercury levels in other cereal products are slightly higher, ranging from <1 to 17 μg/kg (MAFF, 1987) with a mean of <2 μg/kg. These results are consistent with other studies which have found levels of 2 μg/kg (Ocker and Hack, 1975), <0.4 (Varo *et al.*, 1980), 8 μg/kg (Tkachuk and Kuzina, 1983), 5–8 μg/kg (Wiersma *et al.*, 1986), and 0.4–4.5 μg/kg (Cuadrado *et al.*, 1985). The representative concentrations for the bread and other cereals groups are 0.6 μg/kg and 2 μg/kg, respectively.

Fruit and fruit products

Very little mercury is found in fruit and seeds; levels are lower than in vegetables, particularly leafy ones (De Temmerman *et al.*, 1986; Palusova *et al.*, 1991). MAFF has recorded levels of <1 μg/kg in fresh fruit, and other studies have found a similarly low range of 1–2 μg/kg (Barudi and Bielig, 1980; Wiersma *et al.*, 1980) and 1.4–5.6 μg/kg (Barghigiani and Ristori, 1994). The representative value used in this assessment is 1 μg/kg. The levels of mercury determined by MAFF (1987) in apple juices were particularly high compared with the fresh fruit. This was due to the high LOD of the method of analysis used. Therefore, it has been assumed that the mercury levels in fruit products are the same as for fresh fruit (1 μg/kg).

Beverages

The beverages group includes dried coffee, soft drinks and tea (MAFF, 1987). The concentrations of mercury in this food group are very low, 0.5 μg/kg (MAFF, 1982).

Fish

MAFF (1987) have found relatively high levels of mercury in fish from UK coastal waters that are influenced by industrial and municipal discharges (Thames and Mersey Estuaries). Regular monitoring has shown a general reduction in mercury concentrations in all species of fish caught in UK waters, especially in those fished in industrialised areas (Murray and Portman, 1984; MAFF, 1987). Shellfish showed very low levels of mercury (with the exception of whelks, which are of a minor importance in terms of exposure to the general population) (MAFF, 1987). A survey of imported fish and shellfish products found mercury levels lower than those found in local products (MAFF, 1987).

The representative concentrations used in this assessment are all MAFF data from **Total Diet Studies,** with the values being 140 μg/kg for shellfish (prawns), 40 μg/kg for freshwater fish (Trout), 120 μg/kg for canned fish (Tuna) and 220 μg/kg for sea fish (Cod). The evaluation of human exposure to mercury via fish consumption has been based on the assumption that the mercury in fish is almost entirely present in the methyl form. [Authors, such as Huckabee *et al.* (1979) and Galal-Gorchev (1993), have used the same assumption.]

Drinking water

A survey by the Department of Agriculture and Fisheries for Scotland (DAFS, 1973) found a range of between <0.01 and <0.03 μg/l in drinking water in Scotland. Several other studies have found similar levels in drinking water, for example, mean levels of between 0.01 and 0.05 μg/l (Stock and Cucuel, 1934), and a range of 0 to 0.3 μg/l (Palusova *et al.*, 1991). The representative value for drinking water selected for this assessment was 0.03 μg/l as it represents the highest mean concentration measured in UK drinking water.

Breast milk

MAFF found levels to be less than 3 μg/kg in all samples, and less than 1 μg/kg in 66 per cent of samples in a comprehensive survey of mercury levels in human milk in the UK. Other studies have found mean levels of 2 μg/kg (Schramel *et al.*, 1988). It is assumed that most of the mercury present in breast milk is in the inorganic form (Skerfving, 1988; Galal-Gorchev, 1993). Skerfving (1974) reported that 20 per cent of the total mercury in breast milk is methylmercury and 80 per cent is inorganic. Bakir *et al.* (1973), Inskip and Piotrowski (1984) reported that the concentrations of mercury in breast milk are approximately 8 per cent of the maternal blood concentrations, **whilst the Australian Department of Primary Industry reported levels in breast milk to be 3-5% of levels in maternal blood.** The representative concentration used for human milk in this assessment is 2 μg/kg (or 2 ng/ml).

6.5 INTAKE AND EXPOSURE – ESTIMATES FOR DIFFERENT AGE GROUPS

(See Appendices 6.5–6.8).

The data on mercury levels in selected representative foodstuffs, drinking water, urban soil and urban air have been used together with information on the average consumption rates of different foods in the UK, the consumption rate for drinking water and soil, and the average breathing rates to calculate the annual intake of mercury for the average individual in the general population in five age groups. The annual intake is obtained by multiplying the amount consumed, or breathed, by the average arsenic concentration in the media. It has been assumed that:

(i) All mercury in air is in the elemental form.

(ii) All mercury in soil, terrestrials foods and drinking water is in the inorganic form.

(iii) All mercury in fish is in the methyl form.

Table 6.1 Intake of total and methyl mercury for different age groups (μg/kg bw/day)

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Total	0.30	0.06	0.07	0.07	0.08	0.08
Methyl	0.06	0.03	0.04	0.04	0.05	0.05

Table 6.2 Percentage contribution to total mercury intake from different sources

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Diet	99	88	83	88	91	90
Soil		0	3	1	0	0
Water		1	1	1	1	1
Air	1	11	14	11	8	8

As can be seen from Table 6.2, the diet makes up the vast majority of the intake (90 per cent) and breathing air (8 per cent) is a significant source of mercury intake. Soil and water represent negligible sources of intake.

Table 6.3 Percentage contributions to total mercury intake for the three chemical forms

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Inorganic	99	35	35	35	36	36
Organic	0	54	51	54	56	55
Elemental	1	11	14	11	8	9

Most mercury intake is in the organic form (55 per cent), although a significant contribution comes from the intake of inorganic mercury. This is because the methyl form is present in high levels in seafood, comprising the majority of the intake from the diet, but a significant amount originates from the terrestrial diet.

Exposure to the body is calculated in terms of the exposure to the blood, and is calculated by multiplying the intakes by the absorption factors for the given route of exposure. As mentioned previously it has been assumed that all the mercury present in terrestrial foods and drinking water is present in the form of inorganic mercury, that the inhaled mercury is in the form of elemental mercury vapour, and that the mercury present in aquatic foods is in the organic (methylmercury) form. The respective absorption factors taken from Langley (1993) are well established values used in the US EPA risk assessment process, and are 0.15 (inorganic mercury in the GI tract), 0.75 (elemental mercury in the lungs) and 0.95 (methylmercury in the GI tract).

Table 6.4 Exposure to total mercury (µg/kg bw/day)

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Total (µg/kg/bw)	0.04	0.04	0.04	0.05	0.05	0.05

The significance of the intakes of mercury from the diet (in the methyl form) and the elemental form (from air) are further increased by the efficient absorption of these forms to the blood. The chief sources of exposure throughout the lifetime are the diet (90 per cent) and the inhalation of air (10 per cent). Drinking water (0.3 per cent) and direct ingestion of soil (0.06 per cent) make negligible contributions to lifetime exposure.

Table 6.5 Percentage contribution to mercury intake from different sources

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Diet	94	86	82	92	90	90
Soil		0	0	0	0	0
Water		0	0	1	0	0
Air	6	14	17	7	9	10

Table 6.6 Relative contributions (per cent) to total mercury exposure of the three chemical forms

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT	LIFETIME
Inorganic	94	5	6	5	8	8
Organic	0	81	77	81	83	82
Elemental	6	14	17	14	9	10

As can be seen from Table 6.6, methylmercury is the major source of lifetime exposure to mercury (82 per cent), illustrating the importance to human exposure of bioaccumulation of mercury in the aquatic food chain.

Comparison with other estimates of intake and exposure

As shown in Table 6.1, the total intake for the adult population in the UK is 0.08 μg/kg bw/day, or approximately 5.6 μg/day for a 70 kg adult, of which approximately 5 μg/day is from the diet. Intakes of mercury in the national diet are 2–3 μg/day in the UK (MAFF, 1984) and the dietary intake calculated in this study is slightly higher due to the use of conservative estimates of mercury levels in the various foodstuffs. According to a previous assessment carried out by the British Ministry of Agriculture (1971), the daily intake of mercury is, in general, lower than 10 μg (OECD, 1974). The average dietary intake of adults in the UK was estimated by Sherlock *et al.* (1982) to be 3 μg/day, but the intake in coastal communities receiving discharges high in mercury could reach 14.5 μg/day. In Sweden and Japan, the mean daily intake for populations consuming greater than average amounts of fish were estimated to be 9 μg/day and 50 μg/day, respectively. The contribution of fish and shellfish to total mercury intake (55 per cent) calculated in this study is higher than that calculated by some other studies, for example, 35 per cent (Galal-Gorchev, 1993).

The intake during breast-feeding is several times higher (per kg body weight) than during the rest of the lifetime when the infant may receive a proportionate intake greater than that of an individual eating contaminated seafood. However, as the mercury is predominantly in the inorganic form in breast milk, the absorption of mercury by the breast-feeding infant will be far less efficient than the absorption of methylmercury from contaminated fish. Estimates of the absorption of mercury from breast milk give an absorption factor of 14 per cent. However, as the foetus and infant are more sensitive to the toxic effects of mercury than adults (Smart, 1961; Bland and Egan, 1963; GESAMP, 1986; WHO, 1990, 1991; Gerhardsson *et al.*, 1994), exposure during this breast-feeding period and during pregnancy may be very important. Consequently, pregnant women are advised to avoid the consumption of fish rich in mercury, particularly during early stages of the foetus development (Johansson *et al.*, 1991; Lindquist *et al.*, 1991; Svensson *et al.*, 1992).

The excretion of methylmercury in new-born babies is slower than in adults (Clarkson *et al.*, 1985), and the central nervous system of the infant is more susceptible to the effects of mercury than adults (Marsh *et al.*, 1980; Clarkson *et al.*, 1985; Kjellstrom *et al.*, 1986). Several studies have found mercury concentrations in the blood of new-born babies to be 50–80 per cent higher than in their mothers' blood, and the blood of members of the general population (Pitkin *et al.*, 1976; Fujita and Takabatake, 1977; Reynolds, 1979; Skerfving, 1988).

The general population is primarily exposed to mercury through the diet, although mercury present in air, water and soil might contribute to human exposure (WHO, 1990). The exposure of the general population to mercury is more affected by frequent fish consumption than by other sources, such as air, dental fillings and other food stuffs (Airey, 1984). Many studies have found fish and shellfish to be the main source of both total and methylmercury (Cuadrado *et al.*, 1995; Swedish Expert Commission, 1971; WHO, 1972; Norberg *et al.*, 1985; Clarkson *et al.*, 1988; MAFF, 1987; WHO, 1990; Fitzgerald and Clarkson, 1991; Lindqvist *et al.*, 1991; ATSDR, 1992; Elinder *et al.*, 1994; Gerhardsson *et al.*, 1994; Svensson *et al.*, 1995). The results from this study agree with others in terms of the contribution to exposure (approximately 80 per cent) made by fish and shellfish (Cuadrado *et al.*, 1995; Airey, 1984).

Other source of exposure – Amalgam teeth fillings

The mercury content in dental amalgam is approximately 50 per cent by weight (Espevik and Mjor, 1982; ATSDR, 1992) and several studies have demonstrated that fillings release some mercury vapour, causing exposure to elemental mercury vapour (Gay *et al.*, 1979; Svare *et al.*, 1981; Abraham *et al.*, 1984; Brune *et al.*, 1985; Vimy and Lorscheider, 1985; Clarkson *et al.*, 1988; Skare *et al.*, 1990; Akesson *et al.*, 1991; WHO, 1991; Elinder *et al.*, 1994; Halbach, 1994). The action of chewing increases the rate of release (ATSDR, 1992).

A relationship between mercury levels in urine and amalgam have been reported in some studies (Langworth *et al.*, 1988; Akesson *et al.*, 1991), but others (Kroncke *et al.*, 1991) could not find a similar relationship. Svensson *et al.* (1992) found a significant correlation between the number of teeth with amalgam fillings and the concentration of mercury in plasma and urine, and Drasch *et al.* (1989) reported a correlation with mercury concentration in kidney from the general population. Other studies have found correlations between the mercury level in the brain tissue of cadavers from the general population with the number of amalgam fillings (Eggleston and Nylander, 1987; Schiele, 1988; Weiner and Nylander, 1993).

An expert panel in the US recently concluded that amalgam fillings pose no significant health risk (ATSDR, 1992). However, earlier studies have reported cases of oral and skin manifestations and allergic enteritis caused by mercury in amalgam dental fillings (Spreng, 1963; Feuerman, 1975). Despite these instances, the number of cases is very small compared with the number of people with amalgam dental fillings (Berlin, 1986).

It was concluded that to make accurate quantitative estimations of the mercury release from amalgam, and the resulting uptake of mercury by the body is difficult (Enwonwu, 1987; Friberg and Nylander, 1987; Langan *et al.*, 1987; Olsson and Bergman, 1987; Clarkson *et al.*, 1988).

6.6 BODY BURDEN AND BLOOD CONCENTRATIONS – ESTIMATES FOR INDIVIDUALS OF DIFFERENT AGES

The body burden has been estimated using the standard relationship between body concentration, retention time in the body, body mass and intake, summarised below.

$C = T/M \times F$ Where C = Concentration in the body (μg/kg)
T = Retention time in the body (days)
M = Body mass (kg)
F = Exposure (μg/day)

The retention times of organic, elemental and inorganic forms of mercury in the body vary and, therefore, the concentrations in the body are calculated separately and then summed to give the total mercury body burden. The half-lives selected from the literature are 40 days (inorganic), 70 days (organic) and 60 days (elemental) and these correspond to retention times of 60, 100 and 87 days, respectively (Bennett, 1981).

Example
The average body mass for adults is 70 kg (ICRP, 1975) and the annual exposures are 108 μg (inorganic), 128 μg (elemental) and 1,143 μg (organic), corresponding to daily exposures of 0.30 μg/day, 0.35 μg/day and 3.13 μg/day, respectively. From the above equation therefore it is possible to calculate each chemical species concentration in the body as a whole, these being 0.25 μg/kg (inorganic), 0.44 μg/kg (elemental) and 4.47 μg/kg (organic), which gives a whole body concentration of total mercury of 5.17 μg/kg and a body burden of 362 μg.

The data used in the calculation of body burdens are summarised in Table 6.7

Table 6.7 Data used for the calculation of body burden and concentration

	0–3 MTHS	3 MTHS–2 YRS	2–7 YRS	7–14 YRS	ADULT
Exposure (μg/day)					
Inorganic	0.21	0.03	0.06	0.10	0.30
Organic		0.40	0.79	1.58	3.13
Elemental	0.01	0.07	0.18	0.26	0.35
Total	0.01	0.49	1.02	1.95	3.78
Body content – total (μg/kg)	**2.75**	**3.90**	**4.07**	**4.35**	**5.17**
Body Burden (μg)	**14**	**47**	**98**	**187**	**362**

Comparison with measured body burdens and body concentrations
The blood is a good indicator of general mercury exposure (Elinder *et al.*, 1994) and the mercury concentration in blood can reflect recent exposure as well as the body burden derived from earlier exposure. Experimental studies in man have shown that under steady-state conditions, the mercury level in blood is linearly correlated to the intake of methylmercury (WHO, 1972; Grandjean *et al.*, 1994) and to the level of methylmercury in the brain (Berlin, 1986).

The blood concentration can be calculated if it is assumed that the average 70 kg adult contains 4.55 litres of blood (ICRP, 1975) and that 5–10 per cent of the mercury body burden is present in the blood (WHO, 1990).

A body burden of 362 μg in the average adult equates to a mean blood concentration of 4.0–8.0 μg/kg, consistent with reports by WHO (1990) which state that the mean concentration of total mercury in whole blood (in the absence of consumption of fish with high methylmercury contents) of a range of 5–10 μg/l.

The WHO has devised a similar formula to calculate the blood concentration from daily intake (not exposure) which can be used to compare the current estimate of exposure and the resultant body burden. The rate of excretion of mercury is directly proportional to the simultaneous body burden and can be expressed by a single biological elimination half-time WHO (1976). The concept of a single-compartment model (elimination of mercury from the body occurs in one phase only) has been used for the modelling of continuous exposure. The model is based on the estimates of the biological half-life in blood (Inskip and Piotrwski, 1985). In the WHO model it is assumed that a normal 70 kg adult has a blood mass of approximately 5 kg and that 5 per cent of the body burden is found in the blood (Inskip and Piotrwski, 1985). In steady-state, the concentration of mercury in whole blood in adults, in μg/l is approximately numerically equal to the daily intake in μg/day (WHO, 1976). As the current intake for adults has been estimated to be 5.6 μg/day (Table 6.1), this would indicate that the average adult in the UK would have a blood concentration of 5.6 μg/l, which again is in the range proposed by the WHO for non-exposed adults.

6.7 TOXICOLOGICAL ASSESSMENT

Toxic effects of methylmercury in humans are mainly associated with neurological disturbances, as it affects the central nervous system by causing irreversible cell damage and destruction (Amin-Zaki *et al.*, 1979; Fitzgerald and Clarkson, 1991; ATSDR, 1992; Jonnalagadda and Prasada Rao, 1993). Methylmercury exposure can produce damage in the liver and kidneys, but only after being biodegraded to inorganic mercury (MAFF, 1971; Piotrowski and Coleman, 1980; Berlin, 1986; ATSDR, 1992; Jonnalagadda and Prasada Rao, 1993). Direct exposure to inorganic mercury causes kidney damage (Piotrowski and Coleman, 1980; Jonnalagadda and Prasada Rao, 1993).

Even low concentrations of mercury may be considered potentially harmful, and there is uncertainty regarding the extent to which chronic exposure to low levels of mercury may lead to behavioural or intellectual impairment. It has been firmly established that the foetus and infant are more sensitive than the adult to mercury exposure.

For example, the foetus is more susceptible to brain damage from methylmercury as it is believed to inhibit cell division and neuronal migration (Choi *et al.*, 1978; Fitzgerald and Clarkson, 1991; ATSDR, 1992). Consequently, pregnant women are advised to avoid the consumption of fish rich in mercury, particularly during early stages of pregnancy (Johansson *et al.*, 1991; Lindquist *et al.*, 1991; Svensson *et al.*, 1992). In addition, a higher frequency of severe symptoms and damage is seen among persons younger than 5 years (Australian Department of Primary Industry, 1979).

Studies from New Zealand have indicated that adverse effects in infants can occur at maternal blood levels of about 40–80 µg/l (Elinder *et al.*, 1994), and the Canadian federal and provincial authorities have prescribed a safe level of mercury in blood of 50 µg/kg (D'Itri and D'Itri, 1970). Therefore the current levels are only about 5 times lower than those associated with human health effects at this critical stage of development.

Clinically observed effects occur at body concentrations of 500 to 800 µg/kg (WHO, 1976); therefore using this measure of exposure and the results of this assessment, current exposure to mercury for the general population appears to be well below the levels of exposure associated with adverse effects.

The FAO has jointly set a provisional tolerable daily intake (TDI) of 300 µg for mercury, of which not more than 200 µg should be organic mercury (equivalent to daily intakes for adults of 42 µg/day and 28 µg/day respectively). The lifetime daily intake calculated in this study for total mercury is 5.13 µg/day, and for methylmercury is 2.84 µg/day.

6.8 DERIVATION OF ENVIRONMENT ASSESSMENT LEVELS (EALS)

The critical value used in this assessment is the PTWI put forward by the FAO/WHO of 300 µg (total mercury) and 200 µg (methylmercury). This is equivalent to a daily intake of 42 µg for total mercury and 28 µg for methylmercury, and these correspond to TDIs of 0.6 µg/kg bw/day and 0.4 µg/kg bw/day for an average adult individual (mean body weight of 70 kg).

The current intake of total mercury and methylmercury (µg/kg bw/day) can be found in Table 6.1.

The TDIs are equivalent to maximum allowable lifetime exposure and are used to derive a maximum allowable intake for mercury, which may then be equated to maximum allowable environmental levels. The TDI for total mercury is approximately 7.5 times higher than current lifetime daily intake, and for methylmercury the margin is approximately 8. Environmental Assessment Levels were calculated for the four media from the following background environmental levels. Both particulate and total mercury concentrations are used for the EALs for air; total mercury concentration in air has been estimated from Bertorelli (1994), assuming that particulate mercury constitutes about 5 per cent of total atmospheric mercury concentrations in background air.

Current Background Levels

	RANGE	MID-RANGE
Air (total)	1.7–5.2 ng/m^3	3.5
Air (particulate)	0.086–0.26 ng/m^3	0.17
Soil	0.01–0.06 mg/kg	0.04
Fresh water	0.045 µg/l	
Coastal sea water	0.002–0.015 µg/l	0.01

EALs

	EAL RANGE	MID-RANGE
Air (total)	13–39 ng/m^3	26
Air (particulate)	0.65–1.95 ng/m^3	1.28
Soil	0.08–0.45 mg/kg	0.3
Fresh water	0.34 µg/l	
Coastal sea water	0.015–0.11 µg/l	0.08

However, as mentioned earlier there is a smaller margin of safety between the current blood levels in the general population, and the levels in maternal blood which may give rise to adverse effects in the infant. The current upper range of the blood concentration in adults in the UK has been estimated to be 7.9 µg/l, and the lower range of concentrations in maternal blood which have been indicated to cause adverse effects in the infant is 40 µg/l. This would give an acceptable safety margin of only 5, rather than the value of 7.5 used above. Using this lower value, the following EALs could be derived.

	EAL RANGE	MID-RANGE
Air (total)	9–26 ng/m^3	17
Air (particulate)	0.43–1.30 ng/m^3	0.85
Soil	0.05–0.30 mg/kg	0.20
Fresh water	0.23 µg/l	
Coastal sea water	0.01–0.10 µg/l	0.05

6.9 ENVIRONMENTAL STANDARDS FOR MERCURY

The current EALs for mercury in air and the EQSs for mercury in water are:

Air	1,000 ng/m^3 (inorganic)
	100 ng/m^3 (mercury as alkyl mercury)
Fresh water	1 µg/l (total insoluble and soluble)
Coastal water	0.3 µg/l (dissolved)

The current EAL for mercury in air (1,000 ng/m^3) is considerably higher (approximately 60 times) than the proposed EAL derived using exposure commitment. Given that the estimates of exposure and the TDI are well established, it may be appropriate to review the current EAL **with respect to** long-term protection to human health. The current data used for the estimation of background levels, and therefore the EALs, are given as particulate mercury, and it has been assumed that this form constitutes approximately 5 per cent of the total air mercury concentration. This may well be a conservative estimate, but nevertheless it appears that it may be appropriate to investigate the current EAL.

The difference between the current EQS and the proposed EAL is less (a factor of approximately 3) and, given the conservative bias given to this assessment, it may be sufficiently stringent to protect human health. The upper tolerable limit for mercury in drinking water is also 1 µg/l (WHO, 1976). Certainly the route of exposure from fresh water

via the consumption of freshwater fish and drinking water is minor in comparison with the exposure of marine origin. **This route of exposure is still of considerable importance with respect to the protection of human health, and therefore should be monitored closely.**

The difference between the proposed EAL for coastal water and the current EQS is similar to that for fresh water, but given the importance of the ingestion of marine foods to total human exposure, this value may deserve further scrutiny. Again the conservative bias of the assessment may mean that the current EQS is sufficiently stringent to protect human health.

Nriagu (1981) proposed a threshold value for mercury concentration in soil of up to 2 mg/kg. The EAL proposed for soil is approximately one order of magnitude lower than this proposed threshold value **and, dependent upon the results from a more complete data set in the future becoming available, this standard may require further discussion and investigation.**

6.10 SUMMARY

1. Data on levels of mercury in environmental media are generally good, and the selected background concentrations can be considered to be representative of the background levels in the UK. However, the levels in UK air are measured as particulate mercury, and given the importance of the vapour phase in the atmosphere, the background levels of total mercury in air may be open to some debate.

2. There are good, consistent data from a number of sources on the levels of mercury in various foods, and the estimates of intake and exposure calculated compare well with previous studies.

3. The major source of exposure to mercury for the UK general population is from the consumption of seafood (80 per cent).

4. The daily intake of 0.08 μg/kg bw/day for the lifetime of the average individual is probably representative of the general UK population.

5. Well established measures of tolerable exposure to mercury exist in the form of Tolerable Weekly Intakes of 300 μg (total mercury) and 200 μg (methylmercury). The levels of mercury in maternal blood are also an important measure of acceptable exposure, given the significant toxicological importance of mercury exposure to the foetus.

6. Maximum acceptable environmental levels are between 5 and 7.5 times higher than current levels, and EALs have been calculated on this basis.

7. The EAL calculated for air is significantly lower than that currently in force, and there is a smaller difference between the proposed EALs for fresh water and coastal water and the respective EQSs.

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7. EXPOSURE COMMITMENT ASSESSMENT FOR POLYCYCLIC AROMATIC HYDROCARBONS

7.1 INTRODUCTION

At present it is not appropriate to calculate Environmental Assessment Levels (EALs) for polycyclic aromatic hydrocarbons (PAHs) using Exposure Commitment for a number of reasons. Firstly there are a large number of PAH compounds present in the environment, but most studies have concentrated on a limited number, therefore it is not possible to establish daily exposure to ∑PAHs. This problem may be overcome in the future if it becomes possible to relate concentrations of a particular compound, or group of compounds to ∑PAH concentrations in foodstuffs and environmental media. Secondly there is still considerable uncertainty regarding the pathways of PAHs in the environment, and their uptake to the human body. Thirdly, there is no Tolerable Daily Intake (TDI) or critical concentration in a target organ, to which current intakes or estimated tissue concentrations of ∑PAHs or individual compounds (if available) could be compared. There are, however, good data for several individual PAH compounds in environmental media and food, which could be used to estimate daily intake of these individual compounds.

Polycyclic aromatic hydrocarbons are ubiquitous environmental contaminants derived mainly from anthropogenic sources (specifically the burning of fossil fuels) and long-range atmospheric processes are the key to dispersing PAHs through the environment (Jones *et al.*, 1989). They are listed by the US Environmental Protection Agency (US EPA) and the EC as priority pollutants, and some workers have suggested that individual PAH compounds can be used as indicators of particular combustion processes or fuels (e.g., Coronene emissions are relatively high from vehicle emissions, while benzo[a]pyrene (B[a]P) and benzo[e]pyrene (B[e]P) are often associated with coal combustion).

Based on a structure of fused benzene rings they form a large group of organic chemicals that are highly lipophilic and therefore likely to accumulate in animal tissues. Several have been found to be carcinogenic and/or mutagenic in animal studies, while exposure to these compounds has been associated with various types of human cancer. The metabolites of some of the larger PAHs – B[a]P is the prime example – are carcinogenic, although laboratory experiments have not generally been matched to realistic environmental concentrations and pathways (Law and Biscaya, 1994). They are known also to accumulate in vegetation.

A major problem, caused by the large number of PAH compounds and by limitations of the analytical methods applied to studies of human intake and exposure, is that different studies have investigated different compounds, but often reported the results as ∑PAHs. Therefore comparisons of ∑PAH intakes may not be very meaningful. However, for those PAHs for which comparisons are possible, good agreement exists in the mean daily intake figures obtained. In most cases B[a]P has been determined as a leading substance for all PAHs (Maier, 1991).

7.2 ENVIRONMENTAL TRANSPORT AND BEHAVIOUR

Polycyclic aromatic hydrocarbons comprise a vast number of compounds (a total of 333 PAHs has been suggested, taking account of chemical structure permutations) based on benzene units linked together in conjugated ring systems. They do not generally occur as single substances but in combination, and the profile of compounds found can provide information on the source (Law and Biscaya, 1994). The ubiquity of PAHs results from long-range atmospheric transport (McVeety and Hites, 1988).

Studies that have investigated the trends in atmospheric deposition in the UK have found that PAH fluxes began to increase from stable background levels around the turn of the century, and rose to a maximum between 1950 and 1970. The deposition flux has decreased steadily since then, and is now approximately half the maximum (Sanders *et al.*, 1993). Over 53,000 tonnes of ∑PAHs (sum of 12 individual compounds) are estimated to reside in the UK environment, with soil being the major reservoir (Wild and Jones, 1995). This burden is still increasing at the present time, principally through retention by soils, but even studies which have looked at concentrations of a relatively large number of PAHs (Wild and Jones, 1995) acknowledge that there may be many other PAH compounds emitted to the UK environment, where they may reside as contaminants.

Individual PAHs differ substantially in their physical and chemical properties, with the transfer and turnover of lower molecular weight PAHs more rapid than the other group members; there is evidence, for example, that levels of phenanthrene in UK soils have decreased since the 1960s, but that soil concentrations of B[a]P and other heavier PAHs have continued to increase.

Those PAHs with less than four benzene rings exist in the atmosphere exclusively in the vapour phase, while those with more than four benzene rings are present almost exclusively in the particulate phase. They are relatively unstable in air, compared with other organic compounds, such as PCDD/Fs, and transformation processes result in lower concentrations of parent compounds. Compounds in the vapour phase are more susceptible to degradation, and undergo aerosol adsorption as they are transported in air (Broman *et al.*, 1991). It has been concluded that for point sources, distance from the source is most important factor affecting the composition of vapour phase PAHs.

The direct contribution of PAHs to the mutagenicity of ambient air particles may be small in comparison with compounds produced as a result of reactions with other atmospheric species. There may be substantial chemical conversion after emission (De Raat, 1988), as PAHs in the atmosphere react with compounds such as ozone and nitrogen dioxide, especially when exposure is carried out in the presence of light. The resulting products, particularly those following nitration, are often mutagenic.

The exhaust fumes from motor vehicles form a major source of environmental PAHs, particularly in urban areas. The decision to reduce lead in petrol is a cause for concern among some workers, due to the increase in aromatic content of the unleaded petrol, which leads to significant increases in the PAH content of the exhaust.

Benzo[a]pyrene has been regarded for many years as one of the most important PAHs because of its carcinogenic properties, and has often been used as an indicator of the presence of carcinogenic PAHs in air, despite its relative instability (Masclet *et al.*, 1986). However, its usefulness has often been questioned as, although it is one of the most potent PAH carcinogens, it only accounts for a minor proportion of the ∑PAH burden in the atmosphere.

Polycyclic aromatic hydrocarbons incorporated in the soil may be biologically degraded, photo-degraded, volatilized, or stored permanently. Biodegradation is the key process, although some of the low-molecular weight PAHs may be volatilized to the atmosphere (Wild and Jones, 1993). Persistence in the soil environment is positively correlated to the number of benzene rings in the compound molecule, and this pattern is apparent because the high molecular weight compounds are less prone to degradation, leaching and volatilization (Wild *et al.*, 1992). Some of the high molecular weight compounds have been found to persist in the soil for many years. Soils amended with sewage sludge may also have elevated PAH levels (Jones *et al.*, 1989).

In soils most PAHs are strongly adsorbed on to organic matter, thus making them unavailable for plant uptake or leaching to ground water. Direct deposition of particle-bound compounds from the atmosphere is the main source of PAHs in the aerial parts of plants although some retention of vapour-phase PAHs on the waxy leaf cuticle may occur (Wild and Jones, 1992). Contamination of plant parts with soil particles is also generally considered to be a possible source of PAHs in plants. Although direct deposition from the atmosphere is the main source of PAHs in above ground plant tissues, the mixture of PAHs is not necessarily the same as that in air, given that a number of processes may alter the proportions of the different PAHs present in plants (Wild *et al.*, 1992).

Most studies in literature reporting plant PAH concentrations, for example, are limited to just a few compounds, or focus on B[a]P alone. This is a major failing given the important differences in physico-chemical properties within this class of compounds. Studies that have investigated the persistence and fate of certain PAHs have found that there is no evidence for the uptake of intact B[a]P, for example, in a number of plant species (Goodin and Webber, 1995). The dominance of ∑PAH in plant tissues by the low molecular weight compounds is well-documented, and this may be due to the fact that lower molecular weight compounds are more susceptible to plant uptake given their higher aqueous solubility and volatility. However, they are also less persistent in the soil so their availability for uptake could be restricted in the longer term.

Plant PAH composition is very insensitive to changes in the underlying soil PAH burden. There are many uncertainties over the significance of PAHs in relation to soil quality and their transfer from soil into the human food chain. It is thought that while many organics cannot penetrate deep into the root tissues they may be able to either enter or adsorb on to the root surface. As PAHs are lipophilic/hydrophobic compounds, strongly associated with organic matter particles in the soil, the potential for plant uptake via the roots is limited, and as their physicochemical properties suggest, translocation of PAHs will be extremely inefficient. Adsorption on to root surfaces, however, will probably be an important process. Even though root uptake is limited there is still concern over elevated levels in root crops grown on soils with high PAH concentrations. It appears that levels in above ground plant parts are not strongly related to soil PAH levels, but that root crop concentrations would be

more closely related. The PAH content in food produce is especially important since diet is the major source of PAHs for the non-smoking general population.

Most PAHs absorbed by vegetation when it falls to the ground are permanently removed from the atmosphere. Although the equilibrium between the atmosphere and vegetation depends on gas-phase PAH concentrations and ambient temperature, most PAHs absorbed by vegetation at the end of the growing season are incorporated into the soil (Simonich and Hites, 1994). At low ambient temperatures gas-phase PAHs partition into vegetation, and at high ambient temperatures some PAHs volatilize and return to the atmosphere. It has been suggested that atmospheric semi-volatile organic compounds such as PAHs undergo a seasonal partitioning cycle with the surface of the earth (Simonich and Hites, 1994).

Atmospheric deposition of PAHs also represents the major input to both fresh waters and the marine environment, where they tend to adsorb to particles and be deposited to the underlying sediments. Differences between atmospheric composition and PAHs in particulate matter in water could be due to the fact that they are susceptible to degradation and transformation processes in water.

Transport to the marine environment also occurs via surface waters, with additional inputs from petroleum spills and discharges from ships (Hellou *et al.*, 1994). Degradation in marine sediments is generally slow, particularly for the higher molecular weight compounds (Law and Biscaya, 1994). PAHs are accumulated (although not biomagnified) by certain shellfish which concentrate PAHs from the marine environment but do not readily metabolise them (Jackson *et al.*, 1993). Polycyclic aromatic hydrocarbons are readily metabolised by fish to polar metabolites which can then be excreted. Greater acute toxicity is associated with lower molecular weight PAHs, and is correlated with aqueous solubility and octanol-water partition coefficients.

Sewage sludges typically contain between 1 and 10 mg/kg of each individual PAH and therefore there is some concern over their fate in the human food-chain when sewage sludge is used for agricultural purposes. Field-based studies have indicated that a residual fraction (composed of the high-molecular weight PAHs) remains in the soil many years after sludge application (Wild *et al.*, 1990), but that whilst the soil PAH burden may increase substantially, increased PAH concentrations in crops grown on these areas, relative to unsludged areas, have not been consistently detected (Wild *et al.*, 1992). This may be due to the fact that sewage sludges increase the amount of organic matter in the soil, thereby increasing the adsorption capacity, therefore PAHs added to the soil in sludge may be less available for plant uptake. Investigations into the uptake of PAHs by certain crops from sludge-amended soils suggest that the risks posed to human health via this route are minimal (Wild and Jones, 1992).

There are few data on the uptake of PAHs from food (Stavric and Klassen, 1994) and there is considerable inter-individual variation in the metabolism and excretion of ingested PAHs (Strickland and Groopman, 1995). Following ingestion by mammals, PAHs do not biomagnify in the same manner as some other organic chemicals, since they tend to be metabolised at the site of entry into the body where they are converted into reactive electrophilic species giving rise to DNA adduct formation. This is considered to be the initial event in chemical carcinogenesis (Baan *et al.*, 1994) and exposure to PAHs has been

associated with various forms of human cancer. Mammary and other fat tissues are storage depots for PAHs from where they may be released slowly.

The main elimination pathway for B[a]P is via the bile and enterohepatic circulation, and finally excretion in the faeces. Benzo[a]pyrene is rapidly cleared from the blood. Reduced solubility, physical adsorption and the formation of chemical adducts between B[a]P and some food ingredients play a role in reducing the adsorption of B[a]P from the gut. Benzo[a]pyrene adsorption from the intestinal tract is affected by dietary components, and this may be a factor that contributes to the lack of an epidemiological correlation between some human cancers and the B[a]P content of foods.

7.3 ENVIRONMENTAL LEVELS

Air

Urban air

A national urban air monitoring scheme in the UK has produced results for urban air at four locations in the UK (London, Manchester, Cardiff and Stevenage). London had the highest annual mean ∑PAH, and the heavier compounds showed a distinct seasonal variation, with winter levels significantly higher than summer levels; diurnal variations in PAH levels in the urban atmosphere have also been observed (Baek *et al.,* 1992). As the PAH profiles were similar at each site, it was concluded that the sources of contamination were common to each site (Halsall *et al.,* 1994). There is good evidence that air quality in London has improved considerably with respect to B[a]P concentrations over the last 50 years despite a huge increase in the use of motor vehicles probably due to the decreasing use of coal for domestic heating (Halsall *et al.,* 1994).

Over 100 PAH compounds have been identified in the organic fraction of urban atmospheric particulates by Baek et al (1992), who observed a similar trend to the UK monitoring results, of higher winter levels in urban air. The most extensive data available are on B[a]P and, although it is one of the principal carcinogenic PAHs, it represents only a small fraction of the total PAH in most circumstances (Baek *et al.,* 1992). There are many limitations to using a B[a]P surrogate method for estimating the risks posed by a mixture of PAH compounds since the proportion of carcinogenic activity attributable to B[a]P in products of incomplete combustion is known to vary among source categories (Thomson *et al.,* 1985). Although the lighter PAHs have weaker carcinogenic/mutagenic properties they are more abundant in the urban atmosphere and react with other pollutants to form more toxic derivatives.

An interesting trend has been noted with low molecular weight compounds which exist in the vapour phase in the atmosphere and generally dominate the ∑PAH burden of the air over the UK (Gardner *et al.,* 1992). Studies carried out in the UK indicate that some of these compounds may be present in rural air at concentrations equal to, or even greater than, those in urban air. It has been suggested that the reason for this is soil degassing. The total PAH concentrations (gas plus particulate phases) in the ambient air of traffic sources average 5 and 8 times higher than the mean values in urban and rural atmospheres (Lee *et al.,* 1995).

Soil

United Kingdom soil ∑PAH concentrations are generally in the range of 100–54,000 μg/kg (Jones *et al.*, 1989), with higher concentrations in urban areas, near point sources and in soils with naturally higher organic matter contents, e.g., peat soils. It should be noted however, that the total concentrations depend on which of the large number of PAH compounds are included in the study. UK studies have also found that although absolute levels show marked differences in urban and rural soils, in general soils show a relatively constant qualitative mixture, although this is affected to some extent by the soil organic matter content (Jones *et al.*, 1989). However, investigations into the levels of PAHs in soils have also been carried out in Norway (Vogt *et al.*, 1987), and found that the profiles in soils near to point sources were different to those at background sites. This is probably a result of the transformation of some of the compounds, particularly those in the vapour phase.

When assessing risk from soils, B[a]P is considered suitable as a guide substance as its content shows a strong correlation to the sum of other PAHs present in the soil, but in order to properly characterise the background contamination of soils with PAH, new reference values will need to be applied (Tebaay *et al.*, 1993).

Water

If PAHs are incorporated into water they are rapidly transferred into the sediments, although concentrations in the water column are likely to reflect water solubility. Polycyclic aromatic hydrocarbons in aquatic sediments originate largely from anthropogenic combustion.

7.4 PAHs IN FOOD

There are several studies that have examined levels of certain PAH compounds in various food stuffs which could be used to estimate daily intake and exposure from the diet in the UK. However, as with other environmental media, different studies have analysed for different PAH compounds and consequently comparisons of ∑PAH intakes are not appropriate. Given the large number of PAH compounds which may be present in the environment it does not seem likely that attempts to quantify daily ∑PAH from the diet would be realistic, or representative of the actual intake.

However, for some of the more commonly studied PAH compounds, such as B[a]P, it may be possible to produce reasonably good estimates or intakes from the diet and other sources. If in the future it becomes possible to accurately relate levels of particular PAH compounds to ∑PAH concentrations, not only in food but also in other environmental media, then it may be possible to carry out an estimate of ∑PAH exposure from the diet and other sources, such as inhalation, soil ingestion and drinking water.

There are significant differences between PAHs in terms of biological activity, and monitoring of foods in particular has concentrated on around ten of the most "important" compounds (Gilbert, 1994). Of more relevance is the contamination of foods of vegetable origin by direct deposition of PAHs on to the growing plant, contamination of processed oils and fats and contamination of foods of marine origin such as shellfish. Polycyclic aromatic hydrocarbons can be accumulated by shellfish and a survey of B[a]P in vegetables in Holland revealed that those with a large leafy surface contained higher PAH concentrations than others (Welling and Kaandorp, 1986).

Certain foods such as charcoal grilled meats, have been shown to contain relatively high levels (Strickland and Groopman, 1995). Levels in retail fish and animal-derived fats and oils are low in comparison with the levels present in retail vegetable oils (Dennis *et al.*, 1991). Elevated levels have been observed in bran samples, possibly due to the fact that it is obtained from the outer part of the seed and is therefore much more susceptible to environmental contamination.

Studies have shown that the length of cooking time affects the mutagenic activity of PAHs in the fat of foods consumed by humans, such as lamb (Nakano and Fukushima, 1994), with mutagenic activity decreasing with increasing heating time. Certain meat products, especially smoked ones may have elevated PAH levels (De Vos *et al.*, 1990). During curing and smoking substances such as PAHs can be transferred to the food or can be produced during treatment (Muller, 1991)

Root vegetables are in close contact with soil, and PAHs may enter the root tissues. Carrots, for example, may be able to take up organic compounds in specialised oil channels. Studies have found the highest concentrations in the lipid rich peel of carrots. Higher concentrations of PAHs have been detected in cereal-derived products containing higher levels of edible oils, such as biscuits and cakes (Dennis *et al.*,1991) than in bread, for example.

Diet or some specific components in food could play a significant role in influencing and reducing the bioavailability of B[a]P. Any food seems to retard the availability of uptake of B[a]P from the gastro-intestinal tract (GI tract). Water, in which B[a]P is not soluble, may reduce the transfer of B[a]P from the food particles to the intestinal mucosa. In contrast, oil probably enhances B[a]P transfer to the intestinal wall (Stavric and Klassen, 1994).

No PAHs at all were found in drinking water in a Dutch study of dietary intake of PAHs (De Vos *et al.*, 1981).

7.5 INTAKE AND EXPOSURE – SUMMARY OF PREVIOUS STUDIES.

These studies can be divided into those that have looked at a number of PAHs and those studies which have concentrated on one particular PAH, most commonly B[a]P.

Dietary intake has been identified as the principal route of human exposure to PAHs for non-smokers (Santodonato *et al.*, 1981; Shuker and Bennett, 1988), with plant-based foodstuffs constituting approximately 50 per cent of the total PAH intake in a typical UK diet (Dennis *et al.*, 1982). Examination of exposure to PAHs from a number of routes have shown that the dietary contribution is highly important in determining the PAH-DNA adduct load in the blood, a marker of exposure (Strickland *et al.*, 1991).

Further studies of the UK diet have indicated that cereals, oils and fats contribute approximately one third of the PAH **dietary** intake, with fruit, sugars and vegetables providing much of the remainder (Dennis *et al.*, 1983). The total dietary intake of PAHs was estimated to be 3.7 μg per person per day, with B[a]P contributing some 0.25 μg per person per day. Dennis et al (1991) identified margarine as the chief source of PAHs in the oils and fats group, and cereal products containing vegetable oils as an ingredient in the cereals group,

clearly demonstrating the importance of the cereals group and the oils/fats group to the dietary intake of PAHs. The consumption of PAH-containing food products and active smoking account for 99 per cent of total PAH intake, but passive smoking and the inhalation of ambient air are relatively unimportant for total PAH intake (both account for less than 1 per cent) according to Vanrooij et al (1994).

In contrast to ∑PAHs, approximately 80 per cent of the dietary intake of B[a]P comes from the cereals group and the oils/fats group **(Shuker and Bennett, 1988)**. Benzo[a]pyrene has sometimes been used as an indicator of overall PAH contamination by some workers, but as for other environmental media, there are limitations to the use of using B[a]P as a surrogate method of estimating intake (and therefore, risk) from a mixture of PAHs. Despite this, it is interesting to note that studies undertaken in the Netherlands (de Vos *et al.*, 1990; Vaessen *et al.*, 1998) and the UK (Dennis *et al.*, 1983; Dennis *et al.*, 1991) have produced remarkably similar results for the estimation of intake of B[a]P.

Hattemeyer-Frey and Travis (1991) estimated that the food chain contributes approximately 97 per cent of the total daily intake of B[a]P. The daily intake of B[a]P from foods in the USA is reported to be in the range of 0.16–1.6 μg/day (Santodonato *et al.*, 1981); due to the uncertainty surrounding the relative contribution of B[a]P to total PAH intake it is not possible to compare the B[a]P intake with data from the Netherlands, for example, where the reported range for daily ∑PAH intake is 1.1–22.5 μg per person (Vaessen *et al.*, 1988).

An attempt to evaluate the risks to health from environmental exposure to PAHs in Canada concentrated on 5 compounds which are classified as probably carcinogenic: benzo[a]pyrene, benzo[b]fluoranthene (B[b]F), benzo[j]fluoranthene (B[j]F), benzo[k]fluoranthene (B[k]F) and indeno (1,2,3-cd) pyrene (IND). However, available data were inadequate to assess exposure through ingestion, or carcinogenic potency of a wide range of PAHs in the GI tract. They did produce relative carcinogenic potency factors for the four compounds relative to B[a]P: B[b]F 0.06, B[j]F 0.05, B[k]F 0.04 and IND 0.12 (Meek *et al.*, 1994).

Estimated intake through breast-feeding

No data have identified with levels of PAHs in human milk. Therefore it has not been possible to estimate the intake of PAHs through breast-feeding. However, the presence of vegetable oils as an ingredient in infant milk formula appears to increase the PAH level. The mean B[a]P level of 0.49 μg/kg was four times higher than that found in skimmed milk, but the mean value in the reconstituted diluted infant formula is low, approximately 0.1 μg/l (Dennis *et al.*, 1991).

Smoking

Examination of the importance of tobacco smoking to B[a]P exposure, seems to show that for the average/moderate smoker, in the general population, more B[a]P is consumed from food than from smoking (Stavric and Klassen, 1994). The importance of the inhalation pathway is significantly less, with the maximum uptake of B[a]P from ambient air about one order of magnitude less than the uptake via food (Kramers and van der Heijden, 1988), but in some cases, probably due to indoor combustion sources, the intake of B[a]P from food and air can be quite similar.

Smokers get an additional 16 per cent B[a]P from smoking (Hattemeyer-Frey and Travis, 1991).

Food packaging

There is some disagreement as to the importance of packaging of foods with respect to an additional source of exposure to the human population. **PAHs in food originate predominately from environmental sources**, although according to some workers there may be additional inputs from food packaging and food processing (De Vos *et al.*, 1990; Lo and Sandi, 1978). More recent studies suggest that some food preparation may reduce the PAH contents of certain foods, and that food packaging does not contaminate the produce (Wild and Jones, 1991).

7.6 TOXICOLOGICAL ASSESSMENT

At least 18 PAHs are of environmental concern, **identified by inclusion in either** the US EPA's list of priority pollutants or on a tentative list for general reporting of PAH emissions proposed by the Commission of the EC (Baek *et al.*, 1992). Health concerns regarding PAHs focus on their metabolic transformation by aquatic and terrestrial organisms into mutagenic, carcinogenic and teratogenic agents such as dihydrodiol epoxides. These metabolites bind to and disrupt DNA and RNA, and it is this disruption of genetic material which is the basis for tumour formation.

Whilst epidemiological studies on occupational exposure have shown an increase in cancer incidence among workers exposed to PAHs (IARC, 1983), little is known about human cancer risk to the widespread distribution of low levels of PAHs in food products and polluted air (Van Rooij *et al.*, 1994). Animal experiments have shown a very strong carcinogenic and mutagenic action for many PAHs, and it has been found that the PAH derivatives of photo-oxidation, or of PAH reactions with other atmospheric pollutants are more toxic than the original PAH.

Exposure to certain PAHs by inhalation through cigarette smoking, or contact through occupational exposure of the lungs or skin can result in the development of cancer, but despite the apparently strong carcinogenic potential of these chemicals in tests with laboratory animals, no epidemiological evidence has been found to date, linking their presence in foods to human cancers (Stavric and Klassen, 1994).

In addition to work on other PAHs, epidemiological studies attempting to determine whether elevated levels of B[a]P in food are a causative factor in some human cancers have failed to establish a link. The lack of correlation between cancer incidence and intake of dietary PAHs could be due to some "protective" or "detoxification" mechanism.

There are, as far as we are aware, no standards or guidelines for the Tolerable Daily Intake of either individual PAHs, or total PAHs. As has been stressed earlier, the relevance of using a "Total PAH" measurement is limited due to the large number of PAH compounds which may exist, but for which there are no data. Assessments of individual compounds may prove useful indicators, but would obviously fail to take into account the potential exposure and risk from all PAHs in the environment. Unlike PCDD/Fs, for which there is a well established International Toxic Equivalents, the estimation of risk from a mixture of PAH compounds is

still very difficult to quantify. Toxicological assessments have also found that there are inadequate data to assess toxic or carcinogenic potency for a wide range of PAHs in the human body, and in the GI tract in particular.

7.7 Derivation of Environmental Assessment Levels

At present it is not appropriate to set EALs for ∑PAHs using Exposure Commitment for the following reasons:

1. No measure of "acceptable" exposure or total daily intake with which to compare current intakes are available for either individual compounds or mixtures of PAHs. There are, as far as we are aware, no standards or guidelines for the Tolerable Daily Intake of either individual PAHs, or total PAHs. Toxicological assessments have also found that there are inadequate data to assess toxic or carcinogenic potency for a wide range of PAHs in the human body, and in the GI tract in particular.

2. Lack of comparable estimates for ∑PAH intake and exposure due to the large number of PAH compounds, and the variation in the compounds analysed by different studies and referred to as ∑PAHs. Comparisons of ∑PAH intakes may not be very meaningful.

3. There may be many other PAH compounds emitted to the UK environment, where they may reside as contaminants. Given the large number of PAH compounds which may be present in the environment, it does not seem likely that attempts to quantify daily ∑PAH from the diet, or other sources, would be realistic, or representative of the actual intake.

4. The importance of indoor air, and the contribution of domestic combustion sources, as well as motor vehicle traffic, has still not been quantified, although in some cases the intake of B[a]P from food and air can be quite similar.

5. No data on body burdens and tissue levels exist as PAHs are not accumulated in the same manner as some other organic chemicals, but are metabolised at the site of entry into the body.

6. There may be substantial chemical conversion after emission. Formation of derivatives, particularly from atmospheric transformation processes following nitration, which are more toxic and mutagenic, mean that the direct PAH contribution to the mutagenicity of air particulates is small in comparison to the indirect contribution brought about by these transformation processes.

7. Higher PAHs have weaker carcinogenic/mutagenic properties but are more abundant in the urban atmosphere and react with other pollutants to form more toxic derivatives.

7.8 ENVIRONMENTAL STANDARDS FOR PAHS

In the Netherlands the interim goal is to reduce the annual average concentration of B[a]P to 5 ng/m^3 (Baek *et al.*, 1992). The German Federal Government has proposed a guideline limit of 10 ng/m^3 for the annual B[a]P concentration. None of the urban sites monitored in the UK had an annual mean near this limit, the highest being Manchester with 1.82 ng/m^3.

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8. PREDICTION OF CONTAMINANT CONCENTRATIONS IN VARIOUS FOODSTUFFS FROM ATMOSPHERIC CONCENTRATIONS

8.1 PCDD/Fs IN BEEF AND MILK

The animal-based foodstuffs, such as meat and milk products, are the major sources of dietary intake of semi-volatile lipophilic environmental contaminants such as polychlorinated dibenzo-p-dioxins and polychlorinated dibenzo-p-furans (PCDD/Fs), and estimates from a number of studies have found that they contribute at least 50 per cent to the total daily intake of PCDD/F Toxic Equivalents. Therefore measurements or estimates of the levels of these compounds in such foods are crucial in developing an accurate picture of human exposure. A number of investigations have attempted to link environmental concentrations with PCDD/F levels in meat and milk by using mathematical models.

Lorber *et al.* (1994) developed a model which calculates the concentration in soil and herbage from data on atmospheric concentrations of the individual congeners and their physico-chemical characteristics, and have related these levels to beef fat concentrations by using a bioconcentration model. This model has been applied to the UK data gathered in this study. The estimated background air concentrations have been used and the resulting predicted soil, herbage and beef concentrations have been compared with measured data from the UK. The assumptions used are those documented and discussed in Lorber *et al.* (1994), and therefore no detailed further discussion of the derivation of certain parameters is provided in this assessment. In addition, the concentration in whole milk has been estimated assuming that PCDD/Fs are equally distributed in the fat of cattle, and therefore beef fat and milk fat concentrations are the same. The fresh weight concentrations will vary due to the variation in fat content of beef (20 per cent) and whole milk (4 per cent).

The starting point for the model is the total air concentration of each congener, and these have been estimated by taking the mean level measured in UK urban air and dividing by a factor of 6 to give the background air concentration (see Appendix 8.1). It has been found that mean levels in urban air are approximately 6 times higher than the levels in background air.

From this starting point a number of stages are involved in estimating the levels in soil, various forms of vegetation consumed by cattle, and eventually in the beef tissue itself.

8.2 ATMOSPHERIC PARTITIONING – PARTICULATE AND VAPOUR PHASES

PCDD/Fs may be present in either form in the atmosphere, and this will determine the behaviour of the compound in the atmosphere in terms of the pathway to plant material. The derivation of these factors is discussed in Lorber *et al.* (1994), and the general trend is for the more highly chlorinated congeners to be present as a higher fraction of the particulate form. The fractions are detailed in Appendix 8.1.

8.3 PARTICULATE DEPOSITION ON TO VEGETATION

The steady-state situation relating the plant concentration to the particulate air concentration is represented by two equations.

$$Cppa = \frac{Fd + Rw\ Fw}{Kw\ Yj}$$

Where
- Cppa = Concentration in vegetation (ng/kg)
- Fd = Dry deposition rate (ng/m^2/yr)
- Rw = Retention of wet deposition on plants (fraction)
- Fw = Wet deposition rate (ng/m^2/yr)
- Kw = 1st order weathering dissipation constant (1/year)
- Yj = Dry matter yield of crop (kg/m^2)

The dry deposition rate (Fd) used in the above calculation is derived from the particulate air concentration using the following relationship.

$$Fd = \frac{Cpa\ Vd\ Ij}{1{,}000}$$

Where
- Cpa = Air concentration (particulate) (pg/m^3)
- Vd = Dry deposition velocity (m/yr)
- Ij = Fraction of particulates intercepted
- 1,000 = Unit conversion (pg to ng)

Deposition velocity (Vd)
The value used by Lorber *et al.* (1994) is 0.2 cm/s, which is consistent with the dry deposition velocities measured for PCDD/Fs in the field. The figure used by Bennett (1981) for the deposition rate of ambient aerosols is 0.5 cm/s. However, the former value (equivalent to a deposition rate of 63,072 m/yr) is the preferred value.

Fraction of particulates intercepted (Ij)
This value will vary for different plant types, and is taken from Fries and Paustenbach (1990). The values used are 0.35 for grass, and 0.62 for hay, silage and grains.

Wet deposition rate (Fw)
It has been found that the wet deposition rate is approximately the same as the dry deposition rate in field studies in the USA. Therefore, for the purpose of this model it is assumed that Fw = Fd.

Fraction of wet deposition adhering to vegetation (Rw)
The recommended fraction is between 0.1 and 0.3 (McKone and Ryan, 1989), and field studies have indicated a range of 0.24 to 0.37 for particles on a range of vegetation (Hoffman *et al.*, 1992). The figure used by Lorber *et al.* (1994) was 0.3.

Weathering constant (Kw)

It is assumed that dry deposition fully adheres to plant surfaces while deposition is occurring. Weathering constant models the loss of the particle bound concentration from the plant due to wind, rain or weathering processes, after deposition has occurred. The value used by Lorber *et al.* (1994) was calculated from a half-life of 14 days. This value may be too long given experimental results, which indicate a median of 10 days (Baes *et al.*, 1984). The half-life (days) is related to the weathering constant by the following equation:

$$Kw = \frac{1 \times 365}{(T½ \div 1n\,(2))}$$

The half-lives of 14 days and 10 days give weathering constants of 18.1 and 25.3, respectively.

8.4 PARTICULATE DEPOSITION TO SOIL

It is assumed that all particulates deposited reach the soil directly or via weathering removal of particles from vegetation surfaces. The soil concentration is related to the air concentration via the deposition rates, and the relationship is represented by the following equation:

$$Cs = \frac{Fd + Fw}{Ks\,M}$$

Where Cs = Soil concentration (ng/kg)
Fd = Dry deposition rate (ng/m^2/yr)
Fw = Wet deposition rate (ng/m^2/yr)
Ks = 1st order dissipation constant for soil (1/year)
M = Mass of mixing soil per cubic metre (kg/m^2)

The dissipation constant for the soil (Ks) is derived from the half-life in the soil using the following relationship.

$$Ks = \frac{1}{(T½ \div 1n\,(2))}$$

The suggested half-life of TCDD in soil is in the order of 10 years (Young, 1983) to 15 years (Fries and Paustenbach, 1990), and would give rise to Ks values of 0.069 and 0.046 respectively. The half-life for different congeners, however, is likely to vary considerably.

The mixing mass of the soil is calculated assuming a mixing depth of 1 cm (as is the situation with non-ploughed land such as pasture) and a soil bulk density of 1.5 g/cm^3. This would result in a soil mixing mass of 15 kg/m^2.

8.5 VAPOUR PHASE TRANSFERS TO VEGETATION

Plant concentrations are related to vapour phase atmospheric concentrations by the relationship between a mass-based biotransfer factor, summarised in the equation below:

$$Cvpa = \frac{Bvpa \; Cva \; VGag}{1{,}000 \; da}$$

Where
- Cvpa = Plant concentration due to uptake of vapour phase (ng/kg)
- Bvpa = Mass-based air-lear biotransfer factor
- Cva = Vapour phase air concentration (pg/m^3)
- VGag = Bulkiness correction factor
- da = Density of air (kg/m^3)
- 1000 = Units conversion factor (pg to ng)

The VGag is an empirical correction factor which reduces the vegetation concentration, taking into account that the Bvpa was developed for the transfer of air-borne contaminants into leaves rather than bulky above ground vegetation. The VGag value for grass can be assumed to be 1.00, but for the hay/silage/grains mixture a value of 0.5 was used by Lorber *et al.* (1994), but this second value is the subject of considerable uncertainty.

Calculation of the Bvpa

The Bvpa is calculated from the volumetric air-leaf biotransfer factor (Bvol) which in turn is derived from physicochemical data for the compound. The Bvol is related to these parameters by the following equation:

$$\log Bvol = 1.065 \log Kow - \log Kaw - 1.602$$

Where
- Bvol = Bacci volumetric air-leaf transfer factor
- Kow = Octanol/water partition coefficient
- Kaw = Air/water partition coefficient
- -1.602 = Empirical correction factor

Kaw is calculated from the following data:

$$Kaw = \frac{H}{RT}$$

Where
- H = Henry's Law Constant for the compound (atm-m^3/mol)
- R = Ideal Gas Constant (8.205 x 10-5 atm-m^3/mol oK)
- T = Temperature (298.1 oK)

The log Kow values can be found in Appendix 8.1, together with the Henry's Law Constant for each congener.

The empirical correction factor of -1.602 is introduced to account for the influence of photodegradation on the vegetation concentration of the compound. The original relationship was derived by Bacci *et al.* (1992) for TCDD under conditions that would not account for

photodegradation. Subsequent work (McCrady and Maggards, 1993) has found that the relationship overestimates TCDD plant concentrations by a factor of about 40. Therefore Lorber *et al.* (1994) introduced the empirical constant to the equation in order to account for photo-degradation. The rates of photodegradation of the various congeners is still not certain, although it is thought that the higher chlorinated congeners may undergo reductive dechlorination to more toxic derivatives.

The Bvol value calculated can then be used to derive the Bvpa for grass from the equation.

$$Bvpa = \frac{1.19 \times Bvol}{0.15 \times 770}$$

Where 1.19 = The density of air (g/l)

0.15 = The correction factor for dry/wet weight (assuming plants are 15 per cent dry matter)

770 = The grass leaf density (770 g/l)

The Bvpa for hay/silage/grain is derived from a similar relationship, but using different values for the dry/wet weight (30 per cent dry matter) and the leaf (tissue) density (890 g/l).

$$Bvpa = \frac{1.19 \times Bvol}{0.3 \times 890}$$

The concentration (Cvpa) in the two vegetation types can then be calculated.

8.6 PLANT UPTAKE FROM THE SOIL

Concentrations of TCDD in above-ground plant parts can be estimated from soil concentrations using the equation derived by Travis and Arms (1998) using the octanol/water partitioning coefficient log Kow.

$$\log Bv = \log Bs + 1.588 - 0.578 \log Kow$$

Where Bv = Concentration of organic in above ground matter (dry weight)

log Bs = Concentration of organic in soil (ng/kg)

The calculated concentrations can be converted to fresh weight concentrations using the assumptions that grass is 15 per cent dry matter and hay/silage/grain is 30 per cent dry matter.

Total plant concentration

The total concentration in the plant tissue is simply the sum of the concentrations contributed from the particulate-phase air concentrations, the vapour phase air concentrations and the concentration as a result of plant uptake from the soil.

$$Ctot = Cvpa + Cppa + Bv$$

8.7 BIOCONCENTRATION MODEL

A mass balance study on the dietary intake of PCDD/F congeners in relation to milk fat concentrations (McLachlan *et al.*, 1990) has produced Bioconcentration Factors (BCFs) for individual congeners which are listed in Appendix 8.1. These can be used to calculate the concentrations in beef fat, assuming that the beef fat BCFs are the same as those for milk fat. (Most studies assume that PCDD/Fs are fairly uniformly distributed to fat tissue in humans and animals).

The fat concentration can be calculated from the following equation:

$$Cfat = (BCF \times DF \times Bs \times C)soil + (BCF \times DF \times C)grass + (BCF \times DF \times C)hay/grain$$

Where
- BCF = Bioconcentration factor for compound
- DF = Fraction of cattle diet that is soil/grass/hay
- Bs = Bioavailability of contaminant in soil relative to vegetation
- C = Average concentration in soil/grass/hay (ng/kg)

From studies of the uptake of PCDD/Fs from soil and vegetation it has been estimated that the bioavailability ratio of soil:vegetation is 0.65:1 (Lorber *et al.*, 1994).

Cattle ingest substantial amounts of soil when grazing, and it has been estimated that the proportion of the cattle diet that is soil is between 4 and 6 per cent (Lorber *et al.*, 1994; Wild *et al.*, 1994). It has been assumed in the Lorber *et al.* (1994) study that the remainder of the diet is divided equally between pasture grass and hay/silage/grain. Once the fat concentration has been calculated the concentration in meat is estimated by assuming that beef is 19 per cent fat, and that whole milk is 4 per cent fat (Lorber *et al.*, 1994; Wild *et al.*, 1994).

8.8 DISCUSSION

In all of the following discussion it is assumed that the environmental levels and concentrations in the foodstuffs are general background levels and therefore comparable in terms of the predictive capacity of the model. A full calibration of the model using measured data for all media from a single study would be necessary to validate the assumptions made in this assessment. The measured soil and herbage concentrations come from a semi-rural site in the UK (Kjeller, 1991), and the measured levels in meat and whole milk come from the UK survey of PCDD/Fs in the diet (MAFF, 1992) and are the representative values for congeners used in the exposure commitment assessment.

Prediction of soil concentrations

The model performs reasonably well for most PCDD/F congeners, and in terms of the ΣTEQ concentrations, it is quite close to measured data (underestimating soil concentrations by approximately one third). The underestimation is most noticeable for the higher chlorinated congeners, with the predicted value for OCDF being 20 times lower than the measured value. This may be a result of a number of factors including underestimation of the half-life of congeners in soil (10 years) and the deposition velocity of ambient aerosol particles

(0.2 cm/s). Other studies have estimated these values to be 15 years for soil half-life (Fries and Paustenbach, 1990) and 0.5 cm/s for the deposition velocity (Bennett, 1981).

Table 8.1 Measured and predicted soil concentrations (ng/kg)

	Soil (predicted)	Soil (measured)	Measured/ predicted ratio
2,3,7,8-TCDD	0.12	0.09	0.79
1,2,3,7,8-PCDD	0.12	0.27	2.17
1,2,3,4,7,8-HxCDD	0.22	0.35	1.58
1,2,3,6,7,8-HxCDD	0.35	0.61	1.76
1,2,3,7,8,9-HxCDD	0.36	0.47	1.32
1,2,3,4,6,7,8-HpCDD	5.41	5.65	1.04
OCDD	10.51	23.41	2.23
2,3,7,8-TCDF	0.33	1.03	3.14
2,3,4,7,8-PCDF	0.35	0.92	2.61
1,2,3,7,8-PCDF	0.26	1.08	4.21
1,2,3,4,7,8-HxCDF	1.54	1.37	0.89
1,2,3,7,8,9-HxCDF	0.14		
1,2,3,6,7,8-HxCDF	0.81	0.84	1.04
2,3,4,6,7,8-HxCDF	0.86	0.66	0.76
1,2,3,4,6,7,8-HpCDF	2.97	4.25	1.43
1,2,3,4,7,8,9-HpCDF	0.40	0.42	1.04
OCDF	0.27	5.20	19.59
ΣTEQ	**0.93**	**1.41**	**1.5**

Prediction of herbage (grass) concentrations

The model overestimates the levels in herbage by a factor of about 2.7 and there may be a number of reasons for this. Although the model attempts to account for weathering and photodegradation, there is still a great deal of uncertainty regarding the values used. The predicted and measured ΣTEQ concentrations in herbage are 0.235 ng/kg and 0.09 ng/kg fresh weight, respectively (assuming the vegetation is 10 per cent dry matter). The representative value for leafy green vegetables used in the exposure assessment, and taken from measured data (MAFF, 1992), was almost 6 times lower than the predicted herbage concentrations. Therefore there is still considerable scope for the investigation and modelling of the direct deposition of airborne organic contaminants on to crops as a means of entry to the human food chain, and therefore as a potential source of exposure to the human population.

Prediction of beef concentrations

The model appears to perform very well in estimating ΣTEQ concentrations in beef using the UK data. In particular, estimates of the more toxic, less highly chlorinated congeners are very close to the measured levels. The estimates for the more highly-chlorinated congeners, however, are not as convincing, with the measured OCDD levels in beef around 40 times higher than predicted levels, but as OCDD has a very low toxicity equivalency factor (1,000 times less toxic than TCDD), the effect on the ΣTEQ value is negligible. In the case of OCDF the difference is even more significant, being over 3 orders of magnitude. These discrepancies demonstrate that the model may not be widely applicable to organic compounds with similar physicochemical characteristics similar to those of the more highly chlorinated

PCDD/Fs. It should be noted that the Bioconcentration Factors used in the model will greatly affect the final predicted concentration in the fat, and therefore the meat and milk concentrations.

Table 8.2 Measured and predicted herbage concentrations (ng/kg)

	Herbage (predicted)	Herbage (measured)	Measured/ predicted ratio
2,3,7,8-TCDD	0.29	0.03	0.11
1,2,3,7,8-PCDD	0.51	0.14	0.28
1,2,3,4,7,8-HxCDD	0.72	0.14	0.20
1,2,3,6,7,8-HxCDD	0.37	3.00	8.19
1,2,3,7,8,9-HxCDD	0.27	1.40	5.26
1,2,3,4,6,7,8-HpCDD	20.40	5.90	0.29
OCDD	2.64	24.00	9.10
2,3,7,8-TCDF	2.10	0.46	0.22
2,3,4,7,8-PCDF	1.46	0.20	0.14
1,2,3,7,8-PCDF	1.29	0.18	0.14
1,2,3,4,7,8-HxCDF	1.39	0.32	0.23
1,2,3,7,8,9-HxCDF	0.25	0.02	0.09
1,2,3,6,7,8-HxCDF	1.63	0.16	0.10
2,3,4,6,7,8-HxCDF	1.14	0.15	0.13
1,2,3,4,6,7,8-HpCDF	2.19	1.90	0.87
1,2,3,4,7,8,9-HpCDF	0.25	0.14	0.57
OCDF	0.07	2.00	30.19
ΣTEQ	**2.35**	**0.88**	**0.4**

Table 8.3 Measured and predicted concentrations in beef (ng/kg fresh weight)

	Meat (predicted)	Meat (measured)	Measured/ predicted ratio
2,3,7,8-TCDD	0.13	0.17	1.3
1,2,3,7,8-PCDD	0.21	0.24	1.1
1,2,3,4,7,8-HxCDD	0.14	0.17	1.2
1,2,3,6,7,8-HxCDD	0.09	0.54	5.9
1,2,3,7,8,9-HxCDD	0.05	0.26	4.7
1,2,3,4,6,7,8-HpCDD	0.73	4.25	5.8
OCDD	0.23	8.80	38.9
2,3,7,8-TCDF	0.19	0.23	1.2
2,3,4,7,8-PCDF	0.45	0.13	0.3
1,2,3,7,8-PCDF	0.09	0.29	3.1
1,2,3,4,7,8-HxCDF	0.37	0.24	0.6
1,2,3,7,8,9-HxCDF	0.05	0.21	3.9
1,2,3,6,7,8-HxCDF	0.34	0.21	0.6
2,3,4,6,7,8-HxCDF	0.22	0.23	1.0
1,2,3,4,6,7,8-HpCDF	0.11	0.31	2.9
1,2,3,4,7,8,9-HpCDF	0.03	0.26	8.5
OCDF	0.00	5.58	2552.8
ΣTEQ	**0.614**	**0.628**	**1.0**

Prediction of milk concentrations

A similar pattern is observed with milk ΣTEQ levels, but the model generally overestimates the ΣTEQ concentration. In the case of OCDD and OCDF, however, the model again underestimates concentrations by factors of approximately 20 and 1,000, respectively.

Table 8.4 Measured and predicted concentrations in milk (ng/kg fresh weight)

	Milk (predicted)	Milk (measured)	Measured/ predicted ratio
2,3,7,8-TCDD	0.03	0.02	0.8
1,2,3,7,8-PCDD	0.04	0.04	1.0
1,2,3,4,7,8-HxCDD	0.03	0.03	0.9
1,2,3,6,7,8-HxCDD	0.02	0.06	3.5
1,2,3,7,8,9-HxCDD	0.01	0.03	2.7
1,2,3,4,6,7,8-HpCDD	0.15	0.25	1.7
OCDD	0.05	1.00	22.1
2,3,7,8-TCDF	0.04	0.01	0.3
2,3,4,7,8-PCDF	0.09	0.05	0.6
1,2,3,7,8-PCDF	0.02	0.01	0.5
1,2,3,4,7,8-HxCDF	0.07	0.03	0.4
1,2,3,7,8,9-HxCDF	0.01	0.01	1.0
1,2,3,6,7,8-HxCDF	0.07	0.02	0.3
2,3,4,6,7,8-HxCDF	0.04	0.02	0.5
1,2,3,4,6,7,8-HpCDF	0.02	0.04	1.9
1,2,3,4,7,8,9-HpCDF	0.01	0.01	1.7
OCDF	0.00	0.43	983.6
ΣTEQ	**0.123**	**0.091**	**0.7**

Summary

The model may well be a useful basis for a predictive method to link environmental levels to concentrations in these key foodstuffs. It appears to perform particularly well for compounds with the physico-chemical characteristics of the less chlorinated PCDD/Fs. The BCF values are also crucial in estimating milk and meat concentrations, and can only be obtained from experimental mass-balance data on livestock. More data on the behaviour and properties of the individual PCDD/Fs in soil and vegetation are needed to verify the model.

8.9 USE OF PREDICTED LEVELS IN MILK AND MEAT TO ESTIMATE TOTAL DAILY INTAKE

It may be possible to estimate the total daily intake of PCDD/F TEQs from the predicted values generated by the model if a number of qualified assumptions are made.

1. The concentration in beef is representative of the whole meat group, and the fat content of meat in general is 20 per cent (i.e., pork, lamb, offal, poultry and other meat products). This is a fairly conservative estimate as most studies have found that beef levels are generally higher than levels in the other meat products. The consumption rate for this group is simply the sum of the consumption rates for the individual foodstuffs used in the exposure assessment (50.81 kg/yr).

2. The levels in various dairy products can be estimated by using the fat content of each foodstuff. The other dairy products considered are skimmed milk, cheese, butter, cream, ice cream and yoghurt and the fat contents are 2 per cent, 35 per cent, 80 per cent, 25 per cent, 3.5 per cent and 3.0 per cent, respectively. Whole milk is assumed to be 4 per cent fat (Wild *et al.*, 1994).

3. Milk and meat contribute 50 per cent of the total daily intake of PCDD/Fs from all sources.

4. The concentration in the fat of livestock is that calculated by the model, i.e., 3.07 ng TEQ/kg.

Results

The derived concentrations for the various foodstuffs and the calculated intakes are shown in Table 8.5. The total intake from meat and dairy products calculated is approximately 58 ng/year, and using assumption 3 above, it follows that total intake would be approximately 116 ng, equivalent to a daily intake of 4.5 pg TEQ/kg bw/day for a 70 kg adult. The current total daily intake for the general adult population calculated from measured data is 2.5 pg TEQ/kg bw/day. It can be seen that the model and the assumptions used provide a conservative estimate of daily PCDD/F TEQ intake, but one that falls in the range 2 to 10 pg TEQ/kg bw/day suggested by some workers as a reasonable range for unexposed populations world-wide.

Table 8.5 Estimated intake of PCDD/F TEQs from predicted levels in milk and beef

	TEQ (ng/kg)	Consumption (kg/yr)	Intake (ng/yr)
Meat	0.61	50.81	31.22
Milk	0.12	50.24	6.17
Skimmed milk	0.06	48.07	2.95
Cheese	1.08	5.88	6.32
Butter	2.46	2.16	5.31
Cream	0.77	6.21	4.77
Ice cream	0.11	10.85	1.17
Yoghurt	0.09	0.89	0.08
Total			**57.99**

In particular it may be that the assumption regarding the levels in all meat products is overly conservative, as most other meats and poultry contain less fat (i.e., less than 20 per cent) than beef. However, if a particular substance with physical and chemical characteristics to PCDD/Fs, particularly the less chlorinated congeners, is assumed to partition in environmental media in the same way as PCDD/Fs, it may be possible to estimate the total daily intake of the substance (and therefore daily exposure) with a reasonable degree of certainty from measured background concentrations in air, using the model derived by Lorber *et al.* (1994) and the above assumptions. Provisional Environmental Assessment Levels could then be derived from the Tolerable Daily Intake of the substance in the same way as has been carried out for PCDD/Fs.

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9. DISCUSSION

Multimedia transport models, such as Exposure Commitment, are useful tools for regulating exposure to pollutants. They allow regulators to take into account the relative importance of different pathways, which is crucial as these will vary for the wide range of environmental contaminants to which humans are exposed.

This study has endeavoured to assess the feasibility of using Exposure Commitment as a tool in setting environmental standards which take into account long-term human exposure, and its associated risk to human health. It is clear from the growing body of literature that Exposure Commitment (also referred to as exposure assessment, integrated exposure assessment, multimedia exposure assessment) is now considered by many to be a fundamental part of the process of environmental regulation, not only in the derivation of environmental standards, but also in its application to site-specific risk assessments.

Integrated exposure assessments that quantify human exposure from all sources have assumed greater importance over the last 5–10 years, and increasingly play a crucial role in environmental regulation. Examples of their use can be found in Canada and the USA, where quantitative exposure assessments are used to estimate risk from environmental exposure to a number of substances.

The concept of determining current exposure levels, and comparing these with acceptable levels in order to back-calculate acceptable environmental levels is now becoming widespread (Jones *et al.*, 1991). Good examples in the literature include a multimedia exposure analysis by Travis and Hattemeyer-Frey (1991) which found that existing air quality standards for 2,3,7,8-TCDD were several times higher than those proposed taking into account lifetime human exposure from all sources. The results are comparable to those from this study, and those derived in Germany using a similar method (Prinz *et al.*, 1993). These conclusions clearly demonstrate the advantages of Exposure Commitment, and similar methodologies.

A wide range of investigations have attempted to assess exposure to human populations from environmental exposure, and the review of these studies has highlighted a number of points that require consideration.

It must be accepted that any assessment of exposure to the general population cannot account for individual exposures, which may vary considerably depending on the personal activities, lifestyle and diet of the individual. In this study, exposure to the average urban individual has been calculated as firstly, these people are generally exposed to the highest levels of pollutants (although this may depend on a number of factors such as the geographical origin of food consumed in the diet) and secondly, the majority of the UK population lives in urban areas. Obviously there will be regions with naturally higher environmental levels of certain substances, but it is not possible within the scope of this study to account for regional differences, or for regional differences in dietary habits. As an example, individuals consuming a high-fat diet will be exposed to greater quantities of lipophilic organic contaminants, such as PCDD/Fs, and as the fat content of the diet varies regionally, so the average intake of such contaminants vary.

One of the key assumptions is that environmental compartments are in equilibrium, so that the steady-state concentrations in various compartments can be used to define the transfer factors between them, effectively integrating the numerous physical and chemical processes. A number of situations exist where this may not be applicable, e.g., for certain metals, as the total soil concentration increases so the relative availability of the metal decreases. In terms of deriving environmental standards this is not a particular problem, as an increase in soil concentrations may not lead to a corresponding increase in the levels in food crops, and therefore the increase in human exposure may be limited. However, certain parameters such as pH greatly affect the availability of metals in the soil, and decreasing pH in soils will mean that food crops may accumulate certain metals to a greater degree, increasing exposure to the human population from this source.

Further, it is assumed that the environmental levels in different compartments are in equilibrium, but even for contaminants that occur naturally it may be that this assumption is not fully justified. Elevated atmospheric concentrations, even over a long period of time may not be reflected in elevated soil concentrations, for example. In the case of cadmium, which has a very long half-life in the soil (possibly more than 300 years) it is possible that soil concentrations have not yet reached equilibrium with current atmospheric concentrations, and will continue to rise even if there is no further increase in background atmospheric levels (this may be particularly important as cadmium is readily accumulated by food crops). Similarly for PCDD/Fs, increasing soil concentrations may result in higher levels in livestock following consumption of pasture contaminated with soil particles, and higher human exposure through dairy and meat products.

Reliable values for measures of acceptable levels of exposure, such as Tolerable Daily Intakes, are crucial in assessing the risk from background exposure to the general population. Values derived from toxicological experiments or epidemiological studies are still subject to considerable uncertainty, particularly when long-term, low-dose exposures are considered. The TDIs cannot allow for inter-individual variation in response to particular exposures, or greater than additive interactions of exposure to a number of substances.

All routes of exposure should be considered due to the natural variability of the contaminants under consideration, but in this respect there are still a number of restrictions imposed by our current knowledge. For example, indoor air may have considerably higher concentrations of certain contaminants than ambient air, but the lack of available data on indoor levels means that most assessments, including those made in this study, are completed using ambient outdoor air concentrations and therefore may underestimate the contribution from the inhalation pathway, **which may lead to EALs that are not sufficiently stringent to protect human health effectively.**

Similarly, there is very little information on the dermal absorption of environmental contaminants at background concentrations from the atmosphere over long periods of time. Due to the lack of data available it has not been possible to quantify this route for any of the substances under investigation, but in some circumstances this route may be an important source of exposure.

The combination of the original Exposure Commitment method developed by the Monitoring and Assessment Research Centre (MARC), the techniques used by a number of academic

workers (e.g., Duarte Davidson and Jones, 1994; Wild *et al.*, 1994) and the method for the assessment of risk from environmental exposure used by the Canadian authorities, has produced a flexible but clearly defined protocol for the assessment of exposure to environmental contaminants in the UK. The method is far from perfect, and there are still significant gaps in our knowledge, but it is clear that Exposure Commitment can play an important role in the development of environmental regulation in the UK. The key to its use for the derivation of EALs is clearly the quantification of exposure and the comparison of these current estimates with levels of exposure that are considered to be acceptable in toxicological terms for long-term exposure. The proposed protocol also allows comparison of the relative importance of exposure from inhalation of ambient air, the ingestion of soil and food, and from drinking water. Its consistency is vital, but so too is flexibility in its application (e.g., due to variable data availability) so that Exposure Commitment may be applied to the wide range of substances under regulation.

However, the problem of actually measuring environmental levels of contaminants is one of the most important areas of uncertainty when applying the exposure commitment technique. If results are lower than the Limit of Detection, the question of what value should be assigned to the results becomes an important issue. If the LOD is used then this will result in an overestimate of the current environmental levels and therefore will result in higher EALs, which may not be sufficiently stringent to fulfil the regulatory objective. Conversely, if zero is used then the EALs will become too stringent. Advances in analytical techniques, and improved monitoring programmes will help to address this problem, but are unlikely to solve it.

The question of temporal and spatial variation in environmental levels of contaminants also becomes important, particularly given the limited data sets available in terms of extensive geographical coverage and long-term trend analysis of background concentrations of environmental contaminants. In addition, more recent trends in environmental levels have not been accounted for, but in some cases these may take many years to become fully apparent through changes in body burdens and tissue concentrations in the general population.

Despite these limitations, and despite the danger of predicting an EAL that is too high to protect the general population, in nearly all cases the values derived in this study are lower that the environmental standards currently in force.

Usually information from data bases with typical concentrations of a contaminant in different environmental media must be relied upon, and may be collected from scientific literature or national monitoring programmes. As Exposure Commitment can proceed on a relatively limited data base in comparison to dynamic models, it may become highly useful to regulatory authorities when little data are available on certain compounds in the environment.

A number of mathematical models exist which use emission data and the physico-chemical properties of organic compounds to predict environmental levels, and levels in certain key food groups. Whilst there is still uncertainty regarding the accuracy of these models, the data they generate can be used to provide initial indications of exposure using Exposure Commitment, and these coupled with toxicological information may be used to calculate provisional EALs. Environmental monitoring for a wider range of contaminants in the future

is critical and will provide the necessary data to refine provisional EALs. Furthermore, as data on specific compounds become available it may be possible to carry out exposure assessments for those of particular interest or toxicological significance. Data on levels in human tissues and behaviour in the body are, however, crucial to the process of developing EALs from Exposure Commitment.

It has been possible to not only estimate total exposure and identify key sources and crucial periods for lifetime exposure, but also to generate data such as the Lifetime Daily Intake (equivalent to the US EPA Lifetime Average Daily Dose) which can be used for comparison with toxicological data. The identification of critical periods of exposure, e.g., PCDD/Fs during breast-feeding, or mercury exposure to the foetus during pregnancy, also allows a more comprehensive assessment of the risk to the whole population.

Exposure Commitment may also form the basis of assessments for site-specific investigations. For example, if a particular site is releasing a discharge of a substance to a controlled water used for water supply purposes, but the contribution to exposure from drinking water for the general population is negligible, then it is **possible** that the discharge will have little effect on the total human exposure, despite elevated levels in the receiving water. **However, other environmental regulatory objectives will be critical to controlling releases to the environment, and a low risk of human exposure to the general population will be only one of a number of considerations when applying regulatory processes.** (Other routes such as elevated soil and crop concentrations following irrigation should not be ignored). In this way, the Exposure Commitment for the general population can be used to form part of a risk assessment for a target population.

Environmental Assessment Levels have been calculated for five priority pollutants. Good estimates of current exposure have been generated for PCDD/Fs, cadmium, nickel, arsenic and mercury. With the exception of PCDD/Fs **there are reliable data on typical background levels for a number of these substances in most** environmental media. There are also widely-accepted toxicological measures of "tolerable" exposure for PCDD/Fs, cadmium, arsenic and mercury (although the TDI for PCDD/Fs in particular is still the subject of considerable debate). In the absence of a TDI for nickel, a conservative substitute value has been used.

Environmental Assessment Levels calculated for cadmium, arsenic and mercury can be proposed with a reasonable degree of certainty. The use of derived values for background concentrations of PCDD/Fs in environmental media make the EALs for this group of substances less certain, as does the use of a substitute TDI for nickel. These uncertainties may be overcome by environmental monitoring in the first case, and an established TDI in the second case.

It has not been possible to quantify exposure to $\sum$PAHs, or find a measure of acceptable levels of exposure, and so EALs have not been derived. This does, however, highlight the two requirements which are fundamental to deriving EALs using Exposure Commitment: a quantitative estimate of total exposure, and a toxicological measure of acceptable exposure.

The main route of exposure to PCDD/Fs is through the consumption of meat and dairy products, following ingestion of contaminated soil and pasture by livestock, and subsequent

accumulation in their fat tissue. The EALs derived are similar to those from other multimedia assessments, and the assessment clearly demonstrates the usefulness of a Toxic Equivalent system when assessing exposure to a group of chemicals of wide ranging chemical and physical properties. In the absence of any UK standards for PCDD/Fs, the proposed EALs may be useful until further environmental monitoring makes reliable data on background levels available.

Human exposure to cadmium comes mainly from its uptake from soil by plants, and a significant portion of the final exposure originates from direct anthropogenic inputs due to the use of phosphate fertilisers. The proposed EALs are similar to those EALs/EQSs already in force.

The EALs calculated for nickel are considerably lower than the current values. The assessment will be improved if/when a TDI becomes available, and it will then be possible to compare EALs derived by Exposure Commitment with existing standards.

The main sources of exposure to arsenic are drinking water and crops. Given that plants may form an environmental barrier with respect to human exposure (due to the phytotoxicity of arsenic at low concentrations), the drinking water route is of primary concern. The current freshwater EQS is 3 times higher than the proposed EAL. The proposed EAL for air is considerably lower than the current EAL, but the main pathway of concern would appear to be atmospheric deposition on to fresh waters, which **is** regulated by a stringent EQS in fresh water.

The vast majority of human exposure to mercury comes via the consumption of seafood, therefore the EQS for coastal water is of primary concern; the current EQS is higher **than the EAL calculated in this study**. **The proposed EAL for air is also considerably lower than the current EAL.**

It is difficult to make suggestions with respect to the EALs proposed for soil as there are very few environmental standards relating to soil quality. The proposed values may act as guidelines for further investigation. It should be noted that exceedence of an EAL derived from Exposure Commitment in one medium on a local basis may not necessarily lead to intolerable levels of exposure to the population in that area. For instance, if exposure to a substance is mainly derived from seafood of distant origin, and the inhalation pathway contributes only a small fraction of total exposure (e.g., 0.5 per cent) then a local increase in ambient air concentrations **may** not have a significant effect on total lifetime exposure. (If, however, the levels rose to high levels nearing those in cases of occupational exposure, then local toxicological or carcinogenic effects in the lung may need to be considered).

In terms of the feasibility of using Exposure Commitment for deriving EALs for a wide range of compounds, it is apparent that each compound needs to be considered on its own merits, with respect to the availability of data on environmental levels and the toxicological significance. However, given the data available for a number of compounds in the UK diet (fundamental to the assessment of exposure) and other environmental media, it can certainly be considered for the following substances in addition to the five contaminants for which EALs have been derived in this study: copper, zinc, lead, aluminum, antimony, chromium, cobalt, indium, thallium, tin. The use of predictive models and the growing range of organic

compounds monitored in the environment will make derivation of standards using Exposure Commitment suitable for these substances.

Appendix 1.1 MARC Summary Exposure Commitment Assessments

MARC has carried out a number of exposure commitment assessments for a range of environmental pollutants, the main pathway contributions and other key points which arose from the assessments are summarised.

Lead
Receptor – blood: Ingestion 95 per cent; Inhalation 5 per cent.
Approximately 50 per cent of total dietary intake may be due to contamination during food processing. Estimated background levels in blood compared well with general survey measurements from the general population background levels.

Cadmium
Receptor – kidney: Ingestion 99.1 per cent; Inhalation 0.9 per cent.
Estimated a direct source to soil (the addition to soil of cadmium in phosphate fertilisers). Estimated total background levels in kidney tissue compared reasonably with measured levels. Renal damage occurs when levels in the kidney cortex exceed ~200 ug g^{-1} 10 times higher than levels in background exposed individuals. Additional sources of cadmium, e.g., smoking, occupational exposure, may increase these levels significantly.

Arsenic
Receptor – whole body: Ingestion 99.9 per cent; Inhalation 0.1 per cent.
The relationships between concentrations in air, soil and diet were very tentative. Inputs to water from air and direct sources were too uncertain to estimate. The calculated body burden compared reasonably with measured values. Considerable uncertainties in estimates of arsenic intake rates, absorption, distribution and retention in the body tissues, so more data are required before the transfer relationships and exposure commitment can be determined with more confidence.

Mercury
Receptor – whole body: Ingestion (aquatic) 99.6 per cent; Ingestion (terrestrial) 0.3 per cent; Inhalation 0.1 per cent.
Mercury in sediments is converted from the inorganic form to methylmercury which is important with regard to uptake by biota. Inorganic mercury was considered for the inhalation and terrestrial ingestion pathways, and methylmercury for the aquatic ingestion pathway. The estimated average concentration of mercury in the body was much less than the results of tissue analysis. The calculated body burden is quite dependent on the assumed mean effective retention time of methylmercury in the body. Additional measurements of the mercury content in tissues and of the mean residence times would be useful.

Nickel
Receptor – whole body: Ingestion 98.5 per cent; Inhalation 1.5 per cent.
Inferred soil residence time was very uncertain. Systemic effects are associated with nickel carbonyl form, and there are few consistent links between nickel concentration and harmful effects. The assessment dealt with the association between the environment and man of total nickel, but associations between specific nickel compounds may be derived when chemical data on their environmental behaviour are available.

Tin

Receptor – bone: Ingestion 97 per cent; Inhalation 3 per cent.

For the ingestion pathway the association between tin in air and the dietary intake was not well founded. The estimate of tin levels in bone agree with measured levels. Assessment of specific compounds of tin are required, and toxic levels must be better known to complete the assessment of hazard from environmental exposure to tin.

Copper

Receptor – whole body: Ingestion 99.99 per cent; Inhalation 0.01 per cent.

The copper content of drinking water is quite variable so the contribution from this source will vary accordingly. Additional determinations of copper concentrations in food and drinking water, plus measured body burdens would help improve formulation of the exposure commitment for copper.

Vanadium

Receptor – bone: Ingestion 89 per cent; Inhalation 11 per cent.

The estimated contribution to the vanadium content of the body from the inhalation pathway varied due to the difference in background levels of vanadium in rural and urban air. An individual living in a rural environment has a body burden derived mainly from the diet (80 per cent). An individual from an urban environment has an estimated body burden derived approximately equally from diet and inhalation pathways. Estimated body burdens agreed with measured levels.

Antimony

Receptor – liver: Ingestion 99.6 per cent; Inhalation 0.4 per cent.

In the urban environment the estimated contribution to total body burden of the inhalation pathway is ~2 per cent, while in the rural environment this contribution is less than 0.5 per cent. Levels of antimony in food are not well established, so the value used for daily dietary intake is tentative. The estimate of mean retention time in the liver was also provisional. The estimated body burden was comparable to measured levels.

PCBs

Receptor – whole body: Ingestion (terrestrial) 87.1 per cent; Ingestion (aquatic) 12.8 per cent; Inhalation 0.01 per cent; Drinking water <0.001 per cent.

Estimates of transfer factors from representative background levels should be generally relevant and may be applied to more specific cases of exposure. Estimated levels assume continued exposure at levels reported in the past. Levels in the environment and man may well decrease in time due to the reductions in PCB use and release to the environment.

Selenium

Receptor – whole body: Ingestion 99.99 per cent; Inhalation 0.01 per cent.

Soil residence time is only tentatively formed. The estimated mean body concentration compares well with measured levels. Additional data on retention times in soil and the body would reduce the uncertainties, and analysis for specific selenium compounds is required.

Chromium

Receptor – bone: Ingestion 97 per cent; Inhalation 3 per cent.

Estimated body burden was less than normally measured levels. Underestimate of dietary intake or other sources of intake may be the reason for the difference. A major source of uncertainty is the estimated retention time of chromium in the body.

Aluminium

Receptor – bone: Ingestion 99.98 per cent; Inhalation 0.02 per cent.

The estimated body burden was similar to measured levels in normal healthy adults. Some of the associations were tentative and further measurement of environmental levels, dietary intake rate and tissue levels is required.

HCB

Receptor – adipose tissue: Ingestion 99.98 per cent; Inhalation 0.02 per cent.

Diet type is an important factor, as high fat foods will provide the main source of HCB in the diet. The estimated body burden compared well with measured levels. An increased contribution from inhalation in populations near higher emissions would be unlikely to increase the exposure commitment to a high degree. Gaps in the data on distribution of HCB in the human tissues and intake via inhalation have required assumptions to be made.

Appendix 1.2 Food, soil and drinking water consumption rates and breathing rates for various ages

	Adult	7–14 yrs	2–7 yrs	3 mths–2 yrs
Beef	7.38	3.69	1.85	0.92
Pork	4.05	2.03	1.01	0.51
Lamb	3.85	1.93	0.96	0.48
Liver	0.81	0.41	0.20	0.10
Kidney	0.29	0.15	0.07	0.04
Meat products	23.31	11.66	5.83	2.91
Poultry	11.12	5.56	2.78	1.39
Whole milk	46.70	23.35	11.68	5.84
Skimmed and semi-skimmed	48.10	24.05	12.03	6.01
Cheese	5.88	2.94	1.47	0.74
Butter	2.16	1.08	0.54	0.27
Milk products	6.21	3.11	1.55	0.78
Ice cream	4.85	2.43	1.21	0.61
Yoghurt	6.01	3.01	1.50	0.75
Cream	0.89	0.45	0.22	0.11
Vegetable oil	2.39	1.20	0.60	0.30
Animal oil	7.98	3.99	2.00	1.00
Cabbage	10.17	5.09	2.54	1.27
Lettuce	6.32	3.16	1.58	0.79
Peas	11.00	5.50	2.75	1.38
Potatoes	47.37	23.69	11.84	5.92
Onions	13.00	6.50	3.25	1.63
Carrots	13.00	6.50	3.25	1.63
Tomatoes	13.00	6.50	3.25	1.63
Fresh fruit	31.96	15.98	7.99	4.00
Fruit products	16.95	8.48	4.24	2.12
Sugar and preserves	10.61	5.31	2.65	1.33
Bread	39.24	19.62	9.81	4.91
Other cereals	36.63	18.32	9.16	4.58
Beverages	43.07	21.54	10.77	5.38
Freshwater fish	1.64	0.82	0.41	0.21
Marine	4.46	2.23	1.12	0.56
Canned	1.06	0.53	0.27	0.13
Shellfish	0.21	0.11	0.05	0.03
Soil ingestion	0.009	0.018	0.04	0.00
Drinking water	730	365	183	91
Air	8,030	6,000	4,000	1,500

* All consumption rates in kg/yr except: drinking water (litres per year) and air (cubic metres per year).

Appendix 2.1 Concentrations in urban air in the UK (pg/m^3)

	Cardiff	London	Manchester	Stevenage	Mean	Maximum
2,3,7,8-TCDD	0.04	0.03	0.05	0.03	0.04	0.05
1,2,3,7,8-PCDD	0.04	0.04	0.03	0.03	0.04	0.04
1,2,3,4,7,8-HxCDD	0.07	0.04	0.12	0.04	0.07	0.12
1,2,3,6,7,8-HxCDD	0.11	0.04	0.12	0.04	0.08	0.12
1,2,3,7,8,9-HxCDD	0.06	0.04	0.13	0.04	0.07	0.13
1,2,3,4,6,7,8-HpCDD	0.32	0.67	0.51	0.03	0.38	0.67
OCDD	1.50	1.64	1.70	1.20	1.51	1.70
2,3,7,8-TCDF	0.14	0.03	0.14	0.03	0.09	0.14
2,3,4,7,8-PCDF	0.04	0.04	0.06	0.03	0.04	0.06
1,2,3,7,8-PCDF	0.03	0.04	0.19	0.03	0.07	0.19
1,2,3,4,7,8-HxCDF	0.19	0.08	0.31	0.06	0.16	0.31
1,2,3,7,8,9-HxCDF	0.04	0.04	0.04	0.03	0.04	0.04
1,2,3,6,7,8-HxCDF	0.08	0.06	0.95	0.05	0.29	0.95
2,3,4,6,7,8-HxCDF	0.08	0.07	0.13	0.05	0.08	0.13
1,2,3,4,6,7,8-HpCDF	0.28	0.26	0.45	0.21	0.30	0.45
1,2,3,4,7,8,9-HpCDF	0.19	0.06	0.40	0.19	0.21	0.40
OCDF	0.23	0.37	0.23	0.12	0.24	0.37

Data for four sites from Duarte Davidson *et al.*, 1994

Appendix 2.2 Derived concentrations in rural air in the UK (pg/m^3)

2,3,7,8-TCDD	9.38	E-03	2.50	E-03
1,2,3,7,8-PCDD	8.75	E-03	2.33	E-03
1,2,3,4,7,8-HxCDD	1.69	E-02	4.50	E-03
1,2,3,6,7,8-HxCDD	1.94	E-02	5.17	E-03
1,2,3,7,8,9-HxCDD	1.69	E-02	4.50	E-03
1,2,3,4,6,7,8-HpCDD	9.56	E-02	2.55	E-02
OCDD	3.78	E-01	1.01	E-01
2,3,7,8-TCDF	2.13	E-02	5.67	E-03
2,3,4,7,8-PCDF	1.06	E-02	2.83	E-03
1,2,3,7,8-PCDF	1.81	E-02	4.83	E-03
1,2,3,4,7,8-HxCDF	4.00	E-02	1.07	E-02
1,2,3,7,8,9-HxCDF	9.38	E-03	2.50	E-03
1,2,3,6,7,8-HxCDF	7.13	E-02	1.90	E-02
2,3,4,6,7,8-HxCDF	2.06	E-02	5.50	E-03
1,2,3,4,6,7,8-HpCDF	7.50	E-02	2.00	E-02
1,2,3,4,7,8,9-HpCDF	5.25	E-02	1.40	E-02
OCDF	5.94	E-02	1.58	E-02

Appendix 2.3 Concentrations in semi-rural and urban soil in the UK (ng/kg)

	Semi-rural	Urban
2,3,7,8-TCDD	0.094	0.7*
1,2,3,7,8-PCDD	0.27	2.4*
1,2,3,4,7,8-HxCDD	0.35	3.85
1,2,3,6,7,8-HxCDD	0.61	6.71
1,2,3,7,8,9-HxCDD	0.47	5.17
1,2,3,4,6,7,8-HpCDD	5.65	62.15
OCDD	23.41	469*
2,3,7,8-TCDF	1.03	11.33
2,3,4,7,8-PCDF	0.92	10.12
1,2,3,7,8-PCDF	0.54	5.94
1,2,3,4,7,8-HxCDF	0.685	7.535
1,2,3,7,8,9-HxCDF	0.01	0.11
1,2,3,6,7,8-HxCDF	0.84	9.24
2,3,4,6,7,8-HxCDF	0.66	7.26
1,2,3,4,6,7,8-HpCDF	4.25	46.75
1,2,3,4,7,8,9-HpCDF	0.42	4.62
OCDF	5.2	40*

* Congener-specific data for urban soil were available only for 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, OCDD and OCDF (Creaser *et al.*, 1990). The levels for the other congeners in urban soils were derived by multiplying the semi rural concentrations by a factor of 11.

Data for semi rural soils are from Kjeller *et al.*, 1991

Appendix 2.4 Concentrations in treated drinking water and raw river water (from Sweden) (pg/m^3)

	River water	Predicted	Drinking water
2,3,7,8-TCDD	22	3	23
1,2,3,7,8-PCDD	32	4	19
1,2,3,4,7,8-HxCDD	34	5	27
1,2,3,6,7,8-HxCDD	67	9	24
1,2,3,7,8,9-HxCDD	45	6	29
1,2,3,4,6,7,8-HpCDD	225	31	120
OCDD	1,395	190	170
2,3,7,8-TCDF	24	3	17
2,3,4,7,8-PCDF	16	2	14
1,2,3,7,8-PCDF	20	3	11
1,2,3,4,7,8-HxCDF	24	3	24
1,2,3,7,8,9-HxCDF	18	2	29
1,2,3,6,7,8-HxCDF	22	3	23
2,3,4,6,7,8-HxCDF	20	3	24
1,2,3,4,6,7,8-HpCDF	107	15	11
1,2,3,4,7,8,9-HpCDF	44	6	30
OCDF	255	35	59

Note: The drinking water sample was filtered through a charcoal bed before chlorination. The data for the raw river water are the mean levels measured in two Swedish rivers.

Data taken from Rappe *et al.*, 1989

Appendix 2.5 Congener-specific PCDD/F levels in foods and environmental media, and intake data (adults)

Congener	TEF	Food Types – Consumption (kg/yr) and PCDD/F Levels (ng/kg)															
		Meat	Offal	Meat products	Poultry	Whole milk	Skimmed milk	Cheese	Butter	Milk products	Ice cream	Cream	Eggs	Fats & oils	Green vegetables	Potatoes	Root vegetables
		15.28	**1.10**	**23.31**	**11.12**	**50.24**	**48.07**	**5.88**	**2.16**	**6.21**	**10.85**	**0.89**	**9.51**	**10.38**	**27.49**	**47.41**	**39.00**
2,3,7,8-TCDD	1	0.17	0.12	0.04	0.10	0.02	0.01	0.05	0.15	0.06	0.002	0.15	0.05	0.21	0.01	0.01	0.01
1,2,3,7,8-PeCDD	0.5	0.24	0.16	0.06	0.10	0.04	0.02	0.04	0.41	0.11	0.001	0.07	0.08	0.29	0.01	0.01	0.01
1,2,3,4,7,8-HxCDD	0.1	0.17	0.08	0.05	0.16	0.03	0.01	0.03	0.25	0.07	0.001	0.07	0.08	0.13	0.00	0.01	0.03
1,2,3,6,7,8-HxCDD	0.1	0.54	0.26	0.16	0.48	0.06	0.02	0.07	0.58	0.16	0.002	0.14	0.18	0.28	0.01	0.02	0.08
1,2,3,7,8,9-HxCDD	0.1	0.26	0.05	0.05	0.09	0.03	0.01	0.04	0.23	0.07	0.002	0.07	0.06	0.23	0.01	0.01	0.02
1,2,3,4,6,7,8-HpCDD	0.01	4.25	5.35	1.70	2.50	0.25	0.10	0.47	1.94	0.41	0.010	0.45	0.85	4.50	0.13	0.18	0.44
OCDD	0.001	8.80	26.00	16.50	3.50	1.00	0.72	2.40	3.70	2.50	0.050	3.15	3.80	35.00	0.91	1.30	2.20
2,3,7,8-TCDF	0.1	0.23	0.06	0.11	0.19	0.01	0.01	0.04	0.09	0.07	0.005	0.29	0.11	0.24	0.02	0.03	0.13
2,3,4,7,8-PeCDF	0.5	0.13	0.40	0.06	0.07	0.05	0.02	0.10	0.89	0.10	0.003	1.10	0.08	0.55	0.01	0.02	0.04
1,2,3,7,8-PeCDF	0.05	0.29	0.01	0.07	0.04	0.01	0.01	0.01	0.05	0.03	0.001	0.17	0.02	0.11	0.01	0.01	0.01
1,2,3,4,7,8-HxCDF	0.1	0.24	0.24	0.10	0.07	0.03	0.01	0.05	0.46	0.04	0.002	1.05	0.07	0.21	0.01	0.01	0.05
1,2,3,7,8,9-HxCDF	0.1	0.21	0.25	0.05	0.04	0.01	0.01	0.02	0.03	0.02	0.001	0.04	0.01	0.07	0.01	0.01	0.01
1,2,3,6,7,8-HxCDF	0.1	0.21	0.01	0.05	0.04	0.02	0.01	0.03	0.45	0.04	0.001	0.24	0.04	0.20	0.01	0.01	0.01
2,3,4,6,7,8-HxCDF	0.1	0.23	0.25	0.04	0.05	0.02	0.01	0.04	0.43	0.04	0.002	0.06	0.03	0.17	0.01	0.01	0.01
1,2,3,4,6,7,8-HpCDF	0.01	0.31	3.85	0.22	1.11	0.04	0.02	0.10	0.33	0.18	0.004	0.07	0.11	0.99	0.04	0.03	0.16
1,2,3,4,7,8,9-HpCDF	0.01	0.26	0.12	0.02	0.06	0.01	0.01	0.03	0.05	0.05	0.001	0.07	0.03	0.13	0.01	0.01	0.02
OCDF	0.001	5.58	1.50	2.65	0.52	0.43	0.02	0.10	0.13	0.32	0.006	0.07	0.04	2.30	0.04	0.13	0.17
Total PCDD/F (ng/kg)		22.1	38.7	21.9	9.1	2.1	1.0	3.6	10.1	4.2	0.1	7.2	5.6	45.6	1.2	1.8	3.4
TEQ (ng/kg)		0.63	0.64	0.20	0.34	0.09	0.04	0.16	1.07	0.22	0.01	0.94	0.19	0.88	0.03	0.04	0.08
Intake (ng/yr)		337	43	511	101	103	49	21	22	26	1	6	53	473	34	86	132
Intake (ng TEQ/yr)		9.6	0.7	4.8	3.7	4.6	2.0	0.9	2.3	1.4	0.1	0.8	1.9	9.2	0.9	1.9	3.1
Contribution (TEQ)		15.2%	1.1%	7.6%	5.9%	7.2%	3.2%	1.5%	3.7%	2.1%	0.1%	1.3%	2.9%	14.5%	1.4%	3.0%	4.9%

Appendix 2.5 (cont.) Congener-specific PCDD/F levels in foods and environmental media, and intake data (adults)

	Fruit & products	Sugar & preserves	Bread & cereals	Beverages	Fish	Soil	Water (ng/l)	Air (ng/m^3)	Congener-specific Intake					TEQ Intake	
									Diet (ng/yr)	Soil (ng/yr)	Water (ng/yr)	Air (ng/yr)	Total (ng/yr)	Total (ng TEQ/yr)	Contribution to total TEQ (%)
	48.88	**10.61**	**75.87**	**43.07**	**7.37**	**0.009**	**731**	**8,030**							
2,3,7,8-TCDD	0.01	0.01	0.01	0.001	0.13	0.50	2.3 E-05	5.0 E-05	**13.7**	**0.0**	**0.02**	**0.4**	**14**	**14.1**	**22.4**
1,2,3,7,8-PeCDD	0.01	0.01	0.02	0.001	0.20	0.50	1.9 E-05	4.0 E-05	**19.4**	**0.0**	**0.01**	**0.3**	**20**	**9.9**	**15.6**
1,2,3,4,7,8-HxCDD	0.02	0.02	0.01	0.000	0.04	0.37	2.7 E-05	1.2 E-04	**14.0**	**0.0**	**0.02**	**1.0**	**15**	**1.5**	**2.4**
1,2,3,6,7,8-HxCDD	0.04	0.04	0.02	0.001	0.08	0.62	2.4 E-05	1.2 E-04	**37.5**	**0.0**	**0.02**	**1.0**	**38**	**3.8**	**6.1**
1,2,3,7,8,9-HxCDD	0.03	0.03	0.01	0.001	0.04	0.48	2.9 E-05	1.3 E-04	**16.6**	**0.0**	**0.02**	**1.0**	**18**	**1.8**	**2.8**
1,2,3,4,6,7,8-HpCDD	0.40	0.40	0.26	0.011	0.32	27.00	1.2 E-04	6.7 E-04	**295.1**	**0.2**	**0.09**	**5.4**	**301**	**3.0**	**4.8**
OCDD	2.45	2.45	1.98	0.078	2.62	1.43	1.7 E-04	1.7 E-03	**1603.1**	**0.0**	**0.12**	**13.7**	**1,617**	**1.6**	**2.6**
2,3,7,8-TCDF	0.08	0.08	0.01	0.002	1.21	0.95	1.7 E-05	1.4 E-04	**35.2**	**0.0**	**0.01**	**1.1**	**36**	**3.6**	**5.8**
2,3,4,7,8-PeCDF	0.03	0.03	0.03	0.001	0.66	0.93	1.4 E-05	6.0 E-05	**30.0**	**0.0**	**0.01**	**0.5**	**31**	**15.3**	**24.2**
1,2,3,7,8-PeCDF	0.02	0.02	0.01	0.001	0.18	1.06	1.1 E-05	1.9 E-04	**13.0**	**0.0**	**0.01**	**1.5**	**15**	**0.7**	**1.2**
1,2,3,4,7,8-HxCDF	0.02	0.02	0.01	0.001	0.08	1.27	2.4 E-05	3.1 E-04	**19.6**	**0.0**	**0.02**	**2.5**	**22**	**2.2**	**3.5**
1,2,3,7,8,9-HxCDF	0.01	0.01	0.00	0.001	0.04	0.01	2.9 E-05	4.0 E-05	**9.5**	**0.0**	**0.02**	**0.3**	**10**	**1.0**	**1.6**
1,2,3,6,7,8-HxCDF	0.01	0.01	0.01	0.001	0.07	0.82	2.3 E-05	9.5 E-04	**13.3**	**0.0**	**0.02**	**7.6**	**21**	**2.1**	**3.3**
2,3,4,6,7,8-HxCDF	0.01	0.01	0.01	0.001	0.08	0.73	2.4 E-05	1.3 E-04	**13.2**	**0.0**	**0.02**	**1.0**	**14**	**1.4**	**2.3**
1,2,3,4,6,7,8-HpCDF	0.06	0.06	0.06	0.003	0.09	4.13	1.1 E-05	4.5 E-04	**60.5**	**0.0**	**0.01**	**3.6**	**64**	**0.6**	**1.0**
1,2,3,4,7,8,9 HpCDF	0.01	0.01	0.01	0.001	0.09	0.38	3.0 E-05	4.0 E-04	**11.7**	**0.0**	**0.02**	**3.2**	**15**	**0.1**	**0.2**
OCDF	0.17	0.17	0.13	0.003	0.20	15.00	5.9 E-05	3.7 E-04	**239.4**	**0.1**	**0.04**	**3.0**	**243**	**0.2**	**0.4**
Total PCDD/F (ng/kg)	3.3	3.3	2.6	0.1	6.1	56.2	0.001	0.006							
TEQ (ng/kg)	0.06	0.06	0.05	0.003	0.74	2.12	6.2 E-05	3.2 E-04							
Intake (ng/yr)	164	35	196	5	45	0.5	0.5	47							
Intake (ng TEQ/yr)	2.7	0.6	3.8	0.1	5.5	0.0	0.0	2.6	60.4	0.0	0.0	2.6	**63.1**		
Contribution (TEQ)	4.3%	0.9%	6.0%	0.2%	8.7%	0.03%	0.07%	4.1%	**95.8%**	**0.03%**	**0.1%**	**4.1%**	**100%**		

Appendix 2.6 Congener-specific PCDD/F levels in foods and environmental media, and intake data – children (7–14 yrs)

		Food Types – Consumption (kg/yr) and PCDD/F levels (ng/kg)															
		Meat	Offal	Meat products	Poultry	Whole milk	Skimmed milk	Cheese	Butter	Milk products	Ice cream & yoghurt	Cream	Eggs	Fats & oils	Green vegetables	Potatoes	Root vegetables
		7.64	**0.55**	**11.66**	**5.56**	**25.12**	**24.04**	**2.94**	**1.08**	**3.11**	**5.43**	**0.45**	**4.76**	**5.19**	**13.75**	**23.71**	**19.50**
2,3,7,8-TCDD	1	0.17	0.12	0.04	0.10	0.02	0.01	0.05	0.15	0.06	0.002	0.15	0.05	0.21	0.01	0.01	0.01
1,2,3,7,8-PeCDD	0.5	0.24	0.16	0.06	0.10	0.04	0.02	0.04	0.41	0.11	0.001	0.07	0.08	0.29	0.01	0.01	0.01
1,2,3,4,7,8-HxCDD	0.1	0.17	0.08	0.05	0.16	0.03	0.01	0.03	0.25	0.07	0.001	0.07	0.08	0.13	0.00	0.01	0.03
1,2,3,6,7,8-HxCDD	0.1	0.54	0.26	0.16	0.48	0.06	0.02	0.07	0.58	0.16	0.002	0.14	0.18	0.28	0.01	0.02	0.08
1,2,3,7,8,9-HxCDD	0.1	0.26	0.05	0.05	0.09	0.03	0.01	0.04	0.23	0.07	0.002	0.07	0.06	0.23	0.01	0.01	0.02
1,2,3,4,6,7,8-HpCDD	0.01	4.25	5.35	1.70	2.50	0.25	0.10	0.47	1.94	0.41	0.010	0.45	0.85	4.50	0.13	0.18	0.44
OCDD	0.001	8.80	26.00	16.50	3.50	1.00	0.72	2.40	3.70	2.50	0.050	3.15	3.80	35.00	0.91	1.30	2.20
2,3,7,8-TCDF	0.1	0.23	0.06	0.11	0.19	0.01	0.01	0.04	0.09	0.07	0.005	0.29	0.11	0.24	0.02	0.03	0.13
2,3,4,7,8-PeCDF	0.5	0.13	0.40	0.06	0.07	0.05	0.02	0.10	0.89	0.10	0.003	1.10	0.08	0.55	0.01	0.02	0.04
1,2,3,7,8-PeCDF	0.05	0.29	0.01	0.07	0.04	0.01	0.01	0.01	0.05	0.03	0.001	0.17	0.02	0.11	0.01	0.01	0.01
1,2,3,4,7,8-HxCDF	0.1	0.24	0.24	0.10	0.07	0.03	0.01	0.05	0.46	0.04	0.002	1.05	0.07	0.21	0.01	0.01	0.05
1,2,3,7,8,9-HxCDF	0.1	0.21	0.25	0.05	0.04	0.01	0.01	0.02	0.03	0.02	0.001	0.04	0.01	0.07	0.01	0.01	0.01
1,2,3,6,7,8-HxCDF	0.1	0.21	0.01	0.05	0.04	0.02	0.01	0.03	0.45	0.04	0.001	0.24	0.04	0.20	0.01	0.01	0.01
2,3,4,6,7,8-HxCDF	0.1	0.23	0.25	0.04	0.05	0.02	0.01	0.04	0.43	0.04	0.002	0.06	0.03	0.17	0.01	0.01	0.01
1,2,3,4,6,7,8-HpCDF	0.01	0.31	3.85	0.22	1.11	0.04	0.02	0.10	0.33	0.18	0.004	0.07	0.11	0.99	0.04	0.03	0.16
1,2,3,4,7,8,9-HpCDF	0.01	0.26	0.12	0.02	0.06	0.01	0.01	0.03	0.05	0.05	0.001	0.07	0.03	0.13	0.01	0.01	0.02
OCDF	0.001	5.58	1.50	2.65	0.52	0.43	0.02	0.10	0.13	0.32	0.006	0.07	0.04	2.30	0.04	0.13	0.17
Total PCDD/F (ng/kg)		22.1	38.7	21.9	9.1	2.1	1.0	3.6	10.1	4.2	0.1	7.2	5.6	45.6	1.2	1.8	3.4
TEQ (ng/kg)		0.63	0.64	0.20	0.34	0.09	0.04	0.16	1.07	0.22	0.01	0.94	0.19	0.88	0.03	0.04	0.08
Intake (ng/yr)		169	21	255	51	52	25	11	11	13	1	3	27	237	17	43	66
Intake (ng TEQ/yr)		4.8	0.3	2.4	1.9	2.3	1.0	0.5	1.2	0.7	0.0	0.4	0.9	4.6	0.4	1.0	1.5
Contribution (TEQ)		14.9%	1.1%	7.4%	5.8%	7.1%	3.1%	1.5%	3.6%	2.1%	0.1%	1.3%	2.9%	14.2%	1.4%	3.0%	4.8%

Appendix 2.6 (cont.) Congener-specific PCDD/F levels in foods and environmental media, and intake data - child (7-14 yrs)

	Fruit & products	Sugar & preserves	Bread & cereals	Beverages	Fish	Soil	Water (ng/l)	Air (ng/m^3)	Congener-specific Intake				TEQ Intake		
									Diet (ng/yr)	Soil (ng/yr)	Water (ng/yr)	Air (ng/yr)	Total (ng/yr)	Total (ng TEQ/yr)	Contribution to total TEQ (%)
	24.44	**5.31**	**37.94**	**21.54**	**3.69**	**0.018**	**365**	**6,000**							
2,3,7,8-TCDD	0.01	0.01	0.01	0.001	0.13	0.50	2.3 E-05	5.0 E-05	**6.8**	**0.0**	**0.01**	**0.3**	**7**	**7.2**	**22.2**
1,2,3,7,8-PeCDD	0.01	0.01	0.02	0.001	0.20	0.50	1.9 E-05	4.0 E-05	**9.7**	**0.0**	**0.01**	**0.2**	**10**	**5.0**	**15.4**
1,2,3,4,7,8-HxCDD	0.02	0.02	0.01	0.000	0.04	0.37	2.7 E-05	1.2 E-04	**7.0**	**0.0**	**0.01**	**0.7**	**8**	**0.8**	**2.4**
1,2,3,6,7,8-HxCDD	0.04	0.04	0.02	0.001	0.08	0.62	2.4 E-05	1.2 E-04	**18.7**	**0.0**	**0.01**	**0.7**	**19**	**1.9**	**6.0**
1,2,3,7,8,9-HxCDD	0.03	0.03	0.01	0.001	0.04	0.48	2.9 E-05	1.3 E-04	**8.3**	**0.0**	**0.01**	**0.8**	**9**	**0.9**	**2.8**
1,2,3,4,6,7,8-HpCDD	0.40	0.40	0.26	0.011	0.32	27.00	1.2 E-04	6.7 E-04	**147.6**	**0.5**	**0.04**	**4.0**	**152**	**1.5**	**4.7**
OCDD	2.45	2.45	1.98	0.078	2.62	1.43	1.7 E-04	1.7 E-03	**801.7**	**0.0**	**0.06**	**10.2**	**812**	**0.8**	**2.5**
2,3,7,8-TCDF	0.08	0.08	0.01	0.002	1.21	0.95	1.7 E-05	1.4 E-04	**17.6**	**0.0**	**0.01**	**0.8**	**18**	**1.8**	**5.7**
2,3,4,7,8-PeCDF	0.03	0.03	0.03	0.001	0.66	0.93	1.4 E-05	6.0 E-05	**15.0**	**0.0**	**0.01**	**0.4**	**15**	**7.7**	**23.9**
1,2,3,7,8-PeCDF	0.02	0.02	0.01	0.001	0.18	1.06	1.1 E-05	1.9 E-04	**6.5**	**0.0**	**0.00**	**1.1**	**8**	**0.4**	**1.2**
1,2,3,4,7,8-HxCDF	0.02	0.02	0.01	0.001	0.08	1.27	2.4 E-05	3.1 E-04	**9.8**	**0.0**	**0.01**	**1.9**	**12**	**1.2**	**3.6**
1,2,3,7,8,9-HxCDF	0.01	0.01	0.00	0.001	0.04	0.01	2.9 E-05	4.0 E-05	**4.7**	**0.0**	**0.01**	**0.2**	**5**	**0.5**	**1.5**
1,2,3,6,7,8-HxCDF	0.01	0.01	0.01	0.001	0.07	0.82	2.3 E-05	9.5 E-04	**6.7**	**0.0**	**0.01**	**5.7**	**12**	**1.2**	**3.8**
2,3,4,6,7,8-HxCDF	0.01	0.01	0.01	0.001	0.08	0.73	2.4 E-05	1.3 E-04	**6.6**	**0.0**	**0.01**	**0.8**	**7**	**0.7**	**2.3**
1,2,3,4,6,7,8-HpCDF	0.06	0.06	0.06	0.003	0.09	4.13	1.1 E-05	4.5 E-04	**30.2**	**0.1**	**0.00**	**2.7**	**33**	**0.3**	**1.0**
1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.01	0.001	0.09	0.38	3.0 E-05	4.0 E-04	**5.8**	**0.0**	**0.01**	**2.4**	**8**	**0.1**	**0.3**
OCDF	0.17	0.17	0.13	0.003	0.20	15.00	5.9 E-05	3.7 E-04	**119.7**	**0.3**	**0.02**	**2.2**	**122**	**0.1**	**0.4**
Total PCDD/F (ng/kg)	3.3	3.3	2.6	0.1	6.1	56.2	0.001	0.006							
TEQ (ng/kg)	0.06	0.06	0.05	0.003	0.74	2.12	6.2 E-05	3.2 E-04							
Intake (ng/yr)	82	18	98	2	23	1.0	0.2	35							
Intake (ng TEQ/yr)	1.4	0.3	1.9	0.1	2.7	0.0	0.0	1.9	30.2	0.0	0.0	1.9	**32.2**		
Contribution (TEQ)	4.3%	0.9%	5.9%	0.2%	8.5%	0.12%	0.07%	6.0%	**93.8%**	**0.1%**	**0.1%**	**6.0%**	**100%**		

Appendix 2.7 Congener-specific PCDD/F levels in foods and environmental media, and intake data – children (2–7 years)

		Food Types – Consumption (kg/yr) and PCDD/F Levels (ng/kg)															
		Meat	Offal	Meat products	Poultry	Whole milk	Skimmed milk	Cheese	Butter	Milk products	Ice cream & yoghurt	Cream	Eggs	Fats & oils	Green vegetables	Potatoes	Root vegetables
		3.82	**0.28**	**5.83**	**2.78**	**12.56**	**12.02**	**1.47**	**0.54**	**1.56**	**2.72**	**0.23**	**2.38**	**2.60**	**6.88**	**11.86**	**9.75**
2,3,7,8-TCDD	1	0.17	0.12	0.04	0.10	0.02	0.01	0.05	0.15	0.06	0.002	0.15	0.05	0.21	0.01	0.01	0.01
1,2,3,7,8-PeCDD	0.5	0.24	0.16	0.06	0.10	0.04	0.02	0.04	0.41	0.11	0.001	0.07	0.08	0.29	0.01	0.01	0.01
1,2,3,4,7,8-HxCDD	0.1	0.17	0.08	0.05	0.16	0.03	0.01	0.03	0.25	0.07	0.001	0.07	0.08	0.13	0.00	0.01	0.03
1,2,3,6,7,8-HxCDD	0.1	0.54	0.26	0.16	0.48	0.06	0.02	0.07	0.58	0.16	0.002	0.14	0.18	0.28	0.01	0.02	0.08
1,2,3,7,8,9-HxCDD	0.1	0.26	0.05	0.05	0.09	0.03	0.01	0.04	0.23	0.07	0.002	0.07	0.06	0.23	0.01	0.01	0.02
1,2,3,4,6,7,8-HpCDD	0.01	4.25	5.35	1.70	2.50	0.25	0.10	0.47	1.94	0.41	0.010	0.45	0.85	4.50	0.13	0.18	0.44
OCDD	0.001	8.80	26.00	16.50	3.50	1.00	0.72	2.40	3.70	2.50	0.050	3.15	3.80	35.00	0.91	1.30	2.20
2,3,7,8-TCDF	0.1	0.23	0.06	0.11	0.19	0.01	0.01	0.04	0.09	0.07	0.005	0.29	0.11	0.24	0.02	0.03	0.13
2,3,4,7,8-PeCDF	0.5	0.13	0.40	0.06	0.07	0.05	0.02	0.10	0.89	0.10	0.003	1.10	0.08	0.55	0.01	0.02	0.04
1,2,3,7,8-PeCDF	0.05	0.29	0.01	0.07	0.04	0.01	0.01	0.01	0.05	0.03	0.001	0.17	0.02	0.11	0.01	0.01	0.01
1,2,3,4,7,8-HxCDF	0.1	0.24	0.24	0.10	0.07	0.03	0.01	0.05	0.46	0.04	0.002	1.05	0.07	0.21	0.01	0.01	0.05
1,2,3,7,8,9-HxCDF	0.1	0.21	0.25	0.05	0.04	0.01	0.01	0.02	0.03	0.02	0.001	0.04	0.01	0.07	0.01	0.01	0.01
1,2,3,6,7,8-HxCDF	0.1	0.21	0.01	0.05	0.04	0.02	0.01	0.03	0.45	0.04	0.001	0.24	0.04	0.20	0.01	0.01	0.01
2,3,4,6,7,8-HxCDF	0.1	0.23	0.25	0.04	0.05	0.02	0.01	0.04	0.43	0.04	0.002	0.06	0.03	0.17	0.01	0.01	0.01
1,2,3,4,6,7,8-HpCDF	0.01	0.31	3.85	0.22	1.11	0.04	0.02	0.10	0.33	0.18	0.004	0.07	0.11	0.99	0.04	0.03	0.16
1,2,3,4,7,8,9-HpCDF	0.01	0.26	0.12	0.02	0.06	0.01	0.01	0.03	0.05	0.05	0.001	0.07	0.03	0.13	0.01	0.01	0.02
OCDF	0.001	5.58	1.50	2.65	0.52	0.43	0.02	0.10	0.13	0.32	0.006	0.07	0.04	2.30	0.04	0.13	0.17
Total PCDD/F (ng/kg)		22.1	38.7	21.9	9.1	2.1	1.0	3.6	10.1	4.2	0.1	7.2	5.6	45.6	1.2	1.8	3.4
TEQ (ng/kg)		0.63	0.64	0.20	0.34	0.09	0.04	0.16	1.07	0.22	0.01	0.94	0.19	0.88	0.03	0.04	0.08
Intake (ng/yr)		84	11	128	25	26	12	5	5	7	0	2	13	119	9	21	33
Intake (ng TEQ/yr)		2.4	0.2	1.2	0.9	1.1	0.5	0.2	0.6	0.3	0.0	0.2	0.5	2.3	0.2	0.5	0.8
Contribution (TEQ)		14.5%	1.1%	7.2%	5.7%	6.9%	3.0%	1.4%	3.5%	2.1%	0.1%	1.3%	2.8%	13.9%	1.3%	2.9%	4.6%

Appendix 2.7 (cont.) Congener-specific PCDD/F levels in foods and environmental media, and intake data – children (2–7 years)

	Fruit & products	Sugar & preserves	Bread & cereals	Beverages	Fish	Soil	Water (ng/l)	Air (ng/m^3)	Congener-specific Intake					TEQ Intake	
									Diet (ng/yr)	Soil (ng/yr)	Water (ng/yr)	Air (ng/yr)	Total (ng/yr)	Total (ng TEQ/yr)	Contribution to total TEQ (%)
	12.22	**2.66**	**18.97**	**10.77**	**1.85**	**0.037**	**183**	**4000**							
2,3,7,8-TCDD	0.01	0.01	0.01	0.001	0.13	0.50	2.3 E-05	5.0 E-05	**3.4**	**0.0**	**0.00**	**0.2**	**4**	**3.6**	**22.1**
1,2,3,7,8-PeCDD	0.01	0.01	0.02	0.001	0.20	0.50	1.9 E-05	4.0 E-05	**4.9**	**0.0**	**0.00**	**0.2**	**5**	**2.5**	**15.2**
1,2,3,4,7,8-HxCDD	0.02	0.02	0.01	0.000	0.04	0.37	2.7 E-05	1.2 E-04	**3.5**	**0.0**	**0.00**	**0.5**	**4**	**0.4**	**2.4**
1,2,3,6,7,8-HxCDD	0.04	0.04	0.02	0.001	0.08	0.62	2.4 E-05	1.2 E-04	**9.4**	**0.0**	**0.00**	**0.5**	**10**	**1.0**	**6.0**
1,2,3,7,8,9-HxCDD	0.03	0.03	0.01	0.001	0.04	0.48	2.9 E-05	1.3 E-04	**4.2**	**0.0**	**0.01**	**0.5**	**5**	**0.5**	**2.8**
1,2,3,4,6,7,8-HpCDD	0.40	0.40	0.26	0.011	0.32	27.00	1.2 E-04	6.7 E-04	**73.9**	**1.0**	**0.02**	**2.7**	**78**	**0.8**	**4.7**
OCDD	2.45	2.45	1.98	0.078	2.62	1.43	1.7 E-04	1.7 E-03	**401.2**	**0.1**	**0.03**	**6.8**	**408**	**0.4**	**2.5**
2,3,7,8-TCDF	0.08	0.08	0.01	0.002	1.21	0.95	1.7 E-05	1.4 E-04	**8.8**	**0.0**	**0.00**	**0.6**	**9**	**0.9**	**5.7**
2,3,4,7,8-PeCDF	0.03	0.03	0.03	0.001	0.66	0.93	1.4 E-05	6.0 E-05	**7.5**	**0.0**	**0.00**	**0.2**	**8**	**3.9**	**23.6**
1,2,3,7,8-PeCDF	0.02	0.02	0.01	0.001	0.18	1.06	1.1 E-05	1.9 E-04	**3.3**	**0.0**	**0.00**	**0.8**	**4**	**0.2**	**1.2**
1,2,3,4,7,8-HxCDF	0.02	0.02	0.01	0.001	0.08	1.27	2.4 E-05	3.1 E-04	**4.9**	**0.0**	**0.00**	**1.2**	**6**	**0.6**	**3.8**
1,2,3,7,8,9-HxCDF	0.01	0.01	0.00	0.001	0.04	0.01	2.9 E-05	4.0 E-05	**2.4**	**0.0**	**0.01**	**0.2**	**3**	**0.3**	**1.5**
1,2,3,6,7,8-HxCDF	0.01	0.01	0.01	0.001	0.07	0.82	2.3 E-05	9.5 E-04	**3.3**	**0.0**	**0.00**	**3.8**	**7**	**0.7**	**4.3**
2,3,4,6,7,8-HxCDF	0.01	0.01	0.01	0.001	0.08	0.73	2.4 E-05	1.3 E-04	**3.3**	**0.0**	**0.00**	**0.5**	**4**	**0.4**	**2.3**
1,2,3,4,6,7,8-HpCDF	0.06	0.06	0.06	0.003	0.09	4.13	1.1 E-05	4.5 E-04	**15.1**	**0.2**	**0.00**	**1.8**	**17**	**0.2**	**1.0**
1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.01	0.001	0.09	0.38	3.0 E-05	4.0 E-04	**2.9**	**0.0**	**0.01**	**1.6**	**5**	**0.0**	**0.3**
OCDF	0.17	0.17	0.13	0.003	0.20	15.00	5.9 E-05	3.7 E-04	**59.9**	**0.6**	**0.01**	**1.5**	**62**	**0.1**	**0.4**
Total PCDD/F (ng/kg)	3.3	3.3	2.6	0.1	6.1	56.2	0.001	0.006							
TEQ (ng/kg)	0.06	0.06	0.05	0.003	0.74	2.12	6.2 E-05	3.2 E-04							
Intake (ng/yr)	41	9	49	1	11	2.1	0.1	23							
Intake (ng TEQ/yr)	0.7	0.1	0.9	0.0	1.4	0.1	0.0	1.3	15.1	0.1	0.0	1.3	**16.5**		
Contribution (TEQ)	4.1%	0.9%	5.7%	0.2%	8.3%	0.48%	0.07%	7.8%	**91.7%**	**0.5%**	**0.1%**	**7.8%**	**100%**		

Appendix 2.8 Congener-specific PCDD/F levels in foods and environmental media, and intake data – children (3 mths–2 yrs)

		Food Types – Consumption (kg/yr) and PCDD/F Levels (ng/kg)															
		Meat	Offal	Meat products	Poultry	Whole milk	Skimmed milk	Cheese	Butter	Milk products	Ice cream & yoghurt	Cream	Eggs	Fats & oils	Green vegetables	Potatoes	Root vegetables
		1.91	**0.14**	**2.92**	**1.39**	**6.28**	**6.01**	**0.74**	**0.27**	**0.78**	**1.36**	**0.12**	**1.19**	**1.30**	**3.44**	**5.93**	**4.88**
2,3,7,8-TCDD	1	0.17	0.12	0.04	0.10	0.02	0.01	0.05	0.15	0.06	0.002	0.15	0.05	0.21	0.01	0.01	0.01
1,2,3,7,8-PeCDD	0.5	0.24	0.16	0.06	0.10	0.04	0.02	0.04	0.41	0.11	0.001	0.07	0.08	0.29	0.01	0.01	0.01
1,2,3,4,7,8-HxCDD	0.1	0.17	0.08	0.05	0.16	0.03	0.01	0.03	0.25	0.07	0.001	0.07	0.08	0.13	0.00	0.01	0.03
1,2,3,6,7,8-HxCDD	0.1	0.54	0.26	0.16	0.48	0.06	0.02	0.07	0.58	0.16	0.002	0.14	0.18	0.28	0.01	0.02	0.08
1,2,3,7,8,9-HxCDD	0.1	0.26	0.05	0.05	0.09	0.03	0.01	0.04	0.23	0.07	0.002	0.07	0.06	0.23	0.01	0.01	0.02
1,2,3,4,6,7,8-HpCDD	0.01	4.25	5.35	1.70	2.50	0.25	0.10	0.47	1.94	0.41	0.010	0.45	0.85	4.50	0.13	0.18	0.44
OCDD	0.001	8.80	26.00	16.50	3.50	1.00	0.72	2.40	3.70	2.50	0.050	3.15	3.80	35.00	0.91	1.30	2.20
2,3,7,8-TCDF	0.1	0.23	0.06	0.11	0.19	0.01	0.01	0.04	0.09	0.07	0.005	0.29	0.11	0.24	0.02	0.03	0.13
2,3,4,7,8-PeCDF	0.5	0.13	0.40	0.06	0.07	0.05	0.02	0.10	0.89	0.10	0.003	1.10	0.08	0.55	0.01	0.02	0.04
1,2,3,7,8-PeCDF	0.05	0.29	0.01	0.07	0.04	0.01	0.01	0.01	0.05	0.03	0.001	0.17	0.02	0.11	0.01	0.01	0.01
1,2,3,4,7,8-HxCDF	0.1	0.24	0.24	0.10	0.07	0.03	0.01	0.05	0.46	0.04	0.002	1.05	0.07	0.21	0.01	0.01	0.05
1,2,3,7,8,9-HxCDF	0.1	0.21	0.25	0.05	0.04	0.01	0.01	0.02	0.03	0.02	0.001	0.04	0.01	0.07	0.01	0.01	0.01
1,2,3,6,7,8-HxCDF	0.1	0.21	0.01	0.05	0.04	0.02	0.01	0.03	0.45	0.04	0.001	0.24	0.04	0.20	0.01	0.01	0.01
2,3,4,6,7,8-HxCDF	0.1	0.23	0.25	0.04	0.05	0.02	0.01	0.04	0.43	0.04	0.002	0.06	0.03	0.17	0.01	0.01	0.01
1,2,3,4,6,7,8-HpCDF	0.01	0.31	3.85	0.22	1.11	0.04	0.02	0.10	0.33	0.18	0.004	0.07	0.11	0.99	0.04	0.03	0.16
1,2,3,4,7,8,9-HpCDF	0.01	0.26	0.12	0.02	0.06	0.01	0.01	0.03	0.05	0.05	0.001	0.07	0.03	0.13	0.01	0.01	0.02
OCDF	0.001	5.58	1.50	2.65	0.52	0.43	0.02	0.10	0.13	0.32	0.006	0.07	0.04	2.30	0.04	0.13	0.17
Total PCDD/F (ng/kg)		22.1	38.7	21.9	9.1	2.1	1.0	3.6	10.1	4.2	0.1	7.2	5.6	45.6	1.2	1.8	3.4
TEQ (ng/kg)		0.63	0.64	0.20	0.34	0.09	0.04	0.16	1.07	0.22	0.01	0.94	0.19	0.88	0.03	0.04	0.08
Intake (ng/yr)		42	5	64	13	13	6	3	3	3	0	1	7	59	4	11	17
Intake (ng TEQ/yr)		1.2	0.1	0.6	0.5	0.6	0.2	0.1	0.3	0.2	0.0	0.1	0.2	1.1	0.1	0.2	0.4
Contribution (TEQ)		14.9%	1.1%	7.4%	5.8%	7.1%	3.1%	1.5%	3.6%	2.1%	0.1%	1.4%	2.9%	14.2%	1.4%	30%	4.8%

Appendix 2.8 (cont.) Congener-specific PCDD/F levels in foods and environmental media, and intake data – children (3 mths–2 yrs)

	Fruit & products	Sugar & preserves	Bread & cereals	Beverages	Fish	Soil	Water (ng/l)	Air (ng/m^3)	Congener-specific Intake					TEQ Intake	
									Diet (ng/yr)	Soil (ng/yr)	Water (ng/yr)	Air (ng/yr)	Total (ng/yr)	Total (ng TEQ/yr)	Contribution to total TEQ (%)
	6.11	**1.33**	**9.49**	**5.39**	**0.93**	**0.000**	**91**	**1,500**							
2,3,7,8-TCDD	0.01	0.01	0.01	0.001	0.13	0.50	2.3 E-05	5.0 E-05	**1.7**	**0.0**	**0.00**	**0.1**	**2**	**1.8**	**22.2**
1,2,3,7,8-PeCDD	0.01	0.01	0.02	0.001	0.20	0.50	1.9 E-05	4.0 E-05	**2.4**	**0.0**	**0.00**	**0.1**	**2**	**1.2**	**15.4**
1,2,3,4,7,8-HxCDD	0.02	0.02	0.01	0.000	0.04	0.37	2.7 E-05	1.2 E-04	**1.8**	**0.0**	**0.00**	**0.2**	**2**	**0.2**	**2.4**
1,2,3,6,7,8-HxCDD	0.04	0.04	0.02	0.001	0.08	0.62	2.4 E-05	1.2 E-04	**4.7**	**0.0**	**0.00**	**0.2**	**5**	**0.5**	**6.0**
1,2,3,7,8,9-HxCDD	0.03	0.03	0.01	0.001	0.04	0.48	2.9 E-05	1.3 E-04	**2.1**	**0.0**	**0.00**	**0.2**	**2**	**0.2**	**2.8**
1,2,3,4,6,7,8-HpCDD	0.40	0.40	0.26	0.011	0.32	27.00	1.2 E-04	6.7 E-04	**36.9**	**0.0**	**0.01**	**1.0**	**38**	**0.4**	**4.7**
OCDD	2.45	2.45	1.98	0.078	2.62	1.43	1.7 E-04	1.7 E-03	**200.8**	**0.0**	**0.02**	**2.6**	**203**	**0.2**	**2.5**
2,3,7,8-TCDF	0.08	0.08	0.01	0.002	1.21	0.95	1.7 E-05	1.4 E-04	**4.4**	**0.0**	**0.00**	**0.2**	**5**	**0.5**	**5.7**
2,3,4,7,8-PeCDF	0.03	0.03	0.03	0.001	0.66	0.93	1.4 E-05	6.0 E-05	**3.8**	**0.0**	**0.00**	**0.1**	**4**	**1.9**	**23.9**
1,2,3,7,8-PeCDF	0.02	0.02	0.01	0.001	0.18	1.06	1.1 E-05	1.9 E-04	**1.6**	**0.0**	**0.00**	**0.3**	**2**	**0.1**	**1.2**
1,2,3,4,7,8-HxCDF	0.02	0.02	0.01	0.001	0.08	1.27	2.4 E-05	3.1 E-04	**2.5**	**0.0**	**0.00**	**0.5**	**3**	**0.3**	**3.6**
1,2,3,7,8,9-HxCDF	0.01	0.01	0.00	0.001	0.04	0.01	2.9 E-05	4.0 E-05	**1.2**	**0.0**	**0.00**	**0.1**	**1**	**0.1**	**1.5**
1,2,3,6,7,8-HxCDF	0.01	0.01	0.01	0.001	0.07	0.82	2.3 E-05	9.5 E-04	**1.7**	**0.0**	**0.00**	**1.4**	**3**	**0.3**	**3.8**
2,3,4,6,7,8-HxCDF	0.01	0.01	0.01	0.001	0.08	0.73	2.4 E-05	1.3 E-04	**1.7**	**0.0**	**0.00**	**0.2**	**2**	**0.2**	**2.3**
1,2,3,4,6,7,8-HpCDF	0.06	0.06	0.06	0.003	0.09	4.13	1.1 E-05	4.5 E-04	**7.6**	**0.0**	**0.00**	**0.7**	**8**	**0.1**	**1.0**
1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.01	0.001	0.09	0.38	3.0 E-05	4.0 E-04	**1.5**	**0.0**	**0.00**	**0.6**	**2**	**0.0**	**0.3**
OCDF	0.17	0.17	0.13	0.003	0.20	15.00	5.9 E-05	3.7 E-04	**30.0**	**0.0**	**0.01**	**0.6**	**31**	**0.0**	**0.4**
Total PCDD/F (ng/kg)	3.3	3.3	2.6	0.1	6.1	56.2	0.001	0.006							
TEQ (ng/kg)	0.06	0.06	0.05	0.003	0.74	0	6.2 E-05	3.2 E-04							
Intake (ng/yr)	20	4	25	1	6	0.0	0.1	9							
Intake (ng TEQ/yr)	0.3	0.1	0.5	0.0	0.7	0.0	0.0	0.5	7.6	0.0	0.0	0.5	**8.1**		
Contribution (TEQ)	4.2%	0.9%	5.9%	0.2%	8.5%	0.00%	0.07%	6.0%	**94.0%**	**0.0%**	**0.1%**	**6.0%**	**100%**		

Appendix 2.9 Concentrations in human milk on a fat weight basis (ng/kg fat)

Congener	UK	Germany	Belgium	Netherlands
2,3,7,8-TCDD	6.5	3.3	4.2	5.2
1,2,3,7,8-PCDD	14	7	11	18
1,2,3,4,7,8-HxCDD	67	10	8	10
1,2,3,6,7,8-HxCDD	75	40	39	75
1,2,3,7,8,9-HxCDD	10	9	9	11
1,2,3,4,6,7,8-HpCDD	76	50	121	112
OCDD	303	255	555	627
2,3,7,8-TCDF	1.4	2.4	3.3	3.1
2,3,4,7,8-PCDF	25	36	35	24
1,2,3,7,8-PCDF	0.5	0.9	1.4	0.8
1,2,3,4,7,8-HxCDF	8	15	16	7
1,2,3,7,8,9-HxCDF	8	15	8	6
1,2,3,6,7,8-HxCDF	0		0	
2,3,4,6,7,8-HxCDF	4	6	7	3
1,2,3,4,6,7,8-HpCDF	10	13	12	16
1,2,3,4,7,8,9-HpCDF	0			
OCDF	6.8	6.7	0	0.8
TEQ	**44.5**	**35.4**	**37.9**	**39.6**
Body Burden (ng TEQ)	**723**	**575**	**616**	**644**

Source: Rappe *et al.*, 1992

Appendix 2.10 Exposure to PCDD/Fs during the 3-month breast-feeding period

	Breast milk		Air		Total	
	Daily intake (ng)	Daily intake (ng TEQ/day)	Daily intake (ng)	Daily intake (ng TEQ/day)	Exposure (ng)	Exposure (ng TEQ)
2,3,7,8-TCDD	0.142	0.142	4.00 E-05	4.00 E-05	**12.8**	**12.8**
1,2,3,7,8-PCDD	0.306	0.153	3.20 E-05	1.60 E-05	**27.5**	**13.8**
1,2,3,4,7,8-HxCDD	1.464	0.146	9.60 E-05	9.60 E-06	**131.7**	**13.2**
1,2,3,6,7,8-HxCDD	1.638	0.164	9.60 E-05	9.60 E-06	**147.5**	**14.7**
1,2,3,7,8,9-HxCDD	0.218	0.022	1.04 E-04	1.04 E-05	**19.7**	**2.0**
1,2,3,4,6,7,8-HpCDD	1.660	0.017	5.36 E-04	5.36 E-06	**149.5**	**1.5**
OCDD	6.619	0.007	1.36 E-03	1.36 E-06	**595.8**	**0.6**
2,3,7,8-TCDF	0.031	0.003	1.12 E-04	1.12 E-05	**2.8**	**0.3**
2,3,4,7,8-PCDF	0.546	0.273	4.80 E-05	2.40 E-05	**49.2**	**24.6**
1,2,3,7,8-PCDF	0.011	0.001	1.52 E-04	7.60 E-06	**1.0**	**0.0**
1,2,3,4,7,8-HxCDF	0.181	0.018	2.48 E-04	2.48 E-05	**16.3**	**1.6**
1,2,3,7,8,9-HxCDF	0.170	0.017	3.20 E-05	3.20 E-06	**15.3**	**1.5**
1,2,3,6,7,8-HxCDF	0.000	0.000	7.60 E-04	7.60 E-05	**0.1**	**0.0**
2,3,4,6,7,8-HxCDF	0.079	0.008	1.04 E-04	1.04 E-05	**7.1**	**0.7**
1,2,3,4,6,7,8-HpCDF	0.208	0.002	3.60 E-04	3.60 E-06	**18.7**	**0.2**
1,2,3,4,7,8,9-HpCDF	0.000	0.000	3.20 E-04	3.20 E-06	**0.0**	**0.0**
OCDF	0.149	0.000	2.96 E-04	2.96 E-07	**13.4**	**0.0**
Total		**0.97**		**0.02**		**87.5**

Appendix 2.11 Estimated fat tissue concentrations and body burden at various ages

Age (years)	0	0.25	2	7	14	20	25	30	35	40	45	50	55	60	65	70
Fat (ng TEQ/kg)																
Average	2.4	52	35	21	22	33	33	33	34	34	34	35	35	35	35	35
Male	2.3	52	34	21	26	36	33	31	32	33	34	34	34	34	34	35
Female	2.4	52	37	21	19	30	32	35	35	34	35	35	35	36	36	36
Body Burden (ng TEQ)																
Breast-feeding mother	0.89	89	88	112	198	375	322	430	491	525	545	556	562	566	568	569
Average	0.89	87	87	111	197	375	459	507	535	550	559	564	566	568	569	569

Appendix 2.12 Concentrations in human tissues on a fat weight basis (ng/kg fat)

Tissue Type	Blood		Adipose Tissue		
Congener	USA[1]	Germany[1]	Germany[2]	USA[3]	Canada[4]
2,3,7,8-TCDD	5.2	3.6	7.2	6.5	4.2
1,2,3,7,8-PCDD	21	14	21	10	14
1,2,3,4,7,8-HxCDD	13	9	19	10	15
1,2,3,6,7,8-HxCDD	84	61	89	66	137
1,2,3,7,8,9-HxCDD	15	11	12	12	17
1,2,3,4,6,7,8-HpCDD	187	94	101	110	136
OCDD	1,174	596	591	838	500
2,3,7,8-TCDF	3.1	2.5	2.5	1.9	2.2
2,3,4,7,8-PCDF	13	30	40	9	10
1,2,3,7,8-PCDF	2.8	6.5	0.4	0.2	1.9
1,2,3,4,7,8-HxCDF	15	15	15	7	11
1,2,3,7,8,9-HxCDF	1.2	1.2	0.7	0.3	3.3
1,2,3,6,7,8-HxCDF	14	14	16	53	10
2,3,4,6,7,8-HxCDF	3.6	3.6	4.7	0.5	0.8
1,2,3,4,6,7,8-HpCDF	36	21	20	18	19
1,2,3,4,7,8,9-HpCDF	1.8	1.0	0.8	0.7	0.6
OCDF	4.2	5.5	0.4	1.2	3.7
TEQ	40.7	39.6	55.4	33.4	38.0
Body burden (ng TEQ)	661	643	901	543	617

Sources:

(1) Schecter *et al.*, 1991
(2) Beck *et al.*, 1994
(3) Orban *et al.*, 1994
(4) Teschke *et al.*, 1992

Appendix 3.1 Cadmium levels in various meat foodstuffs

	RANGE	MEAN	SOURCE
Beef	<2–180	<30	MAFF, 1983
		1.3	Niemi *et al.*, 1991
		<5	Falandysz, 1991
		6	Falandysz, 1993
	<1–20	4	Vos *et al.*, 1987
	<1–1	1	Jorhem *et al.*, 1991
		1–<5	Vos *et al.*, 1987
Pork	<6–180	<30	MAFF, 1983
		1.5	Niemi *et al.*, 1991
Lamb	<2–90	<20	MAFF, 1983
		2	Schulzschroeder, 1991
Liver	70–230	130	MAFF, 1983
		28–61	Niemi *et al.*, 1991
		16–140	Falandysz, 1991
		120	Falandysz, 1993
	7–360	110	Vos *et al.*, 1987
	1–200	70	Jorhem *et al.*, 1991
		89	Vos *et al.*, 1988
		271	Schulzschroeder, 1991
Kidney	40–2,700	480	MAFF, 1983
		170–350	Niemi *et al.*, 1991
		610	Falandysz, 1993
	30–3,600	520	Vos *et al.*, 1987
		830	Schmidt *et al.*, 1988
	31–3,500	390	Jorhem *et al.*, 1991
		290	Vos *et al.*, 1988
		547	Schuzschroeder, 1991
Poultry	<10–270	<50	MAFF, 1983
	6–14	10	Falandysz, 1991
	6–27	12	Piekacz, 1976

Appendix 3.2 Cadmium levels in various vegetable foodstuffs

	RANGE	MEAN	SOURCE
Cabbage	<10–30	<10	MAFF, 1983
	2–150	6–40	Page *et al.*, 1981
		41	Ryan *et al.*, 1982
		44	Kaferstein *et al.*, 1979
Lettuce	<10–390	<60	MAFF, 1983
		17–34	Kuo and Huang, 1993
	1–198	12–93	Page *et al.*, 1981
	1–160	26	Wolnik *et al.*, 1983
		17	Wolnik *et al.*, 1985
		17	Shacklette, 1980
	19–60	34	Feeney *et al.*, 1984
		14	Muller and Anke, 1994
		33	Giordano *et al.*, 1979
Peas	<10–60	<30	MAFF, 1983
		27	Dowdy and Larson, 1975
	<4–15	10	Zurerzcosano *et al.*, 1991
		6	Ryan *et al.*, 1982
Potatoes	<10–60	<30	MAFF, 1983
		30–50	McLaughlin *et al.*, 1994
	2–182	34	Wolnik *et al.*, 1983
	1–202	50	Kaferstein *et al.*, 1979
		48	Ryan *et al.*, 1982
Onions	<10–40	<20	MAFF, 1983
	<2–90	6–80	Page *et al.*, 1981
	2–54	10	Wolnik *et al.*, 1983
		5	Shacklette, 1980
Carrots	<10–110	<50	MAFF 1983
	2–132	28	Wolnik *et al.*, 1983
		16	Shacklette, 1980
		27	Feeney *et al.*, 1984
		89	Giordano *et al.*, 1979
	10–64	46	Page *et al.*, 1987
Tomatoes	<10–40	<10	MAFF, 1983
	1–80	10–30	Page *et al.*, 1981
	3–48	17	Wolnik *et al.*, 1983
	6–50	22	Feeney *et al.*, 1984
		7	Shacklette, 1980
		8	Kunsch *et al.*, 1994

Appendix 3.3 Cadmium levels in foodstuffs and environmental media

	Concentration
Beef	6
Pork	1.5
Lamb	2
Liver	271
Kidney	830
Meat products	3
Poultry	12
Whole milk	1
Skimmed and semi-skimmed	1
Cheese	1
Butter	1
Milk products	1
Ice cream	1
Yoghurt	1
Cream	1
Eggs	16
Vegetable oil	33
Animal oil	3
Cabbage	44
Lettuce	93
Peas	27
Potatoes	50
Onions	80
Carrots	89
Tomatoes	30
Fresh fruit	3.7
Fruit products	3.7
Sugar and preserves	3.5
Bread	30
Other cereals	30
Beverages	2
Freshwater fish	29
Marine	170
Canned	20
Shellfish	940
Soil ingestion	3,000
Drinking water	1
Air	0.009

* All values in µg/kg (fresh weight) except: soil (µg/kg dry weight), drinking water (µg/l) and air ($\mu g/m^3$).

Appendix 3.4 Cadmium: adult intake and exposure from various sources

	INTAKE (µg/yr)	TOTAL INTAKE (%)	EXPOSURE (BLOOD) (µg/yr)	EXPOSURE (%)
Beef	44	0.37	3	0.36
Pork	6	0.05	0	0.05
Lamb	8	0.07	0	0.06
Liver	220	1.86	13	1.80
Kidney	241	2.04	14	1.97
Meat products	70	0.59	4	0.57
Poultry	133	1.13	8	1.09
Whole milk	47	0.40	3	0.38
Skimmed milk	48	0.41	3	0.39
Cheese	6	0.05	0	0.05
Butter	2	0.02	0	0.02
Milk products	6	0.05	0	0.05
Ice cream	5	0.04	0	0.04
Yoghurt	6	0.05	0	0.05
Cream	1	0.01	0	0.01
Eggs	152	1.29	9	1.24
Vegetable oil	79	0.67	5	0.64
Animal oil	24	0.20	1	0.20
Cabbage	447	3.79	27	3.66
Lettuce	588	4.97	35	4.81
Peas	297	2.51	18	2.43
Potatoes	2,369	20.04	142	19.37
Onions	1,040	8.80	62	8.50
Carrots	1,157	9.79	69	9.46
Tomatoes	390	3.30	23	3.19
Fresh fruit	118	1.00	7	0.97
Fruit products	63	0.53	4	0.51
Sugar and preserves	37	0.31	2	0.30
Bread	1,177	9.96	71	9.63
Other cereals	1,099	9.30	66	8.99
Beverages	86	0.73	5	0.70
Freshwater fish	48	0.40	3	0.39
Marine	758	6.42	45	6.20
Canned	21	0.18	1	0.17
Shellfish	197	1.67	12	1.61
Total Diet	**10,990**	**92.98**	**659**	**89.87**
Soil Ingestion	**27**	**0.23**	**2**	**0.22**
Drinking Water	**730**	**6.18**	**44**	**5.97**
Air	**72**	**0.61**	**29**	**3.94**
Total Intake	**11,819**	**100.00**	**734**	**100.00**

Appendix 3.5 Cadmium: child (7–14 yrs) intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (BLOOD) (µg/yr)	EXPOSURE (%)
Beef	22	0.37	1	0.35
Pork	3	0.05	0	0.05
Lamb	4	0.06	0	0.06
Liver	110	1.84	7	1.75
Kidney	120	2.02	7	1.92
Meat products	35	0.59	2	0.56
Poultry	67	1.12	4	1.06
Whole milk	23	0.39	1	0.37
Skimmed milk	24	0.40	1	0.38
Cheese	3	0.05	0	0.05
Butter	1	0.02	0	0.02
Milk products	3	0.05	0	0.05
Ice cream	2	0.04	0	0.04
Yoghurt	3	0.05	0	0.05
Cream	0	0.01	0	0.01
Eggs	76	1.27	5	1.21
Vegetable oil	39	0.66	2	0.63
Animal oil	12	0.20	1	0.19
Cabbage	224	3.75	13	3.57
Lettuce	294	4.92	18	4.68
Peas	149	2.49	9	2.37
Potatoes	1,184	19.84	71	18.88
Onions	520	8.71	31	8.29
Carrots	579	9.69	35	9.22
Tomatoes	195	3.27	12	3.11
Fresh fruit	59	0.99	4	0.94
Fruit products	31	0.53	2	0.50
Sugar and preserves	19	0.31	1	0.30
Bread	589	9.86	35	9.38
Other cereals	549	9.21	33	8.76
Beverages	43	0.72	3	0.69
Freshwater fish	24	0.40	1	0.38
Marine	379	6.35	23	6.04
Canned	11	0.18	1	0.17
Shellfish	99	1.65	6	1.57
Total Diet	**5,495**	**92.07**	**330**	**87.58**
Soil Ingestion	**54**	**0.90**	**3**	**0.86**
Drinking Water	**365**	**6.12**	**22**	**5.82**
Air	**54**	**0.90**	**22**	**5.74**
Total Intake	**5,968**	**100.00**	**376**	**100.00**

Appendix 3.6 Cadmium: child (2–7 yrs) intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (BLOOD) (µg/yr)	EXPOSURE (%)
Beef	11	0.36	1	0.34
Pork	2	0.05	0	0.05
Lamb	2	0.06	0	0.06
Liver	55	1.78	3	1.67
Kidney	60	1.96	4	1.83
Meat products	17	0.57	1	0.53
Poultry	33	1.08	2	1.02
Whole milk	12	0.38	1	0.36
Skimmed milk	12	0.39	1	0.37
Cheese	1	0.05	0	0.04
Butter	1	0.02	0	0.02
Milk products	2	0.05	0	0.05
Ice cream	1	0.04	0	0.04
Yoghurt	2	0.05	0	0.05
Cream	0	0.01	0	0.01
Eggs	38	1.24	2	1.16
Vegetable oil	20	0.64	1	0.60
Animal oil	6	0.19	0	0.18
Cabbage	112	3.64	7	3.41
Lettuce	147	4.78	9	4.48
Peas	74	2.41	4	2.26
Potatoes	592	19.24	36	18.05
Onions	260	8.45	16	7.92
Carrots	289	9.40	17	8.82
Tomatoes	98	3.17	6	2.97
Fresh fruit	30	0.96	2	0.90
Fruit products	16	0.51	1	0.48
Sugar and preserves	9	0.30	1	0.28
Bread	294	9.56	18	8.97
Other cereals	275	8.93	16	8.37
Beverages	22	0.70	1	0.66
Freshwater fish	12	0.39	1	0.36
Marine	190	6.16	11	5.78
Canned	5	0.17	0	0.16
Shellfish	49	1.60	3	1.50
Total Diet	**2,747**	**89.29**	**165**	**83.74**
Soil Ingestion	**111**	**3.61**	**7**	**3.38**
Drinking Water	**183**	**5.93**	**11**	**5.56**
Air	**36**	**1.17**	**14**	**7.31**
Total Intake	**3,077**	**100.00**	**197**	**100.00**

Appendix 3.7 Cadmium: child (3 mths–2 yrs) intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (BLOOD) (µg/yr)	EXPOSURE (%)
Beef	6	0.37	0	0.36
Pork	1	0.05	0	0.05
Lamb	1	0.07	0	0.06
Liver	27	1.86	2	1.76
Kidney	30	2.04	2	1.93
Meat products	9	0.59	1	0.56
Poultry	17	1.13	1	1.07
Whole milk	6	0.39	0	0.38
Skimmed milk	6	0.41	0	0.39
Cheese	1	0.05	0	0.05
Butter	0	0.02	0	0.02
Milk products	1	0.05	0	0.05
Ice cream	1	0.04	0	0.04
Yoghurt	1	0.05	0	0.05
Cream	0	0.01	0	0.01
Eggs	19	1.29	1	1.22
Vegetable oil	10	0.67	1	0.63
Animal oil	3	0.20	0	0.19
Cabbage	56	3.78	3	3.60
Lettuce	73	4.97	4	4.72
Peas	37	2.51	2	2.39
Potatoes	296	20.02	18	19.04
Onions	130	8.79	8	8.36
Carrots	145	9.78	9	9.30
Tomatoes	49	3.30	3	3.14
Fresh fruit	15	1.00	1	0.95
Fruit products	8	0.53	0	0.50
Sugar and preserves	5	0.31	0	0.30
Bread	147	9.95	9	9.46
Other cereals	137	9.29	8	8.83
Beverages	11	0.73	1	0.69
Freshwater fish	6	0.40	0	0.38
Marine	95	6.41	6	6.09
Canned	3	0.18	0	0.17
Shellfish	25	1.67	1	1.59
Total Diet	**1,374**	**92.92**	**82**	**88.34**
Soil Ingestion	**0**	**0.00**	**0**	**0.00**
Drinking Water	**91**	**6.17**	**5**	**5.87**
Air	**14**	**0.91**	**5**	**5.79**
Total Intake	**1,478**	**100.00**	**93**	**100.00**

Appendix 3.8 Intake of cadmium from breast-feeding

SOURCE	INTAKE (ng/day)	EXPOSURE (ng/day)	EXPOSURE (%)	INTAKE (%)
Breast milk	60.0	3.6	89	55
Air	7.4	3.0	11	45

Air concentration is 9 ng/m^3, absorption in GI tract is 6 per cent and in lungs 40 per cent.

Appendix 3.9 Estimated renal cortex concentrations and body burden at various ages

Age (years)	0	0.25	2	7	14	20	30	40	50	60	70
Renal cortex concentration (µg/g)	0.25	0.23	1.2	3.9	8.2	12.2	20.2	26.6	31.5	35.3	38.2
Body burden (mg)	0.01	0.01	0.2	1.1	3.3	6.9	11.7	15.4	18.3	20.5	22.2
Whole kidney burden (µg)	3.8	4.4	57	356	1,098	2,298	3,909	5,147	6,099	6,832	7,395
Kidney mass (g)	23	29	71	138	202	283	290	290	290	290	290

Appendix 4.1 Nickel concentrations in selected vegetable crops (µg/kg fresh weight)

VEGETABLE	LEVEL (µg Ni/kg fresh weight)	SOURCE
Cabbage	270	McIlveen and Negusanti, 1994
	130	Wolnik *et al.*, 1985
	60	Veien and Andersen, 1986
	<50	Smart and Sherlock, 1987
Lettuce	1,400	McIlveen and Negusanti, 1994
	1,000	Veien and Andersen, 1986
Peas	300	Veien and Andersen, 1986
Potatoes	170	McIlveen and Negusanti, 1994
	60	Veien and Andersen, 1986
	10	Flint and Packirisamy, 1995
Onion	460	McIlveen and Negusanti, 1994
	35	Wolnik *et al.*, 1985
	100	Veien and Andersen, 1986
	<40	MAFF, 1985
Carrots	120	McIlveen and Negusanti, 1994
	40	Veien and Andersen, 1986
	62	Wolnik *et al.*, 1985
	<60	Smart and Sherlock, 1987
	<10	MAFF, 1985
Tomatoes	70	McIlveen and Negusanti, 1994
	110	Wolnik *et al.*, 1985
	90	Veien and Andersen, 1986

Appendix 4.2 Nickel concentrations in various foodstuffs and environmental media (µg/kg fresh weight)

Beef	50
Pork	60
Lamb	90
Liver	50
Kidney	250
Meat products	70
Poultry	70
Whole milk	20
Skimmed milk	20
Cheese	20
Butter	20
Milk products	20
Ice cream	20
Yoghurt	20
Cream	20
Eggs	20
Vegetable oil	90
Animal oil	90
Cabbage	50
Lettuce	1,400
Peas	300
Potatoes	170
Onions	40
Carrots	10
Tomatoes	110
Fresh fruit	200
Fruit products	30
Sugar and preserves	130
Bread	125
Other cereals	300
Beverages	8
Freshwater fish	1,700
Marine	100
Canned	40
Shellfish	1,500
Soil ingestion	1,000,000
Drinking water	10
Air	0.012

* All values are in µg/kg (fresh weight) except drinking water (µg/l) and air ($\mu g/m^3$).

Appendix 4.3 Annual nickel intake and exposure for adults (µg/yr)

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	369	0.50	18.45	0.35
Pork	243	0.33	12.15	0.23
Lamb	347	0.47	17.33	0.33
Liver	41	0.05	2.03	0.04
Kidney	73	0.10	3.63	0.07
Meat products	1632	2.20	81.59	1.53
Poultry	778	1.05	38.92	0.73
Whole milk	934	1.26	46.70	0.88
Skimmed milk	962	1.29	48.10	0.90
Cheese	118	0.16	5.88	0.11
Butter	43	0.06	2.16	0.04
Milk products	124	0.17	6.21	0.12
Ice cream	97	0.13	4.85	0.09
Yoghurt	120	0.16	6.01	0.11
Cream	18	0.02	0.89	0.02
Eggs	190	0.26	9.51	0.18
Vegetable oil	215	0.29	10.76	0.20
Animal oil	718	0.97	35.91	0.67
Cabbage	509	0.68	25.43	0.48
Lettuce	8,848	11.91	442.40	8.31
Peas	3,300	4.44	165.00	3.10
Potatoes	8,053	10.84	402.65	7.56
Onions	520	0.70	26.00	0.49
Carrots	130	0.17	6.50	0.12
Tomatoes	1,430	1.92	71.50	1.34
Fresh fruit	6,392	8.60	319.60	6.00
Fruit products	509	0.68	25.43	0.48
Sugar and preserves	1,379	1.86	68.97	1.30
Bread	4,905	6.60	245.25	4.61
Other cereals	10,989	14.79	549.45	10.32
Beverages	345	0.46	17.23	0.32
Freshwater fish	2,788	3.75	139.40	2.62
Marine	446	0.60	22.30	0.42
Canned	42	0.06	2.12	0.04
Shellfish	315	0.42	15.75	0.30
Total Diet	**57,920**	**77.94**	**2,896**	**54.41**
Soil Ingestion	**9,000**	**12.11**	**450.00**	**8.45**
Drinking Water	**7,300**	**9.82**	**1,971.00**	**37.03**
Air	**96**	**0.13**	**5.78**	**0.11**
Total Intake	**74,317**	**100.00**	**5,322.79**	**100.00**

Appendix 4.4 Annual nickel intake and exposure for 7–14 yrs (µg/yr)

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	185	0.36	9.23	0.28
Pork	122	0.24	6.08	0.18
Lamb	173	0.34	8.66	0.26
Liver	20	0.04	1.01	0.03
Kidney	36	0.07	1.81	0.05
Meat products	816	1.61	40.79	1.22
Poultry	389	0.77	19.46	0.58
Whole milk	467	0.92	23.35	0.70
Skimmed milk	481	0.95	24.05	0.72
Cheese	59	0.12	2.94	0.09
Butter	22	0.04	1.08	0.03
Milk products	62	0.12	3.11	0.09
Ice cream	49	0.10	2.43	0.07
Yoghurt	60	0.12	3.01	0.09
Cream	9	0.02	0.45	0.01
Eggs	95	0.19	4.76	0.14
Vegetable oil	108	0.21	5.38	0.16
Animal oil	359	0.71	17.96	0.54
Cabbage	254	0.50	12.71	0.38
Lettuce	4,424	8.73	221.20	6.63
Peas	1,650	3.26	82.50	2.47
Potatoes	4,026	7.94	201.32	6.03
Onions	260	0.51	13.00	0.39
Carrots	65	0.13	3.25	0.10
Tomatoes	715	1.41	35.75	1.07
Fresh fruit	3,196	6.31	159.80	4.79
Fruit products	254	0.50	12.71	0.38
Sugar and preserves	690	1.36	34.48	1.03
Bread	2,453	4.84	122.63	3.67
Other cereals	5,495	10.84	274.73	8.23
Beverages	172	0.34	8.61	0.26
Freshwater fish	1,394	2.75	69.70	2.09
Marine	223	0.44	11.15	0.33
Canned	21	0.04	1.06	0.03
Shellfish	158	0.31	7.88	0.24
Total Diet	**28,960**	**57.14**	**1448**	**43.38**
Soil Ingestion	**18,000**	**35.52**	**900.00**	**26.96**
Drinking Water	**3,650**	**7.20**	**985.50**	**29.53**
Air	**72**	**0.14**	**4.32**	**0.13**
Total Intake	**50,682**	**100.00**	**1,337.83**	**100.00**

Appendix 4.5 Annual nickel intake and exposure for 2–7 yrs (µg/yr)

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	92	0.17	4.61	0.15
Pork	61	0.11	3.04	0.10
Lamb	87	0.16	4.33	0.14
Liver	10	0.02	0.51	0.02
Kidney	18	0.03	0.91	0.03
Meat products	408	0.76	20.40	0.66
Poultry	195	0.36	9.73	0.32
Whole milk	234	0.44	11.68	0.38
Skimmed milk	241	0.45	12.03	0.39
Cheese	29	0.06	1.47	0.05
Butter	11	0.02	0.54	0.02
Milk products	31	0.06	1.55	0.05
Ice cream	24	0.05	1.21	0.04
Yoghurt	30	0.06	1.50	0.05
Cream	4	0.01	0.22	0.01
Eggs	48	0.09	2.38	0.08
Vegetable oil	54	0.10	2.69	0.09
Animal oil	180	0.34	8.98	0.29
Cabbage	127	0.24	6.36	0.21
Lettuce	2,212	4.15	110.60	3.60
Peas	825	1.55	41.25	1.34
Potatoes	2,013	3.77	100.66	3.28
Onions	130	0.24	6.50	0.21
Carrots	33	0.06	1.63	0.05
Tomatoes	358	0.67	17.88	0.58
Fresh fruit	1,598	3.00	79.90	2.60
Fruit products	127	0.24	6.36	0.21
Sugar and preserves	345	0.65	17.24	0.56
Bread	1,226	2.30	61.31	2.00
Other cereals	2,747	5.15	137.36	4.47
Beverages	86	0.16	4.31	0.14
Freshwater fish	697	1.31	34.85	1.14
Marine	112	0.21	5.58	0.18
Canned	11	0.02	0.53	0.02
Shellfish	79	0.15	3.94	0.13
Total Diet	**14,480**	**27.14**	**724**	**23.59**
Soil Ingestion	**37,000**	**69.35**	**1,850.00**	**60.27**
Drinking Water	**1,825**	**3.42**	**492.75**	**16.05**
Air	**48**	**0.09**	**2.88**	**0.09**
Total Intake	**53,353**	**100.00**	**1,069.63**	**100.00**

Appendix 4.6 Annual nickel intake and exposure for 3 mths–2 yrs (µg/yr)

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	46	0.56	2.31	0.38
Pork	30	0.37	1.52	0.25
Lamb	43	0.53	2.17	0.36
Liver	5	0.06	0.25	0.04
Kidney	9	0.11	0.45	0.07
Meat products	204	2.50	10.20	1.67
Poultry	97	1.19	4.87	0.80
Whole milk	117	1.43	5.84	0.96
Skimmed milk	120	1.47	6.01	0.99
Cheese	15	0.18	0.74	0.12
Butter	5	0.07	0.27	0.04
Milk products	16	0.19	0.78	0.13
Ice cream	12	0.15	0.61	0.10
Yoghurt	15	0.18	0.75	0.12
Cream	2	0.03	0.11	0.02
Eggs	24	0.29	1.19	0.20
Vegetable oil	27	0.33	1.34	0.22
Animal oil	90	1.10	4.49	0.74
Cabbage	64	0.78	3.18	0.52
Lettuce	1,106	13.54	55.30	9.07
Peas	413	5.05	20.63	3.38
Potatoes	1,007	12.32	50.33	8.26
Onions	65	0.80	3.25	0.53
Carrots	16	0.20	0.81	0.13
Tomatoes	179	2.19	8.94	1.47
Fresh fruit	799	9.78	39.95	6.56
Fruit products	64	0.78	3.18	0.52
Sugar and preserves	172	2.11	8.62	1.41
Bread	613	7.50	30.66	5.03
Other cereals	1,374	16.81	68.68	11.27
Beverages	43	0.53	2.15	0.35
Freshwater fish	349	4.27	17.43	2.86
Marine	56	0.68	2.79	0.46
Canned	5	0.06	0.27	0.04
Shellfish	39	0.48	1.97	0.32
Total Diet	**7,240**	**88.61**	**362**	**59.40**
Soil Ingestion	**0**	**0.00**	**0.00**	**0.00**
Drinking Water	**913**	**11.17**	**246.38**	**40.43**
Air	**18**	**0.22**	**1.08**	**0.18**
Total Intake	**8,171**	**100.00**	**609.46**	**100.00**

Appendix 4.7 Nickel intake during the 3-month breast-feeding period

	Daily intake (ng)	Daily exposure (ng)	Intake (%)	Exposure (%)	Total exposure (μg)
Breast milk	12,750	638	99.9	99.9	57.38
Air	10	1	0.1	0.1	0.05

* Assuming concentration in milk is 17 ng/l.

Appendix 5.1 Levels of arsenic in various vegetables

VEGETABLE	LEVEL (µg As/kg fresh weight)	SOURCE
Cabbage	<10–40	Pyles and Woolson, 1982
	14–28	Horler, 1989
	<10	MAFF, 1982
Lettuce	12	Pyles and Woolson, 1982
	11	Wiersma *et al.*, 1986
	15	Jelinek and Corneliussen, 1980
	20	MAFF, 1982
Peas	<10 µg	MAFF, 1982
Potatoes	1–20	Pyles and Woolson, 1982
	13	Wiersma *et al.*, 1986
	8	Jelinek and Corneliussen, 1980
	<10	MAFF, 1982
Onions	No data: assumed to be the same as carrots	
Carrots	22	Wiersma *et al.*, 1986
	20	Jelinek and Corneliussen, 1980
	<10	MAFF, 1982
Tomatoes	1–8	Pyles and Woolson, 1982
	1	Wiersma *et al.*, 1986
	<10	MAFF, 1982

Appendix 5.2 Total arsenic levels in foodstuffs and environmental media

	Concentration (µg/kg)
Beef	9
Pork	1
Lamb	1
Liver	17
Kidney	48
Meat products	4
Poultry	1
Whole milk	0.32
Skimmed milk	0.48
Cheese	0.4
Butter	0.4
Milk products	0.4
Ice cream	0.4
Yoghurt	0.4
Cream	0.4
Eggs	50
Vegetable oil	20
Animal oil	4
Cabbage	10
Lettuce	20
Peas	10
Potatoes	10
Onions	10
Carrots	10
Tomatoes	8
Fresh fruit	14
Fruit products	14
Sugar and preserves	50
Bread	50
Other cereals	50
Beverages	11
Freshwater fish	1,200
Marine	1,600
Canned	600
Shellfish	10,900
Soil	40,000
Drinking water	5
Air	0.094

* All values in µg/kg (fresh weight) except: soil (µg/kg dry weight), drinking water (µg/l) and air ($\mu g/m^3$).

Appendix 5.3 Total arsenic: adult intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	66	0.20	65	0.20
Pork	4	0.01	4	0.01
Lamb	4	0.01	4	0.01
Liver	14	0.04	13	0.04
Kidney	14	0.04	14	0.04
Meat products	93	0.28	91	0.29
Poultry	11	0.03	11	0.03
Whole milk	15	0.05	15	0.05
Skimmed and semi-skimmed	23	0.07	23	0.07
Cheese	2	0.01	2	0.01
Butter	1	0.00	1	0.00
Milk products	2	0.01	2	0.01
Ice cream	2	0.01	2	0.01
Yoghurt	2	0.01	2	0.01
Cream	0	0.00	0	0.00
Eggs	476	1.43	466	1.45
Vegetable oil	48	0.14	47	0.15
Animal oil	32	0.10	31	0.10
Cabbage	102	0.31	100	0.31
Lettuce	126	0.38	124	0.39
Peas	110	0.33	108	0.34
Potatoes	474	1.43	464	1.45
Onions	130	0.39	127	0.40
Carrots	130	0.39	127	0.40
Tomatoes	104	0.31	102	0.32
Fresh fruit	447	1.35	438	1.37
Fruit products	237	0.72	233	0.73
Sugar and preserves	531	1.60	520	1.62
Bread	1,962	5.91	1,923	6.00
Other cereals	1,832	5.52	1,795	5.60
Beverages	474	1.43	464	1.45
Freshwater fish	1,968	5.93	1,929	6.02
Marine	16,056	48.39	15,735	49.12
Canned	636	1.92	623	1.95
Shellfish	2,289	6.90	2,243	7.00
Total Diet	**28,417**	**85.64**	**27,849**	**86.93**
Soil Ingestion	**360**	**1.08**	**353**	**1.10**
Drinking Water	**3,650**	**11.00**	**1,577**	**11.17**
Air	**755**	**2.27**	**257**	**0.80**
Total Intake	**33,182**	**100.00**	**32,035**	**100.00**

Appendix 5.4 Inorganic arsenic: adult intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	50	0.42	49	0.44
Pork	3	0.03	3	0.03
Lamb	3	0.02	3	0.03
Liver	10	0.09	10	0.09
Kidney	10	0.09	10	0.09
Meat products	70	0.59	69	0.62
Poultry	8	0.07	8	0.07
Whole milk	11	0.09	11	0.10
Skimmed milk	17	0.15	17	0.15
Cheese	2	0.01	2	0.02
Butter	1	0.01	1	0.01
Milk products	2	0.02	2	0.02
Ice cream	1	0.01	1	0.01
Yoghurt	2	0.02	2	0.02
Cream	0	0.00	0	0.00
Eggs	357	3.01	349	3.14
Vegetable oil	36	0.30	35	0.32
Animal oil	24	0.20	23	0.21
Cabbage	76	0.64	75	0.67
Lettuce	95	0.80	93	0.84
Peas	83	0.70	81	0.73
Potatoes	355	3.00	348	3.13
Onions	98	0.82	96	0.86
Carrots	98	0.82	96	0.86
Tomatoes	78	0.66	76	0.69
Fresh fruit	336	2.83	329	2.96
Fruit products	178	1.50	174	1.57
Sugar and preserves	398	3.36	390	3.51
Bread	1,472	12.43	1,442	12.97
Other cereals	1,374	11.60	1,346	12.10
Beverages	355	3.00	348	3.13
Freshwater fish	1,476	12.46	1,446	13.01
Marine	0	0.00	0	0.00
Canned	0	0.00	0	0.00
Shellfish	0	0.00	0	0.00
Total Diet	**7,077**	**59.76**	**6,936**	**62.36**
Soil Ingestion	**360**	**3.04**	**353**	**3.17**
Drinking Water	**3,650**	**30.82**	**3,577**	**32.16**
Air	**755**	**6.37**	**257**	**2.31**
Total Intake	**11,842**	**100.00**	**11,122**	**100.00**

Appendix 5.5 Total arsenic: child (7–14 yrs) intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	33	0.19	33	0.20
Pork	2	0.01	2	0.01
Lamb	2	0.01	2	0.01
Liver	7	0.04	7	0.04
Kidney	7	0.04	7	0.04
Meat products	47	0.27	46	0.28
Poultry	6	0.03	6	0.03
Whole milk	7	0.04	7	0.04
Skimmed milk	12	0.07	11	0.07
Cheese	1	0.01	1	0.01
Butter	0	0.00	0	0.00
Milk products	1	0.01	1	0.01
Ice cream	1	0.01	1	0.01
Yoghurt	1	0.01	1	0.01
Cream	0	0.00	0	0.00
Eggs	238	1.37	233	1.40
Vegetable oil	24	0.14	23	0.14
Animal oil	16	0.09	16	0.09
Cabbage	51	0.29	50	0.30
Lettuce	63	0.36	62	0.37
Peas	55	0.32	54	0.32
Potatoes	237	1.37	232	1.40
Onions	65	0.38	64	0.38
Carrots	65	0.38	64	0.38
Tomatoes	52	0.30	51	0.31
Fresh fruit	224	1.29	219	1.32
Fruit products	119	0.69	116	0.70
Sugar and preserves	265	1.53	260	1.56
Bread	981	5.66	961	5.79
Other cereals	916	5.29	897	5.40
Beverages	237	1.37	232	1.40
Freshwater fish	984	5.68	964	5.81
Marine	8,028	46.36	7,867	47.36
Canned	318	1.84	311	1.88
Shellfish	1,145	6.61	1,122	6.75
Total Diet	**14,209**	**82.05**	**13,924**	**83.83**
Soil Ingestion	**720**	**4.16**	**706**	**4.25**
Drinking Water	**1,825**	**10.54**	**1,788**	**10.77**
Air	**564**	**3.26**	**192**	**1.15**
Total Intake	**17,318**	**100.00**	**16,610**	**100.00**

Appendix 5.6 Inorganic arsenic: child (7–14 yrs) intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	25	0.42	24	0.45
Pork	2	0.03	1	0.03
Lamb	1	0.02	1	0.03
Liver	5	0.09	5	0.09
Kidney	5	0.09	5	0.09
Meat products	35	0.59	34	0.63
Poultry	4	0.07	4	0.08
Whole milk	6	0.09	5	0.10
Skimmed milk	9	0.15	8	0.16
Cheese	1	0.01	1	0.02
Butter	0	0.01	0	0.01
Milk products	1	0.02	1	0.02
Ice cream	1	0.01	1	0.01
Yoghurt	1	0.02	1	0.02
Cream	0	0.00	0	0.00
Eggs	178	3.02	175	3.22
Vegetable oil	18	0.30	18	0.32
Animal oil	12	0.20	12	0.22
Cabbage	38	0.65	37	0.69
Lettuce	47	0.80	46	0.86
Peas	41	0.70	40	0.74
Potatoes	178	3.01	174	3.21
Onions	49	0.82	48	0.88
Carrots	49	0.82	48	0.88
Tomatoes	39	0.66	38	0.70
Fresh fruit	168	2.84	164	3.03
Fruit products	89	1.51	87	1.61
Sugar and preserves	199	3.37	195	3.59
Bread	736	12.45	721	13.28
Other cereals	687	11.62	673	12.39
Beverages	178	3.01	174	3.21
Freshwater fish	0	0.00	0	0.00
Marine	0	0.00	0	0.00
Canned	0	0.00	0	0.00
Shellfish	0	0.00	0	0.00
Total Diet	**2,801**	**47.39**	**2,745**	**50.54**
Soil Ingestion	**720**	**12.18**	**706**	**12.99**
Drinking Water	**1,825**	**30.88**	**1,789**	**32.93**
Air	**564**	**9.54**	**192**	**3.53**
Total Intake	**5,910**	**100.00**	**5,430**	**100.00**

Appendix 5.7 Total arsenic: child (2–7 yrs) intake and exposure from various sources

	Intake (µg/yr)	intake (%)	Exposure (µg/yr)	exposure (%)
Beef	17	0.17	16	0.17
Pork	1	0.01	1	0.01
Lamb	1	0.01	1	0.01
Liver	3	0.03	3	0.04
Kidney	3	0.04	3	0.04
Meat products	23	0.24	23	0.24
Poultry	3	0.03	3	0.03
Whole milk	4	0.04	4	0.04
Skimmed milk	6	0.06	6	0.06
Cheese	1	0.01	1	0.01
Butter	0	0.00	0	0.00
Milk products	1	0.01	1	0.01
Ice cream	0	0.00	0	0.01
Yoghurt	1	0.01	1	0.01
Cream	0	0.00	0	0.00
Eggs	119	1.20	117	1.23
Vegetable oil	12	0.12	12	0.12
Animal oil	8	0.08	8	0.08
Cabbage	25	0.26	25	0.26
Lettuce	32	0.32	31	0.33
Peas	28	0.28	27	0.29
Potatoes	118	1.20	116	1.23
Onions	33	0.33	32	0.34
Carrots	33	0.33	32	0.34
Tomatoes	26	0.26	25	0.27
Fresh fruit	112	1.13	110	1.16
Fruit products	59	0.60	58	0.62
Sugar and preserves	133	1.34	130	1.38
Bread	491	4.97	481	5.09
Other cereals	458	4.64	449	4.76
Beverages	118	1.20	116	1.23
Freshwater fish	492	4.98	482	5.11
Marine	4,014	40.66	3,934	41.69
Canned	159	1.61	156	1.65
Shellfish	572	5.80	561	5.94
Total Diet	**7,104**	**71.96**	**6,962**	**73.79**
Soil Ingestion	**1,480**	**14.99**	**1,450**	**15.37**
Drinking Water	**913**	**9.24**	**894**	**9.48**
Air	**376**	**3.81**	**128**	**1.35**
Total Intake	**9,873**	**100.00**	**9,435**	**100.00**

Appendix 5.8 Inorganic arsenic: child (2–7 yrs) intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	12	0.30	12	0.32
Pork	1	0.02	1	0.02
Lamb	1	0.02	1	0.02
Liver	3	0.06	3	0.07
Kidney	3	0.06	3	0.07
Meat products	17	0.42	17	0.45
Poultry	2	0.05	2	0.05
Whole milk	3	0.07	3	0.07
Skimmed milk	4	0.10	4	0.11
Cheese	0	0.01	0	0.01
Butter	0	0.00	0	0.00
Milk products	0	0.01	0	0.01
Ice cream	0	0.01	0	0.01
Yoghurt	0	0.01	0	0.01
Cream	0	0.00	0	0.00
Eggs	89	2.14	87	2.27
Vegetable oil	9	0.21	9	0.23
Animal oil	6	0.14	6	0.15
Cabbage	19	0.46	19	0.49
Lettuce	24	0.57	23	0.60
Peas	21	0.49	20	0.53
Potatoes	89	2.13	87	2.26
Onions	24	0.58	24	0.62
Carrots	24	0.58	24	0.62
Tomatoes	20	0.47	19	0.50
Fresh fruit	84	2.01	82	2.14
Fruit products	44	1.07	44	1.13
Sugar and preserves	99	2.39	97	2.54
Bread	368	8.82	361	9.38
Other cereals	343	8.24	337	8.75
Beverages	89	2.13	87	2.26
Freshwater fish	0	0.00	0	0.00
Marine	0	0.00	0	0.00
Canned	0	0.00	0	0.00
Shellfish	0	0.00	0	0.00
Total Diet	**1,400**	**33.59**	**1,372**	**35.69**
Soil Ingestion	**1,480**	**35.50**	**1,450**	**37.72**
Drinking Water	**913**	**21.89**	**894**	**23.26**
Air	**376**	**9.02**	**128**	**3.33**
Total Intake	**4,169?**	**100.00**	**3,845**	**100.00**

Appendix 5.9 Total arsenic: child (3 mths–7 yrs) intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	8	0.20	8	0.20
Pork	1	0.01	1	0.01
Lamb	0	0.01	0	0.01
Liver	2	0.04	2	0.04
Kidney	2	0.04	2	0.04
Meat products	12	0.28	11	0.29
Poultry	1	0.03	1	0.03
Whole milk	2	0.05	2	0.05
Skimmed milk	3	0.07	3	0.07
Cheese	0	0.01	0	0.01
Butter	0	0.00	0	0.00
Milk products	0	0.01	0	0.01
Ice cream	0	0.01	0	0.01
Yoghurt	0	0.01	0	0.01
Cream	0	0.00	0	0.00
Eggs	59	1.43	58	1.46
Vegetable oil	6	0.14	6	0.15
Animal oil	4	0.10	4	0.10
Cabbage	13	0.31	12	0.31
Lettuce	16	0.38	15	0.39
Peas	14	0.33	13	0.34
Potatoes	59	1.43	58	1.46
Onions	16	0.39	16	0.40
Carrots	16	0.39	16	0.40
Tomatoes	13	0.31	13	0.32
Fresh fruit	56	1.35	55	1.38
Fruit products	30	0.71	29	0.73
Sugar and preserves	66	1.60	65	1.63
Bread	245	5.91	240	6.04
Other cereals	229	5.52	224	5.64
Beverages	59	1.43	58	1.46
Freshwater fish	246	5.93	241	6.06
Marine	2,007	48.37	1,967	49.47
Canned	80	1.92	78	1.96
Shellfish	286	6.90	280	7.05
Total Diet	**3,552**	**85.61**	**3,481**	**87.55**
Soil Ingestion	**0**	**0.00**	**0**	**0.00**
Drinking Water	**456**	**11.00**	**447**	**11.25**
Air	**141**	**3.40**	**48**	**1.21**
Total Intake	**4,149?**	**100.00**	**3,976**	**100.00**

Appendix 5.10 Inorganic arsenic: child (3 mths–7 yrs) intake and exposure from various sources

	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	6	0.42	6	0.45
Pork	0	0.03	0	0.03
Lamb	0	0.02	0	0.03
Liver	1	0.09	1	0.09
Kidney	1	0.09	1	0.09
Meat products	9	0.59	9	0.63
Poultry	1	0.07	1	0.08
Whole milk	1	0.09	1	0.10
Skimmed milk	2	0.15	2	0.16
Cheese	0	0.01	0	0.02
Butter	0	0.01	0	0.01
Milk products	0	0.02	0	0.02
Ice cream	0	0.01	0	0.01
Yoghurt	0	0.02	0	0.02
Cream	0	0.00	0	0.00
Eggs	45	3.01	44	3.21
Vegetable oil	4	0.30	4	0.32
Animal oil	3	0.20	3	0.22
Cabbage	10	0.64	9	0.69
Lettuce	12	0.80	12	0.85
Peas	10	0.70	10	0.74
Potatoes	44	3.00	44	3.20
Onions	12	0.82	12	0.88
Carrots	12	0.82	12	0.88
Tomatoes	10	0.66	10	0.70
Fresh fruit	42	2.83	41	3.02
Fruit products	22	1.50	22	1.60
Sugar and preserves	50	3.36	49	3.58
Bread	184	12.41	180	13.23
Other cereals	172	11.59	168	12.35
Beverages	44	3.00	44	3.20
Freshwater fish	185	12.45	181	13.28
Marine	0	0.00	0	0.00
Canned	0	0.00	0	0.00
Shellfish	0	0.00	0	0.00
Total Diet	**885**	**59.70**	**867**	**63.65**
Soil Ingestion	**0**	**0.00**	**0**	**0.00**
Drinking Water	**456**	**30.79**	**447**	**32.83**
Air	**141**	**9.51**	**48**	**3.52**
Total Intake	**4,182**	**100.00**	**1,362**	**100.00**

Appendix 5.11 Exposure to arsenic during a 3-month breast-feeding period

	DAILY INTAKE (ng)	DAILY EXPOSURE (ng)	INTAKE (%)	EXPOSURE (%)
Breast milk	525.0	514.5	87	95
Air	77.3	26.3	13	5

Appendix 6.1 Mercury levels in various foodstuffs and environmental media

Source	Concentration (µg/kg)
Beef	7
Pork	7
Lamb	5
Liver	16
Kidney	19
Poultry	5
Meat products	2.5
Whole milk	1
Skimmed milk	1
Cheese	2
Butter	1
Milk products	1
Ice cream	1
Yoghurt	1
Cream	1
Eggs	4
Vegetable oil	1
Animal oil	1
Cabbage	2
Lettuce	3
Peas	0.75
Potatoes	1
Onions	0.5
Carrots	2.8
Tomatoes	2
Fresh fruit	1
Fruit products	1
Sugar and preserves	1
Bread	0.6
Other cereals	2
Beverages	0.5
Freshwater fish (Trout)	40
Marine fish (Cod)	220
Canned fish (Tuna)	120
Shellfish (Prawns)	140
Soil	400
Drinking Water	0.03
Air	0.02

Appendix 6.2 Levels of mercury in meat (beef, pork and lamb) in mg/kg (fresh weight)

SOURCE	RANGE	MEAN	SOURCE
Beef	0.005–0.022	<0.007	MAFF (1984)
		0.005	Sell *et al.* (1975)
		<0.01	Stabel-Taucher *et al.* (1975)
	0.004–0.014	0.007	Szprengier (1976)
		0.004	Van De Ven *et al.* (1977)
		0.004	Doyle and Spaulding (1978)
	0.004–0.08	0.007–0.008	Juszkiewicz (1978)
	0.004–0.014	0.008	Szprengier (1978)
		<0.002	Nuurtamo *et al.* (1980)
		<0.01	Kramer *et al.* (1983)
		0.001	Van Der Veen *et al.* (1983)
	<0.001–0.016	0.001	Vos *et al.* (1987)
		0.002–0.003	Vreman *et al.* (1986)
	<0.012	0.002	Falandysz and Gajda (1988)
		0.005	Jorhem *et al.* (1991)
		0.011	Neimi *et al.* (1991)
	0.00027–0.0048	0.0012	Falandysz (1992)
Pork	0.005–0.017	<0.007	MAFF (1984)
		0.009	Jorhem *et al.* (1991)
		0.011	Niemi *et al.* (1991)
		0.0019	Falandeysz (1993)
Lamb	0.005–0.02	<0.006	MAFF (1984)
		<0.005	Froschner and Wolf (1977)
		0.005	Knoppler *et al.* (1979)
		<0.002	Nuurtamo *et al.* (1980)
	<0.001–<0.001	<0.001	Falandysz (1987)
		0.001	Vos *et al.* (1988)
		0.0025	Barghigiani and Ristori (1994)

Appendix 6.3 Levels of mercury in offal, poultry and meat products in mg/kg (fresh weight)

SOURCE	RANGE	MEAN	SOURCE
Offal	0.001–0.02	0.007	MAFF (1984)
Liver		0.01	Sell *et al.* (1975)
		0.006	USA-NBS (1976)
		<0.01–0.04	Stabel-Taucher *et al.* (1975)
		0.006	Van De Ven *et al.* (1977)
		0.045	Froschner and Wolf (1977)
		0.01	Doyle and Spaulding (1978)
		0.005	Flanjak and Lee (1979)
		0.015	Knoppler *et al.* (1979)
		0.002–0.003	Nuurtamo *et al.* (1980)
		<0.01	Kramer *et al.* (1983)
		0.002	Van Der Veen (1983)
		0.004–0.016	Froslie *et al.* (1985)
		<0.01	Vaessen and Ellen (1985)
		0.003–0.007	Vreman *et al.* (1986)
		0.005	Falandysz and Gajda (1988)
	0.007–0.014	0.003–0.004	Vos *et al.* (1988)
		0.006–0.015	Jorhem *et al.* (1991)
		0.012	Niemi *et al.* (1991)
	0.00022–0.066	0.002–0.0042	Falandysz *et al.* (1993)
Kidney	0.013–0.058	0.032	Szprengier (1976)
		0.058	Froschner and Wolf (1977)
		0.01	Van De Ven *et al.* (1977)
		0.02	Doyle and Spaulding (1978)
		0.017–0.018	Juszkiewicz (1978)
	0.038–2.200	0.018	Szprengier (1978)
		0.006	Flanjak and Lee (1979)
		0.042	Knoppler *et al.* (1979)
		0.007	Nuurtamo *et al.* (1980)
		<0.01	Kramer *et al.* (1983)
		0.006	Van Der Veen (1983)
		0.011	Vaessen and Ellen (1985)
		0.005–0.009	Vreman *et al.* (1986)
		0.01	Falandysz and Gajda (1988)
	0.001–0.136	0.008–0.009	Vos *et al.* (1991)
		0.010–0.019	Jorhem *et al.* (1991)
		0.014–0.015	Niemi *et al.* (1991)
	0.00049–0.091	0.007–0.012	Falandysz *et al.* (1993)

Appendix 6.4 Levels of mercury in various vegetables in mg/kg

SOURCE	RANGE	MEAN	SOURCE
Cabbage		0.0019	De Temmerman *et al.* (1986)
		<0.002	Barghigiani and Ristori (1994)
Lettuce	0.0005–0.0028	0.0014	Aichberger (1976)
		0.0014	Barudi and Bielig (1980)
	0.001–0.002	0.001	Varo *et al.* (1980)
		0.003	De Temmerman *et al.* (1986)
	0.0005–0.011	0.002	Wiersma *et al.* (1986)
		0.0018	Barghigiani and Ristori (1994)
Peas		0.00075	De Temmerman *et al.* (1986)
Potatoes	0.001–0.004	0.001	MAFF (1971)
	0.0008–0.015	0.004	Bundesgesundheitsanet (1975)
	0.0004–0.010	0.002	Varo *et al.* (1980)
		0.0009	De Temmerman *et al.* (1986)
	<0.0001–0.017	0.003	Wiersma *et al.* (1986)
		0.0022	Barghigiani and Ristori (1994)
Onions		0.0005	De Temmerman *et al.* (1986)
		<0.002	Barghigiani and Ristori (1994)
Carrots	0.0008–0.0028	0.001	Aichberger (1976)
		0.0028	De Temmerman *et al.* (1986)
	0.0006–0.005	0.002	Wiersma *et al.* (1986)
		0.0026	Barghigiani and Ristori (1994)
Tomatoes	0.0003–0.001	0.0006	Aichberger (1976)
		0.01	(UK) Cross *et al.* (1978)
		0.002	Barudi and Bielig (1980)
		0.0017	De Temmerman *et al.* (1986)
	0.0001–0.008	0.0013	Wiersma *et al.* (1986)
		0.0013	Barghigiani and Ristori (1994)

Appendix 6.5 Intakes and exposure to mercury (adults)

SOURCE	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	52	2.42	7.75	0.56
Pork	28	1.33	4.25	0.31
Lamb	19	0.90	2.89	0.21
Liver	13	0.61	1.94	0.14
Kidney	6	0.26	0.83	0.06
Poultry	56	2.60	0.39	0.03
Meat products	58	2.73	8.74	0.63
Whole milk	47	2.19	7.01	0.51
Skimmed milk	48	2.25	7.22	0.52
Cheese	12	0.55	1.76	0.13
Butter	2	0.10	0.32	0.02
Milk products	6	0.29	0.93	0.07
Ice cream	5	0.23	0.73	0.05
Yoghurt	6	0.28	0.90	0.07
Cream	1	0.04	0.13	0.01
Eggs	38	1.78	5.71	0.41
Vegetable oil	2	0.11	0.36	0.03
Animal oil	8	0.37	1.20	0.09
Cabbage	19	0.90	2.90	0.21
Lettuce	19	0.89	2.84	0.21
Peas	8	0.39	1.24	0.09
Potatoes	47	2.22	7.11	0.52
Onions	7	0.30	0.98	0.07
Carrots	36	1.70	5.46	0.40
Tomatoes	26	1.22	3.90	0.28
Fresh fruit	32	1.50	4.79	0.35
Fruit products	17	0.79	2.54	0.18
Sugar and preserves	11	0.50	1.59	0.12
Bread	24	1.10	3.53	0.26
Other cereals	73	3.43	10.99	0.80
Beverages	22	1.01	3.23	0.23
Freshwater fish (Trout)	66	3.07	62.32	4.52
Marine fish (Cod)	981	45.92	932.14	67.56
Canned fish (Tuna)	127	5.95	120.84	8.76
Shellfish (Prawns)	29	1.38	27.93	2.02
Total Diet	**1,951**	**91.29**	**1,247**	**90.41**
Soil Ingestion	**4**	**0.17**	**0.54**	**0.04**
Drinking Water	**22**	**1.02**	**3.29**	**0.24**
Air	**161**	**7.52**	**128.48**	**9.31**
Total Intake	**2,137**	**100.00**	**1,379.69**	**100.00**

Appendix 6.6 Intake and exposure to mercury (7–14 years)

SOURCE	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	26	2.32	2.58	0.28
Pork	14	1.27	1.42	0.16
Lamb	10	0.86	0.96	0.13
Liver	6	0.58	0.65	0.07
Kidney	3	0.25	0.28	0.03
Poultry	28	2.50	0.00	0.02
Meat products	29	2.62	2.91	0.90
Whole milk	23	2.10	2.34	0.26
Skimmed milk	24	2.16	2.41	0.26
Cheese	6	0.53	0.59	0.05
Butter	1	0.10	0.11	0.02
Milk products	3	0.28	0.31	0.05
Ice cream	2	0.22	0.24	0.04
Yoghurt	3	0.27	0.30	0.05
Cream	0	0.04	0.04	0.01
Eggs	19	1.71	1.90	0.21
Vegetable oil	1	0.11	0.12	0.01
Animal oil	4	0.36	0.40	0.04
Cabbage	10	0.87	0.97	0.11
Lettuce	9	0.85	0.95	0.10
Peas	4	0.37	0.41	0.05
Potatoes	24	2.13	2.37	1.04
Onions	3	0.29	0.33	0.04
Carrots	18	1.63	1.82	0.20
Tomatoes	13	1.17	1.30	0.71
Fresh fruit	16	1.44	1.60	0.98
Fruit products	8	0.76	0.85	0.52
Sugar and preserves	5	0.48	0.53	0.06
Bread	12	1.06	1.18	0.13
Other cereals	37	3.29	3.66	0.20
Beverages	11	0.97	1.08	0.12
Freshwater fish (Trout)	33	2.95	31.49	3.46
Marine fish (Cod)	491	44.06	470.98	73.00
Canned fish (Tuna)	64	5.71	61.06	6.72
Shellfish (Prawns)	15	1.32	14.11	1.55
Total Diet	**975**	**87.59**	**612**	**91.71**
Soil Ingestion	**7**	**0.65**	**0.72**	**0.02**
Drinking Water	**11**	**0.98**	**1.10**	**1.20**
Air	**120**	**10.78**	**96.00**	**7.07**
Total Intake	**1,114**	**100.00**	**710.03**	**100.00**

Appendix 6.7 Intakes and exposure to mercury (2–7 years)

SOURCE	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	13	2.20	1.29	0.35
Pork	7	1.21	0.71	0.19
Lamb	5	0.82	0.48	0.13
Liver	3	0.55	0.32	0.09
Kidney	1	0.23	0.14	0.04
Poultry	14	2.36	1.39	0.37
Meat products	15	2.48	1.46	0.39
Whole milk	12	1.99	1.17	0.31
Skimmed and semi-skimmed	12	2.05	1.20	0.32
Cheese	3	0.50	0.29	0.08
Butter	1	0.09	0.05	0.01
Milk products	2	0.26	0.16	0.04
Ice cream	1	0.21	0.12	0.03
Yoghurt	2	0.26	0.15	0.04
Cream	0	0.04	0.02	0.01
Eggs	10	1.62	0.95	0.25
Vegetable oil	1	0.10	0.06	0.02
Animal oil	2	0.34	0.20	0.05
Cabbage	5	0.82	0.48	0.13
Lettuce	5	0.81	0.47	0.13
Peas	2	0.35	0.21	0.06
Potatoes	12	2.01	1.18	0.32
Onions	2	0.28	0.16	0.04
Carrots	9	1.55	0.91	0.24
Tomatoes	7	1.11	0.65	0.17
Fresh fruit	8	1.36	0.80	0.21
Fruit products	4	0.72	0.42	0.11
Sugar and preserves	3	0.45	0.27	0.07
Bread	6	1.00	0.59	0.16
Other cereals	18	3.11	1.83	0.49
Beverages	5	0.92	0.54	0.14
Freshwater fish (Trout)	16	2.79	15.74	4.21
Marine fish (Cod)	245	41.72	235.49	63.04
Canned fish (Tuna)	32	5.41	30.53	8.17
Shellfish (Prawns)	7	1.25	7.06	1.89
Total Diet	**488**	**82.95**	**307**	**82.32**
Soil Ingestion	**15**	**2.52**	**1.48**	**0.40**
Drinking Water	**5**	**0.93**	**0.55**	**0.15**
Air	**80**	**13.61**	**64.00**	**17.13**
Total Intake	**588**	**100.00**	**373.53**	**100.00**

Appendix 6.8 Intakes and exposure to mercury (3 mths–2 yrs)

SOURCE	INTAKE (µg/yr)	INTAKE (%)	EXPOSURE (µg/yr)	EXPOSURE (%)
Beef	6	2.33	0.65	0.36
Pork	4	1.28	0.35	0.20
Lamb	2	0.87	0.24	0.14
Liver	2	0.59	0.16	0.09
Kidney	1	0.25	0.07	0.04
Poultry	7	2.51	0.25	0.14
Meat products	7	2.63	0.73	0.41
Whole milk	6	2.11	0.58	0.33
Skimmed and semi-skimmed	6	2.17	0.60	0.34
Cheese	1	0.53	0.15	0.08
Butter	0	0.10	0.03	0.02
Milk products	1	0.28	0.08	0.04
Ice cream	1	0.22	0.06	0.03
Yoghurt	1	0.27	0.08	0.04
Cream	0	0.04	0.01	0.01
Eggs	5	1.72	0.48	0.27
Vegetable oil	0	0.11	0.03	0.02
Animal oil	1	0.36	0.10	0.06
Cabbage	2	0.87	0.24	0.14
Lettuce	2	0.86	0.24	0.13
Peas	1	0.37	0.10	0.06
Potatoes	6	2.14	0.59	0.33
Onions	1	0.29	0.08	0.05
Carrots	5	1.65	0.46	0.26
Tomatoes	3	1.18	0.33	0.18
Fresh fruit	4	1.44	0.40	0.22
Fruit products	2	0.77	0.21	0.12
Sugar and preserves	1	0.48	0.13	0.07
Bread	3	1.06	0.29	0.17
Other cereals	9	3.31	0.92	0.52
Beverages	3	0.97	0.27	0.15
Freshwater fish (Trout)	8	2.96	7.87	4.43
Marine fish (Cod)	123	44.34	117.74	66.30
Canned fish (Tuna)	16	5.75	15.26	8.60
Shellfish (Prawns)	4	1.33	3.53	1.99
Total Diet	**244**	**88.16**	**153**	**86.33**
Soil Ingestion	**0**	**0.00**	**0.00**	**0.00**
Drinking Water	**3**	**0.99**	**0.27**	**0.15**
Air	**30**	**10.85**	**24.00**	**13.52**
Total Intake	**277**	**100.00**	**177.58**	**100.00**

Appendix 6.9 Exposure to mercury during the 3-month breast-feeding period

Source	Daily intake (µg)	Daily exposure (µg)	Total exposure (µg)	Contribution (%)
Breast milk	1.50	0.21	18.90	94
Air	0.02	0.01	1.18	6
TOTAL	1.52	0.2	20.08	100

Assuming the absorption of mercury in breast milk in the GI tract is 14 per cent, and the concentration is 2 ng/ml.

Appendix 8.1 Congener-specific parameters used in the model

Congener	Concentration (pg/m^3)	Vapour fraction	Particulate fraction	Log Kow	Henry's Constant ($atm\text{-}m^3/mol$)	BCF
2,3,7,8-TCDD	0.006	0.55	0.45	6.64	1.6 E-05	4.32
1,2,3,7,8-PCDD	0.004	0.26	0.74	6.64	2.6 E-06	4.16
1,2,3,4,7,8-HxCDD	0.006	0.07	0.93	7.79	1.2 E-05	2.02
1,2,3,6,7,8-HxCDD	0.008	0.02	0.98	7.79	1.2 E-05	2.24
1,2,3,7,8,9-HxCDD	0.009	0.04	0.96	7.30	1.2 E-05	1.74
1,2,3,4,6,7,8-HpCDD	0.130	0.02	0.98	8.20	7.5 E-06	0.36
OCDD	0.248	0.00	1.00	7.59	7.0 E-09	0.52
2,3,7,8-TCDF	0.027	0.71	0.29	6.53	8.6 E-06	0.94
2,3,4,7,8-PCDF	0.012	0.30	0.70	6.92	6.2 E-06	3.10
1,2,3,7,8-PCDF	0.010	0.42	0.58	6.79	6.2 E-06	0.73
1,2,3,4,7,8-HxCDF	0.039	0.06	0.94	7.30	1.4 E-05	2.34
1,2,3,7,8,9-HxCDF	0.004	0.06	0.94	7.30	6.1 E-06	2.00
1,2,3,6,7,8-HxCDF	0.021	0.11	0.89	7.30	1.0 E-05	2.00
2,3,4,6,7,8-HxCDF	0.022	0.07	0.93	7.30	1.0 E-05	1.78
1,2,3,4,6,7,8-HpCDF	0.073	0.04	0.96	7.90	5.3 E-05	0.41
1,2,3,4,7,8,9-HpCDF	0.010	0.03	0.97	7.90	5.3 E-05	0.99
OCDF	0.006	0.00	1.00	8.80	1.9 E-06	0.20

Appendix 8.2 Media-specific parameters used in the model

Media-specific variables	Grass	Hay/silage	Soil
Fraction dry matter	0.15	0.3	
Leaf density (g/l)	770	890	
Bulk correction	1	0.5	
Fraction of diet	0.47	0.47	0.06
Bioavailability (soil relative to vegetation)			0.65
Fraction of particles intercepted	0.35	0.62	
Dry matter yield of crop (kg/m^2)	0.15	0.63	

Appendix 8.3 General parameters used in the model

General variables	
Dry deposition rate (cm/s)	0.2
Air density (g/l)	1.19
Ideal gas constant ($atm\text{-}m^3/mol\ oK$)	8.21 x 10 -5
Temperature oK	298.1
Dry deposition rate (m/yr)	63,072
Retention of wet deposition on plants (fraction)	0.3
Half-life on vegetation (days)	14
Weathering dissipation constant (1/yr)	18.07
Half-life in soil (years)	10
Weathering dissipation constant (1/yr)	0.0693
Soil mixing depth (cm)	1
Soil bulk density (g/cm^2)	1.5
Soil mixing mass (kg)	15