C: Monitoring and Assessment Research Centre

S: Global Environmental Monitoring System

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EXPOSURE COMMITMENT ASSESSMENTS OF ENVIRONMENTAL POLLUTANTS

Volume 1, Number 1

Technical Report

Prepared by:
MONITORING AND ASSESSMENT RESEARCH CENTRE
Chelsea College, University of London

With the support of:
UNITED NATIONS ENVIRONMENT PROGRAMME and
THE ROCKEFELLER FOUNDATION

The Monitoring and Assessment Research Centre (MARC), Chelsea College, University of London, became operational on 1 July 1975.

The broad objective of the Centre is to develop methods which will assist in the understanding, definition, evaluation and solution of major environmental problems of global, regional and national concern. Increasing international awareness of these problems, such as chemical pollution, depletion of soil, forest-cover and other important natural resources as well as the spread of endemic diseases, has emphasized the need for such an approach. In this way the Centre offers scientific support to the development of environmental monitoring systems and in particular to the Global Environmental Monitoring System [GEMS] of the United Nations Environment Programme.

The Centre's work is funded by the United Nations Environment Programme and The Rockefeller Foundation.

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Exposure commitment assessments of environmental pollutants

Volume 1, Number 1

Exposure commitment concepts and application;
summary exposure assessments for lead, cadmium and arsenic

by B. G. Bennett

A Technical Report (1981)

Prepared by:
Monitoring and Assessment Research Centre
Chelsea College, University of London

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FOREWORD

The assessment of the consequences of releasing pollutants into the environment is a task which occupies numerous governmental, industrial, and research institutions. This is in recognition of the large number and amounts of chemicals utilized by man and the actual or potential adverse impacts of man-made pollutant releases.

The Monitoring and Assessment Research Centre has taken the initiative in applying the exposure commitment method to the assessment of chemical pollutant environmental behaviour. The exposure commitment is a measure of the total exposure to a pollutant substance and a useful means of expressing source-receptor relationships.

The objective of this and subsequent MARC Reports of this title—Exposure Commitment Assessments of Environmental Pollutants—is to discuss the exposure commitment method and to illustrate its application to an expanding list of chemical pollutants. The utility and limitations of the time independent commitment method will be discussed—the concepts and definitions in this issue, and the relevance of the method and its relationship to environmental monitoring in subsequent issues.

Summary assessments of pollutant behaviour and exposures will be presented and updated as new information becomes available. The application of the method is illustrated for some persistent chemicals of general concern. The initial presentations are based on representative pollutant levels and behaviour. More specific applications and the consideration of special pathways and critical groups will be added to this basic framework in future issues and as data become available. Human exposures are of primary concern here, but the discussions and methods also have relevance with regard to exposures of other biosphere components.

It is hoped that this publication will keep interested individuals informed of this area of work at MARC and that it will stimulate discussion regarding the pollutant assessments. The author would welcome comments on pollution problems, recent studies and measurements of sources, levels and special pathways, and suggestions for improved values of the transport parameters.

Exposure commitment concepts and application

Abstract

The Monitoring and Assessment Research Centre (MARC) has been developing and applying the exposure commitment method to the assessment of pollutant transport in the regional and global environment. This paper gives the definitions, notation, and basic concepts of the method and briefly discusses the application to the transfer of pollutants from sources to man. Exposure commitments give a basis for comparing contributions to exposure from various pathways and for estimating equilibrium concentrations resulting from continuing releases. The method provides a convenient framework for expressing source-receptor relationships.

1 Introduction

In order to assess the consequences of releasing pollutants into the environment, it is necessary to understand the behaviour of the contaminants in the various environmental regions, determine the amounts which are ultimately transferred to man and the biosphere, and relate the levels or burdens with harmful effects. The commitment method, a time independent assessment procedure, has been successfully applied by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) to the release of radioactive materials into the environment (UNSCEAR 1977). An adaptation of this method—the exposure commitment method—is being developed and applied at MARC to non-radioactive pollutant releases (Barry 1979, O'Brien 1979, Miller and Buchanan 1979, Wiersma 1979).

Until the mechanisms by which contaminants may cause deleterious effects are properly understood, there is uncertainty regarding the most relevant measure which should be the basis for assessments. For radioactivity, the absorbed dose in tissue, which is the energy imparted per unit mass of tissue, is estimated, and it is assumed that the risk of harmful effects is directly proportional to the accumulated dose. For non-radioactive pollutants, it can only be assumed that the concentrations of the contaminants measured in the receptor organism are most closely associated with risks of effects. There is some recognition that the duration of exposures is also a factor in effects manifestation, although this is not always quantified. Threshold levels may be absent for some substances but present for others; for example, those which are essential nutrients at low levels can be toxic contaminants at higher levels. Even without thresholds, it is uncertain that single, repeated or continuing exposures are of themselves directly or uniquely related to observable effects. Interactions with other substances may enhance or retard the development of consequential changes. In addition, living organisms and populations often adapt to repeated exposures to pollutants.

Given the uncertainties and complexities involved, one can only proceed along the most reasonable lines that can at present be perceived. The basic concepts of the assessment method presented here are the time-integrated concentrations of pollutants in source, pathway, and receptor reservoirs, the transfer functions which relate the changes in exposures between compartments, and the commitments to intake or exposure which are made when pollutants are released into the environment.

These concepts are discussed below, and definitions and procedures are presented to apply the commitment method to the assessment of environmental problems.

2 Concepts and definitions

When a pollutant is released into the environment, it is dispersed along various routes through air, water, soil and the biosphere. The fractional amounts which reach man along different pathways will differ, as will the time course of the exposure. For example, a contaminant emitted into the air may be inhaled within a few hours or it may be deposited on soil, absorbed by plants and included in diet in a persistent fashion for years into the future. The behaviour of the pollutant in all parts of the environment must be analysed.

In considering environmental systems, we are generally concerned with the concentrations of contaminants in subsections (called reservoirs or compartments) of the environment in which the contaminant can be assumed to be reasonably well mixed. The major reservoirs are the atmosphere, oceans, soil, lakes and streams, ground water, diet, and man. It may be useful to make further distinctions, for example, between the troposphere and stratosphere or between the surface and deep ocean. The compartment model must usually be a compromise between a detailed, realistic representation of a system and a simplified, manageable version.

Having formulated a model for an environmental system, the next objective is to determine the relationships between concentrations in the various compartments. Transfers of the contaminant may occur in various directions between compartments and at different rates. A first approach may be to assume that each reservoir is well mixed and that the transfers obey first order kinetics, which implies that a transfer such as from compartment i to compartment j is proportional to the amount of contaminant in compartment i. Mathematically this is expressed

$$\frac{\mathrm{d}Q_i}{\mathrm{d}t} = -k_{ij}Q_i$$

The proportionality factor, k_{ij}, is known as the rate constant. The solution to this equation is

$$Q_i = Q_{0i}\, \mathrm{e}^{-k_{ij}t}$$

for the single pathway. Beginning with the amount Q_{0i}, the initial amount of contaminant in compartment i, retention is described by the exponential function. The average concentration at a particular time is obtained by dividing the amount of contaminant present by the mass or volume of the compartment.

In the general case, the rate of change of compartment contents is described by a long series of losses and gains along numerous pathways. The evaluation of compartment sizes and rate constants is a prime objective in developing a dynamic (i.e. time-dependent) model. In general, a large data base is required as well as an accurate formulation of the environmental model.

An alternative approach to characterize the movement of pollutants through the environment is the exposure commitment method. This method deals with the time-integrated values of transfer and can usually proceed on a much more limited data base. The time-integrated values are a convenient way of expressing transfer results, even for a dynamic model, but the commitment approach does not require the transfer rates to be evaluated or even that the transfer mechanisms be fully understood. It is sufficient in continuing, constant emission situations simply to determine the equilibrium concentrations. It will be shown that this measure is equivalent to the integrated concentration from a single release.

Let us now define some terms which will be useful in the assessment analyses. The average *concentration* of a pollutant in a reservoir is the amount of pollutant per unit volume or mass of the reservoir material. Concentration is sometimes referred to as the level. Usually the concentration is averaged over the whole volume of the reservoir, but in some cases it may refer to a subregion of the reservoir. Examples of expressions of concentrations are thus 2 μg lead per m^3 of air, or 40 μg lead per g of soil in the 0–20 cm depth region.

Exposure is defined as the time integral of the concentration over a specified time. For a pollutant in compartment i, the exposure from time 0 to time T is

$$E_i(T) = \int_0^T C_i(t)\,dt$$

where $C_i(t)$ is the concentration in the compartment at times prior to T. In many cases the integrals are rather easily obtained. For example, if the concentration is maintained at a constant value over a period of time, the

exposure during that interval is simply the product of the concentration and the time. Thus if we are given that the annual average concentration of lead in air is 1 $\mu g\,m^{-3}$, the exposure of air to lead during the year is 1 $\mu g\,y\,m^{-3}$.

The *flux* is a measure of the rate of flow of a substance moving from one compartment to another. It expresses the quantity of the substance passing per unit time. *Intake* is the total quantity of substance transferred up to a specified time. Intake to a specific compartment is thus the integral over the time interval of interest of the flux into that compartment. For example, ingestion rate of lead in diet by man, corresponding to a flux, could average 1 $\mu g\,d^{-1}$. The total annual ingested amount of lead, or intake by this pathway, would be 365 μg. The mathematical expression for the intake is

$$I_b(T) = \int_0^T F_{ab}(t)\,dt$$

where $I_b(T)$ is the intake to time T in compartment B from the flux $F_{ab}(t)$ from compartment A at times prior to T.

Exposure and intake up to a particular time have been defined as the integrals of concentration or flux over the specified time period. When the integrals are extended to cover all time, the quantities obtained are the *exposure commitment* and the *intake commitment*. These give a measure of the intake or exposure due to a finite release of a pollutant for as long as the pollutant may be present in the compartment of interest. If a compartment has an infinite residence time, that is, if it is a sink for a particular pollutant, the exposure commitment to that compartment will be infinite. The exposure commitments to other compartments would be finite. The intake commitment is finite except in those cases where the pollutant circulates indefinitely.

To illustrate the evaluation of the exposure commitment, consider the hypothetical example in Figure 1. The finite release of a pollutant into the environment in a specific year has given rise to a concentration in a compartment of annual average value A in the first year following release, B in the second year, C in the third year, and so on. The units may be μg per m^3 of air in a well mixed region from the non-localized release of 1 tonne of the pollutant. The annual exposures to air in this case are A $\mu g\,y\,m^{-3}$, B $\mu g\,y\,m^{-3}$, etc. The exposure commitment for this example is (A+B+C+D+E) $\mu g\,y\,m^{-3}$ per tonne released.

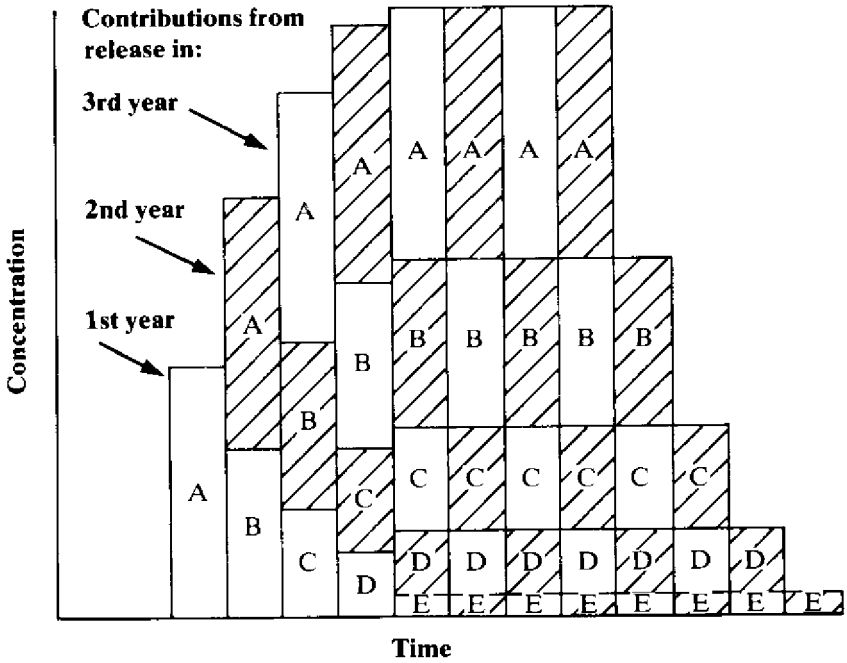


Figure 1 Contributions to the concentration in an environmental compartment due to constant, continuing pollutant releases

For constant, continuing releases, the contributions to concentrations due to release in any particular year are superimposed upon the concentration contributions of previous years' releases, as illustrated in Figure 1. The contributions from alternate year releases are shaded in the figure for clarity. If the emissions continue for a longer period than the time to receive the exposure commitment from a single release, a state of equilibrium is obtained. It is then seen that the annual exposure rate or level is $(A+B+C+D+E)\ \mu g\ m^{-3}$ per tonne per year released. This demonstrates the important feature that the exposure commitment from a finite release of unit magnitude has numerically the same value as the steady-state concentration from a continuing release of unit magnitude rate.

Finite release: exposure commitment per unit release

$$[\mathrm{A}+\mathrm{B}+\mathrm{C}+\mathrm{D}+\mathrm{E}]\frac{\mu\mathrm{g\ y\ m}^{3}}{t},$$

Continuing release: steady-state concentration per unit release rate

$$[\mathrm{A}+\mathrm{B}+\mathrm{C}+\mathrm{D}+\mathrm{E}]\frac{\mu\mathrm{g\ m}^{-3}}{t\mathrm{y}^{-1}},$$

The units, although expressed differently in each situation, are entirely equivalent.

The *transfer function* describes mathematically how the level of a pollutant in a receptor compartment at time t depends on the level in the donor compartment at all previous times. For movement of the pollutant from compartment A to compartment B with transfer function $P_{ab}(t)$, the relationship between the levels at time T is

$$C_b(T)=\int_0^T P_{ab}(T-t)C_a(t)\,\mathrm{d}t$$

Thus, the concentration in compartment B at time T is a weighted sum of concentrations in compartment A at previous times. The weighting is a function of the time since the transfer took place.

The infinite integral of the transfer function is called the *transfer coefficient*, P_{ab}.

$$P_{ab}=\int_0^\infty P_{ab}(T-t)\,\mathrm{d}t$$

This is a very useful parameter in the commitment approach. It can be shown that the transfer coefficient is equal to the ratio of the infinite integrals of the levels in the receptor and donor compartments, which is the ratio of exposure commitments (O'Brien 1979).

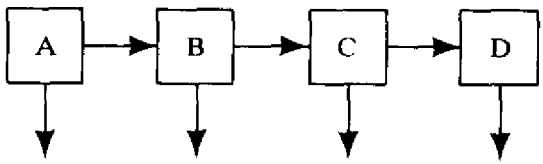
$$P_{ab}=\frac{\int_0^\infty C_b(t)\,\mathrm{d}t}{\int_0^\infty C_a(t)\,\mathrm{d}t}=\frac{E_b}{E_a}$$

In the case of continuing releases, after reaching steady state in compartments other than sinks, the levels are constant, denoted C_a^* and C_b^*. Evaluation of the above equation gives

$$P_{ab} = \frac{C_b^*}{C_a^*}$$

Thus, when there is a state of equilibrium, the transfer coefficient is easily determined, namely, the ratio of steady-state levels in the receptor and donor compartments.

For a chain of compartments in an environmental system the exposure commitment in the receptor is obtained by sequential multiplication of transfer coefficients. Thus for the system of compartments shown here,



the exposure commitment in the final compartment of the chain is

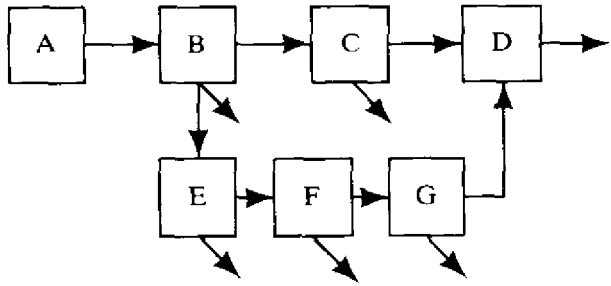
$$\begin{aligned} E_d &= P_{cd} E_c \\ &= P_{cd} P_{bc} E_b \\ &= P_{cd} P_{bc} P_{ab} E_a \end{aligned}$$

or, reordering the factors in the sequence of the transfer,

$$E_d = P_{ab} P_{bc} P_{cd} E_a$$

It is important to note that there are usually losses from each compartment in addition to the transfer along the pathway being studied. These losses may be (1) transfers to other pathways, (2) transfers to sinks, or (3) transformation of pollutant form. Parallel pathways to the same receptor compartment are treated by the principle of superposition, i.e. it is assumed that the systems involved are linear. An example of parallel

pathways is



$$E_d = [P_{ab}P_{bc}P_{cd} + P_{ab}P_{be}P_{ef}P_{fg}P_{gd}]E_a$$

The two products of transfer factors allow the importance of the corresponding pathways to be compared.

Relationships between intake commitments can be formulated in a similar fashion to those for exposure commitments. Fluxes between compartments are partitioned among the various pathways. If the total flux into compartment B is F_{ab}, then the flux out of the compartment into compartment C, F_{bc}, is determined by the convolution relationship

$$F_{bc}(T) = \int_0^T P_{b'c'}(T-t)F_{ab}(t)\,\mathrm{d}t$$

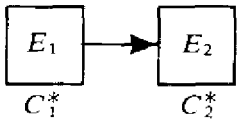
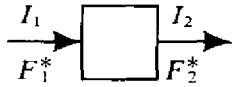
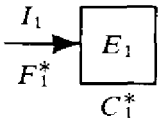
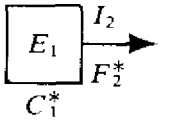
O'Brien (1979) has called the function $P_{b'c'}(T-t)$ the *partitive function* and the infinitive integral of the function the *partitive coefficient*. The partitive coefficient is equal to the ratio of the infinite integrals of the fluxes into and out of a compartment, which is the ratio of intake commitments

$$P_{b'c'} = \frac{\int_0^\infty F_{bc}(t)\,\mathrm{d}t}{\int_0^\infty F_{ab}(t)\,\mathrm{d}t} = \frac{I_c}{I_b}$$

At equilibrium for continuing releases, the partitive coefficient may be evaluated from the ratio of steady-state fluxes

$$P_{b'c'} = \frac{F^*_{bc}}{F^*_{ab}}$$

The analysis of pollutant transfer may proceed either by consideration of transfer coefficients and exposure commitments or of partitive coefficients and intake commitments. In practice, other integral relationships are required, such as between an intake commitment and the resultant exposure commitment. The term *transfer factor* shall be applied to all the various relationships between integral quantities. Transfer coefficients and partitive coefficients are, thus, specific manifestations of what may be more generally referred to as transfer factors. A standard notation for the transfer factor is P_{ij} relating integral quantities in or into compartments i and j. Unprimed subscripts will refer to exposure commitments in the compartment and primed subscripts to intake commitments into the compartment. Examples of the various relationships are given below.

	Finite release	*At steady state*
	$P_{12}=\frac{E_2}{E_1}$	$P_{12}=\frac{C_2^*}{C_1^*}$
	$P_{1'2'}=\frac{I_2}{I_1}$	$P_{1'2'}=\frac{F_2^*}{F_1^*}$
	$P_{1'1}=\frac{E_1}{I_1}$	$P_{1'1}=\frac{C_1^*}{F_1^*}$
	$P_{12'}=\frac{I_2}{E_1}$	$P_{12'}=\frac{F_2^*}{C_1^*}$

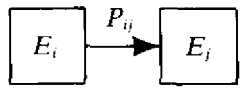
3 Estimation of transfer factors

The evaluation of transfer factors is a primary task in the application of the exposure commitment method. It has been shown above that where steady-state conditions prevail, the transfer factors can be determined by taking the ratio of equilibrium levels or fluxes. Equilibrium can generally be assumed, particularly in remote areas, for naturally occurring substances such as trace metals. Since the levels fluctuate with location and also to some extent with time, values of the equilibrium concentrations should be averaged in space and over sufficiently long time periods to smooth out short-term or local variations in emission patterns.

In some cases it may not be possible to use steady-state values to determine transfer factors. This is so for many man-made compounds, such as pesticides, which do not occur in natural systems or which have been introduced relatively recently, or for which the input has been very variable in amounts, space and time. Even for some pollutants which have a natural background, such as mercury, the natural fluctuations in important reservoirs are quite rapid, and there are important but intermittent emissions from volcanic activity. This makes evaluations of steady-state levels and fluxes very difficult to achieve.

Some specific aspects regarding evaluations of transfer factors are considered below.

(a) Transfer factor relating exposure commitments: P_{ij}

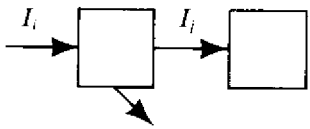


When the pollutant is released over a limited period, it may be possible to obtain a record of concentrations in specific compartments and determine the exposure commitments directly and the transfer factors by ratios of the exposure commitments. Care must be taken to ensure that most of the exposure due to a given release is accounted for. Where data on levels or exposures in intermediate reservoirs are not available, the transfer factors may be estimated to indicate the overall transfer, such as P_{abc} for transfer from compartments A to C through B.

Transfer factors may also be determined if mathematical functions can be determined to describe the changing pollutant levels with time in a series of compartments. This is usually possible when there has been a significantly enhanced release of some pollutant over a relatively short period of a few years, with dispersion processes such that the pollutant

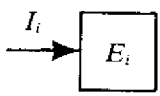
levels are measurable over a longer period of time. This has been the method used to determine transfer factors and to evaluate the dose commitment for ^{90}Sr released during atmospheric nuclear weapons testing (UNSCEAR 1977). The method should also be applicable to many non-radioactive pollutants.

(b) Transfer factor relating intake commitments: $P_{i'j'}$



The considerations of transfer factors with respect to exposure commitments also apply to evaluations based on intake commitments. Direct measurement of steady-state fluxes or of intakes following a specific release may be possible. There are usually several pathways of transfer out of a compartment. The transfer factor then represents a partitioning of the intake amount. A typical measurement of this factor is the fractional absorption of a pollutant substance.

(c) Transfer factor relating an intake commitment to an exposure commitment: $P_{i'i}$



The intake commitment may be the total amount, Q, of the pollutant released by the source. In this case, the transfer factor is

$$P_{i'i} = \frac{E_i}{Q}$$

or for a continuing release rate, q, at steady state

$$P_{i'i} = \frac{C_i^*}{q}$$

The transfer factor $P_{i'i}$ also represents a relationship between the mean residence time of the pollutant in a compartment and the size of the compartment. This is seen by noting that the exposure commitment can be evaluated by dividing the intake commitment by the compartment size M_i (volume or mass) giving concentration, and multiplying by the mean

residence time, $\bar{T}_i$:

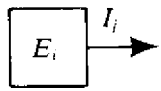
$$E_i = \frac{I_i}{M_i}\bar{T}_i = P_{i'i}I_i$$

Thus

$$P_{i'i} = \frac{\bar{T}_i}{M_i}$$

This is a frequently used association.

(d) Transfer factor relating an exposure commitment to an intake commitment: $P_{ij'}$



The transfer factor $P_{ij'}$ often represents the intake rate of the medium. For example, the exposure commitment to air multiplied by the air inhalation rate gives the intake of the pollutant to lungs.

$$E_1\left(\frac{\mu g\ y}{m^3}\right) \times \underbrace{\text{medium intake rate}}_{P_{12'}}\left(\frac{m^3}{y}\right) = I_2\ (\mu g)$$

The units of the intake rate, which defines the transfer factor, are in this case $m^3\ y^{-1}$. This is equivalent to the fuller expression of the units of the transfer factor determined from the ratio of the intake to exposure commitments, namely, μg per $\mu g\ y\ m^{-3}$. It is important to keep careful track of the units in the expressions to ensure that the sequential relationships are properly formulated.

The relationship $P_{ij'}$ is also the transfer factor which relates the deposition rate to soil from a concentration of pollutant in air. The deposition rate is measured per unit area and the ratio to the air concentration is referred to as the deposition velocity, V_d.

$$V_d = P_{12'} = \frac{I_2(\mu g\ m^{-2})}{E_1\ (\mu g\ y\ m^{-3})}$$

$$= \frac{F^*_{i2}\ (\mu g\ m^{-2}\ y^{-1})}{C^*_1(\mu g\ m^{-3})}$$

The units are expressed slightly differently in the commitment and steady-state formulations; however, it can be seen that they are entirely equivalent. The deposition velocity is just one of a more general list of transfer factors.

4 Further considerations in evaluation of transfer factors

Although determination of the transfer factors is fairly straightforward, there are several areas of complication which should be considered. These include non-linearity, synergistic effects, and heterogeneity.

Linearity implies that the concentration arising from a set of pollutant sources is equal to the sum of concentration contributions from each source considered independently. Increased source emission of a pollutant will increase the concentrations in the relevant compartments by the same factor. The ratios between the steady-state levels will remain constant. The linearity condition is fulfilled when the pollutant compound itself does not induce changes in the involved transfer processes. This is usually the case for low-level or trace occurrence of the pollutant in the environment. At toxic levels, non-linear effects may occur in some subsystems, such as reduced functioning or efficiency in transport processes or in exposure-response relationships.

Synergistic effects are by definition non-linear and thus require separate consideration. The effects may be between two pollutants or between a pollutant and some naturally occurring agent. It may be possible to describe the joint transport of the interacting agents or to determine weighted transfer coefficients for specified source distributions.

The values of the transfer factors depend on the specific set of environmental conditions for which they were derived. Different chemical forms of the pollutant or composition of the media, for example different soil types, may cause transfer to occur to varying extent. Various interactions or changing conditions may require that the values of transfer factors be adjusted. For example, increasing acidity of lakes may cause increased fluxes of heavy metals from the sediments to the water. Some differences in environmental conditions may be judged of minor significance and generalized values of the transfer factors may be used to sufficient approximation.

It is the exceptional case when the environmental compartment is uniformly contaminated by the pollutant substance. Assuming the

compartment as a whole behaves as a well-mixed reservoir is often a sufficiently good approximation. However, there are a number of practical sampling problems in determining the appropriate concentrations and the mean residence times. Concentrations would not be representative in unusual areas of collection or depletion of the pollutant. Gradients in concentrations may occur near interfaces, which must be recognized in specifying the mean concentrations in the media. To the extent that uniform mixing does not occur, there will be differences in compartment parameters depending on the mode of injection of the pollutant into the environment. There may well be important differences in transfer parameters for pollutant and natural background materials. In such cases, it is necessary to study the processes within compartments in greater detail in order to get quantitative estimates of the transfer parameters.

5 Application of the commitment method

The concepts of the exposure commitment method can be made clear by showing the applications in specific pollutant transfer situations. Such examples are included in the summary exposure assessments for lead, cadmium, and arsenic, given elsewhere in this report.

In general, the assessment of pathway transfers begins with a formulation of the main compartmental sequences of the pollutant movement. There is usually a series of parallel pathways from the source to the receptor, for example, inhalation and ingestion of food items from terrestrial and aquatic environments. Figure 2 shows an example of two

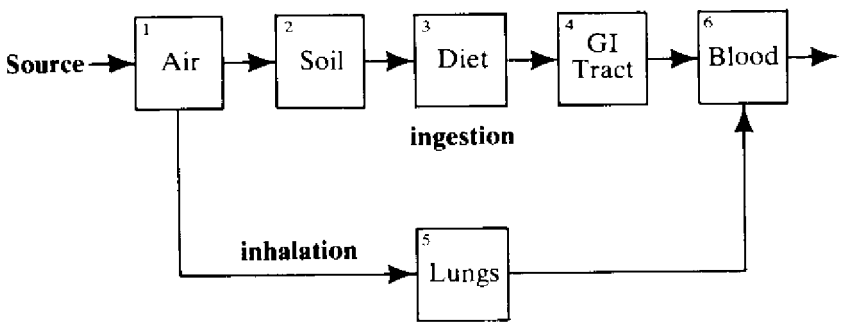


Figure 2 Compartment model for the transfer of a pollutant from atmospheric sources to man

parallel pathways of transfer of a pollutant from general atmospheric sources to the blood of man.

The contributions to exposure commitment in the receptor are obtained by the sequential multiplication of transfer factors. The particular combinations of transfer factors are determined by the availability of data. Use is made of observed equilibrium relationships, for example, between the intake rate of the pollutant in diet and the level in soil. If the compartment is important only in terms of partitioning of the pollutant amounts, it is sufficient to consider only the intake relationships for that compartment. It is not necessary to evaluate the exposure commitment to each compartment.

As an example, consider the inhalation pathway of the system illustrated in Figure 2. Some procedures for evaluation of transfer factors given in Section 3 will be utilized. If the total amount of pollutant Q emitted by the source is known, this defines the intake commitment to air, $I_1 = Q$. The resulting exposure commitment to air, E_1, may be evaluated by measurement or by consideration of the mean residence time of the pollutant in air (Section 3(c)). The transfer factor relating the intake commitment to the exposure commitment is

$$P_{1'1} = \frac{E_1}{Q}$$

The intake commitment to lungs is determined from the exposure commitment to air and the breathing rate (Section 3(*d*)). The transfer factor is equal to the breathing rate and it relates the integral (commitment) quantities by the following formula:

$$P_{15'} = \frac{I_5}{E_1}$$

The lungs may be considered a partitioning compartment, and it will be sufficient to consider the relationship between intake commitment to lungs and to the subsequent compartment (Section 3(b)). The transfer factor is:

$$P_{5'6'} = \frac{I_6}{I_5}$$

While it would have been possible to evaluate the exposure commitment to lungs from the intake amount, the lung mass and the retention time, and then to evaluate the transfer to blood, these steps are combined by simply considering the partitioning ($P_{5'6'}$ evaluated, instead of $P_{5'5}P_{56'}$).

The intake commitment to blood can be related to the exposure commitment to blood by consideration of the mean residence time of the substance in blood and the blood volume (Section 3(c)). The transfer factor is

$$P_{6'6} = \frac{\bar{T}_6}{M_6}$$

or in terms of the commitment quantities

$$P_{6'6} = \frac{E_6}{I_6}$$

The exposure commitment to blood via the inhalation pathway from an amount, Q, released from the source to air is determined from the following sequential multiplication of transfer factors:

$$E_6 = P_{1'1}P_{15'}P_{5'6'}P_{6'6}Q$$

The equivalence of the exposure commitment and the steady-state concentrations, mentioned in Section 2, can be used to evaluate the equilibrium state. The transfer factors have the same numerical values. For example:

$$P_{1'1} = \frac{E_i}{Q}\ \frac{\mu g\ y\ m^{-3}}{t} \quad \text{(finite release)}$$

$$P_{1'1} = \frac{C_1^*}{q}\ \frac{\mu g\ m^{-3}}{t\ y^{-1}} \quad \text{(steady state)}$$

The steady-state concentration in blood from the release rate, q, of the pollutant to air is:

$$C_6^* = P_{1'1}P_{15'}P_{5'6'}P_{6'6}q$$

Evaluation of the contributions from the ingestion pathway are made in a similar manner. Numerical examples are given in the summary exposure assessments for lead, cadmium and arsenic.

The exposure commitment method is a useful means of expressing source-receptor relationships. The exposure commitment to the receptor from a given release or the changes in equilibrium concentrations are evaluated. The contributions from various transfer pathways can be compared. The consequences of different release modes, for example to air or water, can be analysed. Additional discussion of applications of the method, particularly with regard to its relevance and utility in pollutant assessments, is presented in a separate paper (Buchanan and Bennett; to be published).

The evaluation of source strengths and receptor exposures is the first part of pollutant assessment. The second part is to establish exposure-response relationships. The complete assessments, giving source-exposure-effects associations, will be useful in establishing monitoring programmes and in managing the releases of pollutants into the environment.

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LEAD

1 Natural cycle

Lead is an abundantly occurring heavy element. The average content of the earth's crust is 13 $\mu g\, g^{-1}$ (Taylor 1964). The primary form of lead in the natural state is the insoluble sulphide ore, galena, PbS. Other compounds in which lead is found include PbO_2, $PbCO_3$ and $PbSO_4$.

The lead content of soils ranges from about 1 to 500 $\mu g\, g^{-1}$ (Zimdahl and Hassett 1979, Khan 1980). The lowest values are in those of sedimentary or alluvial origin. The highest concentrations of lead occur in the upper horizon of the soil.

The natural lead levels in air are quite low, about 0.5 $ng\, m^{-3}$ (Patterson 1965), originating primarily from wind-blown soil particles with small inputs from volcanic fumes, organic emissions from foliage, forest fires, and sea spray (Table 1).

Lead enters plants by root uptake from soil or by direct deposition from air. Transfer is inefficient and the natural lead levels in plants, animals and man are very low.

2 Anthropogenic sources

Lead is the most widely used non-ferrous metal. It has been mined and manufactured for several thousand years. Emissions of lead by man significantly increased about 250 years ago at the beginning of the Industrial Revolution and again after 1925 when the use of lead in gasoline began. Estimates of current emissions of lead from anthropogenic sources are listed in Table 1. Sixty per cent of the emissions result from gasoline combustion. Localized sources of lead emissions result from metal smelting and refining operations. The release from anthropogenic sources is 18 times that from natural sources.

The trend in emissions from anthropogenic sources has been increasing in recent years: averaging 270, 370, and 430×10^6 $kg\, y^{-1}$ during 1951–60, 1961–70, and 1971–80 respectively (Nriagu 1979). Some countries have begun to limit the use of lead compounds in gasoline.

Table 1 Worldwide emission of lead to the atmosphere[a]

Source	Emission rate (10^6 kg y^{-1})	
Natural		
Windblown dust	16	(0.19–35)[b]
Volcanoes	6.4	(4.2–96)
Vegetation	1.6	(1.6–21)
Forest fires	0.5	(0.04–2.8)
Sea spray	0.02	(0.01–0.05)
Total	24.5	
Anthropogenic[c]		
Gasoline and oil combustion	270	
Non-ferrous metal production	77	
Iron and steel production	50	
Coal combustion	14	
Waste incineration	8.9	
Non-ferrous metal mining	8.2	
Industrial applications	7.4	
Wood combustion	4.5	
Miscellaneous	5.9	
Total	450	

[a] ref. (Nriagu 1979).
[b] range of literature values in brackets.
[c] emission rate during 1975.

Direct sources of lead to soil include lead arsenate insecticides, impurities in fertilizers, and sewage sludge applications. Lead is released to water in liquid effluents from industrial operations and erosion from mining and milling storage and tailing piles.

Various uses of lead can result in sources of transfer to man, such as from painted surfaces, improperly glazed pottery, some pewter containers, improperly soldered cans and electric kettles, lead piping, and food processing operations.

3 Environmental considerations

3.1 Air

The concentrations of lead in urban air range in general from 0.5 to 5 $\mu g\ m^{-3}$. The yearly averages over a five-year period, 1972–77, at the centre of four large cities in Belgium ranged from 0.6 to 1.2 $\mu g\ m^{-3}$ (Kretzschmar, Delespaul and De Rijck 1980). Some reductions in lead concentrations in air have been noted resulting from restricted use of lead in gasoline, such as from 2.8 $\mu g\ m^{-3}$ in 1973–74 to 1.1 $\mu g\ m^{-3}$ in 1976–77 in cities of the Federal Republic of Germany (Jost and Sartorius 1979) and from about 3 $\mu g\ m^{-3}$ in 1970 to 1.5 $\mu g\ m^{-3}$ in 1975 in New York City (Feely, Volchok and Toonkel 1976).

The record of lead concentrations in air at a semirural site in southern England during 1957–74 is illustrated in Figure 1 (Salmon *et al.* 1978). The average level was 0.13 $\mu g\ m^{-3}$. Initial higher levels of lead in air were suppressed by implementation of legislation which limited smoke emissions after 1956. However, 1960–70 was a period of doubling of consumption of gasoline in the U.K. with an increasing average content of lead in the fuel. There are regular seasonal variations in air concen-

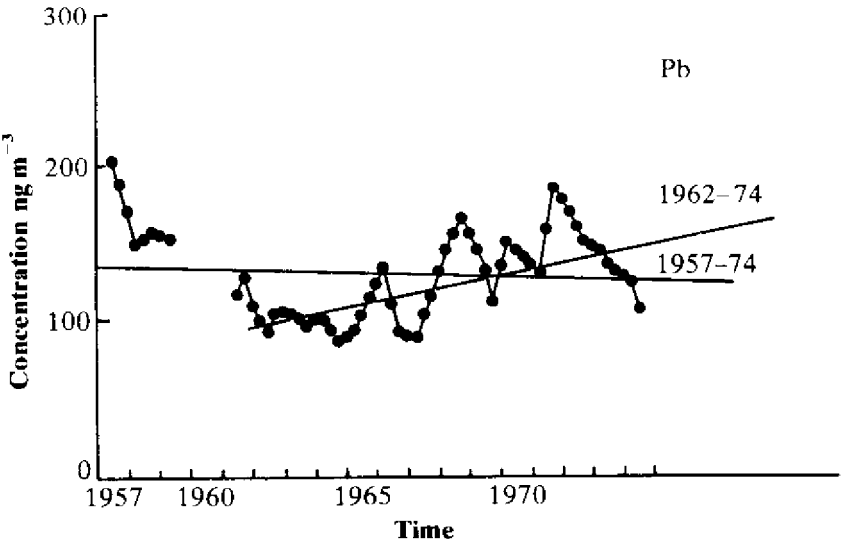


Figure 1 Lead in air at Chilton, Oxfordshire, U.K. (Salmon *et al.* 1978)

trations, with higher levels in the winter due to poorer atmospheric mixing.

Lead in air is associated with particles of submicron size. Fresh exhaust aerosols from automobiles are mostly below 0.15 μm in diameter. Away from roadways there is aggregation and attachment of the primary exhaust lead to the ambient aerosol with a broad peak in particle size between 0.3 and 1 μm (Chamberlain *et al.* 1978). Increased levels of lead in air near busy roadways are noticeable to distances of about 50 m in urban areas and 200 m in rural areas. A theoretical dependence on distance has been formulated, e.g. by Chamberlain (1974).

3.2. Soil

The levels of lead in agricultural soils are typically in the range 20 to 80 μg g^{-1}, with mean values of around 40 to 50 μg g^{-1} (Kahn 1980). The values generally reflect underlying mineralization. The fairly extensive survey results of Archer (1980) are presented in Figure 2. Agricultural

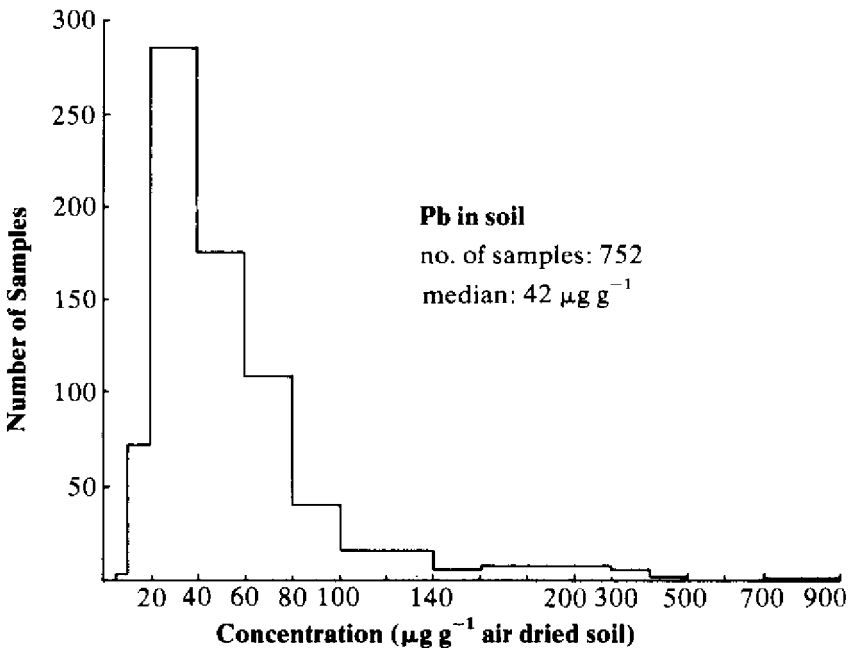


Figure 2 Lead content in agricultural soils of England and Wales (Archer 1980)

soils from 226 farms in England and Wales were analysed. Usually four samples per farm were taken to a depth of 15 cm. Generally, there had been no applications of sewage sludge or other potentially contaminating materials. The median lead concentration was 42 $\mu g\, g^{-1}$.

Input to rural soil of lead from air has been estimated to be 40 to 50 $\mu g\, m^{-2}\, d^{-1}$ (Patterson 1965, Chamberlain 1974). This deposition corresponds to levels in air of 0.15 $\mu g\, m^{-3}$ and a deposition velocity of 0.3 to 0.5 $cm\, s^{-1}$. At this rate of input, it would take 400 years to double the amount of lead in the top 10 cm of soil layer. In general, deposition occurs primarily with rainfall at rural sites (around 75 per cent of total deposition), but dry deposition is more important at urban sites (60 per cent of total deposition) (Feely *et al.* 1976).

Lead pollution of soils from mining and smelting operations is most noticeable locally, where very high levels may build up. Increased input of lead to soil will also be noted in urban areas and along roadways.

More readily soluble lead compounds, such as halides from automobile exhaust, are largely converted to less soluble compounds in soil. Binding to organic matter is important, but there is less interaction with clay minerals (Zimdahl and Hassett 1979). The immobilization of lead in soil is greatest in soils of high cation exchange capacity.

3.3 Plants

Plant uptake of lead depends upon the plant species and the soil conditions. Uptake is greater under conditions of low pH, low cation exchange capacity, low organic matter and low phosphate levels (Zimdahl and Hassett 1979); that is, in those conditions leading to low soil fixation. Some studies report only limited uptake of lead in soil by plants. Others indicate that while root absorption does occur, there is little translocation to shoots. In many cases there may be unclear distinction between the contributions of root uptake, foliar deposition from air, and direct contact or resuspension of soil particles. Tjell, Hovmand and Mosback (1979) estimated that 90 to 99 per cent of the lead in rye-grass from a typical background area in Denmark arose from deposition of atmospheric lead on the plant surface. Foliar deposition is probably less important for many food crops.

Background levels of lead in grass in Denmark are reported to be about 2 $\mu g\, g^{-1}$ on a dry weight basis (Tjell *et al.* 1979). The concentrations increase in the latter part of the growing season as the growth rate

declines, due to decreasing dilution of atmospherically supplied lead with newly grown plant material.

3.4 Diet

Lead concentrations in foods are quite variable, in general ranging from 0.01 to 2.5 mg kg^{-1}. The variability may reflect the contribution of superficial lead to the total present. Washing and peeling may remove some lead, but canning or other processing operations may add to it. In addition, analytical methods are not always substantiated, leaving accuracy in question.

Dietary intake of lead has been estimated to average 200–300 $\mu g\ d^{-1}$ (WHO 1977), with a wider range 100–500 $\mu g\ d^{-1}$ (Tsuchiya 1979) accounting for sampling and analytical differences. A Japanese study indicated little change in intake (~200 $\mu g\ d^{-1}$) between 1950 and 1970 (Tsuchiya 1979). A gradual decrease has been noted in Denmark, however, from 43 $\mu g\ d^{-1}$ in 1962 to 23 $\mu g\ d^{-1}$ in 1976, due primarily to decreasing levels in cereals (Solgaard *et al.* 1978). Lead in fertilizers is being reduced by use of pure ammonia or lead-free ammonium sulphate. In addition, more fertilizer utilization may be reducing lead uptake. The samples in the Danish study had no contact with cooking utensils and also none with chemical agents since the analytical method was optical emission spectrometry. It would be helpful in trace metal studies in general if results could always be presented with convincing evidence that sample contamination has been avoided.

3.5 Water

Lead concentration in deep ocean water is about 0.01–0.02 $\mu g\ \ell^{-1}$ and 0.3 $\mu g\ \ell^{-1}$ in surface ocean water (Chow and Patterson 1966). Dissolved lead in rivers in unpolluted areas is $<0.1\ \mu g\ \ell^{-1}$. Usually reported levels of 1–10 $\mu g\ \ell^{-1}$ are probably inaccurate due to use of insensitive analytical methods (Settle and Patterson 1980). The concentration in rainwater is 10–30 $\mu g\ \ell^{-1}$ but may be 100–500 $\mu g\ \ell^{-1}$ in areas of heavy traffic (Tsuchiya 1979).

Lead in drinking water does not generally exceed 50 $\mu g\ \ell^{-1}$. The mean intake is closer to 20 $\mu g\ d^{-1}$ (NRCC 1978). When lead pipes or tanks are used, the lead concentration may be as high as 3,000 $\mu g\ \ell^{-1}$.

3.6 Miscellaneous

Other sources of lead intake may arise from wine (130–190 $\mu g\,\ell^{-1}$), illicitly distilled whisky (10 $mg\,\ell^{-1}$), tobacco (1–5 μg intake from 20 cigarettes), improperly glazed earthenware vessels particularly for acidic contents, soil, dust and paint, facial cosmetics, coloured newsprint (WHO 1977). The significance of many of these sources, particularly regarding intakes by infants and children, cannot be so generally stated.

4 Metabolism

4.1 Inhalation

Lead-containing particles in ambient air of aerodynamic diameter 0.1 to 1.0 μm have a predicted retention in the pulmonary region of lungs of about 35 per cent (ICRP 1979). Actual measurements of retention in human volunteers range from 10 to 60 per cent (Tsuchiya 1979). Absorption to blood depends on the physicochemical form of the lead. A portion of the deposited amount is cleared by mucociliary action to the gastro-intestinal tract. Marrow, Beiter, Amato and Gibb (1980), utilizing lead aerosols of hydroxide and chloride forms in human experiments, determined that there is nearly complete absorption of the lead retained in lungs. Chamberlain *et al.* (1975) also found high absorption of retained amounts following inhalation of motor exhaust by man but determined that 50 per cent of the lead is taken up from plasma to red cells and retained in blood and the remainder is transferred to extracellular fluids and other sites in the body.

4.2 Ingestion

Absorption of lead in food ranges from 2 to 16 per cent, but may reach 30 to 50 per cent if in solution (NRCC 1978). Children may absorb 45–50 per cent of lead in food. Chamberlain *et al.* (1975) determined that following ingestion, as for inhalation, about 50 per cent of the absorbed amount reaches red blood cells.

Dietary factors can influence absorption from the gastro-intestinal tract; fasting, calcium and iron deficiencies enhance uptake. Intake and absorption may not follow a linear relationship much beyond representative levels. Hammond, O'Flaherty and Gartside (1979) noted that absorption becomes progresssively less as intake increases, perhaps due to adaptive mechanisms.

4.3 Distribution in the body

Lead is transported in blood and distributed primarily to bone. A discussion of metabolic models has been given by Marcus (1979). There is an exchangeable compartment (blood and soft tissues) and a storage compartment (bone). About 90 per cent of the total body burden is found in bone. The concentrations in tissues (men and women over 16) were reported to be 9-34 mg kg^{-1} (wet wt) in bone, 1 mg kg^{-1} in liver, 0.8 mg kg^{-1} in kidney cortex and 0.02 to 0.8 mg kg^{-1} in brain cortex (Barry 1975).

Lead in blood of individuals with only normal background exposure is 10 to 20 μg $d\ell^{-1}$* on average. There is no age difference but slightly higher levels in males (Tsuchiya 1979). There is also a clear difference in levels from urban to suburban to rural, in decreasing order. Higher values are found in populations living near highways and lead smelters.

4.4 Retention times

Retention of lead in soft tissues stabilizes in early adult life and may decrease with age in some tissues. Lead in bone continues to accumulate throughout life. The residence half-time of lead in blood is 16 to 18 days (Chamberlain *et al.* 1978). Other estimates of residence half-times are 19–21 days in blood and soft tissues and about 20 years in bone (Tsuchiya 1979).

5 Effects

Lead may cause both acute and chronic effects, mainly in the haematopoietic, nervous, gastro-intestinal and renal systems. Many steps in haeme synthesis are inhibited by lead, early signs of which, such as decreased levels of ALA-D in erythrocytes, may not of themselves be injurious to the individual. Initial indicator changes occur at levels of 10–60 μg $d\ell^{-1}$ of lead in blood. Anaemia may be apparent at 70–80 μg $d\ell^{-1}$. Neurophysiological effects of lead exposure include mental deterioration, hyperkinetic behaviour, loss of appetite, and encephalopathy, the severity of which may depend on factors such as intensity and duration of exposure and age. Chronic encephalopathy would not be expected to occur below 50–60 μg $d\ell^{-1}$ lead in blood in

* $d\ell$ = decilitre (100 $m\ell$).

children and about 80 $\mu g\ d\ell^{-1}$ in adults (Zielhuis 1975). Dose-response relationships have been formulated by Piotrowski and O'Brien (1979).

6 Literature critique

A substantial literature on lead in the environment and in man has accumulated. A detailed listing of lead data could be misleading, many of the values having uncertain validity or representativeness. The problems in contamination of environmental samples during sampling and analysis have been pointed out by Settle and Patterson (1980). Methods of sampling and analysis have been reviewed by Skogerboe, Hartley, Vogel and Koirtyohann (1979). Comprehensive reviews of lead behaviour and

Table 2 Lead in the environment—summary of representative values

Concentrations	
Atmosphere	
urban	$2\ \mu g\ m^{-3}$
rural	$0.15\ \mu g\ m^{-3}$
remote	$0.01\ \mu g\ m^{-3}$
Lithosphere	
agricultural soil	$50\ \mu g\ g^{-1}$
Hydrosphere	
freshwater	$0.1\ \mu g\ \ell^{-1}$
ocean—surface	$0.3\ \mu g\ \ell^{-1}$
ocean—deep	$0.01\ \mu g\ \ell^{-1}$
Biosphere	
land plants	$0.4\ \mu g\ g^{-1}$
Man	
bone	$9–34\ \mu g\ g^{-1}$
blood	$10–20\ \mu g\ d\ell^{-1}$
Transfer rates	
Intake	
ingestion	$80\ mg\ y^{-1}$
inhalation	
urban	$16\ mg\ y^{-1}$
rural	$1.2\ mg\ y^{-1}$
Absorption	
GI tract	10%
Lungs	
retention	35%
absorption	50%

effects include NAS (1972), EPA (1977), WHO (1977), Chamberlain *et al.* (1978), Grandjean (1978), NRCC (1978), Tsuchiya (1979), Boggess and Wixson (1979), O'Brien, Smith and Coleman (1980).

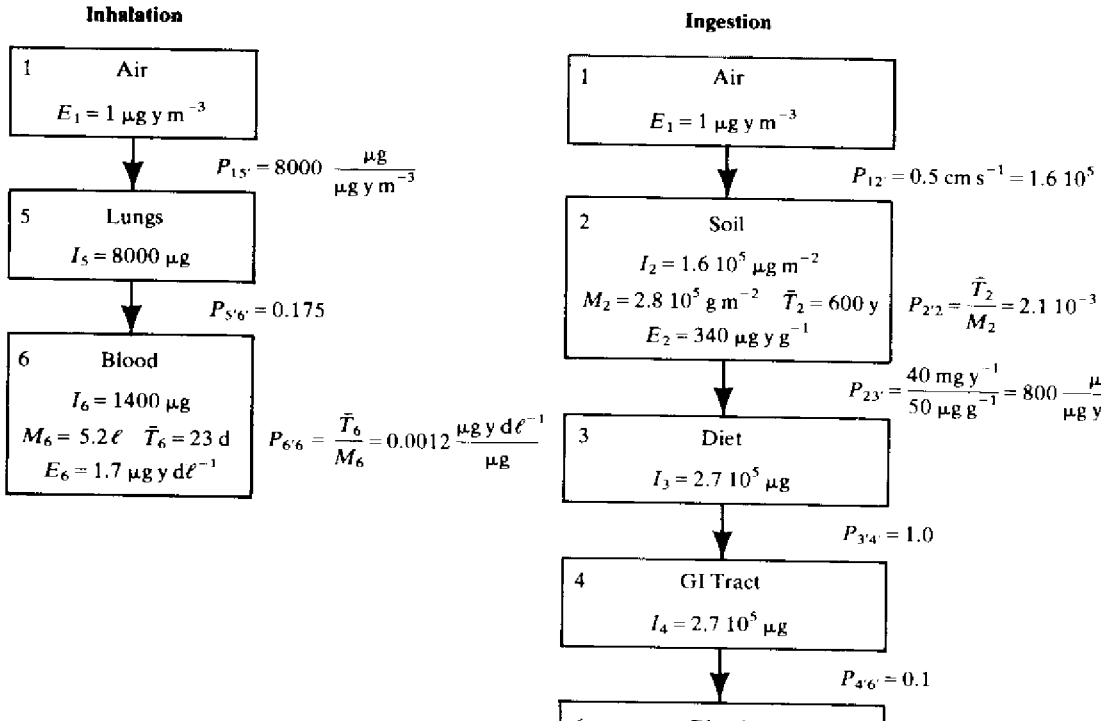
7 Pathway analysis

A summary of the transfer of lead from general atmospheric sources to man is presented in Figure 3. The notation and procedure are that of the exposure commitment method (Bennett 1981). It is convenient to begin with unit exposure commitment to air and determine the resulting exposure commitment to the receptor organ, blood. Blood is not the ultimate receptor organ in the body, but most reports of biological effects have been correlated with lead concentrations in blood. Because of the equivalence between commitment and steady-state analyses, the commitment results can be utilized to determine the contributions to the steady-state concentration of lead in blood from current background concentrations of lead in air.

The basic task in application of the exposure commitment method is the evaluation of transfer factors, P_{ij}, relating exposure or intake commitments in successive reservoirs. To complete the analysis it is necessary to assign representative values to the various parameters. Most of these have been discussed earlier and are listed in Table 2. In a few cases, more arbitrary, tentative values have been selected.

For the inhalation pathway, the main assumptions are the air breathing rate of 22 $m^3\ d^{-1}$ or 8,000 $m^3\ y^{-1}$ ($P_{15'}$), particle retention in the lung of 35 per cent and absorption to blood of 50 per cent ($P_{5'6'} = 0.35 \times 0.5 = 0.175$), residence time of lead in blood of 23 days. The residence time, $\bar{T}_6$, is derived from the half-time estimate of 16 days (Chamberlain *et al.* 1978) and the relationship $\bar{T}_6 = T_{1/2} \div \ln 2$.

The transfer factors for the ingestion pathway are obtained from estimates of the deposition velocity of 0.5 $cm\ s^{-1}$ ($P_{12'}$), mixing depth in soil of 20 cm, residence time in soil of 600 years, an association of 40 $mg\ y^{-1}$ intake rate of lead in unprocessed foods with the background level of lead in agricultural soil of 50 $\mu g\ g^{-1}$ ($P_{23'}$), and absorption to blood from the gastro-intestinal tract of 10 per cent ($P_{4'6'}$). The mixing depth in soil corresponds to the depth of the plough layer. The residence time in soil is very uncertain. A wide range of 400 to 3,000 years was suggested by Bowen (1975). A value of 300 years was used by O'Brien *et al.* (1980). The dietary intake rate of 40 $mg\ y^{-1}$ corresponds to


Inhalation
1 Air
$E_1 = 1\ \mu g\ y\ m^{-3}$
$P_{15'} = 8000\ \frac{\mu g}{\mu g\ y\ m^{-3}}$
5 Lungs
$I_5 = 8000\ \mu g$
$P_{5'6'} = 0.175$
6 Blood
$I_6 = 1400\ \mu g$
$M_6 = 5.2\ \ell \quad \bar{T}_6 = 23\ d$
$E_6 = 1.7\ \mu g\ y\ d\ell^{-1}$
$P_{6'6} = \frac{\bar{T}_6}{M_6} = 0.0012\ \frac{\mu g\ y\ d\ell^{-1}}{\mu g}$
Ingestion
1 Air
$E_1 = 1\ \mu g\ y\ m^{-3}$
$P_{12'} = 0.5\ cm\ s^{-1} = 1.6\ 10^5$
2 Soil
$I_2 = 1.6\ 10^5\ \mu g\ m^{-2}$
$M_2 = 2.8\ 10^5\ g\ m^{-2} \quad \bar{T}_2 = 600\ y$
$E_2 = 340\ \mu g\ y\ g^{-1}$
$P_{2'2} = \frac{\bar{T}_2}{M_2} = 2.1\ 10^{-3}$
$P_{23'} = \frac{40\ mg\ y^{-1}}{50\ \mu g\ g^{-1}} = 800\ \frac{\mu}{\mu g\ y}$
3 Diet
$I_3 = 2.7\ 10^5\ \mu g$
$P_{3'4'} = 1.0$
4 GI Tract
$I_4 = 2.7\ 10^5\ \mu g$
$P_{4'6'} = 0.1$

110 $\mu g\ d^{-1}$, an average value for survey results in the U.K. and U.S.A. (MAFF 1975; Bogen, Welford and Morse 1976).

The assessment shows that for unit exposure commitment to air, the ingestion pathway is more significant in transferring lead to man (33 $\mu g\ y\ d\ell^{-1}$, compared to 1.7 $\mu g\ y\ d\ell^{-1}$ via the inhalation pathway). Similar relationships are inferred for the equilibrium state: from 1 $\mu g\ m^{-3}$ of lead in air, the ingestion pathway contributes 33 $\mu g\ d\ell^{-1}$ of lead in blood and the inhalation pathway contributes 1.7 $\mu g\ d\ell^{-1}$.

The relationships for the current background levels of lead in air are given in Table 3. The background level of lead in rural air is assumed to be 0.15 $\mu g\ m^{-3}$. This is associated with 40 $mg\ y^{-1}$ of lead in diet. Other amounts of lead in diet arise from contamination in processing, drinking water, and miscellaneous sources. It has been assumed that these additional sources could contribute another 40 $mg\ y^{-1}$ to the ingestion pathway. Settle and Patterson (1980) suggested that one-half of dietary lead intake could come from use of lead-soldered cans. The background level

Table 3 Current levels of lead in the background environment and in man

Air	Diet	Blood
Ingestion pathway		
$0.15\ \frac{\mu g}{m^3}$	$\longrightarrow 40\ \frac{mg}{y}$	$\longrightarrow 5\ \frac{\mu g}{d\ell}$
Direct source to diet	$\longrightarrow 40\ \frac{mg}{y}$	$\longrightarrow 5\ \frac{\mu g}{d\ell}$
Inhalation pathway		
$2\ \frac{\mu g}{m^3}$	urban $\longrightarrow 3\ \frac{\mu g}{d\ell}$	
$0.15\ \frac{\mu g}{m^3}$	rural $\longrightarrow 0.3\ \frac{\mu g}{d\ell}$	
	Total	$10\text{–}13\ \frac{\mu g}{d\ell}$
		[Effects level $10\text{–}60\ \frac{\mu g}{d\ell}$]

of lead in urban air is assumed to be 2 $\mu g\ m^{-3}$. From these various levels, the background level of lead in blood is estimated to be 10–13 $\mu g\ d\ell^{-1}$. This agrees with general survey measurements for individuals exposed only to normal background levels.

Some effects of lead in blood, such as changes in chemical constituent levels, can be associated with relatively low concentrations of lead (10–60 $\mu g\ d\ell^{-1}$). More significant, harmful effects to the individual may occur at concentrations greater than 60 $\mu g\ d\ell^{-1}$. There is not a wide margin between background levels of lead in blood and potentially harmful levels.

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CADMIUM

1 Natural cycle

Cadmium is present in the earth's crust at an average concentration of $0.2\ \mu g\ g^{-1}$ (Taylor 1964). The natural concentration in soil normally ranges from 0.01 to $0.7\ \mu g\ g^{-1}$ (Baker, Amacher and Leach 1979). Cadmium is bound in clay and basic soil but is more mobile in sandy and acidic soils. Cadmium is found with zinc in the environment. The two metals have chemical and physical similarities.

Estimates of the global emission of cadmium to the atmosphere from natural sources are listed in Table 1. The major emissions are from volcanoes, vegetation and windblown dusts. Cadmium concentrations in the rural atmosphere range from 0.1 to 6 ng m^{-3} (CEC 1978).

Cadmium enters water from atmospheric deposition and soil erosion. Dissolved levels in freshwaters are $<1\ \mu g\ \ell^{-1}$ and average $0.15\ \mu g\ \ell^{-1}$ in sea water (Fleischer *et al.* 1974).

Plants, both terrestrial and aquatic, readily take up cadmium. Animals and man acquire cadmium primarily through ingestion. It accumulates in the human body with age. Cadmium is considered to be biologically non-essential. It is toxic at higher levels of exposure.

2 Anthropogenic sources

Cadmium is widely used in industrial processes. Typical uses are electroplating (55 per cent of use in U.S.A.), plasticizers (20 per cent), pigment production (12 per cent), nickel–cadmium batteries (5 per cent), miscellaneous uses (8 per cent) (Yost and Miles 1979). Estimates of the global emission of cadmium to the atmosphere in a recent year from anthropogenic sources are listed in Table 1. The trend in global anthropogenic emissions has been increasing, from an average release of 3.4 to 5.4 to 7.4×10^6 kg y^{-1} during 1951–60, 1961–70, and 1971–80 respectively (Nriagu 1979).

Cadmium is present in phosphate fertilizers ($5–100\ \mu g\ g^{-1}$) and in sewage sludges, which are also utilized as fertilizer (Berrow and Burridge 1979, Davis and Coker 1979). A survey of nearly 200 sludges found

Table 1 Worldwide emission of cadmium to the atmosphere[a]

Source		Emission rate (10^6 kg y^{-1})	
Natural			
Volcanoes		0.5	(0.3–8)[b]
Vegetation		0.2	(0.2–3)
Windblown dust		0.1	(0.001–0.2)
Forest fires		0.01	(0.001–0.07)
Sea spray		~0.001	
	Total	0.8	
Anthropogenic[c]			
Non-ferrous metal production		5.3	
Waste incineration		1.4	
Phosphate fertilizer manufacture		0.2	
Wood combustion		0.2	
Iron and steel production		0.07	
Coal combustion		0.06	
Industrial applications		0.05	
Oil and gasoline combustion		0.003	
Non-ferrous metal mining		0.002	
	Total	7.3	

[a] Reference (Nriagu 1979).
[b] range of literature values in brackets.
[c] emission rate during 1975.

cadmium concentrations ranging from 0 to 180 $\mu g\ g^{-1}$ (dry weight) with a median value of 23 $\mu g\ g^{-1}$ (Davis and Coker 1979). The mean concentration of cadmium in sludges receiving no industrial effluents was 7.5 $\mu g\ g^{-1}$ (Davis and Coker 1979).

On a national basis, cadmium has been estimated to reach crop land from the following sources: air deposition (41 per cent), phosphate fertilizers (54 per cent), sludge application (5 per cent) (Yost and Miles 1979). Sludge, where utilized, can contribute 90 per cent of the total cadmium input to soil; however, it is applied to less than 5 per cent of agricultural land in the U.K., for example, (Davis and Coker 1979). Typical application rates are 160 $\mu g\ m^{-2}\ y^{-1}$ from atmospheric deposition (derived from the product of 1 $ng\ m^{-3}$ in rural air and a deposition velocity of 0.5 $cm\ s^{-1}$), 250 $\mu g\ m^{-2}\ y^{-1}$ from inorganic fertilizer (50 $\mu g\ g^{-1}$ in fertilizer times an application rate of 50 $kg\ ha^{-1}\ y^{-1}$), and

3,500 $\mu g\ m^{-2}\ y^{-1}$ from sludge application (7 $\mu g\ g^{-1}$ in sludge (dry weight) times an application rate of 5,000 kg ha $^{-1}$ (dry weight)).

Cadmium is present in fuels: 0.2 to 5 $\mu g\ g^{-1}$ in coal and about 0.3 $\mu g\ g^{-1}$ in heating oil (CEC 1978). Additional sources of cadmium to the environment include dusts from rubber tyre wear, household dust from rubber-backed carpeting (WHO 1979), and slight releases from use of zinc galvanized water pipes, and pottery, ceramic and plastic containers coloured with cadmium pigments (Boudène 1979).

3 Environmental considerations

3.1 Air

The concentrations of cadmium in air depend on the distances from emitting sources. The levels range from 1 to 50 ng m^{-3}, depending on the location and degree of industrialization. Yearly averages of 6 to 21 ng m^{-3} were reported for three large Belgian cities during 1972–77 and 9–54 ng m^{-3} in another located near non-ferrous and ferrous industries (Kretzschmar, Delespaul and De Rijck 1980). A value of 3 ng m^{-3} was determined for a semi-rural site in southern England during 1957–74, although the uncertainty was fairly high (Salmon *et al.* 1978). The general population will normally inhale $<0.5\ \mu g\ d^{-1}$, equivalent to <25 ng m^{-3} in air (Piscator 1979).

3.2. Soil

Cadmium concentrations in soil are generally less than 1 $\mu g\ g^{-1}$ in non-polluted areas. Average values of 0.2 to 0.4 $\mu g\ g^{-1}$ in uncontaminated soil have been suggested with 0.9 $\mu g\ g^{-1}$ in the organic fractions (CEC 1978, Nriagu 1980). The average concentration in soil from 91 samples from farming areas in the U.S.A. was 0.57 $\mu g\ g^{-1}$ (Klein 1972). Cadmium concentrations in contaminated soil may reach 800 $\mu g\ g^{-1}$, as recorded in agricultural soil reclaimed from mine waste at Shipham in the U.K. (Marples and Thornton 1980). The residence time of cadmium in soil is uncertain but may be expected to range up to several hundred years (Nriagu 1980). Bowen (1975) has suggested a residence time of 300 years.

3.3 Plants

Cadmium is more readily taken up by plants than is lead, for example, but a straightforward correlation between plant and soil levels is not always

possible (Boudène 1979). Plant-soil concentration ratios are of the order of 1 to 40 (Bingham and Page 1975). Uptake is greater from more acidic soils. Background levels in plant leaves have been estimated to range from 0.05 to 0.2 $\mu g\ g^{-1}$ (Baker *et al.* 1979). Application of inorganic or sludge fertilizer, adding up to 10 $\mu g\ g^{-1}$ to cadmium in surface soil, increased the cadmium concentrations in grain and potatoes ten- to fifteenfold over background levels (Baker *et al.* 1979). In general, the fruit and seeds of plants contain less cadmium than the leaves (Bingham 1979).

3.4 Diet

Concentrations of cadmium are a few $\mu g\ kg^{-1}$ in basic foods (milk, potatoes, fruits, meat) but may be 50–100 $\mu g\ kg^{-1}$ in grains (wheat, rice) even in non-polluted areas (Friberg *et al.* 1974). In polluted areas, 1,000 $\mu g\ kg^{-1}$ in rice (Japan) was not uncommon. Leafy vegetables, such as spinach and lettuce, readily take up cadmium from soil (Bingham 1979). Shellfish accumulate cadmium to a relatively high degree (0.5 to 1.5 $\mu g\ g^{-1}$) (Boudène 1979), as do also the kidney and liver of mammals and fish. Normal dietary intake is considered to be in the range 15 to 70 $\mu g\ d^{-1}$ (Boudène 1979).

3.5 Water

Levels in water are generally low ($<1\ \mu g\ \ell^{-1}$). Drinking water is, thus, usually only a minor contributor to intake. Dissolved levels may reach 15 $\mu g\ \ell^{-1}$ or higher in polluted areas. Sea water contains between 0.04 and 0.3 $\mu g\ \ell^{-1}$ (Friberg *et al.* 1979).

3.6 Tobacco

The concentration of cadmium in tobacco may be relatively high (1–2 $\mu g\ g^{-1}$), and smokers can possibly absorb as much cadmium from smoke as from food. Intake is 0.1–0.2 μg per cigarette with high absorption (~50 per cent) (Piscator 1979).

4. Metabolism

Absorption of cadmium into blood ranges from 1 to 12 per cent (mean 5 per cent) from the gastro-intestinal tract and from 15 to 50 per cent (mean about 25 per cent) from the lungs. Nutritional status may affect absorp-

tion of dietary intake. Calcium and iron deficiencies may increase absorption.

Cadmium is rapidly cleared from blood and accumulates principally in kidneys and liver. Initially high concentrations may occur in liver following exposure, but in time cadmium in liver is redistributed to the kidneys. The concentration of cadmium in kidneys increases with age, reaching 10 to 50 $\mu g\, g^{-1}$ at age 50. Retention half-time in kidneys is 10 to 40 years (Friberg *et al.* 1974). The average concentration in blood is of the order of 1 $\mu g\, d\ell^{-1}$ or less (Friberg *et al.* 1974).

The body burden of cadmium in adults is normally 9.5 to 40 mg, of which 40 to 80 per cent is in kidneys and liver. It is generally assumed that one-third of the body burden is in kidneys (Friberg *et al.* 1974).

Excretion of cadmium, balancing intake, is of the order of 1–2 $\mu g\, d^{-1}$ in urine and 30–50 $\mu g\, d^{-1}$ in faeces. Absorbed cadmium is excreted mainly in urine. Cadmium in faeces can be used as an indicator of ingestion intake. Excretion of 0.5 $\mu g\, d^{-1}$ of cadmium in urine may be taken to indicate $\sim 10\ \mu g\, g^{-1}$ in renal cortex (Piscator 1979). Kidney damage is probable if excretion is around 15 $\mu g\, d^{-1}$. Blood is a less satisfactory indicator of body burden, reflecting mostly recent exposure.

The placenta accumulates cadmium and acts as a barrier in reducing transfer of cadmium from mother to foetus. Cadmium concentration in newborn blood is 30 to 50 per cent lower than in maternal blood, whereas the concentration in the placenta is tenfold higher than in maternal blood (Lauwerys 1979).

A metabolic model proposed by Kjellström and Nordberg (1978) is generally successful in describing cadmium behaviour in the body. Consideration of age-dependent renal changes improves agreement in model predictions with observations which, in particular, show declining kidney cadmium concentrations after about the age of 50 years (Travis and Haddock 1980).

5 Effects

Kidney is the critical organ for effects from cadmium. In high level inhalation exposures, the lungs may also be affected. Renal tubular dysfunction may occur when the concentration is around 200 $\mu g\, g^{-1}$ in renal cortex (Friberg *et al.* 1974, WHO 1979). Chronic high level exposures can lead to bone damage—decalcification, fractures, deformity.

It has been suggested that cadmium may be implicated in the development of hypertension, but the results of epidemiological studies are inconclusive (Lauwerys 1979). A few studies have indicated that occupational exposure to cadmium oxide may increase the risk of prostate cancer in man (IARC 1973).

6 Literature critique

The earlier literature on cadmium is full of erroneous data on the concentrations in the environment, and particularly in tissues and body fluids. Useful reviews of the cadmium literature have been prepared by Friberg *et al.* (1974, 1979), Commission of the European Communities (1978), Hammons *et al.* (1978), Piscator (1979), Lauwerys (1979), WHO (1979), Nriagu (1980), Boudène (1979), National Research Council Canada (1979) and U.K. Department of the Environment (1980). A critical review of cadmium analytical methodology is available in O'Laughlin *et al.* (1976).

7 Pathway analysis

A summary of the transfer of cadmium from general atmospheric sources to man is presented in Figure 1. The notation and procedure are that of the exposure commitment method (Bennett 1981). It is convenient to begin with unit exposure commitment to air and determine the resulting exposure commitment to the receptor organ, kidneys. Because of the equivalence between commitment and steady-state analyses, the commitment results can be utilized to determine the contributions to the steady-state concentration of cadmium in kidneys from current background concentrations of cadmium in air.

The basic task in application of the exposure commitment method is the evaluation of transfer factors, P_{ij}, relating exposure or intake commitments in successive reservoirs. To complete a tentative assessment, representative values have been assigned to the various parameters based on the foregoing discussion. A summary of some of these values is presented in Table 2.

For the inhalation pathway, the main assumptions are the air breathing rate of 22 $m^3\ d^{-1}$ or 8,000 $m^3\ y^{-1}$ ($P_{15'}$), particle retention in the lung and absorption to blood of 25 per cent of the intake amount ($P_{5'6'}$), distribution of cadmium in blood to kidneys of 30 per cent ($P_{6'7'}$), and residence time in kidneys of 30 y. The transfer factors for the ingestion

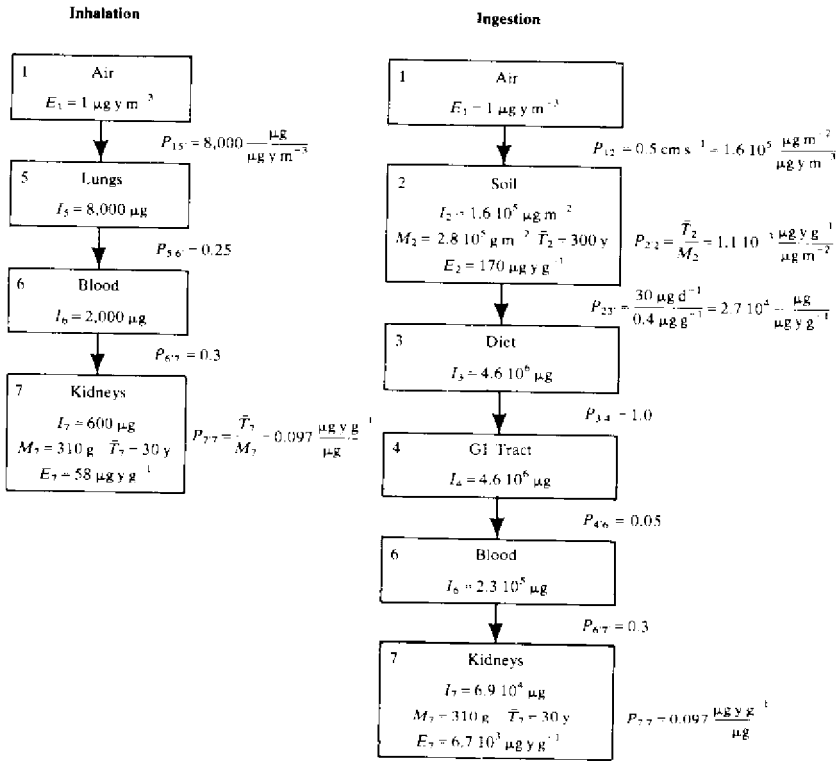

Figure 1 Pathway evaluations for cadmium

pathway are obtained from estimates of the deposition velocity of 0.5 cm s^{-1} ($P_{12'}$), mixing depth in soil of 20 cm, residence time in soil of 300 y, an association of 30 $\mu g\ d^{-1}$ intake rate of cadmium in diet with the background level of cadmium in soil of 0.4 $\mu g\ g^{-1}$ ($P_{23'}$), and absorption to blood from the GI tract of 5 per cent ($P_{4'6'}$).

The assessment shows that for unit exposure commitment to air, the ingestion pathway is more significant in transferring cadmium to man (6700 $\mu g\ y\ g^{-1}$, compared to 58 $\mu g\ y\ g^{-1}$ via the inhalation pathway). Similar relationships are inferred for the equilibrium situation: from 1 $\mu g\ m^{-3}$ of cadmium in air, the ingestion pathway contributes 6,700 $\mu g\ g^{-1}$ of cadmium in kidneys and the inhalation pathway contributes 58 $\mu g\ g^{-1}$.

Table 2 Cadmium in the environment—Summary of representative values

Concentrations	
Atmosphere	
rural	0.1–6 ng m^{-3}
urban	1–50 ng m^{-3}
Lithosphere	
agricultural soil	0.2–0.6 μg g^{-1}
Hydrosphere	
freshwater	<1 μg ℓ^{-1}
marine	0.04–0.3 μg ℓ^{-1}
Biosphere	
land plants	0.05–0.2 μg g^{-1}
Man	
body burden (age 50)	
Europe	20 μg
U.S.A.	30 μg
Japan	50 μg
body burden (newborn)	1 μg
Transfer rates	
Intake	
ingestion	15–70 μg d^{-1}
inhalation	<0.5 μg d^{-1}
Excretion	
faeces	30–50 μg d^{-1}
urine	1–2 μg d^{-1}
bile	small
Absorption factors	
Gastro-intestinal tract	5%
Lungs	25%

The relationships for the current background levels of cadmium in the environment and in man are given in Table 3. The background level of cadmium in rural air is assumed to be 0.001 μg m^{-3}, which from use of the transfer factors is associated with cadmium levels in soil of 0.2 μg g^{-1} and intake in diet of 15 μg d^{-1}. Since the representative intake rate of cadmium in diet is 30 μg d^{-1}, an additional source is suggested. This is also known to exist, namely, the addition to soil of cadmium present in phosphate fertilizers. Additions of fertilizers to agricultural land, increasing the cadmium concentration in soil by 0.2 μg g^{-1}, will contribute an additional 15 μg d^{-1} to cadmium in diet.

Table 3 Current levels of cadmium in the background environment and in man

Air	Soil	Diet	Kidneys
	Ingestion pathway		
$0.001\frac{\mu g}{m^3}$	$\longrightarrow 0.2\frac{\mu g}{g}$	$\longrightarrow 15\frac{\mu g}{d}$	$\longrightarrow 7\frac{\mu g}{g}$
Direct source to soil	$\longrightarrow 0.2\frac{\mu g}{g}$	$\longrightarrow 15\frac{\mu g}{d}$	$\longrightarrow 7\frac{\mu g}{g}$
	Inhalation pathway		
$0.03\frac{\mu g}{m^3}$	urban		$\longrightarrow 1.7\frac{\mu g}{g}$
$0.001\frac{\mu g}{m^3}$	rural		$\longrightarrow 0.06\frac{\mu g}{g}$
	Total		$14\text{–}16\frac{\mu g}{g}$
			$\left[\text{Renal damage level} \sim 200\frac{\mu g}{g}\right]$

The background level of cadmium in urban air is assumed to be 0.03 $\mu g\, m^{-3}$. The inhalation intake contributes less than 15 per cent of the background concentration of cadmium in kidneys. The total background level of cadmium in kidneys is estimated to be 14 $\mu g\, g^{-1}$ for rural residents and 16 $\mu g\, g^{-1}$ for urban residents. The concentrations would be about 50 per cent higher if the cadmium is considered to be localized in the kidney cortex. These values are reasonably confirmed by background measurements.

It is generally assumed that renal damage becomes manifest at cadmium concentrations in kidney cortex of 200 $\mu g\, g^{-1}$ or higher. This is about a factor of 10 higher than the current levels in background exposed individuals. The margin is less for individuals exposed to additional sources, such as from cigarette smoking or from occupational exposure.

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ARSENIC

1 Natural cycle

Arsenic is a semi-metallic element of atomic number 33. It occurs in the earth's crust at average concentration of 1.8 $\mu g\, g^{-1}$ (Taylor 1964). Arsenic is mostly associated with sulphur in nature and forms a number of sulphide minerals. Sedimentary rocks generally contain more arsenic than do igneous rocks (NRCC 1978). Weathering of rocks releases soluble arsenates which migrate under acidic conditions but are immobilized via co-precipitation with hydrous iron oxide and other compounds in neutral or alkaline conditions (NRCC 1978).

Volcanic activity is the principal natural source of arsenic in the atmosphere (Walsh 1979). The estimated emission rates from this and other sources are listed in Table 1. Natural levels of arsenic in air range from about 0.2 to 10 $ng\, m^{-3}$ (NRCC 1978).

Arsenic resembles phosphorus chemically, has −3, +3 and +5 valance states, binds with non-metals and metals, and also forms organic compounds. The toxicity of arsenic compounds is, in decreasing order: arsines, arsenites (inorganic, +3), arsenoxides (organic, +3), arsenates (inorganic, +5), pentavalent organic compounds, arsonium compounds, metallic arsenic.

2 Anthropogenic sources

Arsenic has long been known as a poison and a therapeutic agent. An inorganic arsenic compound, known as Fowler's solution, was widely used for treatment of various ailments before the introduction of antibiotics. Some uses of arsenicals for human treatment remain, mostly as antiparasitic drugs (Fowler, Ishinishi, Tsuchiya and Vahter 1979).

The major current uses of arsenic are in pesticides, herbicides, cotton desiccants, and wood preservatives; in the fabrication of glass and dye-stuffs; and as an additive in alloys to increase hardness and heat resistance.

Recent world production of arsenic has been fairly steady at around 50,000 tonnes per year (NAS 1977). The main producing countries are Sweden, Namibia, France, and Mexico (Pershagen 1979).

The major sources of arsenic emissions to air arise from the smelting of metals, combustion of fuels, and pesticide use (Table 1). Estimated emission factors are 960, 590, and 360 tonnes per 10^6 tonnes of copper, zinc, and lead produced, respectively, and 1.4 tonnes per 10^6 tonnes of coal burned (NAS 1977). The content of arsenic in coal can reach up to 1,500 $\mu g\, g^{-1}$, although average levels are much less, of the order of 5 $\mu g\, g^{-1}$ (Pershagen 1979). The concentration of arsenic in oil shale is 25–75 $\mu g\, g^{-1}$, resulting in high arsenic levels in the oil and solid waste (Chappell 1979).

Arsenic is present in phosphate rock used to manufacture fertilizers and detergents. These uses may contribute to increased arsenic

Table 1 Worldwide emission of arsenic to the atmosphere[a]

Source		Emission rate (10^6 kg y^{-1})
Natural		
Volcanoes		7
Biological activity		0.26
Weathering		0.24
Forest fires		0.16
Sea spray		0.14
	Total	7.8
Anthropogenic		
Copper production		13
Iron and steel production		4.2
Lead and zinc production		2.2
Agricultural chemicals		2.1
Wood fuel		0.60
Agricultural burning		0.56
Coal combustion		0.55
Waste incineration		0.43
Cotton ginning		0.023
Mineral ore mining		0.013
Residual fuel		0.004
	Total	23.7

[a] Reference Walsh *et al.* (1979).

concentrations in river water (IARC 1973). The discharge of water from geothermal power plants has resulted in elevated levels of arsenic in adjacent reservoirs (Pershagen 1979).

Arsanilic acid is sometimes added to swine and poultry feed as a growth-promoting agent, and residual amounts may reach man in diet.

3 Environmental considerations

3.1 Air

Arsenic is present in air mainly in particulate form as arsenic trioxide. Methyl arsines are present immediately above arsenic-treated soils although these compounds are generally rapidly oxidized in the presence of sunlight (NRCC 1978).

An air sampling network in urban areas of the U.S.A. reported levels of <10 to 750 ng m^{-3} for 133 stations during 1964, with an average of 20 ng m^{-3} (Sullivan 1969). Lower values are reported for rural areas of the U.S.A., England and Canada (0.3 to 6 ng m^{-3}) (NAS 1977, NRCC 1978). Values of 1.4 to 1.6 $\mu g\ m^{-3}$ are encountered in the vicinity of copper and other smelters (Fowler, B. A., Ishinishi, N., Tsuchiya, K. and Vahter, M. 1979).

An analysis of the longer-term trend of arsenic particulate concentrations in air has been performed for a semi-rural site in England (Salmon *et al.* 1978). The results are shown in Figure 1. The mean concentration was 5.4 ng m^{-3} with a declining trend over the period 1957–1974. The most significant decline occurred in 1957–61 as a result of legislation restricting industrial smoke emissions. The continuing decline of arsenic in air since 1962 was shown to be correlated with the winter declines in atmospheric black smoke, indicating fuel combustion as a major source of atmospheric arsenic at this location (Salmon *et al.* 1978).

3.2 Soil

Uncontaminated soil generally contains less than 40 $\mu g\ g^{-1}$ of arsenic (Fowler *et al.* 1979). An average background level is about 7 $\mu g\ g^{-1}$ in normal soil (NRCC 1978). However, levels of 100 to 2,500 $\mu g\ g^{-1}$ have been found in the vicinity of copper smelters (Pershagen 1979). Pesticides, herbicides and defoliants may cause arsenic levels to reach 700 $\mu g\ g^{-1}$ in agricultural soils (Bishop and Chisholm 1962).

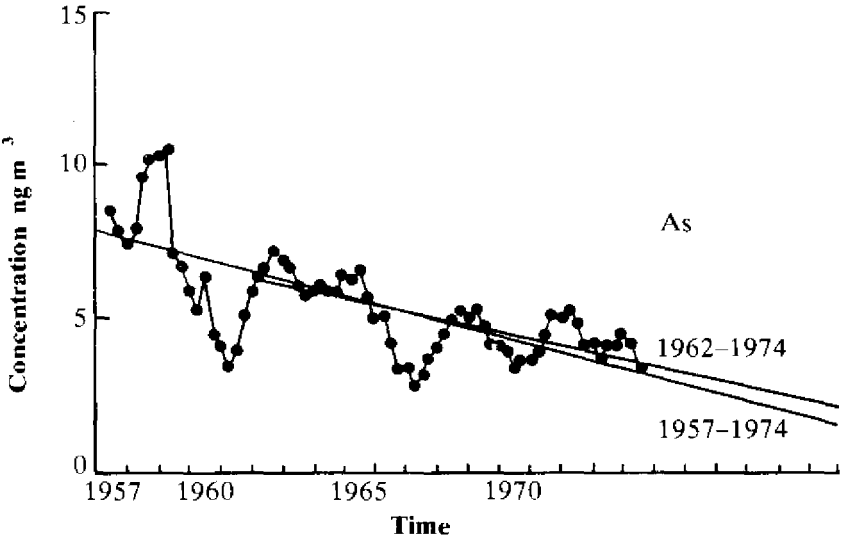

Figure 1 Arsenic in air at Chilton, Oxfordshire, U.K. (Salmon *et al.* 1978)

Arsenic binding capacity of soils varies with the levels of iron and aluminium oxides, clay, organic matter, pH and moisture content (NRCC 1978). Arsenic is most mobile in acidic, sandy loams (soils with low ion exchange capacity). Arsenic in soil due to arsenical applications is persistent with half-times of 6.5 to 16 years (NRCC 1978).

Arsenic can be transformed by soil micro-organisms to volatile organic arsines. Estimates of volatilization loss from soil range from 20 to 60 per cent of applied amounts (NRCC 1978). Microbial activity in soil is greatest when the organic matter content is high.

3.3 Plants

Uptake by plants is related to the concentration of soluble arsenic in the soil, the type of soil and the plant species. Surface contamination resulting from insecticide applications may contribute to the concentrations. Natural arsenic levels in plants seldom exceed 1 to 2 $\mu g\ g^{-1}$ (dry weight) and average about 0.5 $\mu g\ g^{-1}$ (NRCC 1978).

An indication of concentration ratios (plant/soil) is available from an experiment using soya bean plants grown for 60 days in a silt-loam soil containing 4.6 $\mu g\ g^{-1}$ total endogenous arsenic. The concentration in the

plant was 0.22 $\mu g\ g^{-1}$ (dry weight), giving a concentration ratio of 0.05 (Cataldo and Wildung 1978). The value is about a factor of five less for the concentration in the plant expressed on a fresh weight basis. The concentration ratio was 0.15 (dry plant tissue and dry soil) when the soil was treated with soluble arsenic compounds, raising the soil concentration by 2.5 $\mu g\ g^{-1}$ (Cataldo and Wildung 1978).

The highest levels of arsenic in plants occur in the roots, followed by the tops, seeds and fruit (NRCC 1978). High levels in edible parts are generally prevented by prior occurrence of reduced plant growth at higher concentrations of arsenic in soil.

Arsenic accumulates somewhat more in aquatic plants than in land plants. Natural levels may be up to 30 $\mu g\ g^{-1}$ with average concentrations around 3 $\mu g\ g^{-1}$ (dry weight) (NRCC 1978).

3.4 Diet

Most foods contain low levels of arsenic, normally below 0.25 $mg\ kg^{-1}$ (Fowler *et al.* 1979). Soya bean seeds and certain vegetables containing mustard oil, such as cabbage and cress, concentrate arsenic to a greater extent than do other species (Boudène 1979). The chemical form of arsenic in food has not been established.

The arsenic content in fish, particularly marine fish, may range from 1 to 10 $mg\ kg^{-1}$ with values over 100 $mg\ kg^{-1}$ in certain bottom-feeding fish, crustaceans, and seaweed (Fowler *et al.* 1979). Different organic forms are present. Concentration ratios (tissue/water) have been estimated to range from 3 to 30 for freshwater fish and 40 to 3,000 for marine fish (Woolson 1975).

Ingestion of arsenic in diet depends on the seafood content. Estimates range from 30 $\mu g\ d^{-1}$ in Canada, 90 $\mu g\ d^{-1}$ in England, 40 $\mu g\ d^{-1}$ (excluding seafood) to 190 $\mu g\ d^{-1}$ for typical diet in the U.S.A., and 70 to 370 $\mu g\ d^{-1}$ in Japan (NRCC 1978, Fowler *et al.* 1979). A survey of dietary intake in Canada, France, U.K. and U.S.A. indicated a range of 7 to 60 $\mu g\ d^{-1}$, with an average of about 30 $\mu g\ d^{-1}$ (WHO 1973).

3.5 Water

The concentration of arsenic in rivers and lakes varies greatly—generally $<10\ \mu g\ \ell^{-1}$ but sometimes as high as 1 $mg\ \ell^{-1}$ (Fowler *et al.* 1979). The concentrations in groundwater depend on the arsenic content of the bed-rock; examples are $>0.05\ mg\ \ell^{-1}$ in some samples from Nova Scotia,

Canada, where the concentration in bed-rock is high, 0.4 to 1.3 mg ℓ^{-1} in carbonate spring waters in the U.S.A., Romania, U.S.S.R. and New Zealand, up to 1.8 mg ℓ^{-1} in artesian wells in Taiwan, and up to 3.4 mg ℓ^{-1} in Cordoba (Argentina) groundwaters (Fowler *et al.* 1979).

Sea water contains 1 to 5 μg ℓ^{-1} (Fowler *et al.* 1979). Several different forms of arsenic may be present in natural waters, but with highest concentrations of inorganic forms.

Arsenic may persist in natural waters for long periods of time—tens of years in lakes and thousands of years in the ocean (NRCC 1978). Persistence depends on the flow rate of the water. Some lakes treated with aquatic herbicides may return to background levels within one month to one year (NRCC 1978). Arsenic may accumulate in bottom sediments.

3.6 Miscellaneous

Wine made from grapes sprayed with arsenic insecticides or fungicides may contain levels of arsenic up to 0.5 mg ℓ^{-1} in the trivalent inorganic form (Crecelius 1977). Tobacco may contain arsenic if arsenic insecticides have been used.

4 Metabolism

4.1 Absorption

Absorption of arsenic following inhalation is uncertain. Retention of ambient aerosols may be considered to be of the order of 35 per cent. A large fraction is transferred from lungs to blood (50–80 per cent) (NRCC 1978).

Over 80 per cent of the ingested amount of dissolved inorganic trivalent arsenic is absorbed from the gastro-intestinal tract (Fowler *et al.* 1979). Similar absorption is expected for the inorganic pentavalent form. Organic compounds present in seafood are also readily absorbed.

Skin is also a possible route of absorption, as indicated by some occupational accidents (Fowler *et al.* 1979).

4.2 Distribution in the body

Arsenic is cleared from blood plasma at a very high rate and is distributed widely in the body. Highest concentrations initially are in liver and kidney, but the main storage appears to be in bone, muscle, and skin

(Fowler *et al.* 1979). From the data of Liebscher and Smith (1968), who analysed various tissues of normal individuals, the total body content of arsenic is estimated to be of the order of 1 mg (see Table 2).

Biotransformation of arsenic occurs in the body—from trivalent to pentavalent form and from inorganic to methylated forms—which may serve as a detoxifying mechanism (Fowler *et al.* 1979).

Table 2 Concentration of arsenic in tissues and total body content

Tissue	Concentration ($\mu g\ g^{-1}$) Dry*	Wet§	Mass (g)	Body content (μg)
Muscle	0.063	0.013	28,000	370
Bone	0.057	0.047	5,000	240
Skin	0.090	0.034	2,600	89
Blood	0.038	0.007	5,500	40
Liver	0.028	0.008	1,800	14
Lungs	0.082	0.021	570	12
Hair	0.46	0.41	20	8
Nails	0.30	0.28	3	1
Other tissue	0.03	0.008	26,500	210
			Total	984 μg

* Reference (Leibscher and Smith 1968).

§ Tissue water content from (ICRP 1975).

4.3 Retention times

Arsenic is eliminated from the body at a rapid rate. Retention half-times following a single intravenous injection of inorganic arsenic were 2 hours, 8 hours and 8 days (Mealey, Brownall and Sweet 1959). A longer retention half-time in the body has also been suggested: 280 days (Buhler 1973). Small amounts of arsenic are stored several months in ectodermal tissues (hair, nails) (NRCC 1978).

A recent, detailed study of human retention of arsenic has been reported by Pomroy, Charbonneau, McCallough and Tam (1980). Oral doses of inorganic ^{74}As were given to six individuals who were then monitored by whole body counting for up to 103 days. From the combined results three retention components were determined: 65.9 per cent of the intake with a half-time of 2.1 days, 30.4 per cent with 9.5 days and 3.7 per cent with 38.4 days. This gives a weighted half-time of 5.7 days and a mean retention time of 8.2 days.

Organic forms of arsenic have shorter retention times, e.g. a half-time of 2.6 days, corresponding to a mean retention time of 3.7 days, for orally administered arsanilic acid (Calesnick, Wase and Overby 1966).

Excretion of arsenic is mainly via urine—70 per cent in chronic exposures—the remainder in faeces (Fowler *et al.* 1979). Less important routes are via skin, hair, nails and sweat.

5 Effects

At high concentrations arsenic is, of course, toxic. Long-term moderate exposure to arsenic in air, diet or drinking water can result in lesions of the skin and mucous membranes and nervous and respiratory system damage. Disorders of the circulatory system and liver may also occur (Fowler *et al.* 1979).

Epidemiological studies have given associations between exposure to arsenic and skin and lung cancers; however, reliable and consistent induction of cancer in animals via arsenic exposure has not been achieved (Fowler *et al.* 1979, IARC 1973).

6 Literature critique

More and more data on arsenic in the environment are becoming available, with attention focused currently on evaluation of chemical forms. Reviews of arsenic occurrence in the environment and toxicity include: Fowler (1977), Fowler *et al.* (1979), NAS (1977), NRCC (1978), IARC (1973), Pershagen (1979), Lafontaine (1979). A summary of representative data is presented in Table 3.

7 Pathway analysis

The transfer of arsenic to man from general environmental sources occurs by the pathways of inhalation, and ingestion of terrestrial and aquatic foods. The basic compartmental arrangements for the transfer analysis and a summary of the transfer relationships are given in Figure 2. The notation and procedure are that of the exposure commitment method (Bennett 1981). It is convenient to begin with unit exposure commitment to air and determine the resulting exposure commitment to the receptor, considered in this case to be the whole body of man. Because of the equivalence between commitment and steady-state analyses, the commitment results can be utilized to determine the contributions from current background concentrations of arsenic in air, soil, water, and diet to the steady-state concentration of arsenic in the body.

Table 3 Arsenic in the environment
Summary of representative values

Concentrations		
Atmosphere		
rural	0.2–10 ng m^{-3}	
urban	10–750 ng m^{-3}	(20)*
Lithosphere		
agricultural soil	0.1–40 μg g^{-1}	(7)
Hydrosphere		
freshwater	<10 μg ℓ$^{-1}$	
marine	1–5 μg ℓ$^{-1}$	
Biosphere		
land plants	<0.4 μg g^{-1}	(0.1)
aquatic plants	<6 μg g^{-1}	
fish	1–10 μg g^{-1}	
food	<0.25 μg g^{-1}	
Man (body content)	1 mg	
Transfer rates		
Intake		
ingestion	30–370 μg d^{-1}	(40)
inhalation	0.1–4 μg d^{-1}	(0.4)
Absorption		
GI tract	0.8–1.0	
lungs		
retention	0.35	
absorption	0.5–0.8	

* representative mean values in parentheses

The basic task in application of the exposure commitment method is the evaluation of the transfer factors (P_{ij}) relating exposure or intake commitments in successive reservoirs. For the inhalation pathway, the main assumptions are the air breathing rate of 22 m^3 d^{-1} or 8,000 m^3 y^{-1} (P_{15}), particle retention in the lung of 35 per cent and absorption to blood of 80 per cent ($P_{5,6} = 0.35 \times 0.8 = 0.28$), general distribution of arsenic throughout the body and an effective mean residence time of eight days.

For the terrestrial ingestion pathway, the assumptions to obtain relationships between the concentrations in air, soil, and diet are very tentative. The value of the total deposition velocity for arsenic attached to ambient aerosol particles is assumed to be 0.5 cm s^{-1}. The mixing depth in soil is 20 cm and the soil density is 1.4 g cm^{-3}. These are standard assumptions.

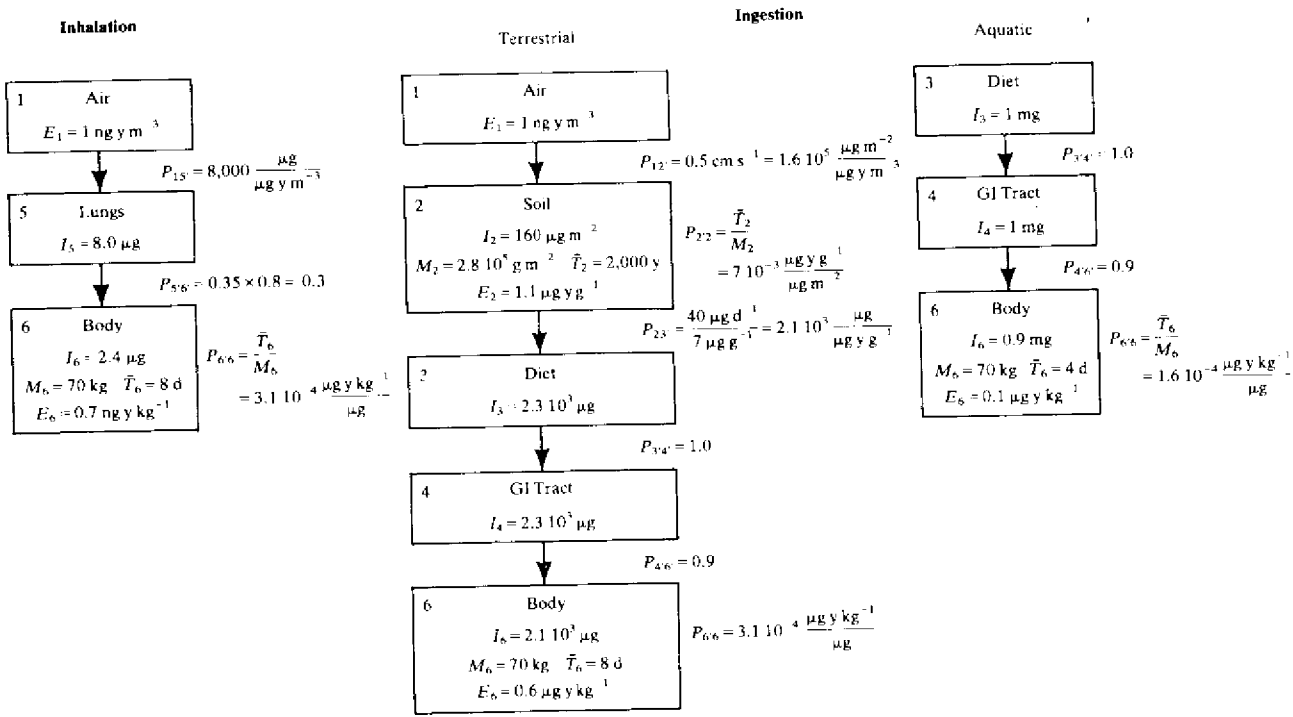

Figure 2 Pathway evaluations for arsenic

Measurements of arsenic deposition at background sites in the U.K. during 1972–75 ranged from 0.8 to 5.5 mg m^{-2} y^{-1} (Cawse 1980). Taking 1 mg m^{-2} y^{-1} as representative of the natural component of deposition (F_2^*), the concentration of arsenic in air (C_1^*) at rural background locations can be inferred.

$$F_2^* = P_{12'} C_1^*$$

$$P_{12'} = 0.5 \text{ cm s}^{-1} = 1.6\ 10^5 \text{ m y}^{-1}$$

$$= 1.6\ 10^5 \frac{\mu\text{g m}^{-2}}{\mu\text{g y m}^{-3}}$$

$$F_2^* = 10^3\ \mu\text{g m}^{-2}\text{ y}^{-1}$$

Therefore $C_1^* = 6$ ng m^{-3}

Further, assuming 7 μg g^{-1} to be the natural concentration of arsenic in soil (C_2^*), the residence time of arsenic in soil ($\bar{T}_2$) can be inferred.

$$C_2^* = \frac{\bar{T}_2}{M_2} F_2^*$$

$$C_2^* = 7\ \mu\text{g g}^{-1}$$

$$F_2^* = 10^3\ \mu\text{g m}^{-2}\text{ y}^{-1}$$

$$M_2 = 1.4 \text{ g cm}^{-3}\ 20 \text{ cm}\ 10^4 \text{ cm}^2 \text{ m}^{-2}$$

$$= 2.8\ 10^5 \text{ g m}^{-2}$$

Therefore, $\bar{T}_2 = 2{,}000$ years.

This value of the residence time of natural arsenic in soil is very tentative. Other natural input pathways to soil, specifically the weathering of rock, which is of the order of $7.8 \text{ g m}^{-2} \text{ y}^{-1} \times 10\ \mu\text{g g}^{-1} = 0.08 \text{ mg m}^{-2} \text{ y}^{-1}$, are minor and have been neglected. The inferred residence time is much longer than the observed residence times of applied arsenicals utilized in agriculture, although it agrees with a preliminary estimate of Bowen (1975). More information is needed to judge the correctness of individual parameters in the above associations. The estimated values of transfer factors are summarized in Figure 2.

To complete the analysis of the terrestrial ingestion pathway, the remaining assumptions are 40 μg d^{-1} dietary intake of arsenic, 90 per cent absorption of arsenic from the gastro-intestinal tract, and a mean residence time in the body of eight days. The transfer factors are

evaluated in Figure 2. The transfer linking dietary intake to the exposure commitment in the body is evaluated as follows:

$$P_{3'6} = P_{3'4'}P_{4'6'}P_{6'6}$$
$$= 1.0 \times 0.9 \times 3.1\ 10^{-4} \frac{\mu g\ y\ kg^{-1}}{\mu g}$$
$$= 2.8\ 10^{-4} \frac{\mu g\ y\ kg^{-1}}{\mu g}$$

The equivalent expression for equilibrium is $P_{3'6} = 0.28\ \mu g\ kg^{-1}$ of arsenic in the body per mg y^{-1} of arsenic intake. This factor would apply to intake of arsenic in terrestrial foods and also drinking water.

For the aquatic ingestion pathway, it is assumed that intake is of organic forms of arsenic. Absorption from the gastro-intestinal tract is assumed to be 90 per cent, but retention in the body is with a four-day mean residence time. The inputs to water from air and from direct releases are too uncertain to estimate transfer factors. The analysis of the aquatic ingestion pathway begins with unit dietary intake of 1 mg y^{-1}. The transfer factors are given in Figure 2. The overall transfer factor from dietary intake to the exposure commitment in the body is:

$$P_{3'6} = P_{3'4'}P_{4'6'}P_{6'6}$$
$$= 1.0 \times 0.9 \times 1.6\ 10^{-4} \frac{\mu g\ y\ kg^{-1}}{\mu g}$$
$$= 1.4\ 10^{-4} \frac{\mu g\ y\ kg^{-1}}{\mu g}$$

and for the equilibrium case is 0.14 $\mu g\ kg^{-1}$ in the body per mg y^{-1} intake. This factor applies to intake of arsenic in organic form, such as in aquatic foods.

The relationships for the current background levels of arsenic in air, soil, diet, and man are given in Table 4. The background level in rural air of 6 ng m^{-3} is associated with the level of 7 $\mu g\ g^{-1}$ in soil and the dietary intake rate of arsenic in terrestrial foods of 15 mg y^{-1}.

The intake rate of arsenic in aquatic foods is determined by the products of representative arsenic concentrations and consumption amounts, being 3 $\mu g\ g^{-1} \times 6{,}000\ g\ y^{-1}$ for marine fish, 15 $\mu g\ g^{-1} \times$

Table 4 Current levels of arsenic in the background environment and in man

	Air	Soil	Diet	Body
	Ingestion pathway			
Terrestrial foods	$6\frac{ng}{m^3}$	$\longrightarrow 7\frac{\mu g}{g}$	$\longrightarrow 15\frac{mg}{y}$	$\longrightarrow 4\frac{\mu g}{kg}$
Aquatic foods*			$33\frac{mg}{y}$	$\longrightarrow 5\frac{\mu g}{kg}$
Drinking water			$0.5\frac{mg}{y}$	$\longrightarrow 0.1\frac{\mu g}{kg}$
	Inhalation pathway			
	$20\frac{ng}{m^3}$	urban		$\longrightarrow 0.01\frac{\mu g}{kg}$
	$6\frac{ng}{m^3}$	rural		$\longrightarrow 0.004\frac{\mu g}{kg}$
			Subtotal§	$4.1\frac{\mu g}{kg}$
			Total	$9\frac{\mu g}{kg}$
				[Effects level 15–340 $\mu g\,kg^{-1}$][2]

* Intake of organic forms of arsenic.
§ Associated with intake of inorganic arsenic.

1,000 g y^{-1} for shellfish, with negligible contribution from ingestion of freshwater fish. The concentrations are approximate means reported by Fowler (1977). The consumption amounts are the U.S.A., European average intakes of fish of 8 kg y^{-1} (ICRP 1975), assumed consisting of 6 kg y^{-1} marine fish, 1 kg y^{-1} freshwater fish, and 1 kg y^{-1} shellfish. The intake rate of arsenic in aquatic foods, 33 mg y^{-1}, is over twice as great as in terrestrial foods. However, with half the retention time, the contribution to the arsenic concentration in the body is about the same as that from the terrestrial ingestion pathway.

The contributions to current body levels of arsenic from drinking water intake and inhalation are also included in Table 4. The representative concentration of arsenic in drinking water is taken to be 1 $\mu g\,\ell^{-1}$ and consumption is assumed to average 1.4 $\ell\,d^{-1}$ (ICRP 1975). The intake rate is, thus, 0.5 mg y^{-1} from drinking water, contributing 0.1 $\mu g\,kg^{-1}$ to the concentration of arsenic in the body. Inhalation of arsenic makes a negligible contribution to the body content. The total estimated concentration of arsenic in the body corresponds to a body burden of about 0.6 mg (9 $\mu g\,kg^{-1} \times 70$ kg).

Close relationships have not been established between arsenic concentrations in tissues and the occurrence of harmful effects. A summary of studies indicates that intakes of arsenic in drinking water of 55 to 1,200 mg y^{-1} have been associated with chronic poisoning (NRCC 1978). Applying the intake to body transfer factor derived above ($P_{3'6}$) gives a range of 15 to 340 $\mu g\,kg^{-1}$ of arsenic in the body from this range of intake rates of arsenic in inorganic form. This indicates that there is not a wide margin separating current levels from harmful effects. There is less concern regarding intake of arsenic in organic form, for which apparently much higher concentrations can be tolerated in the body.

It is recognized that there are considerable uncertainties in the estimates of arsenic intake rates, the absorption, distribution and retention in the body and in the more critical tissues, and the levels at which harmful effects may occur. The above discussion has been presented more to provide a framework of analysis. As more data are acquired, the transfer relationships can be determined and applied with more confidence.

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