Monitoring and Assessment Research Centre
GEMS: Global Environmental Monitoring System

MARC REPORT NUMBER 25

EXPOSURE COMMITMENT ASSESSMENTS OF ENVIRONMENTAL POLLUTANTS

Volume 1, Number 2

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) is an independent international institute undertaking research on major environmental pollution problems. It is located in Chelsea College in the University of London and has been in operation since July 1975.

The objective of the MARC core research programme is to develop and apply techniques for the assessment of pollution problems of global, regional or local significance. The programme is mainly carried out by means of reviews which synthesize existing relevant knowledge from a wide range of disciplines.

MARC currently receives financial support from the United Nations Environment Programme (UNEP), the World Health Organization (WHO) and The Rockefeller Foundation.

The results of the Centre's work are published mainly in the MARC report series as Technical Reports, General Reports and Research Memoranda, or in professional journals.

MARC PUBLICATIONS

MARC TECHNICAL REPORTS

MARC Technical Reports are detailed accounts of the Centre's research programme. Their main purpose is to increase knowledge and understanding of the environment and provide useful data and methods of approach for those who are involved in monitoring and assessment of environmental pollution.

MARC GENERAL REPORTS

MARC General Reports are synoptic reviews of environmental topics relevant to monitoring and assessment. Each is written by a specialist in the field, keeping a sense of perspective across the whole breadth of the subject. Their main purpose is to be usable by those environmental scientists and managers who are not expert in the topic being covered but who need to obtain a broader, multidisciplinary understanding of monitoring and assessment problems.

MARC RESEARCH MEMORANDA

MARC Research Memoranda are short informal reports related to MARC's ongoing work programme. They may include ideas, data and bibliographical material useful to monitoring and assessment. Their main purpose is to act as a forum for wider discussion.

ISBN 0 905918 22 3

Exposure commitment assessments of environmental pollutants

Volume 1, Number 2

Summary exposure assessments for mercury, nickel, tin

by B. G. Bennett

A Technical Report (1981)

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

With the support of:
United Nations Environment Programme and
The Rockefeller Foundation

Abstract

This is the second in a series of reports presenting summary assessments of exposure of man to environmental pollutants. In this report, mercury, nickel and tin are considered. Representative background levels of these pollutants in air, soil, water and diet due to natural and man-made sources are indicated. The intake rates by man are estimated and distribution and retention in the body are discussed. For each pollutant, a pathway analysis is conducted in which quantitative transfer relationships are developed. The contributions of inhalation and ingestion pathways to the current concentrations of the pollutants in man are evaluated.

Previously published in this series:
Volume 1, Number 1 (1981) Exposure commitment concepts and application; summary exposure assessments for lead, cadmium and arsenic.

CONTENTS

MERCURY

1 Natural cycle

Mercury is a metal of atomic number 80. It occurs in nature in several chemical forms, as elemental mercury vapour, mercurous (+1) and mercuric (+2) inorganic compounds and in organic forms. The divalent inorganic form predominates in soil. Soil concentrations range over two orders of magnitude, being highest in mineralized areas. The average abundance of mercury in the earth's crust is 0.08 $\mu g\ g^{-1}$ (Taylor 1964).

Mercury compounds in soil undergo reduction to elemental mercury by bacterial action. This process and the high volatility of mercury leads to considerable degassing from soil. Volcanic activity also introduces mercury into the atmosphere. Estimates of emissions of mercury to the atmosphere are presented in Table 1. It is uncertain whether vaporization from the ocean surface is an important contribution to mercury in the atmosphere. Mercury concentrations in air are much lower over the ocean. Mercury in air is returned to the land and ocean by deposition processes.

Leaching and erosion of mercury in soil contribute to mercury in water and sediments of both the freshwater and marine environments.

Table 1 Worldwide emissions of mercury to the atmosphere*

Source		Emission rate (10^6 kg y^{-1})
Natural		
Soil degassing		17.8
Oceanic emission		7.6
Coastal emission		1.4
Release from biota		0.04
Volcanoes		0.02
	Total	26.8
Anthropogenic		
	Total	10.0

* Reference National Academy of Sciences (1978).

This flux involves inorganic mercury, but much is associated with dissolved and particulate organic matter. Mercury in sediments is in part transformed to alkylated forms, particularly methylmercury which is important with regard to uptake by biota. In terms of quantity, the alkylated forms do not contribute appreciably to the global mercury cycle.

In aquatic systems, fish and shellfish accumulate high levels of methylmercury. Top predatory fish, such as bass and pike in freshwater and swordfish and tuna in marine waters, have high natural levels of mercury. In marine fish the presence of selenium in levels equal to or exceeding the mercury content of the fish may reduce the toxic effects. Selenium appears to immobilize the methylmercury, though the protection mechanism is not clear. For freshwater fish, the selenium levels may be too low to achieve this effect.

2 Anthropogenic sources

Mercury has a wide range of industrial uses. The chloralkali, electrical equipment and paint industries are large consumers. Losses occur from direct uses of mercury and also from energy production, mining and related activities, particularly copper smelting and fossil fuel burning.

Anthropogenic sources are raising the levels of mercury in air, soil, freshwater lakes and streams and ocean estuaries, if not the ocean itself. An estimated 25–30 per cent of the total atmospheric mercury is anthropogenic (NAS 1978). Increases in the levels in lakes, rivers and sediments have been by factors of about two to five. The increases in soil and the ocean are less perceptible. Whether mercury levels in biological organisms have been increased has not been reliably determined. There are large variations and a lack of historical data from a wide range of specimens.

Under favourable environmental conditions, biological and chemical methylation of mercury will occur and even extremely low levels can rapidly bioaccumulate. Methylation rates are a function of the mercury burden, bacterial population, nutrient loading, pH, sedimentation rates and other physiochemical conditions. The local conditions near industrial releases are obviously of concern, but increased mercury levels have also been noted in fish in remote lakes which are poorly buffered and sensitive to acid rain. The changed conditions cause an increased rate of methylation, resulting in greater mercury availability to biota.

Birds that prey on contaminated aquatic organisms, particularly fish, may acquire high levels of organic mercury. Those that live longer have more time to concentrate mercury. At high levels the reproductive rate is affected.

Alkylmercury fungicides used as seed dressings were at one time important sources of mercury in terrestrial food chains. Mercury was transferred first to seed-eating rodents and birds and subsequently to carnivorous birds. Since 1970, several countries have prohibited the use of mercury compounds in seed treatments.

3 Environmental considerations

3.1 Air

Concentrations of mercury in air are of the order of a few ng m^{-3} in remote areas and up to 50 ng m^{-3} in urbanized areas (Berlin 1979). The range of levels in rural areas has been given as 3–9 ng m^{-3} over non-mineralized areas and 7–53 ng m^{-3} over mineralized areas (ICRP 1975).

At a semi-rural site in England, the mercury concentration in air averaged 1.4 ng m^{-3} over the years 1957–74 (Figure 1) (Salmon *et al.* 1978). The initial declining trend in this period is due to clean air

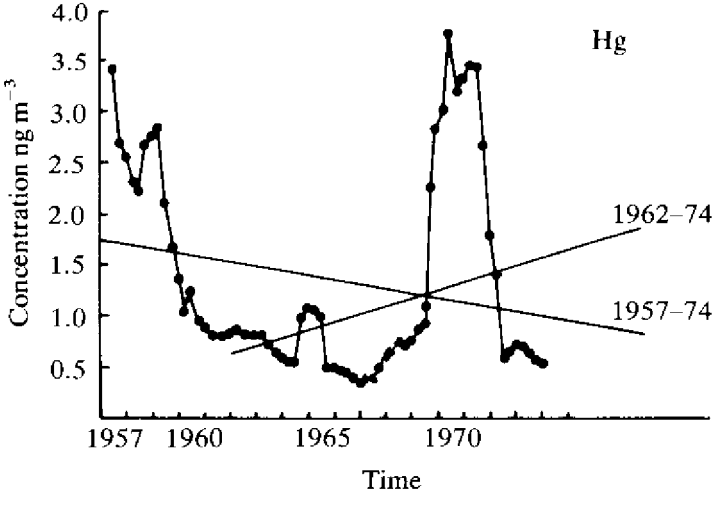


Figure 1 Mercury in air at Chilton, Oxfordshire, U.K. (Salmon *et al.* 1978)

legislation. The large excursion in 1970 has been attributed to possible local use of an agricultural fungicide (Salmon *et al.* 1978).

Other estimates of mercury concentrations in air include: 7 ng m^{-3} in urban areas (range 0.5–50), 4 ng m^{-3} in rural remote continental-areas (range 1–10), 1.5 ng m^{-3} over continental shelf areas, and 0.7 ng m^{-3} over oceanic and polar regions (NAS 1978).

3.2 Soil

The concentrations of mercury in soil are variable but usually rather low. The range is about 10 to 300 ng g^{-1}; however, the levels can exceed 500 μg g^{-1} in mineralized areas (NAS 1978). A representative continental mean concentration is 70 ng g^{-1} (UKDOE 1976, NAS 1978). The residence time of mercury in soil is estimated to be 1,000 years (NAS 1978); however, such an estimate must include considerable recycling. When account is taken of the volatilization of mercury from soil to air, the mean residence time of mercury in soil is reduced to less than one year (Miller and Buchanan 1979).

The concentration of mercury in marine sediments is of the order of 0.05 to 1.2 μg g^{-1} in the open ocean and < 1 μg g^{-1} in coastal regions (NAS 1978). A representative level in sediments of both freshwater and marine areas is 330 ng g^{-1} (NAS 1978). In polluted areas (near chlor-alkali plants) the levels may reach 800 to 1,000 μg g^{-1}.

3.3 Plants

Uptake and circulation in plants of inorganic mercury compounds and organically complexed mercurials adsorbed on clay is limited; however, uptake through the roots of gaseous mercury, such as arises from decaying sulphides, is more efficient (NAS 1978). Plants may acquire mercury more readily from the air than from soil. Typical mercury concentrations in plants grown in agricultural soils are 10–40 ng g^{-1} (wet wt) (UKDOE 1976). Levels in aquatic plants are 30–80 ng g^{-1} in unpolluted waters and up to 40 μg g^{-1} in polluted waters.

3.4 Diet

The results of several surveys of mercury in foods have been summarized (NAS 1978): dairy products, 2–20 ng g^{-1}; meat, fish, poultry, 10–20 ng g^{-1}; grain and cereals, 20–50 ng g^{-1}; potatoes, 6–20 ng g^{-1};

vegetables, 2–10 ng g^{-1}; beverages 2–6 ng g^{-1}; fish, 10–300 ng g^{-1}; large marine fish 200–1,500 ng g^{-1}. Almost all of the mercury in fish is in the methylmercury form. The organic form has not been detected in foods of vegetable origin.

Dietary intake of mercury is dependent on the amounts of fish consumed. Fish consumption may range from 0 to 500 g d^{-1}. The estimated average national intake of total mercury in diet is 5–10 μg d^{-1} in Europe, U.S.S.R. and Canada, 20 μg d^{-1} in the U.S.A., and 40–80 μg d^{-1} in Japan (Boudène 1979).

3.5 Water

Mercury is normally strongly bound to particulate matter in freshwaters and probably mostly in dissolved form in the ocean (NAS 1978). Typical mercury concentrations are 20–60 ng ℓ^{-1} in freshwater and 10–30 ng ℓ^{-1} in the ocean (NAS 1978). Uptake by biota of methylmercury in water is quite efficient, so that even low concentrations in water can lead to higher concentrations in fish.

4 Metabolism

4.1 Absorption

Approximately 80 per cent of inhaled mercury vapour is retained (WHO 1976). Pulmonary retention for other forms is uncertain. Gastro-intestinal absorption of inorganic mercury compounds in food is about 7 per cent but nearly 100 per cent for methylmercury.

Absorbed mercury vapour is rapidly oxidized to the mercuric form. Methylmercury is transformed to some extent to inorganic forms in most organs, but little transformation occurs in the brain.

4.2 Distribution in the body

Mercury accumulates particularly in the renal cortex of the kidneys although it is distributed to most other organs and tissues. Mercury has a special affinity for ectodermal and epithelial cells and glands and is thus preferentially found in the lining of the intestinal tract, skin, hair, salivary and sweat glands, thyroid, liver, kidneys, pancreas, testicles, and prostate. Methylmercury is easily transferred across the placenta; the concentration in foetal blood is equal to that in maternal blood (Koos and Longo 1976).

Tissue concentrations from analysis of large numbers of samples from normal individuals are presented in Table 2. These data are for total mercury; methylmercury comprises 10–15 per cent of the total body content (Kitamura, Sumino, Hayakawa and Shibata 1976). The differences in results reported in the various studies may reflect exposure differences but are more probably analytical variations. The results from

Table 2 Concentration of mercury in human tissues

Tissue	Concentration* ($\mu g\ g^{-1}$)			
	U.K.	U.S.A.	Japan	Sweden
Muscle	0.061 (15)	0.13 (69)	0.057 (27)	
Bone	0.25 (16)			0.043 (12)
Skin	0.76 (18)	0.19 (60)	0.048 (27)	
Liver	0.39 (22)	0.25 (95)	0.42 (30)	0.036 (21)
Blood	0.017 (3)		0.058 (19)	
Kidney	0.35 (20)	0.76 (95)	0.98 (30)	0.088 (21)
Brain	0.22 (21)	0.11 (121)	0.095 (41)	
Lung	0.36 (26)	0.25 (17)	0.070 (28)	0.008 (8)
Teeth	1.94 (59)			
Hair	3.8 (70)		3.4 (53)	
Heart	0.25 (22)	0.10 (57)	0.069 (29)	
Other tissue	0.25 (166)	0.10 (132)	0.07 (164)	

* Number of samples in parentheses.

References: U.K.: Liebscher and Smith (1968); concentrations converted from dry to wet weight with tissue water content from ICRP (1975).

U.S.A.: Mottet and Body (1974).

Japan: Kitamura *et al.* (1976).

Sweden: Brune *et al.* (1980); Lindh *et al.* (1980).

the most recent studies in Sweden (Brune, Nordberg and Wester 1980; Lindh, Brune, Nordberg and Wester 1980) indicate lower concentrations by a factor of 10 compared with earlier studies. More data will be required to establish more accurately the representative levels of mercury in tissues.

4.3 Retention times

Excretion of mercury in urine and faeces is variable, depending on the form of mercury. Methylmercury is excreted mainly via faeces. Additional routes of excretion are by volatilization from the lungs, by sweat and in hair.

Retention times vary in the different organs–from a few days to months. Long retention and thus higher accumulations occur in the brain and kidneys (Berlin 1979). Two phases of clearance of methylmercury from blood have been identified with average half-times of 7.6 hours and 52 days (Kershaw, Dhahir and Clarkson 1980). More rapid clearance of inorganic mercury from blood is observed, with a half-time of the order of 24 to 28 days (Rahola, Hattula, Korolainen and Miettinen 1973). The effective clearance half-time from the body as a whole is about 70 days for methylmercury and 40 days for inorganic mercury (Aberg *et al.* 1969, Miettinen 1973). The mean residence times in the body are thus of the order of 100 d for merthylmercury and 60 d for inorganic compounds ($\bar{T} = T_{\frac{1}{2}} \div \ln 2$).

5 Effects

Mercury has no known metabolic function. Even low concentrations may be considered potentially harmful. Toxic effects of methylmercury in humans are dominated by neurological disturbances. It is not yet clear to what extent chronic low level exposure may lead to behavioural or intellectual impairment. Clinically observable effects in human adults occur at blood levels of 0.2–0.5 $\mu g\, m\ell^{-1}$ and body concentrations of about 0.5–0.8 $\mu g\, g^{-1}$ (WHO 1976). The foetus and infant may be somewhat more sensitive than the adult to methylmercury exposure.

6 Literature critique

A large number of reviews of mercury environmental behaviour and toxicity have been published, particularly since the occurrence of the

poisoning incidents at Minamata Bay in Japan (1953–60) and in Iraq (1972), and the detection of methylmercury in remote lakes of Scandinavia and Canada. General reviews include Friberg and Vostal (1972), World Health Organization (1976), National Academy of Sciences (1978) and Nriagu (1979). Other general reviews with large collections of summarized data were published by the U.K. Department of the Environment (1976), and the National Research Council Canada (1979). Reviews which emphasize the toxicology of mercury include: Koos and Longo (1976), Berlin (1979), Gatti, Macri and Silano (1979), and Piotrowski and Inskip (1981). Reviews of analytical methods are included in WHO (1976), NAS (1978) and NRCC (1979).

7 Pathway analysis

The transfer of mercury to man from general environmental sources occurs by the pathways of inhalation and ingestion of terrestrial and aquatic foods. The basic compartmental arrangement for the transfer analysis is shown in Figure 2. A summary of the transfer relationships for each pathway is given in Figure 3. The notation and procedure are that of the exposure commitment method (Bennett 1981).

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 environmental compartments. The exposure commitment is the time integral of the concentration in a compartment, and the intake commitment is the time integral of the flux of contaminant into the compartment. Primed subscripts in the transfer factor refer to relationships between intake commitments and unprimed subscripts between exposure commitments. Mixed relationships between intake and exposure commitments are also included in the pathway analyses (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 to current levels of mercury in the body from the background concentrations of mercury in air, soil, water, and diet.

A summary of representative levels of mercury in the environment and of intake rates and absorption fractions by man is given in

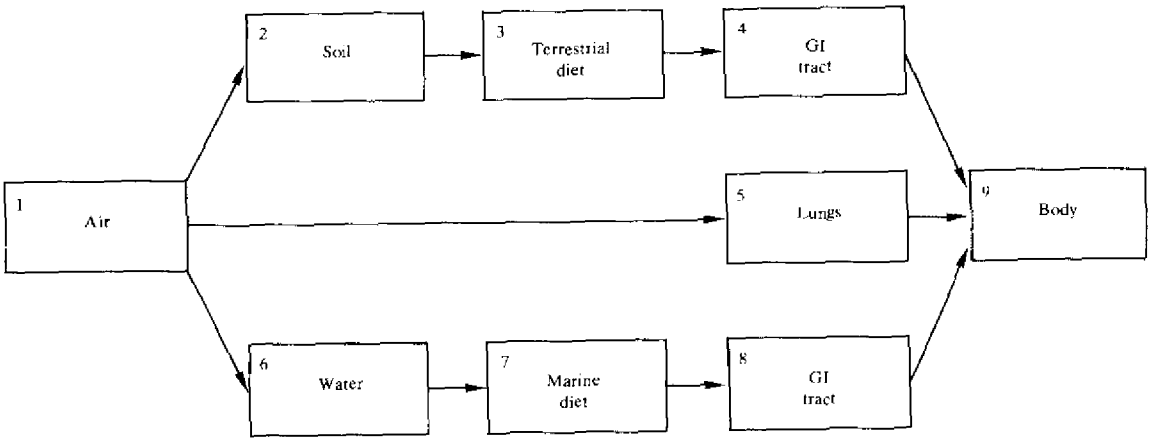


Figure 2 Compartment model for the transfer of mercury from atmospheric sources to man

Table 3. Some additional assumptions are required to complete the pathway analysis. Some of these are very tentatively assigned for the moment. In the analysis it is assumed that inorganic mercury is appropriately considered for the inhalation and terrestrial ingestion pathways and methylmercury for the marine ingestion pathway.

Table 3 Mercury in the environment: summary of representative values

Concentrations		
Atmosphere		
urban	7 ng m^{-3}	(0.5–50)*
rural	4 ng m^{-3}	(1–10)
continental shelf area	1.5 ng m^{-3}	
oceanic and polar regions	0.7 ng m^{-3}	
Lithosphere		
agricultural soil	70 ng g^{-1}	(10–300)
Hydrosphere		
freshwater	40 ng ℓ^{-1}	(20–60)
ocean	30 ng ℓ^{-1}	(10–30)
sediments	330 ng g^{-1}	
Biosphere		
land plants	20 ng g^{-1}	(10–40)
aquatic plants	40 ng g^{-1}	(30–80)
terrestrial animals and birds		
(non-fish-eating)	20 ng g^{-1}	
freshwater fish		
—non-predatory	< 100 ng g^{-1}	(20–200)
—predatory	< 500 ng g^{-1}	(400–1,000)
marine fish		
—non-predatory	< 100 ng g^{-1}	(40–200)
—predatory	300 ng g^{-1}	(200–1,500)
Transfer rates		
Intake		
terrestrial diet	5 μg d^{-1}	
marine diet	10 μg d^{-1}	
inhalation	0.09 μg d^{-1}	
Absorption		
GI tract		
—inorganic mercury	0.05	
—methylmercury	1.0	
lungs	0.7	

* Range of values in parentheses.

Transfer factors for the inhalation pathway are determined from the air intake rate of 22 $m^3 d^{-1}$ (8,000 $m^3 y^{-1}$), retention in the lungs and absorption to blood of 70 per cent of the intake amount, and distribution throughout the body with an effective mean residence time of 60 days. The transfer factors are summarized or evaluated in Figure 3. The lungs are a partitioning compartment and the transfer factor from lungs to the body expresses a ratio of intake commitments ($P_{5'9'}$). Evaluation of the exposure commitment to lungs is not required, although this could have been determined with estimates of the mean residence time of mercury in lungs and the lung mass. The other transfer factors correspond to ratios of intake to exposure commitments ($P_{15'}$) or vice versa ($P_{9'9}$).

The exposure commitment to the body from 1 $\mu g\ y\ m^{-3}$ of mercury in air via the inhalation pathway is thus:

$$E_9 = P_{15'} P_{5'9'} P_{9'9} E_1$$

$$= 8{,}000 \frac{\mu g}{\mu g\ y\ m^{-3}} 0.7 \frac{\mu g}{\mu g} 0.0023 \frac{\mu g\ y\ kg^{-1}}{\mu g} 1\ \mu g\ y\ m^{-3}$$

$$= 13\ \mu g\ y\ kg^{-1}$$

Transfer factors for the terrestrial ingestion pathway are determined from the background mercury levels in air (4 $ng\ m^{-3}$) and soil (70 $ng\ g^{-1}$) and the contribution to the dietary intake of mercury excluding fish (5 $\mu g\ d^{-1}$). The results are shown in Figure 3. The ratio of equilibrium concentrations in soil and air (P_{12}) is equivalent to the ratio of exposure commitments in the two compartments. The units are expressed differently, but they are equivalent (Bennett 1981).

The association between mercury concentrations in soil and diet is not firm, but it completes the chain analysis. Alternatively, one could evaluate the air-diet transfer factor ($P_{13'}$) from the ratio of dietary intake and the background concentration in air:

$$P_{13'} = \frac{5\ \mu g\ d^{-1}}{4\ ng\ m^{-3}} = 4.6\ 10^5 \frac{\mu g}{\mu g\ y\ m^{-3}}$$

This accounts for direct transfers of mercury to diet from air and indirect transfers through soil. It is not necessary to evaluate the separate pathways. The environment has, in effect, combined the separate contributions and it is only necessary to accept the final result.

Transfer from dietary intake to the gastro-intestinal tract is a direct transfer ($P_{3'4'} = 1.0$). Absorption to blood and thus to the body is assumed

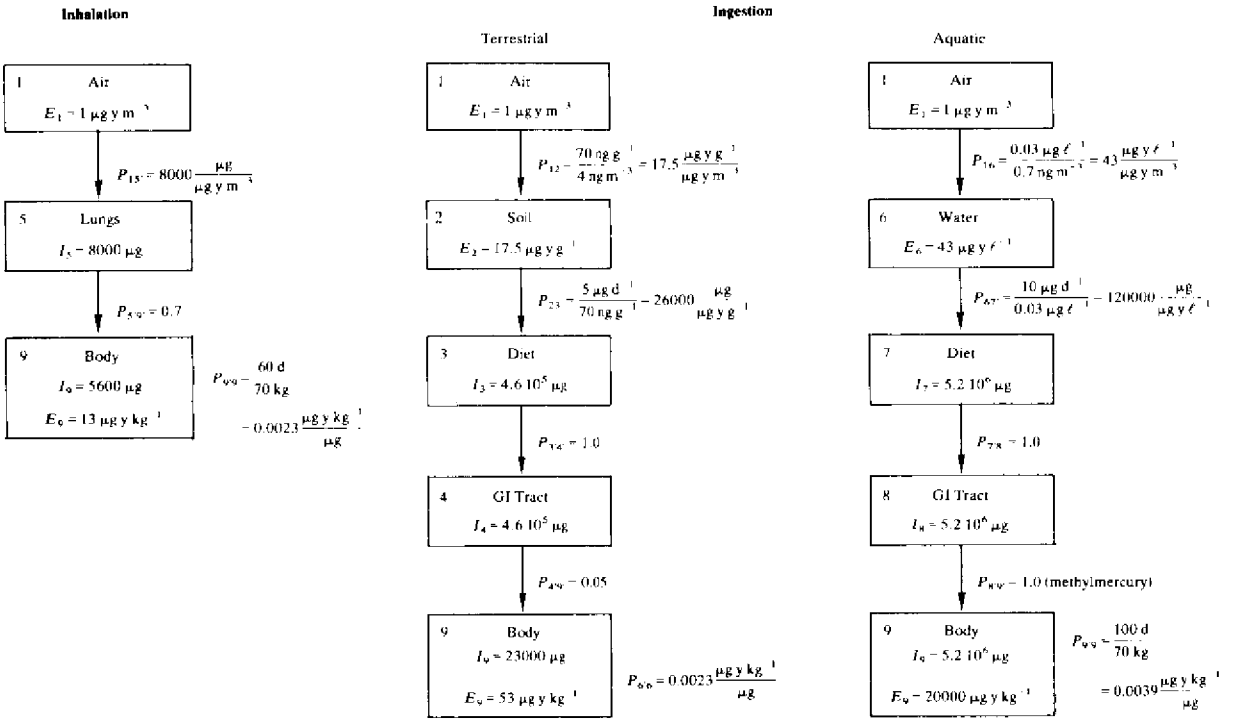


Figure 3 Pathway evaluations for mercury

to be 5 per cent ($P_{4'9'} = 0.05$). It is anticipated that mercury in food is somewhat less available than the directly administered inorganic mercury compounds used in experimental studies.

The exposure commitment to the body from 1 $\mu g\ y\ m^{-3}$ of mercury in air via the terrestrial ingestion pathway is thus:

$$E_9 = P_{13'} P_{3'4'} P_{4'9'} P_{9'9} E_1$$

$$= 4.6 \times 10^5 \frac{\mu g}{\mu g\ y\ m^{-3}} 1.0 \frac{\mu g}{\mu g} 0.05 \frac{\mu g}{\mu g}$$

$$\times 0.0023 \frac{\mu g\ y\ kg^{-1}}{\mu g} 1\ \mu g\ y\ m^{-3}$$

$$= 53\ \mu g\ y\ kg^{-1}$$

The marine ingestion pathway transfer factors are also estimated from representative background levels (0.7 $ng\ m^{-3}$ in oceanic air, 0.03 $\mu g\ \ell^{-1}$ in sea water, 10 $\mu g\ d^{-1}$ intake in diet). Absorption of methylmercury from the gastro-intestinal tract to blood and the body is assumed to be 100 per cent. The mean residence time of methylmercury in the body is 100 d. The exposure commitment to the body from this pathway is:

$$E_9 = P_{16} P_{67'} P_{7'8'} P_{8'9'} P_{9'9} E_1$$

$$= 43 \frac{\mu g\ y\ \ell^{-1}}{\mu g\ y\ m^{-3}} 1.2 \times 10^5 \frac{\mu g}{\mu g\ y\ \ell^{-1}} 1.0 \frac{\mu g}{\mu g} 1.0 \frac{\mu g}{\mu g}$$

$$\times 0.0039 \frac{\mu g\ y\ kg^{-1}}{\mu g} 1\ \mu g\ y\ m^{-3}$$

$$= 20{,}000\ \mu g\ y\ kg^{-1}$$

The current levels of mercury in the environment and the estimated contributions to the concentration in blood are given in Table 4. The inhalation and terrestrial ingestion pathways make relatively small contributions to the concentrations in the body. The marine ingestion pathway determines almost entirely the level of mercury in the body. The average concentration of mercury in the body of 14 $\mu g\ kg^{-1}$ gives an estimated body burden of 980 μg of mercury. The estimated concentration is much less than the results of tissue analyses (Table 2).

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

	Air	Diet	Body
Inhalation pathway			
	$0.004 \frac{\mu g}{m^3}$	⟶	$0.05 \frac{\mu g}{kg}$
Ingestion pathway			
terrestrial	$0.004 \frac{\mu g}{m^3}$	⟶ $5 \frac{\mu g}{d}$ ⟶	$0.2 \frac{\mu g}{kg}$
marine	$0.0007 \frac{\mu g}{m^3}$	⟶ $10 \frac{\mu g}{d}$ ⟶	$14 \frac{\mu g}{kg}$
		Total	$14 \frac{\mu g}{kg}$
		[Effects level 500–800 $\frac{\mu g}{kg}$]	

Closest agreement is with the more recent tissue concentration measurements from Sweden (also given in Table 2). The calculated body burden is quite dependent on the assumed mean effective retention time of methylmercury in the body. Additional measurements of the mercury content in tissues and of the mean residence times would be useful.

At steady state, the analysis (Table 4) indicates that the relationship between methylmercury intake in fish and average body concentration is 1.4 $\mu g\, kg^{-1}$ per $\mu g\, d^{-1}$ intake, whereas for total dietary intake of mercury the relationship is approximately 1 $\mu g\, kg^{-1}$ per $\mu g\, d^{-1}$ intake. Observed relationships between dietary intake and blood levels are about 0.7 to 1 $\mu g\, \ell^{-1}$ per $\mu g\, d^{-1}$ intake rate (WHO 1976).

The pathway analysis of environmental transfer of mercury to man has been intended to provide a framework to link the various environmental compartments and to serve as a basis for further evaluations. Tentative parameter values have been assigned to complete the analysis. It will now be necessary to extend the analysis to consider the potential range of values and uncertainties of the various transfer factors. Special cases of exposure can also be considered as data become available.

References

Aberg, B., Ekman, L., Falk, R., Greitz, U., Persson, G. and Snihs, J. O. 1969 Metabolism of methylmercury (^{203}Hg) compounds in man; excretion and distribution. *Arch. Environ. Health* **19**, 478–484.

Bennett, B. G. 1981 Exposure commitment concepts and application. In: *Exposure Commitment Assessments of Environmental Pollutants*, Volume 1, No. 1, MARC Report No. 23, Monitoring and Assessment Research Centre, Chelsea College, London.

Berlin, M. 1979 Mercury. In: *Handbook on the Toxicology of Metals*. L. Friberg, F. Nordberg and V. Vouk (Eds), Elsevier North-Holland Biomedical Press, Amsterdam, 503–530.

Boudène, C. 1979 Food contamination by metals. In: *Trace Metals: Exposure and Health Effects*, E. Di Ferrante (Ed.), Commission of the European Communities, Pergamon Press, Oxford, 163–183.

Brune, D., Nordberg, G. and Wester, P. O. 1980 Distribution of 23 elements in the kidney, liver and lungs of workers from a smeltery and refinery in north Sweden exposed to a number of elements and of a control group. *Sci. Total Environ.* **16**, 13–35.

Friberg, L. and Vostal, J. (Eds) 1972 *Mercury in the Environment*, CRC Press, Cleveland.

Gatti, G. L., Macri, A. and Silano, V. 1979 Biological and Health effects of mercury. In: *Trace Metals: Exposure and Health Effects*, E. Di Ferrante (Ed.), Commission of the European Communities, Pergamon Press, Oxford, 73–98.

International Commission on Radiological Protection 1975 *Report of the Task Group on Reference Man*, ICRP Report No. 23, Pergamon Press, Oxford.

Kershaw, T. G., Dhahir, P. H. and Clarkson, T. W. 1980 The relationship between blood levels and dose of methylmercury in man. *Arch. Environ. Health* **35**, 28–36.

Kitamura, S., Sumino, K., Hayakawa, K. and Shibata, T. 1976 Mercury content in human tissues from Japan. In: *Effects and Dose Response Relationships of Toxic Metals*, G. Nordberg (Ed.), Elsevier Scientific Publishing Co., Amsterdam, 290–298.

Koos, B. J. and Longo, L. D. 1976 Mercury toxicity in the pregnant woman, fetus and newborn infant: A review. *Amer. J. Obstet. Gynec.* **126**, 390.

Liebscher, K. and Smith, H. 1968 Essential and nonessential trace elements. *Arch. Environ. Health* **17**, 881–890.

Lindh, U., Brune, D., Nordberg, G. and Wester, P. O. 1980 Levels of antimony, arsenic, cadmium, copper, lead, mercury, selenium, silver, tin, and zinc in bone tissue of industrially exposed workers. *Sci. Total Environ.* **16**, 109–116.

Miettinen, J. K. 1973 Absorption and elimination of dietary mercury (Hg^{2+}) and methylmercury in man. In: *Mercury, Mercurials, and Mercaptans*, M. W. Miller and T. W. Clarkson (Eds), Charles C. Thomas, Springfield, Illinois, 233–243.

Miller, D. R. and Buchanan, J. M. 1979 *Atmospheric transport of mercury: exposure commitment and uncertainty calculations*, MARC Report No. 14, Monitoring and Assessment Research Centre, Chelsea College, University of London.

Mottet, N. K. and Body, R. L. 1974 Mercury burden of human autopsy organs and tissues. *Arch. Environ. Health* **29**, 18–24.

National Academy of Sciences 1978 An Assessment of Mercury in the Environment. NRC/NAS, Washington D.C.

National Research Council Canada 1979 Effects of mercury in the Canadian Environment. NRCC Report 16739, Environmental Secretariat, Ottawa.

Nriagu, J. O. (Ed.) 1979 *The Biogeochemistry of Mercury in the Environment.* Elsevier/North-Holland Biomedical Press, Amsterdam.

Piotrowski, J. K. and Inskip, M. J. 1981 *Health effects of methylmercury*: MARC Report No. 24, Monitoring and Assessment Research Centre, Chelsea College, University of London.

Rahola, T., Hattula, T., Korolainen, A. and Miettinen, J. K. 1973 Elimination of free and protein-bound ionic mercury ($^{203}Hg^{++}$) in man. *Ann. Clin. Res.* **5**, 214–219.

Salmon, L., Atkins, D. H. F., Fisher, E. M. R., Healy, C. and Law, D. V. 1978 Retrospective trend analysis of the content of U.K. air particulate material 1957–1974. *Sci. Total Environ.* **9**, 161–200.

Taylor, S. R. 1964 Abundance of chemical elements in the continental crust: a new table. *Geochim. Cosmochim. Acta* **28**, 1273–1285.

U.K. Department of the Environment 1976 *Environmental Mercury and Man*, Pollution Paper No. 10, HMSO, London.

World Health Organization 1976 *Environmental Health Criteria* 1, *Mercury*, World Health Organization, Geneva.

NICKEL

1 Natural cycle

Nickel is a metal of atomic number 28. Its average abundance in the earth's crust is 75 $\mu g\, g^{-1}$ (Taylor 1964). Nickel is particularly concentrated in basic rocks (basalt) ($\sim 150\ \mu g\, g^{-1}$) with much lower concentrations in granite ($\sim 0.5\ \mu g\, g^{-1}$) (Taylor 1964).

Some nickel compounds include nickel oxide (NiO), nickel hydroxide ($Ni(OH)_2$, $Ni(OH)_3$), nickel sulphide (Ni_3S_2), nickel sulphate ($NiSO_4$), nickel chloride ($NiCl_2$), and nickel carbonyl ($Ni(CO)_4$). Natural occurrence of nickel is mainly in sulphide and oxide ores. The nickel content of soil may range widely, from 3 to 1,000 $\mu g\, g^{-1}$, depending on mineral composition (NAS 1975).

The main sources of nickel in the atmosphere are windblown dusts and volcanoes (Table 1). Nickel compounds are relatively soluble, and distribution of the element in the environment becomes widespread. Nickel is found at low concentrations in plants and animals. It is not known to be an essential element for man.

2 Anthropogenic sources

A large amount of nickel, about 40 per cent of total production, is used in steel production (Norseth and Piscator 1979). It is also used in alloys, e.g. coins and domestic utensils, and in electroplating. Nickel hydroxide is used in nickel-cadmium batteries. There has been rapid growth in industrial demand for nickel in recent years. World production in 1975 was 753,000 tonnes (Duke 1980).

Nickel occurs in coal at concentrations ranging in general from 1–70 $\mu g\, g^{-1}$ (mean about 15 $\mu g\, g^{-1}$) (Swaine 1980). Higher concentrations occur in oil. Nickel emission factors for coal and oil combustion and various other industrial activities have been formulated; for example, 0.2 $g\, t^{-1}$ of coal burned (assuming 75 per cent removed through particle control devices) and 30 $g\, t^{-1}$ from residual and fuel oil combustion (USEPA 1973). Estimates of anthropogenic sources of nickel to the atmosphere are listed in Table 1.

Table 1 Worldwide emission of nickel to the atmosphere*

Source		Emission rate (10^6 kg y^{-1})
Natural		
Windblown dusts		4.8
Volcanoes		2.5
Vegetation		0.8
Forest fires		0.2
Meteoric dusts		0.2
Sea spray		0.009
	Total	8.5
Anthropogenic†		
Residual and fuel oil combustion		27
Nickel mining and refining		7.2
Waste incineration		5.1
Steel production		1.2
Industrial applications		1.0
Gasoline and diesel fuel combustion		0.9
Coal combustion		0.7
	Total	43

* Reference Schmidt and Andren (1980).
† Emissions during mid-1970s.

In the conversion of coal to methane, nickel catalysts could be converted to nickel subsulphate or nickel carbonyl and possibly released to the environment. Nickel accumulates along roadways, arising from use of nickel-bearing gasoline and abrasion of metal parts and tyres of vehicles. Nickel is added to agricultural soils by application of sewage sludge. The nickel content of the sludges may range from 50 to 5,000 $\mu g\ g^{-1}$ (NAS 1977).

3 Environmental considerations

3.1 Air

Ambient levels of nickel in air have been reported to be 6 $ng\ m^{-3}$ in non-urban areas of the U.S.A. and 17 to 25 $ng\ m^{-3}$ in urban areas in

summer and winter respectively (NAS 1975). At a semi-rural site in England, the average concentration during 1957–74 was 19 ng m^{-3} with a standard deviation of ±50 per cent (Salmon *et al.* 1978). There were wide variations over the period (< 10 to 50 ng m^{-3}), but no overall trend. There was little difference in average levels in summer and winter.

Yearly averages of nickel in air of three large Belgian cities during 1972–77 ranged from 9 to 34 ng m^{-3} and from 13 to 60 ng m^{-3} in a fourth city (Kretzschmar, Delespaul and De Rijck 1980). The relatively little variation of this data for nickel indicates that diffuse sources (traffic, home heating, distant sources) generally predominate (Kretzschmar *et al.* 1980).

Higher values are recorded in heavily industrialized areas and larger cities, e.g. 140 ng m^{-3} in Meuse Valley in Belgium, 117–175 ng m^{-3} in an industrial town in Czechoslovakia and 150 ng m^{-3} in New York City (Rondia 1979, NAS 1975).

3.2 Soil

The large range of natural nickel in soil is only somewhat limited in considering cultivated soils. Based on several reviews, Berrow and Burridge (1980) suggest a normal range of nickel in cultivated soils of 5–500 μg g^{-1} with a typical level of 50 μg g^{-1}. The distribution of values for 752 soil samples taken from farms in England and Wales by Archer (1980) is shown in Figure 1. Most values were in the range 4–80 μg g^{-1}. An isolated high value was 228 μg g^{-1}. The median value was 26 μg g^{-1}.

3.3 Plants

The concentration of nickel in plants is in the general range of 0.05 to 5 μg g^{-1} (dry wt) (NAS 1977). Nickel is relatively more toxic to plants than are other heavy metals. A level in excess of 50 μg g^{-1} in plants is regarded as toxic, although parts of certain plants may show even higher accumulations (Jaffré, Brooks, Lee and Reeves 1976, Boudène 1979). Plant toxicity is less easily related to soil levels, since pH is an important factor. In nickel amended soils, liming to pH 7 was found to result in an appreciable reduction in nickel toxicity compared with pH values less than 6.5 (Williams 1980).

Levels of nickel in various plant species grown in the same soils vary widely, as may the concentrations in the various parts of a single plant

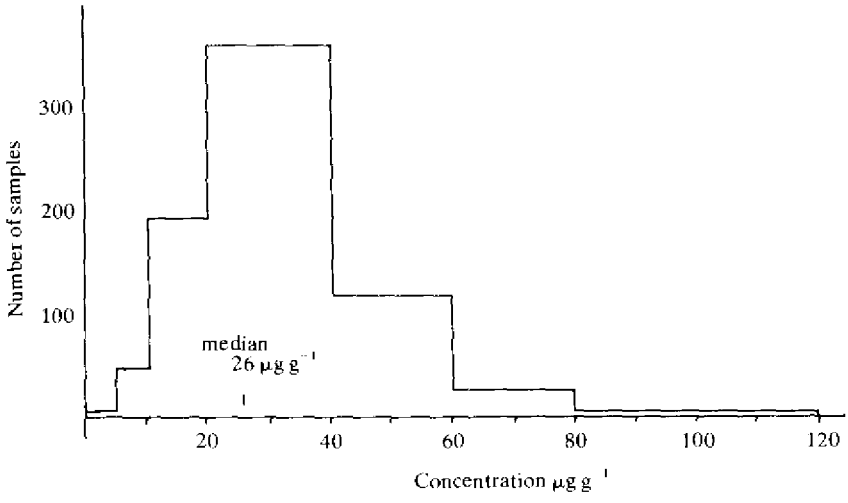


Figure 1 Nickel content of agricultural soils in England and Wales (Archer 1980)

(NAS 1977). Levels in seeds may be three times higher than in stems and leaves (NAS 1977).

3.4 Diet

Data on nickel in foodstuffs are scarce. Available data have been compiled by the National Academy of Sciences (1975) and by Stoeppler (1980). The fresh weight concentrations were: grains, vegetables and fruits 0.02 to 2.7 $\mu g\, g^{-1}$, meats 0.06 to 0.4 $\mu g\, g^{-1}$ and seafood 0.02 to 20 $\mu g\, g^{-1}$. Recent determination of nickel in foods in the Netherlands indicates an average value of 0.5 $\mu g\, g^{-1}$ (wet wt), with certain vegetables such as peas, beans, cabbage, spinach and lettuce between 1 and 3 $\mu g\, g^{-1}$ and fruits, cereals and potatoes between 0.1 and 0.5 $\mu g\, g^{-1}$ (Ellen, van den Bosch-Tibbersma and Douma 1978). Levels in meat and milk are low, between 0.02 and 0.2 $\mu g\, g^{-1}$ (dry wt) and 4 and 25 $\mu g\, \ell^{-1}$ respectively (Boudène 1979).

Aquatic organisms contain relatively larger amounts of nickel, e.g. oysters 1.5 $\mu g\, g^{-1}$ and salmon 1.7 $\mu g\, g^{-1}$ (wet wt) (Boudène 1979). Molluscs may accumulate high levels of nickel if there are high concentrations in the water (Friedricks and Filice 1976).

Estimates of daily intake of nickel in diet are 300–500 μg (Schroeder, Balassa and Tipton 1962), 290 μg (Nodiya 1972), 260 μg (Horak and Sunderman 1973), 165 μg (Myron *et al.* 1978) and 100–300 μg (Clemente, Cigna Rossi and Santaroni 1980). Nickel may be transferred to diet from use of nickel-containing cooking vessels and utensils, from grinding of cereals to flour and hydrogenation processing of oils (Boudène 1979).

3.5 Water

Surveys of nickel in surface waters in Europe and the U.S.A. during 1962–73 gave means of 15 and 19 μg ℓ^{-1} respectively, with maximum levels up to 960 μg ℓ^{-1} (Rondia 1979). Drinking water generally contains less than 10 μg ℓ^{-1} (NAS 1975). In exceptional cases, values up to 75 μg ℓ^{-1} are found and as much as 200 μg ℓ^{-1} near mining areas (Norseth and Piscator 1979).

Levels of nickel in sea water range from 0.1 to 0.5 μg ℓ^{-1} (NAS 1977).

3.6 Miscellaneous

Nickel is found in tobacco, with content of cigarettes ranging from 1 to 3 μg per cigarette (Norseth and Piscator 1979). About 10 to 20 per cent of the nickel is released into the smoke stream. The form in smoke is uncertain, but might possibly be nickel carbonyl (Stahly 1973).

4 Metabolism

4.1 Absorption

Absorption of nickel by the body following intake depends on the chemical form. Data from animal studies indicate that following inhalation, nickel carbonyl is readily absorbed, due to its high lipid solubility, but nickel oxide less so (Sunderman and Selin 1968, Norseth and Piscator 1979). One study showed up to 75 per cent absorption of intratracheal administered nickel chloride (Clary 1975) but another demonstrated only slow removal and thus the potential accumulation of nickel in the lung following chronic exposure to low levels of atmospheric nickel (Williams *et al.* 1980). Following ingestion, about 10 per cent or less of nickel in diet is absorbed (Norseth and Piscator 1979).

Penetration of nickel ions through the skin has been reported (Nielsen 1977).

4.2 Distribution in the body

Animal studies indicate that following intravenous or intratrachael injections, the highest concentrations of nickel are found in kidneys and lungs (NAS 1975). The distribution is similar for nickel carbonyl, which is metabolized to nickel and carbon monoxide. The nickel is oxidized and the carbon monoxide becomes bound to haemoglobin.

Data on human tissue concentrations of nickel are few, as analytical techniques have not always had sufficient sensitivity. Some recent determinations are: lung 15.9 $\mu g\,kg^{-1}$, liver 8.7 $\mu g\,kg^{-1}$ and heart 6.1 $\mu g\,kg^{-1}$ (wet wt) (Myron *et al.* 1978). Higher levels are reported in hair and sweat with some uncertainty. Background levels of nickel in blood serum in a non-exposed population in the U.S.A. were 2.6 $\mu g\,\ell^{-1}$ and 4.6 $\mu g\,\ell^{-1}$ for persons living near a large nickel mine (McNeely, Nechay and Sunderman 1972).

4.3 Retention time

Nickel is cleared rapidly from plasma and excreted predominantly in urine. Excretion rates in a non-exposed population in the U.S.A. were 2.6 $\mu g\,d^{-1}$ and 7.9 $\mu g\,d^{-1}$ for persons living near a large nickel mine (McNeely *et al.* 1972).

Some initial exhalation of absorbed nickel carbonyl occurs via the lungs in the first hours after exposure until it has been degraded, whereupon urinary excretion dominates (Norseth and Piscator 1979).

Animal studies indicate that 80 to 90 per cent of the injected amounts of nickel are excreted within the first three days following exposure (Norseth and Piscator 1979).

From a review of available data, the ICRP has recommended for assessments that it be assumed that 70 per cent of the amount absorbed into blood is excreted with very little delay through the kidneys. The remainder is assumed to be uniformly distributed throughout all organs and tissues of the body and retained with a biological half-life of 1,200 days (ICRP 1981). This retention time has been derived from fairly high estimates of body content (10 μg) and intake rate of nickel (0.4 $mg\,d^{-1}$) (ICRP 1975). Using the most recent estimate of daily intake (~ 170 $\mu g\,d^{-1}$) with fractional absorption and retention in the body of 0.05 and

0.3 respectively, and an estimated body content of nickel of 500 μg (estimated from a mean concentration in tissue of 7 $\mu g\,kg^{-1}$), a mean retention time of 200 days is derived.

5 Effects

Excessive exposure to nickel compounds may cause a variety of local effects, but only nickel carbonyl is associated with systemic effects. Exposure to nickel-containing mists and dusts may cause asthma, pneumoconiosis, and irritation of nasal membranes (Norseth and Piscator 1979). Nickel metal and compounds can have strong sensitizing effects on skin leading to dermatitis. This is particularly the case for direct contact from coins, jewellery, etc. Ingested nickel can aggravate chronic dermatitis. Nickel in general, however, is relatively non-toxic through the oral route, due to limited intestinal absorption.

Initial symptoms of nickel carbonyl exposures are mild nausea, headache, dyspnea and chest pain. These symptoms may disappear, but following a latency period of 0.5 to 5 days, severe pulmonary insufficiency may occur (Norseth and Piscator 1979). As measured by urinary concentrations during the first days after exposure, exposures are classified as mild if the urinary concentrations are less than 100 $\mu g\,\ell^{-1}$ and severe if greater than 500 $\mu g\,\ell^{-1}$ (Sunderman 1971).

Exposures to certain nickel compounds, particularly nickel subsulphide and nickel oxide, as shown in animal studies, can cause lung cancer and cancer of the nasal sinus (IARC 1976).

6 Literature critique

Reviews of nickel behaviour in the environment and effects of excessive exposures in man include National Academy of Sciences (1975), Nielsen (1977), Norseth and Piscator (1979), Rondia (1979) and International Agency for Research on Cancer (1976). The recent compilation and discussion of environmental nickel data contributed by several authors and edited by Nriagu (1980) is particularly comprehensive.

There is still some question regarding the essentiality to man of trace levels of nickel in diet. Maximum recommended levels of nickel as a food contaminant have not been published. An increased cancer risk in individuals occupationally exposed to nickel compounds has been recognized for some years, but uncertainties regarding the active agents and mechanisms of effects prevent reliable risk evaluations.

7 Pathway analysis

The transfer of nickel from general environmental sources to man occurs via the inhalation and ingestion pathways. The basic compartmental arrangement and the transfer relationships are given in Figure 2. The notation and analysis are that of the exposure commitment method (Bennett 1981). It is convenient to begin with unit exposure commmitment to air and determine the resulting exposure commitment to the receptor, considered to be the whole body of man. The commitment results can then be utilized to determine the contributions to the steady-state concentration of nickel in the body from current background levels in air and diet.

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 an initial 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 lungs and absorption to blood of 20 per cent of the intake amount ($P_{5'6'}$), distribution of nickel in blood to body tissues of 30 per cent ($P_{6'7'}$) and residence time in the body of 200 d. The sequential multiplication of transfer factors gives the following exposure commitment to the body from unit exposure commitment to air.

$$
\begin{aligned}
E_7 &= P_{15'}P_{5'6'}P_{6'7'}P_{7'7}E_1 \\
&= 8{,}000\ \frac{\mu g}{\mu g\ y\ m^{-3}}\ 0.2\ \frac{\mu g}{\mu g}\ 0.3\ \frac{\mu g}{\mu g} \\
&\quad \times 7.8\ 10^{-6}\ \frac{\mu g\ y\ g^{-1}}{\mu g}\ 1\ \mu g\ y\ m^{-3} \\
&= 0.004\ \mu g\ y\ g^{-1}
\end{aligned}
$$

For the ingestion pathway, the analysis begins with an assumed deposition velocity of 0.5 $cm\ s^{-1}$ ($P_{12'}$) for nickel attached to ambient aerosol particles. For background concentrations of nickel in air of the order of 20 $ng\ m^{-3}$, this results in a flux rate to surface soil of 3 $mg\ m^{-2}\ y^{-1}$. The

Table 2 Nickel in the environment: summary of representative values

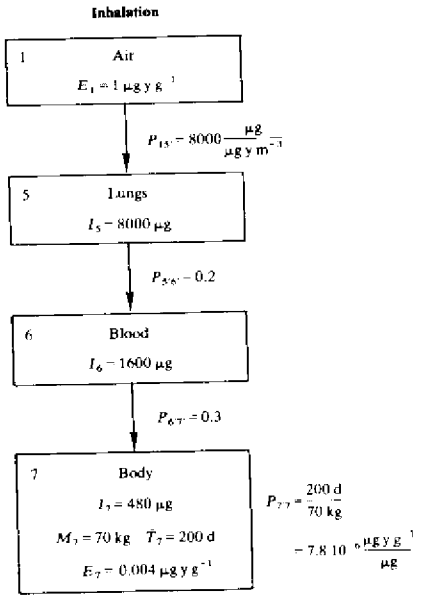
Concentrations		
Atmosphere		
urban	20 ng m^{-3}	(10–60)*
rural	10 ng m^{-3}	(6–20)
Lithosphere		
agricultural soil	30 μg g^{-1}	(4–230)
Hydrosphere		
freshwater	10 μg ℓ^{-1}	(< 10–960)
ocean	0.3 μg ℓ^{-1}	(0.1–0.5)
Biosphere		
land plants	0.5 μg g^{-1}	(0.01–3)
foods	0.5 μg g^{-1}	(0.01–3)
Man		
tissues	7 μg kg^{-1}	(1–15)
blood	3 μg ℓ^{-1}	(1–5)
Transfer rates		
Intake		
ingestion	170 μg d^{-1}	(165–500)
inhalation—urban	0.4 μg d^{-1}	(0.2–1)
—rural	0.2 μg d^{-1}	(0.1–0.4)
Absorption		
GI tract	0.05	(0.01–0.1)
lungs—retention	0.35	(0.3–0.4)
—absorption	0.6	

* Range of values in parentheses.

mixing depth in soil is assumed to be 20 cm, which is the plough layer thickness, and the soil density is 1.4 g cm^{-3}. Input from deposition may be considered the main current source of nickel to the background soil compartment. Then, from the above input rate and the background level of nickel in soil of 26 μg g^{-1} (Figure 1), one infers a mean residence time of nickel in soil of 2,400 y.

The relationship between nickel in the soil compartment and the intake rate via diet is obtained from background equilibrium values. The transfer factor, $P_{23'}$, is evaluated in Figure 2 from the representative dietary intake rate of nickel of 170 μg d^{-1} and the rounded concentration in soil of 30 μg g^{-1}.

Dietary intake of nickel is transferred directly to the gastro-intestinal tract ($P_{3'4'} = 1.0$). Fractional absorption to blood is assumed to be 5 per



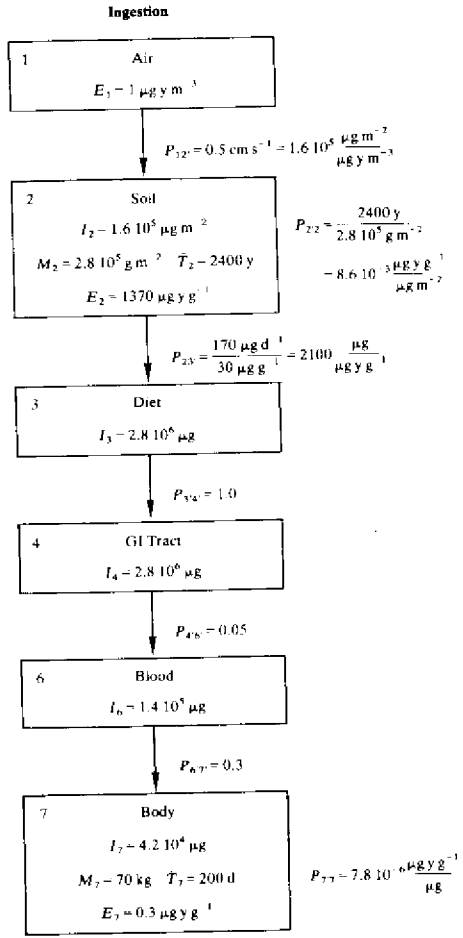


Figure 2 Pathway evaluations for nickel

cent, of which 70 per cent is rapidly excreted and 30 per cent is transferred to body tissues and retained on average for 200 d.

The exposure commitment to the body from unit exposure commitment of nickel in air via the ingestion pathway is obtained from the sequential product of transfer factors.

$$E_7 = P_{12}P_{2'2}P_{23}P_{3'4}P_{4'6}P_{6'7}P_{7'7}E_1$$

$$= 1.6\ 10^{5}\ \frac{\mu g\ m^{-2}}{\mu g\ y\ m^{-3}}\ 8.6\ 10^{-3}\ \frac{\mu g\ y\ g^{-1}}{\mu g\ m^{-2}}\ 2.1\ 10^{3}\ \frac{\mu g}{\mu g\ y\ g^{-1}}$$

$$\times 1.0\ \frac{\mu g}{\mu g}\ 0.05\ \frac{\mu g}{\mu g}\ 0.3\ \frac{\mu g}{\mu g}\ 7.8\ 10^{-6}\ \frac{\mu g\ y\ g^{-1}}{\mu g}\ 1\ \mu g\ y\ m^{-3}$$

$$= 0.3\ \mu g\ y\ g^{-1}$$

At equilibrium, the concentration of 1 $\mu g\ m^{-3}$ of nickel in air can be related to the concentrations of nickel in the body of 0.004 $\mu g\ g^{-1}$ from the inhalation pathway and 0.3 $\mu g\ g^{-1}$ from the ingestion pathway.

The current levels of nickel in air, diet and man are indicated in Table 3. The level of 0.02 $\mu g\ m^{-3}$ of nickel in air contributes an estimated nickel concentration of 0.08 $\mu g\ kg^{-1}$ in the body via the inhalation pathway. This same concentration in air can be associated with representative dietary intake of nickel of 160 $\mu g\ d^{-1}$. This link is not strong, however, as it uses the assumed deposition velocity and the inferred

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

	Air	Diet	Body
Inhalation pathway			
	$0.02\ \frac{\mu g}{m^3}$	⟶	$0.08\ \frac{\mu g}{kg}$
Ingestion pathway			
	$0.02\ \frac{\mu g}{m^3}$	⟶ $160\ \frac{\mu g}{d}$	⟶ $6.8\ \frac{\mu g}{kg}$
	drinking water	$10\ \frac{\mu g}{d}$	⟶ $0.4\ \frac{\mu g}{kg}$
		Total	$7.3\ \frac{\mu g}{kg}$

mean residence time of nickel in soil, the latter parameter in particular being very uncertain.

The dietary intake may be taken as the starting point in the chain, giving an estimated contribution to nickel concentration in the body of 6.8 $\mu g\ kg^{-1}$. Intake of nickel in drinking water gives an additional small contribution to nickel content of the body. The current body burden of nickel is estimated to be 500 μg (7.3 $\mu g\ kg^{-1} \times 70$ kg).

The ingestion pathway is the greatest contributor to the nickel concentration in the body. The low absorption of nickel from the gastrointestinal tract and the limited retention in tissues lead to the general conclusion that toxic effects would be expected only in extreme and unusual circumstances. The relationships developed here give the general associations between total nickel in the environment and in man. Relationships for specific nickel compounds may be useful and can be derived as pertinent data on environmental behaviour become available.

References

Archer, F. C. 1980 Trace elements in soils in England and Wales. In: *Inorganic Pollution and Agriculture*, MAFF Ref. Book 326, H.M.S.O., London, 184–190.

Angino, E., Wixson, B. G. and Smith, I. 1977 Drinking water quality and chronic disease. *Environ. Sci. Technol.* **11**, 660–665.

Bennett, B. G. 1981 Exposure commitment concepts and application. In: Exposure Commitment Assessments of Environmental Pollutants, Volume 1, No. 1, MARC Report No. 23, Monitoring and Assessment Research Centre, Chelsea College, London.

Berrow, M. L. and Burridge, J. C. 1980 Trace element levels in soils: effects of sewage sludge. In: *Inorganic Pollution and Agriculture*, MAFF Ref. Book 326, H.M.S.O., London, 159–183.

Boudène, C. 1979 Food contamination by metals. In: *Trace Metals: Exposure and Health Effects*, E. Di Ferrante (Ed.), Commission of the European Communities, Pergamon Press, Oxford, 163–183.

Clary, J. J. 1975 Nickel chloride induced metabolic changes in the rat and guinea pig. *Toxicol. Appl. Pharmacol.* **31**, 55–65.

Clemente, G. F., Cigna Rossi, L., Santaroni, G. P. 1980 Nickel in foods and dietary intake of nickel. In: *Nickel in the Environment*, J. O. Nriagu (Ed.), John Wiley & Sons, New York, 493–498.

Duke, J. M. 1980 Production and uses of nickel. In: *Nickel in the Environment*, J. O. Nriagu (Ed.), John Wiley & Sons, New York, 51–65.

Ellen, G., van den Bosch-Tibbersma, G. and Douma, F. F. 1978 Nickel content of various Dutch foodstuffs. *Zeits. Lebensm. Untersuch. Forsch.* **166**, 145.

Friedrich, A. R. and Filice, F. P. 1976 Uptake and accumulation of the nickel ion by Mytilus edulis. *Bull. Environ. Contam. Toxicol.* **16**, 750–755.

Horak, W. and Sunderman, F. W., Jr. 1973 Fecal nickel excretion by healthy adults. *Clin. Chem.* **19**, 429–430.

International Agency for Research on Cancer (IARC) 1976 Nickel and nickel compounds. In: *IARC Monographs on the evaluation of carcinogenic risk of chemicals to man*, Vol. 11, IARC, Lyon, 75–112.

International Commission on Radiological Protection (ICRP) 1975 *Report on the Task Group on Reference Man*. ICRP Publication 23, Pergamon Press, Oxford.

International Commission on Radiological Protection (ICRP) 1981 *Limits for intakes of radionuclides by workers*. ICRP Publications 30, Part 2, Pergamon Press, Oxford.

Jaffré, T., Brooks, R. R., Lee, J. and Reeves, R. D. 1976 *Sebertia acuminata*: a hyperaccumulator of nickel from New Caledonia. *Science* **193**, 579–580.

Kretzschmar, J. G., Delespaul, I. and De Rijck, T. 1980 Heavy metal levels in Belgium: a five-year survey. *Sci. Total Environ.* **14**, 85–97.

McNeely, M. D., Nechay, M. W. and Sunderman, F. W., Jr. 1972 Measurements of nickel in serum and urine as indicators of environmental exposure to nickel. *Clin. Chem.* **18**, 992–995.

Myron, D. R., Zimmerman, T. J., Shuler, T. R., Klevay, L. M., Lee, D. R. and Nielsen, F. H. 1978 Intake of nickel and vanadium by humans; a survey of selected diets. *Am. J. Clin. Nutr.* **31**, 527–531.

National Academy of Sciences (NAS) 1975 *Nickel*. National Research Council/National Academy of Sciences, Washington D.C.

National Academy of Sciences (NAS) 1977 *Geochemistry and the environment, Volume II. The relation of other selected trace elements to health and disease*. Washington, D.C.

Nielsen, F. H. 1977 Nickel toxicity. In: *Toxicology of Trace Elements*, R. A. Goyer and M. A. Mehlman (Eds), John Wiley & Sons, New York, 129–146.

Nodiya, P. I. 1972 Cobalt and nickel balances in students of an occupational technical school. *Gig. Sanit.* **37**, 108–109 (in Russian).

Norseth, T. and Piscator, M. 1979 Nickel. In: *Handbook on the Toxicology of Metals*, L. Friberg, F. Nordberg and V. Vouk (Eds), Elsevier/North-Holland Biomedical Press, Amsterdam, 541–553.

Nriagu, J. O. (Ed.) 1980 *Nickel in the Environment*, John Wiley & Sons, New York.

Rondia, D. 1979 Sources, modes, and levels of human exposure to chromium and nickel. In: *Trace Metals: Exposure and Health Effects*, E. Di Ferrante (Ed.), Commission of the European Communities, Pergamon Press, Oxford, 117–134.

Salmon, L., Atkins, D. H. F., Fisher, E. M. R., Healy, C. and Law, D. V. 1978 Retrospective trend analysis of the content of U.K. air particulate material 1957–1974. *Sci. Total Environ.* **9**, 161–200.

Schmidt, J. A. and Andren, A. W. 1980 The atmospheric chemistry of nickel. In: *Nickel in the Environment*, J. O. Nriagu (Ed.), John Wiley & Sons, New York, 93–135.

Schroeder, H. A., Balassa, J. J. and Tipton, I. H. 1962 Abnormal trace metals in man—nickel. *J. Chron. Dis.* **15**, 51–65.

Stahly, E. E. 1973 Some consideration of metal carbonyls in tobacco smoke. *Chem. Ind.* **13**, 620–623.

Stoeppler, M. 1980 Analysis of nickel in biological materials and natural waters. In: *Nickel in the Environment*, J. O. Nriagu (Ed.), John Wiley & Sons, New York, 661–821.

Sunderman, F. W. and Selin, C. E. 1968 The metabolism of nickel-63 carbonyl. *Toxicol. Appl. Pharmacol.* **12**, 207–218.

Sunderman, F. W. 1971 Effect of nickel carbonyl upon hepatic concentrations of adenosine triphosphate. *Res. Commun. Chem. Pathol. Pharm.* **2**, 545–551.

Swaine, D. J. 1980 Nickel in coal and fly ash. In: *Nickel in the environment*, J. O. Nriagu (Ed.), John Wiley & Sons, New York, 67–92.

Taylor, S. R. 1964 Abundance of chemical elements in the continental crust: a new table. *Geochim. Cosmochim. Acta* **28**, 1273–1285.

U.S. Environmental Protection Agency 1973 Emission factors for trace substances. U.S. EPA Report EPA-450/2-73-001.

Williams, J. H. 1980 Effect of soil pH on the toxicity of zinc and nickel to vegetable crops. In: *Inorganic Pollution and Agriculture*, MAFF Ref. Book 326, H.M.S.O., London, 211–218.

Williams, S. J., Holden, K. M., Sabransky, M., Menzel, D. B. 1980 The distributional kinetics of Ni^{2+} in the rat lung. *Toxicol. Appl. Pharmacol.* **55**, 85–93.

TIN

1 Natural cycle

Tin is a metal of atomic number 50. Its average abundance in the earth's crust is 2 $\mu g\ g^{-1}$ (Taylor 1964). Tin has two oxidation states, +2 (stannous) and +4 (stannic) and forms a large number of inorganic and organic compounds.

Tin is mobilized in the environment by the weathering of rocks. The average level in soils is 10 $\mu g\ g^{-1}$, varying from 2 to 200 $\mu g\ g^{-1}$ (Bowen 1966). Natural levels in air are less than 10 $ng\ m^{-3}$. It occurs in freshwater at concentrations of about 0.04 $\mu g\ \ell^{-1}$ and in sea water of 0.2 to 3 $\mu g\ \ell^{-1}$ (Bowen 1966, NAS 1977).

Tin is commonly found in plants and animals, usually at levels less than 1 $\mu g\ g^{-1}$, but it is not thought to be an essential trace element.

2 Anthropogenic sources

Tin is used in tin plating and food cans, accounting for about 85 per cent of total production (Piscator 1979). It is used in solders and alloys. Pewter contains 90–95 per cent tin. Inorganic tin compounds are used in glass making and textile printing. Organotin compounds are used in stabilizers in plastics and have also been utilized as pesticides. Stannous fluoride is added to toothpaste to prevent caries.

World production of tin in 1979 was 245,000 tonnes, increasing on average one per cent per year since 1950 (Bauer 1980). Malaysia is the leading producer, with Thailand, Indonesia, the U.S.S.R., China, Bolivia, the U.K. and Brazil other important producers.

3 Environmental considerations

3.1 Air

Tin and its compounds are of generally low volatility; therefore, tin in the atmosphere may be considered to result mainly from wind-blown dusts and volcanoes and the direct emissions from industries.

Reported concentrations of tin in air are 1.5 $ng\ m^{-3}$ at Sutton in the U.K. (Hamilton 1974), 100 $ng\ m^{-3}$ at Heidelberg, Germany (Bogen

1973) and <3 to 300 ng m^{-3} in the U.S.A., the lowest values being in rural and suburban areas (Tabor and Warren 1958). Much higher concentrations, 3,800–4,400 ng m^{-3} were measured 700 m from an industrial plant in Japan (Piscator 1979).

3.2 Soil

The concentration of tin in soil varies greatly from a few $\mu g\, g^{-1}$ to several hundred $\mu g\, g^{-1}$ (Schroeder, Balassa and Tipton 1964). The levels are usually related to the bedrock from which the soils are derived. Of nearly 900 samples of soil from the U.S.A., only one per cent contained more than 10 $\mu g\, g^{-1}$ (NAS 1977). Levels of tin in soils from mining districts are, of course, much higher.

3.3 Plants

Levels of tin in land plants are generally less than 0.3 $\mu g\, g^{-1}$, except in lichens and mosses (Craig 1980). Marine plants contain tin at an average level of 1 $\mu g\, g^{-1}$ (Craig 1980).

Levels of tin in plants may reflect levels of tin in soil, but the concentrating effect is not great. Concentration ratios are around 1 (NAS 1977). Accumulations from sea water are greater, concentration ratios being 92 for brown algae and 2,900 for plankton (Bowen 1966).

3.4 Diet

Concentrations of tin in foods are usually less than 0.3 $\mu g\, g^{-1}$ for those of vegetable origin and less than 3 $\mu g\, g^{-1}$ for those of animal origin (Boudène 1979). Concentrations in tinned foodstuffs may reach several hundred $\mu g\, g^{-1}$, e.g. tinned asparagus in excess of 500 $\mu g\, g^{-1}$ (Boudène 1979) and high acidity juices of 100–500 $\mu g\, g^{-1}$ (Piscator 1979). Corrosion promoters in foods are nitrates, such as in vegetables produced in intensive farming, suphur dioxide pesticides and naturally occurring sulphur compounds in foods. In addition, atmospheric oxygen operates quite rapidly on opened cans. This contamination is considerably reduced by use of laquered cans.

Dietary intake of tin is estimated to be of the order of 200 $\mu g\, d^{-1}$ (Hamilton and Minski 1972–73) which may be considerably increased if large amounts of canned food are included in diet. Schroeder *et al.* (1964) estimated dietary intake to be 3.6 mg d^{-1}. The above concentration estimates indicate that intake of tin would be less than 2 mg d^{-1}.

3.5 Water

The concentration of tin in freshwater is generally around 1 $\mu g\ \ell l^{-1}$ or less (Piscator 1979). Sometimes higher concentrations are found in drinking water, possibly resulting partly from use of bronze fittings (Piscator 1979).

The general occurrence of organotin compounds in water is not known; however, due to the ease of degradation of these compounds in the aquatic environment, the levels are likely to be low (Craig 1980).

4 Metabolism

4.1 Absorption

Absorption of inhaled tin is uncertain. Absorption of ingested tin is only a few per cent, values for rats being 0.6 per cent (stannic compounds) and 2.8 per cent (stannous compounds) (Piscator 1979). Other studies with mice, rats, dogs and monkeys also indicated low absorption from the gut, less than 5 per cent (Furchner and Drake 1976).

The metabolism of organotin compounds has not been widely studied. Absorption of short-chain alkyltin compounds is greater than the longer-chain compounds. Intestinal absorption of the various compounds is 10 per cent or less in animals. There are no data for humans (Piscator 1979).

4.2 Distribution in the body

Highest concentrations of tin in animals following oral doses are found in kidneys, liver and bone with the main deposit in bone (35–46 per cent of administered dose) (Hiles 1974). In humans, small amounts of tin have been found in most organs, mainly bone, lungs, liver and kidneys. Tissue concentrations are generally less than 1 $\mu g\ g^{-1}$ (wet wt), e.g. 0.8 $\mu g\ g^{-1}$ in rib bone and lungs, 0.4 in liver, 0.2 in kidneys and 5 $\mu g\ \ell^{-1}$ in blood (Hamilton *et al.* 1972–73) and 0.03 to 1.4 $\mu g\ g^{-1}$ (median 0.1 $\mu g\ g^{-1}$) in femur (Lindh *et al.* 1980). Tin is thought to accumulate in lungs with age (Schroeder *et al.* 1964).

Organotin compounds seem to be distributed to liver, kidneys and brain, as evidenced by animal studies (Piscator 1979).

4.3 Retention times

Tin is excreted mainly via urine and to some extent via bile (Piscator 1979). Retention time in soft tissues is short. In animal studies (five

species) 20–60 per cent of the dose (stannous chloride) was found to be excreted in urine and 5–10 per cent in faeces within three days (Furchner and Drake 1976). Following intravenous injections of animals with the isotope ^{113}Sn (radioactive half-life of 115 d), four components of elimination were identified, the longest half-time being about 90 d which corresponds to a biological half-time component of 410 d (Furchner and Drake 1976). The retention function for absorbed tin in humans is assumed to have fractions of 0.2, 0.2 and 0.6 retained with half-times of 4, 25 and 400 d respectively (ICRP 1981). The effective half-time is thus 246 d, giving a mean retention time of 350 d.

5 Effects

Tin is not known to be an essential element in humans but it is thought necessary for growth in rats (Schwarz, Milne and Vinyard 1970). The inorganic and organic tin compounds are generally not highly toxic to man and animals. Gastro-intestinal symptoms (nausea, diarrhoea) may be caused by the consumption of 50 to 100 mg of tin in foods (Piscator 1979). Several outbreaks of food tin poisoning have been reported, with concentrations in the food of 250 to 500 $\mu g\, g^{-1}$ (Piscator 1979). Pneumoconiosis may be caused by long-term inhalation of tin oxide dusts and fumes. Systemic effects of tin exposure observed in animals include neurological damage and renal lesions.

The toxicity of organotin compounds declines as the length of the carbon chain increases. Triethyltin has produced encephalopathy characterized by irritation of white matter, but no damage to nerve cells, as opposed to alkylmercury behaviour (Piscator 1979). The damage is reversible. Other trialkyltin compounds have weaker effects on the brain. Tri- and dibutyltin compounds cause mainly liver and bile duct lesions respectively in animals (Piscator 1979).

6 Literature critique

There are considerable difficulties in obtaining quantitative determinations of low concentrations of tin in environmental samples. A variety of methods have been proposed, but interlaboratory comparison is lacking.

A review of tin toxicity is presented by Piscator (1979). Consideration of organotin compounds in the environment was reviewed by Craig (1980). Microbiological degradation of the organic compounds is rapid,

making persistence and potential environmental problems much less than for methylmercury. Bioconcentration of organotin in mammals does not seem to be important, as elimination is in general quite rapid.

7 Pathway analysis

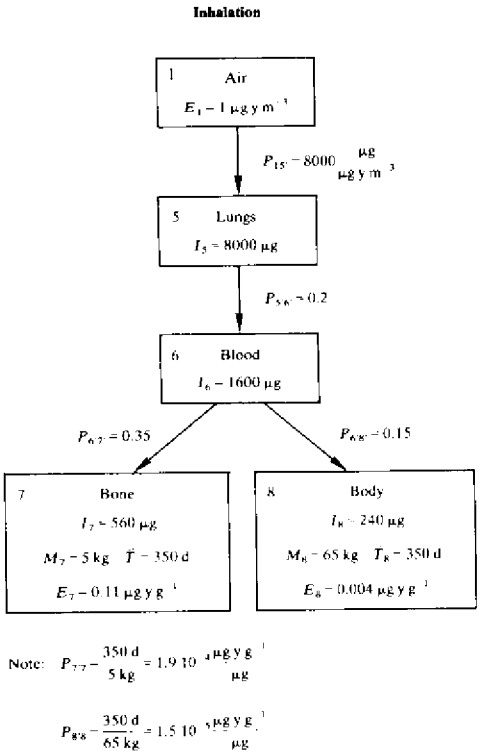
A list of representative values of tin levels in the environment and of transfer rates to man is given in Table 1. Many of the values are uncertain, but it is useful to assign tentative estimates to complete an initial assessment example.

The compartmental model used in assessing the transfer of tin from general atmospheric sources to man is illustrated in Figure 1. The

Table 1 Tin in the environment: summary of representative values

Concentrations		
Atmosphere		
urban	30 ng m^{-3}	(3–300)*
rural	10 ng m^{-3}	(1–100)
Lithosphere		
agricultural soil	10 μg g^{-1}	(2–200)
Hydrosphere		
freshwater	0.04 μg ℓ^{-1}	(<1)
ocean	0.8 μg ℓ^{-1}	(0.2–3)
Biosphere		
land plants	<0.3 μg g^{-1}	
marine plants	1 μg g^{-1}	
food	0.2 μg g^{-1}	(0.1–500)
Man		
tissues	<1 μg g^{-1}	
blood	5 μg ℓ^{-1}	
Transfer rates		
Intake		
ingestion	200 μg d^{-1}	(150–2000)
inhalation—urban	0.7 μg d^{-1}	(0.07–7)
—rural	0.2 μg d^{-1}	(0.02–2)
Absorption		
GI tract	0.02	(0.006–0.05)
lungs—retention	0.35	(0.3–0.4)
—absorption	0.6	

* Range of values in parentheses.



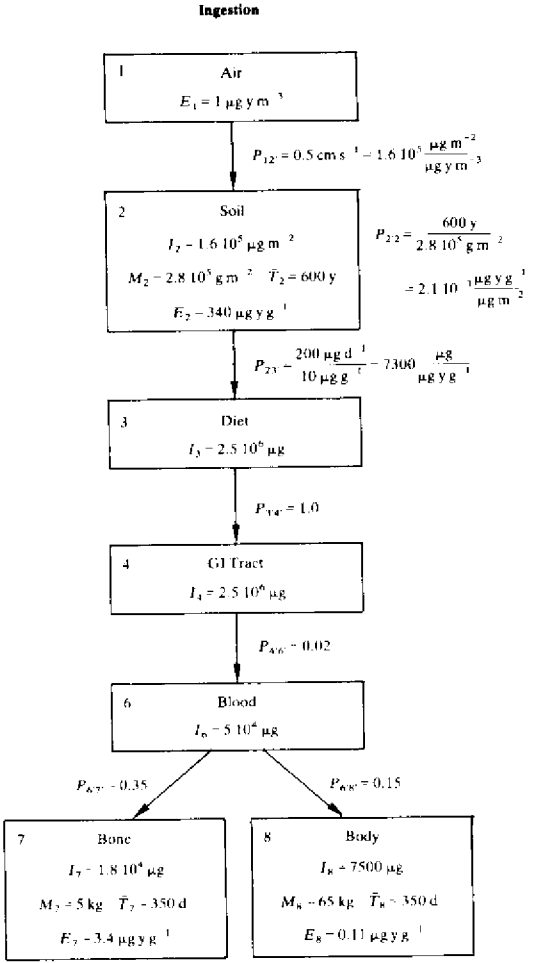


Figure 1 Pathway evaluations for tin

inhalation and ingestion pathways are considered. The parameters for evaluation of the transfer factors, P_{ij}, are included in the diagrams. The transfer factors relate intake and exposure commitments in successive compartments (Bennett 1981).

The assessments begin with unit exposure commitment to air and progress to the receptor organs in man. Exposure commitments are estimated separately for bone and for the remainder of body tissues.

For the inhalation pathway the main assumptions in the exposure evaluation are the breathing rate of 22 $m^3 d^{-1}$ or 8,000 $m^3 y^{-1}$ ($P_{15'}$), retention in the lungs and absorption to blood of 20 per cent of the intake amount ($P_{5'6'}$). Of the amount reaching blood, 50 per cent is assumed rapidly eliminated, 35 per cent is retained in bone and 15 per cent in other body tissues (ICRP 1981). The same effective mean residence time, 350 d, is used for retention in both bone and other tissues.

The exposure commitments to the receptor organs from unit exposure commitment to air are obtained by sequential multiplication of the transfer factors. For the inhalation pathway the results are for bone:

$$
\begin{aligned}
E_7 &= P_{15'} P_{5'6'} P_{6'7'} P_{7'7} E_1 \\
&= 8{,}000 \frac{\mu g}{\mu g\ y\ m^{-3}} 0.2 \frac{\mu g}{\mu g} 0.35 \frac{\mu g}{\mu g} \\
&\quad \times 1.9\ 10^{-4} \frac{\mu g\ y\ g^{-1}}{\mu g} 1\ \mu g\ y\ m^{-3} \\
&= 0.1\ \mu g\ y\ g^{-1}
\end{aligned}
$$

and for other body tissues:

$$
\begin{aligned}
E_8 &= P_{15'} P_{5'6'} P_{6'8'} P_{8'8} E_1 \\
&= 8{,}000 \frac{\mu g}{\mu g\ y\ m^{-3}} 0.2 \frac{\mu g}{\mu g} 0.15 \frac{\mu g}{\mu g} \\
&\quad \times 1.5\ 10^{-5} \frac{\mu g\ y\ g^{-1}}{\mu g} 1\ \mu g\ y\ m^{-3} \\
&= 0.004\ \mu g\ y\ g^{-1}
\end{aligned}
$$

For the ingestion pathway, the association between tin in air and the dietary intake is not well founded; however, as for nickel, preliminary parameters including a mean residence time in soil can be assigned. The deposition velocity for atmospheric particulates is, as before, assumed to be 0.5 cm s^{-1}. The residence time in soil, $\bar{T}_2$, can be determined from the relationship

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

where C_2^* is the concentration of tin in soil, F_2^* is the input rate per unit area, with the asterisks indicating steady-state values, and M is the soil mass per unit area (density 1.4 g cm^{-3} and mixing depth 20 cm) (Bennett 1981). A background concentration of tin in air of 30 ng m^{-3} times the deposition velocity gives an input rate to soil of 4.7 mg m^{-2} y^{-1}. If this is assumed to be the main input source and the soil concentration is 10 μg g^{-1}, the mean residence time of tin in soil, inferred from the above formula, is 600 y.

The ratio of the dietary intake rates of tin to the associated background concentration of tin in soil gives a value for the transfer factor between the soil and diet compartments. Representative dietary intake rate is estimated to be 200 μg d^{-1} and the soil concentration 10 μg g^{-1}. The transfer factor, $P_{23'}$, is evaluated in Figure 1.

Transfer of dietary intake to the gastro-intestinal tract is by direct movement ($P_{3'4'} = 1.0$). Absorption to blood is assumed to be 2 per cent (ICRP 1981). Distribution to tissues and retention is the same as for tin reaching blood following inhalation.

Contributions to the exposure commitments to bone and other tissue of tin via the ingestion pathway are determined from the sequential multiplication of transfer factors:

$$\begin{aligned} E_7 &= P_{12'}P_{2'2}P_{23'}P_{3'4'}P_{4'6'}P_{6'7'}P_{7'7}E_1 \\ &= 1.6\times10^{5}\,\frac{\mu g\ m^{-2}}{\mu g\ y\ m^{-3}}\,2.1\times10^{-3}\,\frac{\mu g\ y\ g^{-1}}{\mu g\ m^{-2}}\,7.3\times10^{3}\,\frac{\mu g}{\mu g\ y\ g^{-1}} \\ &\quad\times 1.0\,\frac{\mu g}{\mu g}\,0.02\,\frac{\mu g}{\mu g}\,0.35\,\frac{\mu g}{\mu g}\,1.9\times10^{-4}\,\frac{\mu g\ y\ g^{-1}}{\mu g}\,1\ \mu g\ y\ m^{-3} \\ &= 3.4\ \mu g\ y\ g^{-1} \end{aligned}$$

$$E_8 = P_{12}P_{2'2}P_{23}P_{3'4}P_{4'6}P_{6'8}P_{8'8}E_1$$

$$= 1.6\times10^{5}\,\frac{\mu g\ m^{-2}}{\mu g\ y\ m^{-3}}\,2.1\times10^{-3}\,\frac{\mu g\ y\ g^{-1}}{\mu g\ m^{-2}}\,7.3\times10^{3}\,\frac{\mu g}{\mu g\ y\ g^{-1}}$$

$$\times 1.0\,\frac{\mu g}{\mu g}\,0.02\,\frac{\mu g}{\mu g}\,0.15\,\frac{\mu g}{\mu g}\,1.5\times10^{-5}\,\frac{\mu g\ y\ g^{-1}}{\mu g}\,1\ \mu g\ y\ m^{-3}$$

$$= 0.1\ \mu g\ y\ g^{-1}$$

These results are for an exposure commitment to air of $1\ \mu g\ y\ m^{-3}$. At steady state the relationships are numerically equal. For example, $1\ \mu g\ m^{-3}$ of tin in air will contribute, at equilibrium, a concentration of $3.4\ \mu g\ g^{-1}$ of tin in bone via the ingestion pathway.

The relationships for current background levels are given in Table 2. A concentration of tin in air of $30\ ng\ m^{-3}$ is assumed representative. This results in a concentration in bone of $3\ ng\ g^{-1}$ via the inhalation pathway and, with the intermediate association of $200\ \mu g\ d^{-1}$ dietary

Table 2 Current levels of tin in the background environment and in man

	Air	Diet	Bone	Body
Inhalation pathway	$0.03\ \frac{\mu g}{m^3}$		$0.003\ \frac{\mu g}{g}$	$0.0001\ \frac{\mu g}{g}$
Ingestion pathway	$0.03\ \frac{\mu g}{m^3}$	$200\ \frac{\mu g}{d}$	$0.1\ \frac{\mu g}{g}$	$0.003\ \frac{\mu g}{g}$
		Total	$0.1\ \frac{\mu g}{g}$	$0.003\ \frac{\mu g}{g}$

intake rate, contributes a concentration in bone of 100 ng g^{-1} via the ingestion pathway. The concentrations are a factor of thirty less in other body tissues. The most significant transfer of tin to the body at equilibrium is via the ingestion pathway.

These estimates are in agreement with the median results of the recent measurements of tin in bone by Lindh *et al.* (1980), but the estimated concentration in other tissue is less than is usually measured. Additional sensitive analytical determinations of tin in tissues may indicate the need for adjustment of some parameters of the calculations. Both measurements and calculations must be extended to specific compounds of tin and toxic levels must be better known to complete the assessment of hazard from environmental exposure to tin.

References

Bauer, W. (Ed.) 1980 *Metal Statistics 1969–1979*, Metallgesellschaft AG, Frankfurt.

Bennett, B. G. 1981 Exposure commitment concepts and application. In: *Exposure Commitment Assessments of Environmental Pollutants*, Volume 1, No. 1, MARC Report No. 23, Monitoring and Assessment Research Centre, Chelsea College, London.

Bogen, J. 1973 Trace elements in atmospheric aerosol in the Heidelberg area, measured by instrumental neutron activation analysis, *Atmos. Environ.* **7**, 1117–1125.

Boudène, C. 1979 Food contamination by metals. In: *Trace Metals: Exposure and Health Effects*, E. Di Ferrante (Ed.), Commission of the European Communities, Pergamon Press, Oxford, 163–183.

Bowen, H. J. M. 1966 *Trace Elements in Biochemistry*, Academic Press, London.

Craig, P. J. 1980 Metal cycles and biological methylation. In: *Handbook of Environmental Chemistry, Vol. 1, Part A, The Natural Environment and Biogeochemical Cycles*, O. Hutzinger (Ed.), Springer-Verlag, Berlin, 169–227.

Furchner, J. E. and Drake, G. A. 1976 Comparative metabolism of radionuclides in mammals—XI. Retention of ^{113}Sn in the mouse, rat, monkey and dog. *Health Phys.* **31**, 219–224.

Hamilton, E. I., Minski, M. J. and Cleary, J. J. 1972–73 The concentration and distribution of some stable elements in healthy human tissues from the United Kingdom, *Sci. Total Environ.* **1**, 341–374.

Hamilton, E. I. and Minski, M. J. 1972–73 Abundance of the chemical elements in man's diet and possible relations with environmental factors, *Sci. Total Environ.* **1**, 375–394.

Hamilton, E. I. 1974 The chemical elements and human morbidity—water, air and places—a study of natural variability, *Sci. Total Environ.* **3**, 3–85.

Hiles, R. A. 1974 Absorption, distribution and excretion of inorganic tin in rats, *Toxicol. Appl. Pharmacol.* **27**, 366–379.

International Commission for Radiological Protection (ICRP) 1981 *Limits for intakes of radionuclides by workers*. ICRP Publication 30, Part 2, Pergamon Press, Oxford.

Lindh, U., Brune, D., Nordberg, G. and Wester, P. O. 1980 Levels of antimony, arsenic, cadmium, copper, lead, mercury, selenium, silver, tin and zinc in bone tissue of industrially exposed workers, *Sci. Total Environ.* **16**, 109–116.

Piscator, M. 1979 Tin. In: *Handbook on the Toxicology of Metals*, L. Friberg, F. Nordberg and V. Vouk (Eds), Elsevier/North-Holland Biomedical Press, Amsterdam, 613–626.

National Academy of Sciences (NAS) 1977 *Geochemistry and the Environment Vol. II. The relation of other selected trace elements to health and disease.* Washington D.C.

Schroeder, H. A., Balassa, J. J. and Tipton, I. H. 1964 Abnormal trace elements in man—tin, *J. Chron. Dis.* **17**, 483–502.

Schwarz, K., Milne, D. B. and Vinyard, E. 1970 Growth effects of tin compounds in rats maintained in a trace element controlled environment, *Biochem. Biophys. Res. Commun.* **40**, 22.

Tabor, E. C. and Warren, W. V. 1958 Distribution of certain metals in the atmosphere of some American cities, *Arch. Ind. Hyg.* **17**, 145–151.

Taylor, S. R. 1964 Abundance of chemical elements in the continental crust: a new table, *Geochim. Cosmochim. Acta* **28**, 1273–1285.

Tipton, I. H., Stewart, P. L. and Dickson, J. 1969 Patterns of elemental excretion in long-term balance studies, *Health Phys.* **16**, 455–462.

List of MARC Reports

All MARC publications are subject to peer review. Titles to date in the series are:

No.

1 *The ozone depletion problem (an example of harm commitment)* by Lester Machta, (out of print)

2 *Vanadium in the environment* by Siv Bengtsson and Germund Tyler, 38 pp £1.00

3 *Suggestions for the development of a hazard evaluation procedure for potentially toxic chemicals* by Robert C. Harriss, 18 pp £1.00

4 *The utility of the Nigerian peasant farmer's knowledge in the monitoring of agricultural resources* by David Barker, Julius Oguntoyinbo and Paul Richards, 55 pp £1.00

5 *Monitoring tropical forests: a review with special reference to Africa* by Timothy J. Synnott, 45 pp (reprint)

6 *Radar design for determining the strength of tropical cyclones in the Bay of Bengal* by Harold W. Baynton, 38 pp £1.00

7 *Atmospheric pathways of sulphur compounds* by D. M. Whelpdale, 39 pp £1.00

8 *Environmental education in the United Kingdom Universities and Polytechnics: a compendium* by Kenneth Guy, Sally Turner and Lesley Williams, 66 pp £1.00

9 *Some methodological issues in the measurement, analysis and evaluation of peasant farmers' knowledge of their environment* by David Barker, 13 pp £1.00

10 *Air concentration and deposition rates from uniform area sources* by Lester Machta, 12 pp £1.00

11 *A handbook to estimate climatological concentration, deposition and horizontal fluxes of pollutants on a regional scale* by Lester Machta, 87 pp £5.00

12 *An introduction to the exposure commitment concept with reference to environmental mercury* by P. J. Barry, 93 pp £2.00

13 *The exposure commitment method with application to exposure of man to lead pollution by* B. J. O'Brien, 88 pp £2.00

14 *Atmospheric transport of mercury: exposure commitment and uncertainty calculations* by D. R. Miller and J. M. Buchanan, 75 pp £2.00

15 *Kinetic and exposure commitment analyses of lead behaviour in a biosphere reserve* by G. B. Wiersma, 41 pp £2.00

Progress reports in environmental monitoring and assessment 1. Lead (16, 17, 18) 1[illegible].00

16 *Lead pollution of the global environment* by B. J. O'Brien, S. Smith and D. O. Coleman

17 *Analysis of the effects of lead in tissue upon human health using dose-response relationships* by J. K. Piotrowski and B. J. O'Brien

18 *The establishment and interpretation of dose-effect relationships for heavy metal pollutants* by J. R. Whitehead

19 *The microcosm: biological model of the ecosystem* by Sidney Draggan, 45 pp £2.00

20 *Environmental hazards of heavy metals: summary evaluation of lead, cadmium and mercury* by J. K. Piotrowski and D. O. Coleman, 42 pp £2.00

21 *Lead in the soil environment* by D. H. Khan, 74 pp £2.00

22 *A preliminary evaluation of WMO-UNEP precipitation chemistry data* by C. C. Wallén, 19 pp £2.00

23 *Exposure commitment assessment of environmental pollutants*, Volume 1, Number 1, by B. G. Bennett, 59 pp £2.00

24 *Health effects of methylmercury* by J. K. Piotrowski and M. J. Inskip, 82 pp £2.00

MONITORING AND ASSESSMENT RESEARCH CENTRE
Chelsea College, University of London
The Octagon Building, 459A Fulham Road, London SW10 0QX

Price
U.K. £2.00
U.S.A. $4.00

ISBN 0 905918 22 3