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EXPOSURE COMMITMENT ASSESSMENTS OF ENVIRONMENTAL POLLUTANTS Volume 2

Technical Report

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TED NATIONS ENVIRONMENT PROGRAMME

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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.

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Exposure commitment assessments of environmental pollutants

Volume 2

Summary exposure assessments for PCBs, selenium, chromium

by B. G. Bennett

A Technical Report (1982)

Prepared by:
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With the support of:
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Abstract

This is the third in a series of reports presenting summary assessments of exposure of man to environmental pollutants. In this report, PCBs, selenium and chromium 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.

Volume 1, Number 2 (1981) Summary exposure assessments for mercury, nickel, tin.

CONTENTS

POLYCHLORINATED BIPHENYLS (PCBs)

1 Natural cycle

Polychlorinated biphenyls (PCBs) are a class of organic compounds produced by chlorination of biphenyl ($C_{12}H_{10}$). There are 10 possible forms – monochlorobiphenyl ($C_{12}H_9Cl$) through decachlorobiphenyl ($C_{12}Cl_{10}$) – but a large number of isomers are possible. The chlorine content ranges from 18 per cent ($C_{12}H_9Cl$) to 75 per cent ($C_{12}Cl_{10}$). Commercial PCB products consist of mixtures of several chlorinated forms.

PCBs are not produced in the environment, either from natural sources or from chemical transformation of other compounds (NAS 1979).

2 Anthropogenic sources

PCBs have been manufactured since 1929. Their primary use has been as coolant-dielectrics in transformers and capacitors, as heat transfer fluids, and as protective coatings for wood. Wider applications include their use in paints, inks and pesticides.

Sources of entry of PCBs into the environment include losses during manufacture and leakage from electrical equipment. Uses in other than closed systems can result in substantial losses to the environment. Mobilization of PCBs from landfills or incinerators are secondary routes of entry to the environment. PCBs are released from waste treatment plants in discharged water and sewage sludge.

There were no restrictions on the use or disposal of PCBs until evidence of human health and environmental hazards became available in 1969 – 70 (NAS 1979). The largest producer in the U.S.A. restricted sales to applications in closed electrical systems in 1971 and ceased production of PCBs in 1977. Other countries have adopted measures to reduce their use and avoid losses to the environment (WHO 1976).

With diminishing industrial use of PCBs, future sources of PCBs to the environment will be from disposal sites, losses from equipment still in use and atmospheric releases. Significant incidents of past exposure include

leakages from electrical equipment in food processing plants, resulting in contaminated rice oil (Japan 1968) and chicken feed (Puerto Rico 1977), and releases from manufacturing plants to rivers (New York 1942-75). Other than such inadvertent contamination or unintended releases, only atmospheric sources would be expected to cause other than local contamination.

PCBs are extremely stable compounds and are not oxidized, reduced or otherwise chemically transformed under environmental conditions. Some high temperature industrial applications could lead to formation of degraded products, such as chlorinated dibenzofurans (CDFs). Light as well as heat can accelerate the transformation of PCBs to CDFs. However, in most uses PCBs are not exposed to sunlight and consequently this decomposition process may be of only little significance. Microbial degradation occurs only on lower chlorinated biphenyls.

The major sink for environmental PCBs is in sediments of aquatic systems. The PCB residues are adsorbed to suspended solids and subsequently incorporated into the sediments. Some recycling may take place due to mechanical or biological processes.

3 Environmental considerations

3.1 Air

PCBs exist in air as a vapour with a smaller but uncertain fraction (around 10 per cent) associated with particulates (Doskey and Andren 1981). Atmospheric PCB concentrations for oceanic and rural continental areas are of the order of 0.05 ng m^{-3} (range 0.002 to 1.6 ng m^{-3}) and for urban areas of 5 ng m^{-3} (range 0.5 to 36 ng m^{-3}) (NAS 1979). A decreasing trend in concentrations in air should eventually become apparent due to reduced use of PCBs, but long-term survey measurements do not exist.

Recent measurements indicate that indoor air in commercial, industrial and residential buildings contains PCBs at concentrations at least one order of magnitude higher than outdoors, due to use of fluorescent lighting, electrical devices and other products containing PCBs (MacLeod 1981). The higher levels of PCBs in urban air may be due to general leakage from buildings as well as from more specific industrial emissions.

Removal of PCBs from outdoor air is by wet and dry deposition. Dry deposition, in particular, is difficult to measure and therefore its significance is uncertain. Revaporization from soil and waters can return PCBs to the air.

3.2 Soil

PCBs are efficiently adsorbed on soils with high organic content and on clays. Volatilization loss may occur from soils of low organic content.

PCBs widely dispersed in air and deposited with rainfall should accumulate in surface soil; however, there is little information on average levels. The concentrations in agricultural soils are generally not detectable. In urban areas of the U.S.A. the average concentration in soil is 0.002 mg kg^{-1} (NAS 1979).

3.3 Plants

PCBs have been measured in plants, for example, 0.02 mg kg^{-1} (fresh wt) (range 0.002 – 0.04 mg kg^{-1}) in vegetation from remote areas of Germany (Klein and Weisgerber 1976), but there is little information on retention from atmospheric deposition, uptake from soil and translocation in the plant.

Significant levels of PCBs have been observed in vascular plants in estuarine waters. Algae exhibit particularly high accumulation capacity and can serve as indicators of contamination in the aquatic environment (Kalmaz and Kalmaz 1979).

3.4 Animals

PCBs are efficiently absorbed by organisms and are resistent to degradation within the body. There are thus very high values of bioaccumulation in tissues, particularly in fish and other aquatic organisms. For example, average concentrations of PCBs in water of 0.0082 μg kg^{-1} and in mature lake trout of 28 mg kg^{-1} in Lake Michigan in the U.S.A. correspond to a concentrations ratio of 3.4×10^6 (Metcalf 1977; US EPA 1972).

PCBs accumulate to higher levels in fish with greater amounts of fat tissue such as carp, catfish, lake trout and salmon. An initial tolerance guide-line of 5 mg kg^{-1} established in the U.S.A. has been exceeded in fish from industrial regions in waters known to have received large amounts of PCBs in wastes. An average concentration of PCBs in freshwater fish of the Great Lakes in the U.S.A. has been estimated to be 3 mg kg^{-1} with generally much lower levels elsewhere in the country (NAS 1979). A concentration of 0.2 mg kg^{-1} has been suggested as representative for marine fish of the North Atlantic, North American shelf region (NAS 1979).

PCBs accumulate in birds and other wildlife depending on dietary characteristics. Levels up to 900 mg kg^{-1} in liver of fish-eating birds have

been noted (Prestt, Jeffries and Moore 1970). A general, representative range of PCB concentrations in wildlife of 0.02 to 0.4 mg kg^{-1} has been suggested (NAS 1979).

3.5 Diet

Surveys of PCBs in foods in Sweden, Canada, the U.S.A. and Japan indicate concentrations generally less than 0.1 mg kg^{-1} (WHO 1976). Fish from contaminated areas contain higher levels, up to 35 mg kg^{-1} but generally less than 3 mg kg^{-1}. High levels have been noted in packaged foods (2 – 11 mg kg^{-1}) due to transfer from packaging materials produced from recycled paper containing carbonless copy paper (WHO 1976). This source should be diminishing as PCB containing paper disappears from use.

Dietary intake of PCBs has been estimated to range from 5 to 100 $\mu g\ d^{-1}$ (WHO 1976). As indicated in Table 1, intake is estimated to be 24 $\mu g\ d^{-1}$ from representative values of PCB concentrations (NAS 1979) and consumption (ICRP 1975). Fish is an important factor in this estimate, contributing 15 $\mu g\ d^{-1}$, but this could be quite variable, depending particularly on the source and amounts of freshwater fish consumed. The estimated intake from other components of diet, about 9 $\mu g\ d^{-1}$, could also be quite variable. Estimated intake of PCBs in meals not containing fish has been reported to be 6, 41 and 84 $\mu g\ d^{-1}$ in Switzerland (Zimmerli and Marek 1973). Fish contribute an average 80 per cent to dietary intake of PCBs in Japan with estimated total intake ranging from 4 to 50 $\mu g\ d^{-1}$ (Tatsukawa 1976). The estimated total dietary intake of 24 $\mu g\ d^{-1}$ (Table 1), which applies to the period when most estimates were made (early to mid 1970s) reflects a balance of widely varying intakes of foods and PCB concentrations.

Table 1 Estimated dietary intake of PCBs

Diet source	Concentration ($\mu g\ kg^{-1}$)	Consumption ($kg\ y^{-1}$)	Intake ($mg\ y^{-1}$)
Plants	9	270	2.4
Livestock	14	55	0.8
Freshwater fish	2,000	2	4.0
Marine fish	200	7	1.4
Drinking water	0.0015	440	0.0007
		Total	8.6

3.6 Water

PCBs in the aquatic environment originate from precipitation, land run-off, industrial and municipal waste discharges. PCBs, particularly the more highly chlorinated forms, have low solubility in water but are strongly adsorbed to sediments. Concentrations of PCBs in freshwaters are low, even if sediments contain appreciable amounts. A general range of PCBs in freshwaters in wider geographical regions is $1-3$ ng ℓ^{-1} (NAS 1979).

PCBs in sediments of freshwater lakes range from 2 to 20 μg kg^{-1} for those receiving atmospheric pollution only and 4 to 320 μg kg^{-1} for lakes also receiving industrial and municipal wastes (NAS 1979). PCBs in sediments of rivers have been 1 to 160 μg kg^{-1} in the U.S.A. during $1971-74$, with much higher levels in some rivers, such as the Hudson River, near industrial outfalls (NAS 1979, Bopp, Simpson, Olsen and Kostyk 1981).

PCB concentrations in the ocean are $2-3$ ng ℓ^{-1} in the North Atlantic and less than one elsewhere (NAS 1979). Levels in the Mediterranean Sea averaged 2 ng ℓ^{-1}, an order of magnitude less than in some tributaries, such as the Po and Adige Rivers in Italy (Galassi and Provini 1981). It has been suggested that measurements of PCBs in the ocean have been biased upward by sampling contamination, and that reported measurements may be too high by at least one order of magnitude (Risebrough, de Lappe and Walker 1976, NAS 1979).

4 Metabolism

4.1 Absorption

PCBs are easily absorbed through ingestion, inhalation or dermal contact. Less chlorinated forms can be metabolized and excreted but more highly chlorinated forms accumulate in body tissues. PCBs cross the placental barrier and are transferred to milk.

4.2 Distribution in the body

PCBs accumulate in adipose tissue. Concentrations in human populations are reported to range from 0.9 to 1.3 mg kg^{-1} in New Zealand, Norway, Canada, U.S.A. and the U.K., 3.6 to 5.0 mg kg^{-1} in Israel, Austria, Japan and Denmark and 6.4 to 7.9 mg kg^{-1} in East and West Germany, as summarized from various reports published during 1974-77 (NAS 1979). Assuming lipids constitute 30 per cent of body weight, an average PCB

concentration of 0.35 mg kg^{-1} body weight has been suggested as representative (NAS 1979). The average content of PCBs in human milk has been reported to be 0.03 mg kg^{-1} (0.9 mg kg^{-1} fat) in Sweden during 1976-77 with no decreasing trend since 1967 (Westöö and Norén 1978).

4.3 Retention time

Evidence of accumulation of PCBs in tissues suggests long retention times, particularly for the more highly chlorinated forms. For the unspecified composition of PCBs in the environment, a mean effective retention time in the body may be estimated from the body content at steady state divided by the intake rate, assuming complete absorption and initial retention. From representative values of these parameters, as discussed above, a body content of 25 mg (0.35 mg kg^{-1} × 70 kg) and dietary intake rate of 8.6 mg y^{-1}, the mean residence time of PCBs in the body is estimated to be about three years.

5 Effects

Acute effects of PCBs are minimal except in some aquatic invertebrates. Low-level exposure of humans to PCBs may cause such non-specific effects as fatigue, abdominal pain, numbness of limbs, swelling of joints and headache (NAS 1979). Additional attributable effects include blood lipid abnormalities, dermatological disorders and cancer. Effects in primates following chronic exposure include early abortions, low birth weights, loss of immunological competence in infants and learning and behavioural deficiencies.

Some effects associated with PCBs may also be due to highly toxic polychlorinated dibenzofurans, which are a trace constituent in PCB mixtures and may also result from metabolic transformations. They may be formed in the environment from photochemical reactions and have been identified in environmental samples (Rappe *et al.* 1981).

6 Literature critique

There is a large amount of results of PCB monitoring studies in the literature, but much less in the way of descriptions of the dynamic behaviour of these compounds. Some difficulties remain in sampling and analytical techniques. Reviews of PCB behaviour and toxicity include NIEHS (1972), Peakall and Risebrough (1975), WHO (1976), USEPA (1976), Roberts, Rogers, Bailey and Rorke (1978), IARC (1978), Kalmaz and Kalmaz (1979)

and NAS (1979).

7 Pathway analysis

PCBs become most widely dispersed in the environment through atmospheric transport and, on a more local or regional scale, following release to water. There is also mobilization of PCBs in soil or landfills but the rates of dispersion and subsequent transfer to biota and man are very difficult to generalize.

A broadly representative pathway analysis is given below, starting from unit exposure commitment to air or water. The exposure commitment of an environmental compartment is the time integral of the concentration in the compartment following a specified release of the pollutant from a source. The time independent exposure commitment method of pollutant assessment has been discussed previously (Bennett 1981).

Transfer factors describe the relationships between exposure or intake commitments in successive compartments (Bennett 1981). The intake commitment is the time integral of the flux into a compartment and corresponds to the cumulative amount of pollutant transferred along a particular pathway. The notation used for the transfer factor is P_{ij} for transfer from compartment i to compartment j. Primed subscripts indicate that intake commitments or steady-state fluxes are involved in the relationship and unprimed subscripts refer to exposure commitments or steady-state concentrations (Bennett 1981).

A summary of representative levels of PCBs in the environment is presented in Table 2. These are broad averages for unspecified mixtures of PCBs. It is to be expected that more highly chlorinated forms become most prevalent in compartments further along the pathway chains. More recent measurements of environmental concentrations should be indicating lower values reflecting reduced production and use of PCBs.

The compartmental arrangements for the pathway analysis and evaluation of the transfer factors are given in Figure 1. The initial transfer factors in the chains – from air to plants to livestock and from water to fish – are ratios of equilibrium concentrations with values taken from Table 2. The ratios of concentrations are equivalent to ratios of exposure commitments with time added to the numerator and denominator of the units in the latter expressions (Bennett 1981).

Because of the uncertainty of PCB concentrations in the ocean, the transfer factor from water to fish has been taken to be the same as in

Table 2 PCBs in the environment: Summary of representative values*

Concentrations			
Atmosphere			
urban		5 ng m^{-3}	(0.5 – 40)§
rural		0.05 ng m^{-3}	(0.002 – 2)
Lithosphere			
soil		~0.2 ng kg^{-1}	(0.02 – 2000)
Hydrosphere			
freshwater		2 ng ℓ^{-1}	(<1 – 30)
ocean		~0.2 ng ℓ^{-1}	(<0.2 – 9)
Biosphere			
plants		9 μg kg^{-1}	(2 – 40)
wildlife		~90μg kg^{-1}	(20 – 400)
livestock		14 μg kg^{-1}	(2 – 100)
freshwater fish		2000 μg kg^{-1}	(20 – 50000)
marine fish		200 μg kg^{-1}	
man		350 μg kg^{-1}	(300 – 2000)
Transfer Rates			
Intake			
ingestion	– terrestrial	3 mg y^{-1}	
	– aquatic	5 mg y^{-1}	
inhalation	– urban	0.04 mg y^{-1}	
	– rural	0.0004 mg y^{-1}	
Absorption			
GI tract		1.0	
lungs		0.5	

* Reference (NAS 1979)

§ Range of values in parentheses

freshwater, i.e. a concentration ratio of 10^6. If the representative concentration of PCBs in marine fish is taken to be 0.2 mg kg^{-1}, one would then infer an average concentration in ocean water of 0.2 ng ℓ^{-1}.

The transfer factor relating to concentration of PCBs in the food item to the dietary intake rate (or equivalently the exposure commitment in food to the intake commitment) corresponds to the consumption rate. Consumption rates have been taken to be 55 kg y^{-1} (meat), 270 kg y^{-1} (plant foods), 2 kg y^{-1} (freshwater fish) and 7 kg y^{-1} (marine fish and shellfish). It is recognized that consumption rates are different in various geographical regions (ICRP 1975).

The transfer factor relating the concentrations of PCBs in the body to the dietary intake rate ($P_{4,5}$) is given by the ratio of the mean residence time of

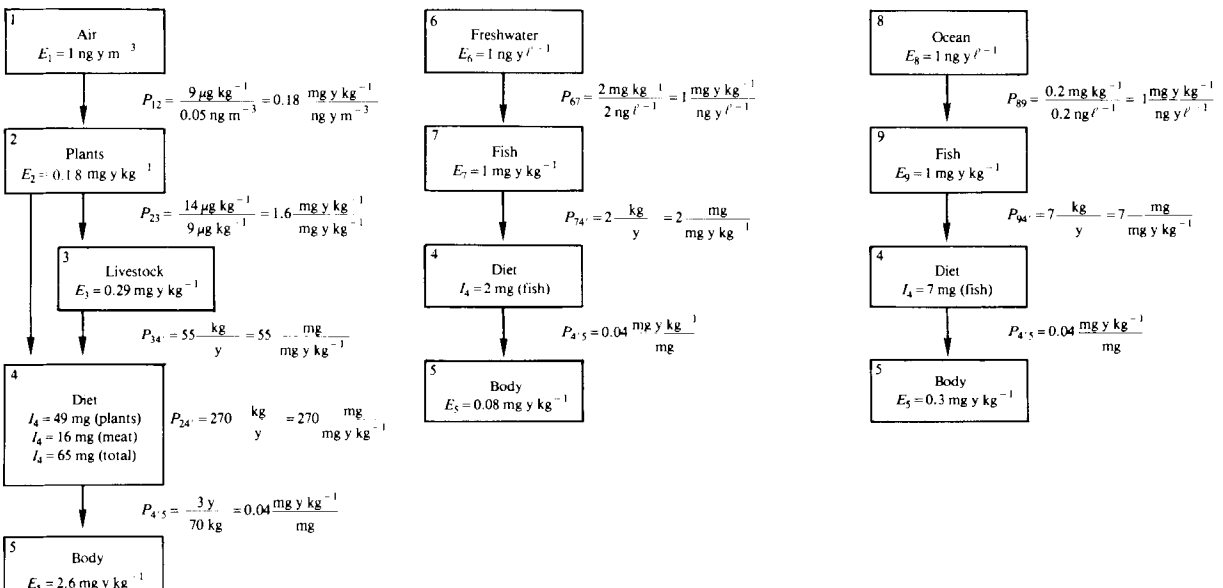


Figure 1 Pathway evaluations for PCBs

PCBs in the body (3 y) and the body mass (70 kg). This is a frequently used relationship of the exposure commitment method (Bennett 1981). It is assumed that PCBs are completely absorbed from diet. Concentrations are expressed in terms of body weight, although the distribution of PCBs is primarily to adipose tissue. Assuming adipose tissue to be 30 per cent of body weight (NAS 1979), concentrations in adipose tissue are higher by a factory of 3.3.

The exposure commitment to the receptor compartment is determined by the sequential product of transfer factors and the exposure commitment of the initial compartment. Thus, for the terrestrial ingestion pathway the exposure commitment to the body of man (E_5) from unit exposure commitment to air (E_1 = 1 ng y m^{-3}) is

$$E_5 = P_{12}\,(P_{23}\,P_{34} + P_{24})\,P_{45}\,E_1 = 2.6 \text{ mg y kg}^{-1}$$

The contributions from the two parallel pathways, ingestion of plants and ingestion of meat, are added. At equilibrium, the relationship is 1 ng m^{-3} in air associated with 2.6 mg kg^{-1} in the body. For the aquatic pathways the equilibrium relationships are 0.08 mg kg^{-1} in the body (freshwater fish pathway) and 0.3 mg kg^{-1} in the body (marine fish pathway) from 1 ng ℓ^{-1} of PCBs in water.

The relationships between current levels of PCBs in the environment and in man, assuming equilibrium in recent years, are shown in Table 3. The greatest contribution to dietary intake and concentrations in the body is the ingestion of freshwater fish. Less by over a factor of two, is the ingestion of marine fish. The total concentration of PCBs in the body is estimated to be 0.35 mg kg^{-1}, corresponding to a body burden of 25 mg. This is assumed to apply to representative, non-specific background exposures and excludes individuals who at one time or another may have had access to more contaminated foods.

Secondary pathways may also contribute to the concentrations of PCBs in the body. Inhalation of air, assuming 5 ng m^{-3} of PCBs in urban air, a breathing rate of 22 m^3 d^{-1}, retention and absorption of inhaled particles/vapour of 50 per cent, and a mean residence time of PCBs in the body of 3 y, contributes 0.8 μg kg^{-1} to the concentration in the body. Intake of PCBs in drinking water, assuming 2 ng ℓ^{-1} in water, consumption of 1.2 ℓ d^{-1} and complete absorption, contributes 0.04 μg kg^{-1} to the PCB concentration in the body. Both of these pathways are negligible compared with the above considered pathways.

Table 3 Current levels of PCBs in the background environment and in man*

	Air	Water	Fish	Diet	Man
Terrestrial pathway					
	$0.05\ \frac{ng}{m^3}$	———	———	$3.2\ \frac{mg}{y}$ ⟶	$0.13\ \frac{mg}{kg}$
Aquatic pathways					
Freshwater		$2\ \frac{ng}{\ell}$ ⟶	$2\ \frac{mg}{kg}$ ⟶	$4\ \frac{mg}{y}$ ⟶	$0.16\ \frac{mg}{kg}$
Marine		$0.2\ \frac{ng}{\ell}$ ⟶	$0.2\ \frac{mg}{kg}$ ⟶	$1.4\ \frac{mg}{y}$ ⟶	$0.06\ \frac{mg}{kg}$
				Total§	$0.35\ \frac{mg}{kg}$

* Secondary pathways:
inhalation (5 ng m^{-3} in urban air) → 0.8 μg kg^{-1} (man)
drinking water (2 ng ℓ^{-1}) → 0.04 μg kg^{-1} (man)

§Approximate effects level:

$$0.07\ \frac{mg\ d^{-1}}{kg\ body\ wt} \times 70\ kg \times 365\ \frac{d}{y} \times 0.04\ \frac{mg\ y\ kg^{-1}}{mg} = 70\ \frac{mg}{kg}$$

Effects of PCBs in man seem to occur at intake rates of about 0.07 mg d^{-1} kg^{-1} body weight (WHO 1976). This corresponds to 4.9 mg d^{-1} or 1.8 g y^{-1} for the 70 kg adult. Applying the transfer factor $P_{4'5}$, corresponding to the mean retention time of 3 y, the equilibrium concentration in the body would be 70 mg kg^{-1}. This effects level concentration is 200 times greater than the current estimated background concentration for most individuals.

This analysis provides a framework for analysis of the behaviour of PCBs in the environment and their transfer to man. Estimates of the transfer factors obtained from representative background levels should be generally relevant and may be applied to more specific cases of exposure. The estimated concentrations of PCBs in man assume continued exposure at levels reported in the recent past. Evidence may yet be acquired indicating lower concentrations of PCBs in the environment and in man, reflecting reduced use and release to the environment of these compounds.

References

Bennett, B.G. 1981 Exposure commitment concepts and application. In: *Exposure Commitment Assessments of Environmental Pollutants.* Vol. 1, No. 1, MARC Report 23, Monitoring and Assessment Research Centre, Chelsea College, University of London, 1 – 17.

Bopp, R.F., Simpson H.J., Olsen, C.R., and Kostyk, N. 1981 Polychlorinated biphenyls in sediments of the tidal Hudson River, New York. *Environ. Sci. Technol.* **15,** 210 – 216.

Doskey, P.V. and Andren, A.W. 1981. Modeling the flux of atmospheric polychlorinated biphenyls across the air/water interface. *Environ Sci. Technol.* **15,** 705 – 711.

Galassi, S, and Provini, A. 1981 Chlorinated pesticides and PCBs contents of the two main tributaries into the Adriatic Sea. *Sci Total Environ.* **17,** 51 – 57.

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

International Agency for Research on Cancer 1978 Polychlorinated Biphenyls. In: *IARC Monographs on the Evaluation of the Carinogenic Risk of Chemicals to Humans, Vol.* **18,** Lyon, 43 – 103.

Kalmaz, E.V. and Kalmaz, G.D. 1979 Transport, distribution and toxic effects of polychlorinated biphenyls in ecosystems: review, *Ecol. Model.* **6,** 223 – 251.

Klein, W. and Weisgerber, I. 1976 PCBs and environmental contamination *Environ. Qual. Safety* **5,** 237.

MacLeod, K.E. 1981 Polychlorinated biphenyls in indoor air. *Environ. Sci. Technol.* **15,** 926 – 928.

Metcalf, R.L. 1977 Biological fate and transformation of pollutants in water. In: *Fate of Pollutants in the Air and Water Environments, Part II* I. H. Suffet (Ed.), John Wiley & Sons Inc., New York, 195 – 221.

National Academy of Sciences, 1979 *Polychlorinated Biphenyls,* National Research Council, NAS, Washington DC..

National Institute of Environmental Health Sciences, 1972 Conference on PCBs. *Environ Health Perspect.* **1,** 1 – 85.

Peakall, D.B. and Risebrough, R.W. 1975 PCBs and their environmental effects. *CRC Crit. Rev. Environ. Control* **5,** 469 – 508.

Prestt, I., Jeffries D.J. and Moore, N.W. 1970 Polychlorinated biphenyls in wild birds in Britain and their avian toxicity, *Environ. Pollut.* **1,** 3 – 26.

Rappe, C., Buser H.R., Stalling, D.L., Smith, L.M. and Dougherty, R.C. 1981 Identification of polychlorinated dibenzofurans in environmental samples *Nature.* **292,** 524 – 526.

Risebrough, R.W., de Lappe, B.W., Walker, W. II. 1976 Transfer of higher molecular weight chlorinated hydrocarbons to the marine environment. In: *Marine Pollutant Transfer,* H.L. Windom, R.A. Duce (Eds), D.C. Heath and Co., Lexington, Massachusetts, 261 – 321.

Roberts, J.R., Rodgers, D.W., Bailey, J.R. and Rorke, M.A. 1978 *Polychlorinated biphenyls: Biological Criteria for an Assessment of their Effects on Environmental Quality.* National Research Council Canada, Report NRCC 16077, Ottawa.

Taksukawa, R. 1976 PCB pollution of the Japanese environment. In: P.C.B. Poisoning and Pollution, K. Higuchi (Ed), Kodansha Ltd, Tokyo, 147-179.

U.S. Environmental Protection Agency 1976 *National Conference on Polychlorinated Biphenyls.* USEPA Report EPA – 560/6 – 75 – 004, Washington, DC.

U.S. Environmental Protection Agency 1972 An evaluation of DDT and dieldrin in Lake Michigan. Ecol. Res. Serv. Report No. EPA R3 – 72 – 003, Washington, D.C.

Westöö, G. and Norén, K. 1978 Organochlorine contaminants in human milk, Stockholm 1967-1977, *Ambio* **7**, 62 – 64.

World Health Organization 1976 *Polychlorinated Biphenyls and terphenyls*. Environmental Health Criteria 2, Geneva.

Zimmerli, B. and Marek, B. 1973 *Die Belastung der Schweizerischen Bevölkerung mit Pestiziden*. Mitt. Lebensm. Unters. Hyg. **64**, 459 – 479.

SELENIUM

1 Natural cycle

Selenium, the element of atomic number 34, is widely distributed in trace amounts in the environment. Its average concentration in the earth's crust is 0.05 $\mu g\ g^{-1}$ (Taylor 1964). Much of its occurrence is associated with sulphide minerals. Higher concentrations of selenium are found in some sedimentary rocks leading to regionally higher concentrations in soil. Atmospheric selenium arises from volcanic emissions, windblown dusts, and volatile release from plants, animals and soils. Volatile organic selenium compounds are formed by micro-organisms in soils and sediments.

Selenium belongs to the VIa group of elements of the periodic table, including oxygen and sulphur, which are lighter, and tellurium and polonium, which are heavier. Selenium in its chemical behaviour is essentially non-metallic. Elemental selenium is not soluble in water but many of its salts (selenites and selenates) are soluble. Elemental selenium is easily oxidized.

There are wide variations in uptake by plants of selenium in soil. It has been shown to be an essential element for animals (Schwartz and Foltz 1957); diseases have been reported in domestic animals and also in man as a result of selenium deficiency, but it is toxic at higher levels of intake. Methylated selenium compounds result from biological transformation of selenium and have low toxicity.

2 Anthropogenic sources

Selenium is used in the electrical industry, particularly in photoelectric cells and also in semiconductors and rectifiers. Other uses are in the glass industry, pigment manufacture, chemical and rubber industries, stainless steel production, lubricants, fungicides and feed additives.

Most commercial selenium is obtained as a recovered by-product in the refining of sulphide ores of other metals, such as copper. Selenium is present in coal at concentrations ranging from 0.1 to 4 $\mu g\ g^{-1}$ (Shamberger 1981).

Emissions of selenium to air from anthropogenic sources result primarily from fossil fuel combustion, estimated to be about 62 per cent of the total emissions, and secondarily from non-ferrous metal smelting and refining

(26 per cent), glass and ceramics manufacturing (5 per cent) and fuel oil combustion (5 per cent) (NAS 1976).

Selenium may be found in waste water streams of ore refineries, coal fired power plants and industrial plants in which selenium is utilized.

3 Environmental considerations

3.1 Air

Selenium concentrations in air are of the order of a few ng m^{-3}, the levels depending particularly on coal burning power plants in the vicinity. The average concentration of selenium in air at a semi-rural site in England during 1957-74 (Figure 1) was 1.3 ng m^{-3} (1.6 in winter and 0.9 in summer) (Salmon *et al.* 1978). There was a decline in levels due to clean air legislation followed by an only slightly decreasing trend since 1960. The overall trend matches that of black smoke, and it is likely that most of the selenium in air at this site originates from fuel burning rather than industrial processes. It has been pointed out that the average ratio of selenium to sulphur in air is 10^{-4}, the same ratio as in fossil fuels (Hashimoto, Hwang and Yanagisawa 1970).

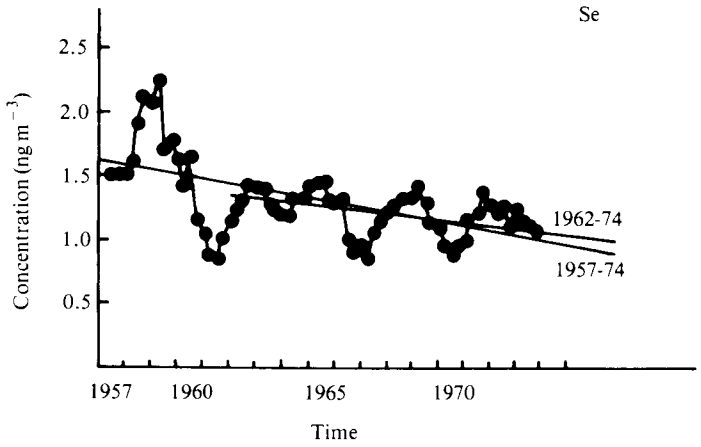


Figure 1 Selenium in air at Chilton, Oxfordshire, U.K. (Salmon *et al.* 1978)

The good correlation between selenium and sulphur in air determined by Priest, Navarre and Ronneau (1981) allowed an extrapolated minimum background level (at the zero level for sulphur) of selenium in rural European air of 0.17 ng m^{-3} to be estimated. The residual selenium level may be attributed to sources not associated with sulphur, such as volatile releases from plants and micro-organisms. At a more remote European site (Jungfraujoch, Switzerland) the background selenium concentration in air was reported to be 0.013 ng m^{-3} (Adams, Van Craen and Van Espen 1980).

Selenium in air in urban areas has been measured. The mean result for Glasgow during a 13-month period in 1972-73 was 3.3 ng m^{-3} (McDonald and Duncan 1979). The range for two-day determinations was large: 0.01 to 27.7 ng m^{-3}. Measurement results for several urban sites in the U.S.A. are generally similar (NAS 1976).

3.2 Soil

Selenium concentrations in soil are commonly 1 to 10 $\mu g\ g^{-1}$, but are extremely variable, ranging from 0.1 $\mu g\ g^{-1}$ in selenium-deficient areas, such as New Zealand, to 1,200 $\mu g\ g^{-1}$ in seleniferous areas in Ireland (Glover, Levander, Parizek and Vouk 1979). A continental-wide survey of selenium in soil in the United States (912 samples) gave a range of 0.1 to 4.3 $\mu g\ g^{-1}$ and a geometric mean of 0.3 $\mu g\ g^{-1}$ (Shacklette, Boerngen and Keith 1974). Based on several reviews, Berrow and Burridge (1980) suggest a normal range of selenium in cultivated surface soils of 0.1 to 2 $\mu g\ g^{-1}$ with a typical level of 0.5 $\mu g\ g^{-1}$.

Selenium is bound in acid soils as ferric selenite of very low solubility. Soils rich in iron contribute to selenium binding and reduce its availability to vegetation. In alkaline soils, more selenium is present in the soluble selenate form available to plants.

3.3 Plants

There is a wide range of selenium uptake ability by plants (Rosenfeld and Beath 1964). The tolerance or toxicity to selenium is quite variable (Brown and Shrift 1982). Some plants grow only in soils of high selenium content and accumulate concentrations of up to a few thousand $\mu g\ g^{-1}$. These are generally non-food plants, but occasionally unusually high concentrations of selenium are reported in some legumes, nuts and mushrooms (Vokal-Borek 1979). Secondary selenium absorbing plants contain up to 50 – 500 $\mu g\ g^{-1}$. Most crop plants, grains and grasses rarely contain more than 30 $\mu g\ g^{-1}$,

the general range being 0.05 to 1 $\mu g\ g^{-1}$ (Vokal-Borek 1979). Most of the selenium in these plants is associated with plant protein (NAS 1976).

In general, soils of higher selenium content produce plants of higher selenium content, with 10 to 40 per cent of the concentration in soil to be expected in the plant (Koljonen 1978). However, because of the importance of the form of selenium in soil and the species differences, such relationships can be only loosely applied.

3.4 Diet

Most dietary intake of selenium occurs via plant foods, the main exception being fish and certain seafoods. There is considerable variability in levels in plants due to soil conditions, whereas more consistent levels are contained in animal tissues to meet the nutritional needs of the animal. The ranges of selenium concentrations in foods sampled in the U.S.A. were 0.03 to 0.6 $\mu g\ g^{-1}$ in grain products, 0.004 to 0.04 in most vegetables and fruits, 0.01 to 0.07 in milk, cheese and egg white, 0.2 in egg yolk, 0.1 to 0.3 in meats, 0.4 in beef liver and marine fish, and 0.6 in seafoods (wet wt basis) (Morris and Levander 1970). Additional values for milk, eggs and meat from Norway and a review for other areas are given by Karlsen, Norheim and Frøslie (1981).

The chemical form of selenium in foods has not been well established. There are considerable losses of volatile selenium compounds in foods during cooking (Ganapathy, Joyner, Sawyer and Häfner 1978). It has been noted that the availability of selenium in foods following ingestion is higher for those of plant origin than for those of animal origin (Cantor, Scott and Noguchi 1975). Selenium in fish, for example, which is formed in stable complexes with metals, notably mercury, is much less available. A protective effect of selenium against mercury toxicity has been noted in this circumstance, but the biochemical basis for such an interrelationship is not clear.

Estimates of average daily intake of selenium in diet are 6 – 70 $\mu g\ d^{-1}$ in New Zealand (Robinson 1976), 7 – 50 $\mu g\ d^{-1}$ in Italy (Cigna Rossi, Clemente and Santaroni 1976) 30 – 60 in Finland (Varo and Koivistoinen 1981), 60 $\mu g\ d^{-1}$ in the U.K. (Thorn, Robertson and Buss 1978), 60 – 220 $\mu g\ d^{-1}$ in the U.S.A. (Schroeder, Frost and Balassa 1970, Welsh, Holden, Wolf and Levander 1981), 100 – 220 $\mu g\ d^{-1}$ in Canada (Thompson, Erdody and Smith 1975), 90 – 210 $\mu g\ d^{-1}$ in Japan (Sakurai and Tsuchiya 1975), and 220 $\mu g\ d^{-1}$ in Venezuela (Mondragon and Jaffe 1976). The minimum dietary

requirement has been variously and tentatively estimated to be 20 to 120 μg d^{-1} (Stewart, Griffiths, Thompson and Robinson 1978, NAS 1976) and a minimal toxic selenium intake amount may be 500 μg d^{-1} (Sakurai and Tsuchiya 1975).

An example of the distribution of daily selenium intake determinations for a particular area (eastern U.S.A.) is shown in Figure 2. The results, for 132 daily diet composites self-selected by 22 individuals, are slightly skewed by a few relatively high values, but 80 per cent of the data were within 50 to 200 μg d^{-1}. The median intake was 74 μg d^{-1}. The concentrations of selenium in the composite samples were 0.04 ± 0.02 μg g^{-1} (wet wt) and 0.24 ± 0.13 μg g^{-1} (dry wt) (Welsh *et al.* 1981).

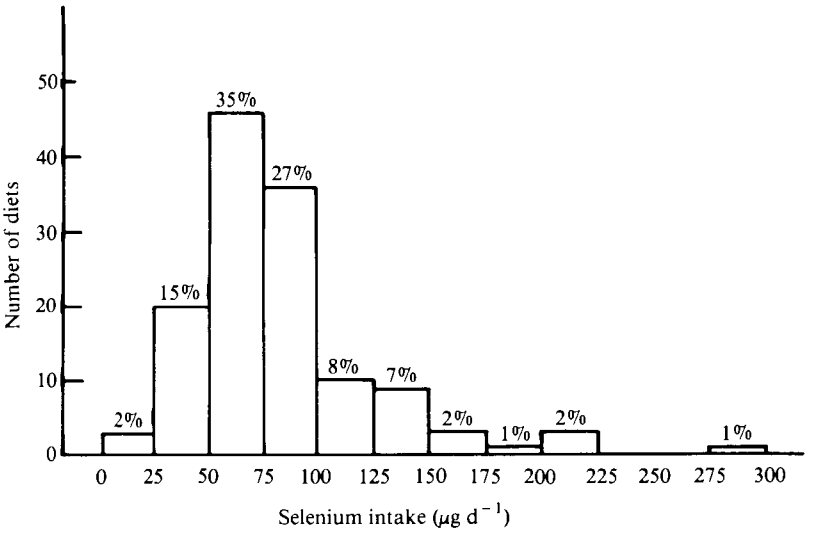


Figure 2 Distribution of selenium in 132 daily diet composites from the eastern U.S.A. (Welsh *et al.* 1981)

3.5 Water

Relatively low levels of selenium are found in most surface and ground waters, generally less than 10 μg ℓ^{-1}. In the presence of iron, selenium precipitates as insoluble ferric selenite at pH of the water less than 7. At

higher pH, about 8, the selenites may be oxidized to soluble selenates thereby increasing the concentrations in water up to 10 - 400 $\mu g\ \ell^{-1}$ (Glover *et al.* 1979).

In a review of recent surveys of selenium in freshwater, Bowen (1979) gives a median concentration of 0.2 $\mu g\ \ell^{-1}$ with a range of 0.02 to 1 $\mu g\ \ell^{-1}$. Irrigation drainage from seleniferous soils may result in water concentrations up to 400 $\mu g\ \ell^{-1}$ and in some cases over 2,000 $\mu g\ \ell^{-1}$ (NAS 1976). Precipitation of selenium with metal hydroxides usually greatly reduces the levels in larger rivers and lakes. A representative concentration of selenium in sea water is 0.2 $\mu g\ \ell^{-1}$ (range 0.05 – 0.2) (Bowen 1979).

4 Metabolism

4.1 Absorption

There is little quantitative information on the absorption of selenium compounds through the lungs or skin. Ingested selenium is readily absorbed, though with some dependence on chemical form. Organic selenium compounds, such as selenomethionine, may be incorporated directly into tissue proteins and accumulate to high levels (Vokal-Borek 1979, Scott 1973). Absorption of inorganic selenite may be limited by available selenium binding sites in tissues, although biotransformation to organic forms occurs which are then incorporated into tissue proteins (Glover *et al.* 1979). In general, absorption of selenites, selenates and organic selenium compounds is efficient. Metal selenides and elemental selenium are poorly absorbed (Vokal-Borek 1979). It is estimated that about 80 per cent of dietary selenium is absorbed (Stewart *et al.* 1978).

4.2 Distribution in the body

Absorbed selenium is widely distributed by blood to organs and tissues, the largest amounts occurring in the liver and kidneys. In a study of selenium in human tissues from Japan, somewhat higher concentrations were found in lungs with about equal concentrations in kidneys, spleen, pancreas, muscle and brain (Yukawa, Suzuki-Yasumoto, Amano and Terai 1980). The concentration ranges were of the order 0.5 to 5 $\mu g\ g^{-1}$ (wet wt). In another study from Sweden the results were 0.7 $\mu g\ g^{-1}$ (0.4 to 1.0 $\mu g\ g^{-1}$) in kidneys, 0.2 $\mu g\ g^{-1}$ (0.1 to 0.5 $\mu g\ g^{-1}$) in liver, 0.1 $\mu g\ g^{-1}$ (0.04 to 0.2 $\mu g\ g^{-1}$) in lungs, and 0.06 $\mu g\ g^{-1}$ (0.06 to 0.1 $\mu g\ g^{-1}$) in bone of non-occupationally exposed individuals (Brune, Nordberg and Wester 1980, Lindh, Brune, Nordberg and Wester 1980). Regional differences are expected, reflecting variable

dietary selenium intake. This has been shown for selenium in blood, the mean result being 0.21 $\mu g\ m\ell^{-1}$ from various locations throughout the U.S.A. (Allaway, Kubota, Losee and Roth 1968). A nearly normal distribution of selenium in blood was found for 626 samples from South Dakota in the U.S.A. which includes seleniferous areas, with a mean and standard deviation of $0.26 \pm 0.06\ \mu g\ m\ell^{-1}$ (Howe 1979). Whole body selenium content is estimated to be approximately 3 to 6 mg from studies performed in New Zealand (Stewart *et al.* 1978) and 13 mg from a general review of reported results (ICRP 1975).

4.3 Retention times

Selenium is excreted primarily in urine in man and monogastric animals. In ruminants, microbial reduction of selenium compounds to insoluble forms occurs following ingestion which reduces absorption and makes faeces the major route of excretion. Excretion via the lungs occurs at higher levels of selenium intake. The exhaled volatile compound, dimethylselenide, is an intermediate product of selenium metabolism which is exhaled only when its rate of formation exceeds the rate of further methylation to trimethylselenonium ion, a urinary selenium metabolite (Glover *et al.* 1979).

The rate of excretion is described by several components, a rapid first phase which may be dose dependent, an intermediate phase, and a longer phase representing slower whole-body turnover of selenium. The half-time of the third phase corresponded to 27 days in the rat, increasing to 70 days with a selenium deficient diet (Ewan, Pope and Baumann 1967). The long-term phase in man, determined from studies in New Zealand, where dietary intake of selenium is very low, was 103 days for sodium selenite and 234 days for selenomethionine (Griffiths, Stewart and Robinson 1976). For general retention estimates, suggested retention half-times for man are 3, 30 and 150 days with component fractions of 0.1, 0.4 and 0.5 respectively (ICRP 1980).

5 Effects

Effects of selenium may result from either deficient or excessive amounts. Deficiency diseases have been noted mostly in domestic animals, including muscle degeneration, liver damage, slow growth and reduced fertility. Some of these effects can be prevented by selenium supplements to the diet. Deficiency effects may also occur in man. Recently, selenium has been associated with Keshan disease, a cardiomyopathy that effects children in certain areas of China. Dietary supplementation with selenium has successfully been used in preventing this disease. (Chinese Academy of

Medical Sciences 1979). Other epidemiological studies have shown associations between selenium intake and reduced mortality rates from heart disease (Shamberger 1981).

Selenium toxicity has been observed in animals from consumption of plants containing high levels of selenium. Acute poisonings most often involve vascular damage and haemorrhage in various tissues and organs. Chronic poisonings, which may be of the blind staggers type or a degenerative alkali disease, follow ingestion of feed at levels greater than 5 $\mu g\ g^{-1}$ (NAS 1976).

Adverse effects of selenium in man occurring in areas of high dietary selenium intake include pathological changes of nails, gastro-intestinal disorders, skin discoloration and tooth decay, but other possible causes of these conditions cannot be excluded (Glover *et al.* 1979). Studies of the possible carcinogenicity of selenium or of its protective effects against cancer have not been conclusive (IARC 1975).

6 Literature critique

There is a wide literature on selenium toxicity and deficiencies in health of animals and man and on the numerous interactions of selenium and other substances such as sulphates, phosphates, mercury, cadmium, arsenic and other metals, which mutually affect their distribution in the body, retention and toxicity. The mechanisms of interactions are complex and not yet fully understood. Recent reviews of such studies include Diplock (1976), Vokal-Borek (1979) and Brown and Shrift (1982). Reviews of selenium in soils and plants include Bisbjerg (1972), Lewis (1976), and Peterson, Benson and Zieve (1981). General reviews of selenium in the environment and biological effects include NAS (1976), Glover *et al.* (1979), Vokal-Borek (1979) and Shamberger (1981). A review of analytical methods was given by Shendrikar (1974).

7 Pathway analysis

The transfer of selenium from general environmental sources to man occurs via the inhalation and ingestion pathways. The pathway analysis, as presented below, utilizes the exposure commitment method (Bennett 1981). The exposure commitment is the time integral of the concentration in an environmental medium. The intake commitment is the time integral of the flux of a substance into a compartment. The concepts, notation and procedures of the method have been presented previously (Bennett 1981).

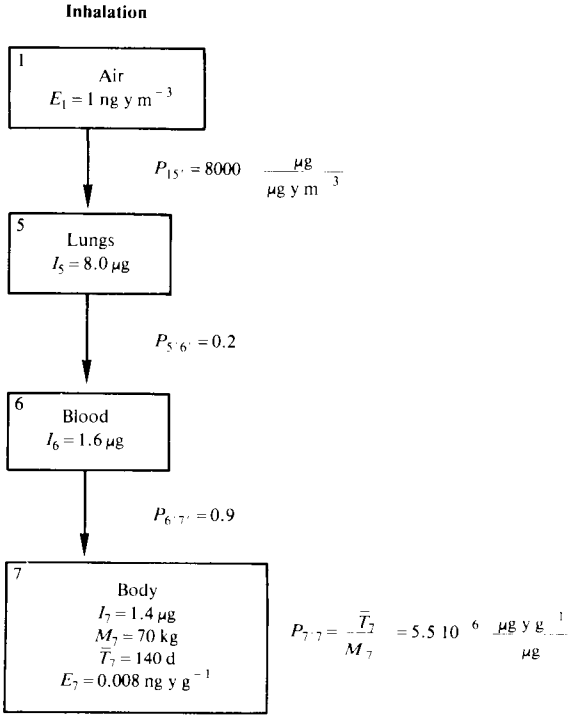
The basic task in the application of the exposure commitment method is the evaluation of transfer factors, P_{ij}, relating exposure or intake commitments in successive environmental compartments. To complete an initial assessment, representative values have been assigned to the concentrations and transfer rates of selenium in the environment. A summary of these values is given in Table 1.

The compartmental arrangement representing the transfer pathways and the evaluations of transfer factors are shown in Figure 3. It is convenient to begin with unit exposure commitment to air and determine the resulting

Table 1 Selenium in the environment: summary of representative values

Concentrations		
Atmosphere		
urban	3 ng m^{-3}	(0.01 – 30)*
rural	1.3 ng m^{-3}	(0.01 – 3)
Lithosphere		
agricultural soil	0.4 μg g^{-1}	(0.1 – 2)
Hydrosphere		
freshwater	0.2 μg ℓ^{-1}	(0.02 – 10)
ocean	0.2 μg ℓ^{-1}	(0.05 – 0.2)
Biosphere		
primary accumulator plants	1,000 μg g^{-1}	(100 – 5000)
secondary accumulator plants	100 μg g^{-1}	(10 – 500)
food crops	0.1 μg g^{-1}	(0.05 – 1)
Man		
tissues	0.1 μg g^{-1}	(<0.05 – 5)
blood	0.2 μg m ℓ^{-1}	(0.1 – 0.4)
Transfer rates		
Intake		
ingestion	70 μg d^{-1}	(10 – 220)
inhalation – urban	0.07 μg d^{-1}	(0.0002 – 0.7)
– rural	0.03 μg d^{-1}	(0.0002 – 0.07)
Absorption		
GI tract	0.8	
lungs – retention	0.35	
– absorption	~0.6	

* range of values in parentheses



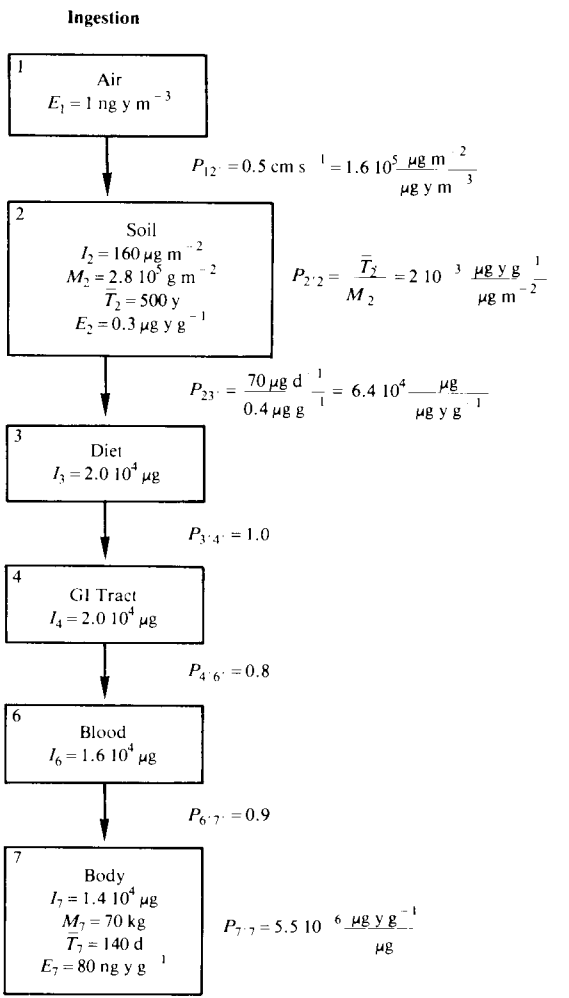


Figure 3 Pathway evaluations for selenium

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 selenium in the body from current background levels in air and diet.

For the inhalation pathway, the main assumptions are the air breathing rate of 22 $m^3\ d^{-1}$ or 8,000 $m^3\ y^{-1}$ ($P_{15'}$), retention in the lungs and absorption to blood of 20 per cent of the intake amount ($P_{5'6'}$), distribution of selenium in blood to the body tissues of 90 per cent ($P_{6'7'}$) and residence time in the body of 140 days. Retention of ambient airborne particulates in the pulmonary region of the lungs is of the order of 35 per cent. Absorption of selenium compounds from retained particles is uncertain. Tentatively, the combined retention-absorption transfer factor is taken to be 0.2 ($P_{5'6'}$). Of the amount transferred to blood, an estimated 90 per cent is distributed to tissues and retained longer than a few days. The retention components are assumed to have half-times of 30 and 150 days (section 4.3). Accounting for the distribution between the two components (40 per cent and 50 per cent respectively) gives an effective half-time of about 100 days and a mean retention time of 140 days.

The sequential multiplication of transfer factors gives the following exposure commitment to the body (E_7) via the inhalation pathway from unit exposure commitment to air (E_1).

$$E_7 = P_{15'} P_{5'6'} P_{6'7'} P_{7'7} E_1$$

$$= 8\ \frac{\mu g}{ng\ y\ m^{-3}}\ 0.2\ \frac{\mu g}{\mu g}\ 0.9\ \frac{\mu g}{\mu g}\ 5.5\ 10^{-3}\ \frac{ng\ y\ g^{-1}}{\mu g}\ 1\ ng\ y\ m^{-3}$$

$$= 0.008\ ng\ y\ g^{-1}$$

For the ingestion pathway, the analysis begins with the deposition velocity of 0.5 cm s^{-1} ($P_{12'}$). This is the median result derived from selenium measurements in air and total deposition at four sites in England and Wales during 1978 (Cawse 1980), and it is a typical value for trace elements attached to ambient aerosols.

From the product of the deposition velocity and the concentration of selenium in air, of the order of 1.3 ng m^{-3} in rural areas, the deposition rate onto the soil is estimated to be 210 $\mu g\ m^{-2}\ y^{-1}$. This is assumed mixed in the surface soil of density 1.4 g cm^{-3} and depth 20 cm, which is the plough layer thickness. The representative concentration of selenium in agricultural soil

is taken to be 0.4 $\mu g\ g^{-1}$, which is the geometric mean of the extremes of the typical range (0.1 to 2 $\mu g\ g^{-1}$) and in agreement with the results of wider surveys (section 3.2).

The residence time of selenium in soil can be determined from the formula

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

where C_2^* is the concentration in soil, F_2^* is the input rate per unit area, with the asterisks indicating steady-state values, and M_2 is the soil mass per unit area in the surface 20 cm layer. Using the above values of concentration and input (deposition) rate, the mean residence time is inferred to be about 500 y. This estimate is very uncertain, but will be used to complete the pathway analysis until more direct associations become available.

The relationship between selenium in soil and the intake rate via diet is obtained from representative background values: 70 $\mu g\ d^{-1}$ dietary intake rate and 0.4 $\mu g\ g^{-1}$ concentration in soil. The transfer factor between the soil and diet compartments ($P_{2\,3'}$) is evaluated in Figure 3.

Dietary intake of selenium is transferred directly to the gastro-intestinal tract ($P_{3'4'} = 1.0$). Fractional absorption to blood is assumed to be 80 per cent, of which 10 per cent is excreted within a few days and 90 per cent is distributed to tissues and retained with an effective retention time of 140 days.

The exposure commitment to the body via the ingestion pathway from unit exposure commitment to air (1 ng y m^{-3}) is determined from the following sequential product of transfer factors.

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

$$= 160\ \frac{\mu g\ m^{-2}}{ng\ y\ m^{-3}}\ 2\ 10^{-3}\ \frac{\mu g\ y\ g^{-1}}{\mu g\ m^{-2}}\ 6.4\ 10^{4}\ \frac{\mu g}{\mu g\ y\ g^{-1}}$$

$$\times 1.0\ \frac{\mu g}{\mu g}\ 0.8\ \frac{\mu g}{\mu g}\ 0.9\ \frac{\mu g}{\mu g}\ 5.5\ 10^{-6}\ \frac{\mu g\ y\ g^{-1}}{\mu g}\ 1\ ng\ y\ m^{-3}$$

$$= 0.08\ \mu g\ y\ g^{-1}$$

This result in terms of exposure commitments is numerically the same as the relationship between equilibrium concentrations. Thus, for the ingestion

pathway, 1 ng m^{-3} of selenium in air corresponds, at equilibrium, to a concentration of 0.08 $\mu g\ g^{-1}$ of selenium in the body.

The relationships for the current background levels are given in Table 2. The concentrations of selenium in air are taken to be 3 ng m^{-3} in urban areas and 1.3 ng m^{-3} in rural areas. From the product of transfer factors, these values give contributions to selenium in the body of 0.02 ng g^{-1} from the inhalation pathway and 0.1 $\mu g\ g^{-1}$ from the ingestion pathway. The ingestion pathway predominates. The computations for the ingestion pathway could have begun with the dietary intake rate, as the initial part of the pathway including the residence time of selenium in soil is only tentatively formulated.

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

	Air	Soil	Diet	Body
Inhalation pathway	3 $\frac{ng}{m^3}$	———	———	0.00002 $\frac{\mu g}{g}$
Ingestion pathway	1 $\frac{ng}{m^3}$	→ 0.4 $\frac{\mu g}{g}$	→ 70 $\frac{\mu g}{d}$	→ 0.10 $\frac{\mu g}{g}$
				Total 0.1 $\frac{\mu g}{g}$

The estimated mean concentration of selenium in the body of 0.1 $\mu g\ g^{-1}$ corresponds to a whole body content of 7 mg. Concentrations up to an order of magnitude greater could apply to the main organs of accumulation of selenium, according to measurements, and concentrations somewhat lower than the mean would be expected in other tissues.

This completes a framework of the environmental pathway analysis for selenium. Additional measurements are required, particularly of the retention times in soil and the body, and closer associations defining the transfer factors would decrease the uncertainties. The analysis must be made for specific selenium compounds and for more specific exposure conditions, when adequate information becomes available.

References

Adams, F. C., Van Craen, M. J. and Van Espen, P. J. 1980 Enrichment of trace elements in remote aerosols. *Environ. Sci. Technol.*. **14**, 1002 – 1005.

Allaway, W. H., Kubota, J., Losee, F., Roth, M. 1968 Selenium, molybdenum, and vanadium in human blood. *Arch. Environ. Health.* **16**, 342 – 348.

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

Bisbjerg, B. 1972. Studies on selenium in plants and soils. *Riso Report No. 200*, Danish Atomic Energy Commission Research Establishment, Riso, Denmark.

Bowen, H. J. M. 1979 *Environmental Chemistry of the Elements.* Academic Press, London.

Brown, T. A. and Shrift, A. 1982. Selenium: toxicity and tolerance in higher plants. *Biol. Rev.* **57**, 59 – 84.

Brune, D., Nordberg, G., 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.

Cantor, A. H., Scott, M. L., and Noguchi, T. 1975 Biological availability of selenium in feedstuffs and selenium compounds for prevention of exudative diathesis in chicks. *J. Nutr.* **105**, 96 – 105.

Cawse, P. A. 1980 *A survey of atmospheric trace elements in the U.K.: Results for 1978.* U.K. Atomic Energy Authority Report AERE-R 9484, Harwell.

Chinese Academy of Medical Sciences – Keshan Disease Research Group 1979 Observations on effect of sodium selenite in prevention of Keshan disease. *Chin. Med. J.* **92**, 471 – 476.

Cigna Rossi, L., Clemente, G. F., Santaroni, G. 1976 Mercury and Selenium distribution in a defined area and in its population. *Archives Environ. Health.* **31**, 160 – 165.

Diplock, A. T. 1976 Metabolic aspects of selenium action and toxicity. *CRC Crit. Rev. Toxicol.* **4**, 271 – 329.

Ewan, R. C., Pope, A. L., Baumann, C. A. 1967 Elimination of fixed selenium by the rat. *J. Nutr.* **91**, 547 – 554.

Ganapathy, S. N., Joyner, B. T., Sawyer, D. R., and Häfner, K. M. 1978 Selenium content of selected foods. In: *Trace Element Metabolism in Man and Animals, Vol. 3*, M. Kirchgessner (Ed.). Technische University, Munich, 322.

Glover, J., Levander, O., Parizek, J. and Vouk, V. 1979 Selenium. In: *Handbook on the Toxicology of Metals.* L. Friberg, G. F. Nordberg and V. B. Vouk (Eds.). Elsevier/North-Holland Biomedical Press, Amsterdam, 555 – 577.

Griffiths, N. M., Stewart, R. D. H. and Robinson, M. F. 1976 The metabolism of ^{75}Se-selenomethionine in four women. *Brit. J. Nutr.* **35**, 373 – 382.

Hashimoto, Y., Hwang, J. Y. and Yanagisawa, S. 1970 Possible sources of atmospheric pollution of selenium. *Environ. Sci. Technol.* **4**, 157 – 158.

Howe, M. 1979 Selenium in the blood of South Dakotans. *Arch. Environ. Health.* **34**, 444 – 448.

International Agency for Research on Cancer 1975 Selenium and selenium compounds. In: *IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man, Vol. 9.* Lyon, 245 – 260.

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

International Commission on Radiological Protection 1980 Limits for intakes of radionuclides by workers. ICRP Publication 30, Part 2, Pergamon Press, Oxford.

Karlsen, J. T., Norheim, G. and Frøslie, A. 1981. Selenium content of Norwegian milk, eggs and meat. *Acta Agric. Scand.* **31**, 165 – 170.

Koljonen, T. 1978 The availability of selenium as nutrient in different geological environments, with special reference to Finland and Iceland. *Ambio* **7**, 169 – 171.

Lewis, B. G. 1976 Selenium in biological systems and pathways for its volatilization in higher plants. In: *Environ. Biogeochem.* Vol. 1. J. O. Nriagu (Ed.). Ann Arbor Science Publishers, Inc., Ann Arbor, Michigan, 389 – 409.

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.

McDonald, C. and Duncan, J. J. 1979 Atmospheric levels of trace elements in Glasgow. *Atmos. Environ.* **13**, 413 – 417.

Mondragon, M. C. and Jaffe, W. G. 1976 Consumo de selenio en la ciudad de Caracas en comparacion con el de otras ciudades del mundo. *Arch. Latino Am. Nutr.* **26**, 341.

Morris, V. C. and Levander, O. A. 1970 Selenium content of foods. *J. Nutr.* **100**, 1383 – 1388.

National Academy of Sciences 1976 *Medical and Biological Effects of Environmental Pollutants – Selenium*. NAS, Washington, D.C.

Peterson, P. J., Benson, L. M. and Zieve, R. 1981 Metalloids. In: *Effect of Heavy Metal Pollution on Plants* Vol. 1, N. W. Lepp (Ed.). Applied Science Publishers, London, 279 – 342.

Priest, P., Navarre, J. L. and Ronneau, C. 1981 Elemental background concentration of the atmosphere of an industrialized country. *Atmos. Environ.* **15**, 1325 – 1330.

Robinson, M. F. 1976 The moonstone: more about selenium. *J. Human Nutr.* **30**, 79.

Rosenfeld, I. and Beath, O. A. 1964. Chemistry of selenium. In: *Selenium, Geobotany, Biochemistry, Toxicity, and Nutrition*. Academic Press, New York, 299 – 332.

Sakurai, H. and Tsuchiya, K. 1975 A tentative recommendation for maximum daily intake of selenium. *Environ. Physiol. Biochem.* **5**, 107 – 118.

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.

Schroeder, H. A., Frost, D. V. and Balassa, J. J. 1970 Essential trace metals in man: Selenium. *J. Chron. Dis.* **23**, 227 – 243.

Schwartz, K. and Foltz, C. M. 1957 Selenium as an integral part of Factor 3 against dietary necrotic liver degeneration. *J. Am. Chem. Soc.* **79**, 3292.

Scott, M. L. 1973 Nutritional importance of selenium. In: *Organic selenium compounds, their chemistry and biology*. D. L. Klayman and W. H. H. Günther (Eds.). John Wiley, New York, 629 – 661.

Shacklette, H. T., Boerngen, J. G. and Keith, J. R. 1974 Selenium, fluorine, and arsenic in surficial materials of the conterminous United States. *Geological Survey Circular* **692**, U.S. Department of the Interior, Washington, D.C.

Shamberger, R. J. 1981 Selenium in the environment. *Sci. Total Environ.* **17**, 59 – 74.

Shendrikar, A. D. 1974 Critical evaluation of analytical methods for the determination of selenium in air, water and biological materials. *Sci. Total Environ.* **3**, 155 – 168.

Stewart, R. D. H., Griffiths, N. M., Thompson, C. D. and Robinson, M. F. 1978 Quantitative selenium metabolism in normal New Zealand women. *Brit. J. Nutr.* **40**, 45 – 54.

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

Thompson, J. N., Erdody, P. and Smith, D. C. 1975 Selenium content of food consumed by Canadians. *J. Nutr.* **105**, 274.

Thorn, J., Robertson, J. and Buss, D. H. 1978 Trace nutrients. Selenium in British food. *Brit. J. Nutr.* **39**, 391.

Varo, P. and Koivistoinen, P. 1981 Annual variations in the average selenium intake in Finland: cereal products and milk as sources of selenium in 1979/80. *Int. J. for Vitamin and Nutr. Res.* **51**, 79 – 84.

Vokal-Borek, H. 1979 *Selenium.* USIP Report 79-16, University of Stockholm, Institute of Physics, Stockholm.

Welsh, S. O., Holden, J. M., Wolf, W. R. and Levander, O. A. 1981 Selenium in self-selected diets of Maryland residents. *J. Amer. Diet. Assoc.* **79**, 277 – 285.

Yukawa, M., Suzuki-Yasumoto, M., Amano, K., Terai, M. 1980 Distribution of trace elements in the human body determined by neutron activation analysis. *Arch. Environ. Health,* **35**, 36 – 44.

CHROMIUM

1 Natural cycle

Chromium is a metal of atomic number 24. Its average abundance in the earth's crust is 100 $\mu g\ g^{-1}$ (Taylor 1964). It is normally found in the trivalent state as chromite ore. Chromium compounds are generally not readily soluble and mobility is reduced except at low pH in soil and water (Rondia 1979). Chromium, in the form of trivalent compounds and particularly in an organic complex, is an essential trace element in man and animals, but hexavalent compounds (chromates) can be toxic at high concentrations.

2 Anthropogenic sources

Chromium is used in metallurgical and chemical industries including use in alloys and plated materials and in tanning and pigment production (Langård and Norseth 1979). Airborne industrial particulate releases are generally of the trivalent form of chromium. Some occupational settings may involve exposures to chromates in air; for example, fumes from stainless steel welding (Stern 1981). Chromates are easily reduced to trivalent form, but are more stable in water and thus mostly associated with industrial waste water releases. Local air and water pollution by chromates can arise from power plant cooling towers, where chromates are added to the cooling water for corrosion inhibition.

3 Environmental considerations

3.1 Air

Chromium in air has been reported at concentrations of less than 10 ng m^{-3} to about 50 ng m^{-3} in urban areas and usually less than 10 ng m^{-3} in rural areas (NAS 1974). The annual concentrations in 87 urban areas in the U.S.A. generally ranged from 3 to 30 ng m^{-3} during 1976 with mean concentrations for all sites of 4 to 8 ng m^{-3} during 1970-76 (USEPA 1979). The annual average chromium concentration in air in four large Belgian cities during 1972-77 was 14 – 43 ng m^{-3} and 11 – 20 ng m^{-3} in non-urban areas (Kretzschmar, Delespaul and De Rijck 1980). Variability was low indicating mostly diffuse sources of emissions to air.

The mean level of chromium in air at a semi-rural site in England during 1957-74 was 9.4 ng m^{-3} (Salmon *et al.* 1978). Higher levels between 1957 and 1960 were reduced by smoke abatement legislation, indicating important anthropogenic contributions. Levels since 1962 have been fairly constant at around 5 ng m^{-3} (Figure 1). Concentrations of chromium at rural and remote sites in the U.S.A. have been of the order of 3 ng m^{-3} or less during 1970-76 (USEPA 1979).

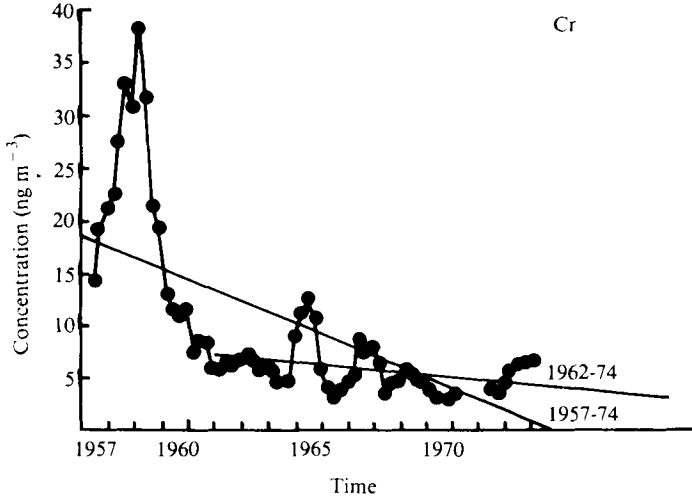


Figure 1 Chromium in air at Chilton, Oxfordshire, U.K. (Salmon *et al.* 1978)

3.2 Soil

Concentrations of chromium in soil range from trace amounts to 250 $\mu g\ g^{-1}$ or more. Soils derived from some ultrabasic (serpentine) rocks have the highest chromium content with concentrations of several thousand $\mu g\ g^{-1}$. Results of over 500 samples of agricultural soil from a generally non-serpentine soil region of the Federal Republic of Germany were mostly in the range 8 – 32 $\mu g\ g^{-1}$ with a mean of 17 $\mu g\ g^{-1}$ (Kick, Bürger and Sommer 1980). In France, the concentration in soil was found most generally to be in the range 30 to 75 $\mu g\ g^{-1}$ (Boudène 1979). A large sampling of soils in the United States gave a mean of 37 $\mu g\ g^{-1}$ (NAS 1974). Little soluble chromium is found; insoluble Fe-Cr mixed hydroxides are formed which are less available for plant uptake (Rondia 1979).

3.3 Plants

Plant uptake of chromium is low. Concentrations in plants are less than the corresponding concentrations in soil (Peterson 1975). Chromium concentrations in plants grown in normal soils range from 0.1 to 5 $\mu g\ g^{-1}$ (Anderson 1981). There are species differences. Among crop plants, higher concentrations are found in cauliflower and root vegetables than in grain plants (Lahouti and Peterson 1979). Uptake of trivalent chromium is greater than chromates, but the trivalent ions are retained at the cell walls and transfer through the plant is limited. Chromate ions remain in a soluble form in roots and are transported to shoots more easily than trivalent ions, though still to a very limited extent (Skeffington, Shewry and Peterson 1976).

Trivalent chromium is not toxic to plants unless present in high concentrations in soil (>500 $\mu g\ g^{-1}$ dry wt). Hexavalent chromium is toxic to plants, but if released to soil it is rapidly reduced to less soluble forms (Cary, Allaway and Olson 1977b). The low availability through root uptake and also foliar absorption of chromates present in cooling tower water has been demonstrated (Parr and Taylor 1980).

3.4 Diet

Considerable variation is found in the concentration of chromium in foods, values ranging from non-detectable to over 500 $\mu g\ kg^{-1}$ (wet wt) (NAS 1974). Chromium in the form of the glucose tolerance factor complex is found predominantly in Brewer's yeast at concentrations of about 2000 $\mu g\ kg^{-1}$. Normal chromium concentration in vegetables is 20 to 50 $\mu g\ kg^{-1}$ (wet wt) and a similar range (but dry wt) in grains and flour (Rondia 1979). Vegetables normally rich in iron tend also to accumulate chromium (Cary, Allaway and Olson 1977a, Rondia 1979). Chromium in cow's milk ranges from 0 to 13 $\mu g\ \ell^{-1}$ with a mean of 3.1 $\mu g\ \ell^{-1}$ (Rondia 1979). Some enhancement of concentrations of chromium in marine animals is thought possible, but no general statement can yet be made (Boudène 1979). Chromium concentrations in freshwater fish are low (< 1 $\mu g\ g^{-1}$ wet wt) and trophic level effects seem to be absent (Elwood, Beauchamp and Allen 1980).

Dietary intake of chromium is estimated to be in the range 30 to 100 $\mu g\ d^{-1}$ (Langård and Norseth 1979). A reported value for Italy is 40 $\mu g\ d^{-1}$ (Clemente, Cigna Rossi and Santaroni 1978). The major contributors are meat and vegetables. Minor sources are fish, vegetable oil and fruit (Langård and Norseth 1979).

3.5 Water

The concentration of chromium in rivers and lakes is usually 1 to 10 $\mu g\ \ell^{-1}$ with lower concentrations in sea water (<0.04 to 0.5 $\mu g\ \ell^{-1}$) (NAS 1974). Reported mean concentrations in the Meuse river in Belgium during 1971-73 was 5 $\mu g\ \ell^{-1}$ but up to 400 $\mu g\ \ell^{-1}$ downstream from an industrial zone (Rondia 1979). Mean chromium concentrations in drinking water in the U.S.A. have been reported to be 0.4 $\mu g\ \ell^{-1}$ (Durfor and Becker 1964) and 2.3 $\mu g\ \ell^{-1}$ (Angino, Wrixson and Smith 1977) and 2.6 and 8.6 $\mu g\ \ell^{-1}$ in two areas in Finland (Punsar 1975).

3.6 Miscellaneous

Cigarettes may contain 0.4 $\mu g\ g^{-1}$ of chromium, but estimates of inhaled amounts are not available (Langård and Norseth 1979).

4 Metabolism

4.1 Absorption

Absorption of chromium following inhalation depends on the chemical form. The more readily soluble hexavalent chromium compounds can be more easily absorbed than the trivalent ones (Langård and Norseth 1979).

Absorption of inorganic chromium from the gastro-intestinal tract is low – less than 1 per cent of trivalent chromium and about 2 per cent of chromates (Langård and Norseth 1979). Absorption of organic forms is higher.

4.2 Distribution in the body

Chromium is cleared rapidly from blood. Distribution in the body depends on the chemical form. Trivalent chromium acetate and citrate are rapidly excreted, whereas chromium chloride and chromates are somewhat more retained, primarily in liver, spleen and bone marrow (Langård and Norseth 1979). Retained chromium is found in the trivalent form.

Chromium is found in the new-born. Tissue concentrations decrease with age, except for lung which retains some amounts from inhaled air (Langård and Norseth 1979). The decreasing concentrations may be due to suboptimal chromium levels in most diets (Anderson 1981). The concentrations of chromium in adults (residents of New York and Chicago) were highest in hair 0.2 – 2 $\mu g\ g^{-1}$, followed by lungs 0.14 – 0.7 $\mu g\ g^{-1}$, liver 0.27 $\mu g\ g^{-1}$, and kidneys 0.09 $\mu g\ g^{-1}$ (Schroeder, Balassa and Tipton 1962).

Analyses of tissues of residents of the U.K. gave similar results for lungs and hair, 0.5 $\mu g\ g^{-1}$, but lower concentrations in other tissues: kidneys and blood 0.03 $\mu g\ g^{-1}$, liver 0.008 $\mu g\ g^{-1}$ and muscle 0.005 $\mu g\ g^{-1}$ (Hamilton, Minski and Cleary 1973). Recent measurements of tissues from non-occupationally exposed individuals from Japan also indicate lower concentrations in soft tissues, from 0.003 to 0.04 $\mu g\ g^{-1}$ in liver, kidney, pancreas, pharynx, aorta, bone marrow, brain and skin, 0.03 to 0.16 $\mu g\ g^{-1}$ in the adrenal glands and 0.02 to 0.3 $\mu g\ g^{-1}$ in lungs (Hyodo, Suzuki, Furuya and Meshizuka 1980).

4.3 Retention times

Chromium compounds are retained in the lungs for varying times prior to absorption or passage to the gastro-intestinal tract. The most soluble compounds are retained on the order of days, with halides and nitrates on the order of weeks and oxides and hydroxides on the order of years (ICRP 1980).

Chromium absorbed into blood following either inhalation or ingestion is excreted mainly via urine, previously reported to be 7-10 $\mu g\ d^{-1}$, but more recent, improved determinations indicate less than 1 $\mu g\ d^{-1}$ (Anderson 1981, Donaldson and Rennert 1981). This excretion rate is consistent with average dietary intake of about 60 $\mu g\ d^{-1}$ with 1 to 2 per cent absorption.

Metabolic behaviour of chromium is assumed to be based on the trivalent chromic form, other forms being reduced to the chromic form soon after absorption (ICRP 1980). With some experience from animal studies (Mertz, Roginski and Reba 1965), retention components for absorbed amounts are estimated to be 30 per cent excreted with a half-time of 0.5 days, 5 per cent transported to bone and retained with a half-time of 1000 days and 2 components distributed to organs and tissues other than bone with fractional amounts and half-times being 40 per cent with 6 days and 25 per cent with 80 days (ICRP 1980).

5 Effects

Human exposure to trivalent chromium is largely via food, from which there is no evidence of adverse effects. Chromium is an essential element in man and animals, playing an important role in insulin metabolism as the glucose tolerance factor. It has been shown to reduce blood low density cholesterol levels, representing a conjectured reduced risk of coronary heart disease (Riales 1979). Hexavalent chromium compounds are toxic and may cause skin and nasal lining ulcerations, bronchial asthma and lung cancer

(Langård and Norseth 1979, Norseth 1981). There are no descriptions of toxic organic chromium compounds.

As with nickel, skin contact with chromium can lead to sensitization and allergic skin reactions. Chromates in cement have caused effects in building industry workers. Traces of chromates in cleaning solutions have on contact caused skin disorders (Rondia 1979).

6 Literature critique

Because of the varying toxicity of the various forms of chromium, elemental analyses of environmental samples are of only limited value. Determinations of chromate levels would be most pertinent. The total daily need of trivalent chromium is uncertain as are also the levels which might be excessive. Some nutritionalists have recommended production of Cr-rich vegetables to increase intake (Rondia 1979).

Reviews of chromium occurrence, metabolism and toxicity include Mertz (1969), IARC (1973), National Academy of Sciences (1974), Underwood (1977), Langård and Norseth (1979) and the papers of a symposium on chromium in nutrition and metabolism edited by Shapcott and Hubert (1979). Accurate analysis of chromium, particularly in biological materials, is difficult because of low concentrations, matrix effects, binding to sample containers and contamination from sampling equipment. Various analytical methods and sensitivities are listed in Hubert (1979), Hancock (1979) and Anderson (1981).

7 Pathway analysis

The analysis of the transfer of chromium from general environmental sources to man is based on the exposure commitment method. The notation and procedure have been presented previously (Bennett 1981). The basic task in application of this method is the evaluation of transfer factors, P_{ij}, relating exposure or intake commitments in successive reservoirs. To provide initial estimates of transfer factors, representative values have been assigned to the various parameters required. A summary of some of these values, namely average concentrations and intake rates, are presented in Table 1. The pathway evaluations are given in Figure 2.

The analyses of the transfers via the inhalation and ingestion pathways begin with unit exposure commitment to air, and the resulting exposure commitments to the receptor organs, bone and the remainder of the body, are determined. Because of the equivalence between commitment and

Table 1 Chromium in the environment: summary of representative values

Concentrations		
Atmosphere		
urban	7 ng m^{-3}	(1 – 50)*
rural	4 ng m^{-3}	(0.3 – 10)
Lithosphere		
agricultural soil	40 μg g^{-1}	(10 – 200)
Hydrosphere		
freshwater	0.2 μg ℓ^{-1}	(0.1 – 10)
ocean	0.1 μg ℓ^{-1}	(0.04 – 0.5)
Biosphere		
land plants	0.5 μg g^{-1}	(0.1 – 3)
food	0.03 μg g^{-1}	(0.01 – 0.3)
Man		
tissues	0.02 μg g^{-1}	(0.003 – 0.3)
Transfer rates		
Intake		
ingestion	60 μg d^{-1}	(30 – 100)
inhalation – urban	0.2 μg d^{-1}	(0.02 – 1)
– rural	0.09 μg d^{-1}	(0.007 – 0.2)
Absorption		
GI tract	0.01	(0.01 – 0.1)
lungs – retention	0.35	(0.3 – 0.4)
– absorption	~0.5	

*Range of values in parentheses

steady-state analyses, the commitment results can be utilized to determine the contributions to the steady-state concentration of chromium in the body from current background concentrations of chromium in air, diet and water.

For the inhalation pathway, the main assumptions are the air breathing rate of 22 $m^3 d^{-1}$ or 8000 $m^3 y^{-1}$ (P_{15}), particle retention in the lung of 35 per cent and absorption to blood of about 50 per cent ($P_{5,6} = 0.35 \times 0.5 = 0.2$). The retention fraction is typical for trace contaminants attached to ambient aerosols, but the absorption fraction is quite uncertain. Absorption depends on the chemical form and may be somewhat overestimated for both chromic and chromate compounds. Chromium absorbed into blood is assumed to be distributed 5 per cent to bone with a retention half-time of 1000 days and 65 per cent to other tissues with an effective half-time of 35 days. The mean residence times are thus, 1400 d in bone and 50 d in other tissues. The remaining 30 per cent of the amount transferred to blood is assumed to be excreted within a day.

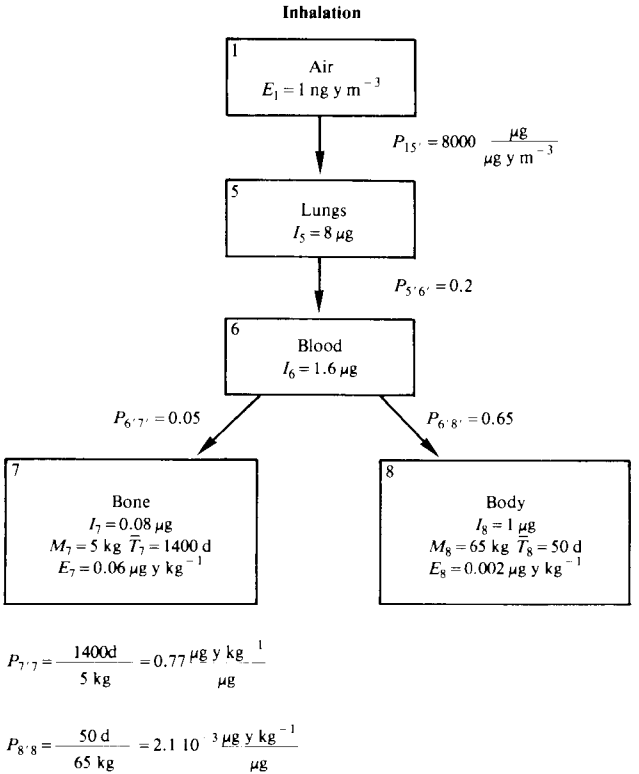


Figure 2 Pathway evaluations for chromium

For the ingestion pathway, the analysis begins with an estimated deposition velocity of 1.5 cm s^{-1} or 4.7 10^5 m y^{-1} ($P_{12'}$). This is the approximate mean of chromium measurements in air and deposition at background sites in the U.K. during 1978 and 1979 (Cawse 1980, 1981). The mean residence time of chromium in soil can be inferred from the relationship

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

where C_2^* is the concentration of chromium in soil, F_2^* is the input rate per unit area, with the asterisks indicating steady-state values, and M_2 is the soil mass per unit area (density 1.4 g cm^{-3} and mixing depth 20 cm) (Bennett 1981). A background concentration of chromium in air of 5 ng m^{-3} times the deposition velocity gives an input rate to soil of 2,400 μg m^{-2} y^{-1}. If this is assumed to be the main input source and the soil concentration is 40 μg g^{-1}, the mean residence time of chromium in soil is estimated from the above formula to be 5,000 years. This is a very uncertain estimate, but Bowen (1975) also inferred a long residence time of 6,000 years for chromium, based on rough estimates of soil inputs and outflows.

The ratio of the dietary intake rate of chromium to the associated background concentration of chromium in soil gives a value of the transfer factor between the soil and diet compartments. Representative dietary intake is estimated to be 60 μg d^{-1} and the soil concentration 40 μg g^{-1}. The transfer factor, $P_{23'}$, is evaluated in Figure 2.

Transfer of dietary intake to the gastro-intestinal tract is by direct transfer ($P_{3'4'} = 1.0$). Absorption to blood is assumed to be 1 per cent for trivalent chromium compounds and 10 per cent for chromates (ICRP 1980). Fractional distribution to bone and other tissues and the retention times are the same as for chromium reaching blood following inhalation.

Contributions to the exposure commitments to bone and other tissues are determined from sequential multiplication of transfer factors. For an exposure commitment of 1 ng y m^{-3} to air (E_1), the results are to bone (E_7)

$$E_7 = P_{15'} P_{5'6'} P_{6'7'} P_{7'7} E_1$$

$$= 8000 \frac{\mu g}{\mu g \text{ y } m^{-3}} \; 0.2 \frac{\mu g}{\mu g} \; 0.05 \frac{\mu g}{\mu g} \; 0.77 \frac{\mu g \text{ y } kg^{-1}}{\mu g} \; 1 \text{ ng y } m^{-3}$$

$$= 0.06 \; \mu g \text{ y } kg^{-1} \text{ (via inhalation)}$$

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

$$= 4.7\ 10^5\ \frac{\mu g\ m^{-2}}{\mu g\ y\ m^{-3}}\ 0.02\ \frac{\mu g\ y\ g^{-1}}{\mu g\ m^{-2}}\ 550\ \frac{\mu g}{\mu g\ y\ g^{-1}}\ 1.0\ \frac{\mu g}{\mu g}$$

$$\times 0.01\ \frac{\mu g}{\mu g}\ 0.05\ \frac{\mu g}{\mu g}\ 0.77\ \frac{\mu g\ y\ kg^{-1}}{\mu g}\ 1\ ng\ y\ m^{-3}$$

$$= 2\ \mu g\ y\ kg^{-1}\ \text{(via ingestion)}$$

For tissues of the body other than bone, the contributions to the exposure commitment (E_8) from unit exposure commitment of air ($E_1 = 1$ ng y m^{-3}) the results are

$$E_8 = P_{15,}P_{5,6,}P_{6,8,}P_{8,8}E_1$$

$$= 8000\ \frac{\mu g}{\mu g\ y\ m^{-3}}\ 0.2\ \frac{\mu g}{\mu g}\ 0.65\ \frac{\mu g}{\mu g}\ 0.0021\ \frac{\mu g\ y\ kg^{-1}}{\mu g}\ 1\ ng\ y\ m^{-3}$$

$$= 0.002\ \mu g\ y\ kg^{-1}\ \text{(via inhalation)}$$

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

$$= 4.7\ 10^5\ \frac{\mu g\ m^{-2}}{\mu g\ y\ m^{-3}}\ 0.02\ \frac{\mu g\ y\ g^{-1}}{\mu g\ m^{-2}}\ 550\ \frac{\mu g}{\mu g\ y\ g^{-1}}\ 1.0\ \frac{\mu g}{\mu g}$$

$$\times 0.01\ \frac{\mu g}{\mu g}\ 0.65\ \frac{\mu g}{\mu g}\ 0.0021\ \frac{\mu g\ y\ kg^{-1}}{\mu g}\ 1\ ng\ y\ m^{-3}$$

$$= 0.07\ \mu g\ y\ kg^{-1}\ \text{(via ingestion)}$$

At steady state the relationships are numerically equal. For example, 1 ng m^{-3} of chromium in air will make, at equilibrium, contributions to the chromium concentration in bone of 0.06 $\mu g\ kg^{-1}$ via the inhalation pathway and 2 $\mu g\ kg^{-1}$ via the ingestion pathway.

The relationships for the current background levels are given in Table 2. Ingestion is the predominant contributor to body concentrations. The values indicate that the body content of chromium is of the order of 60 μg, although additional accumulation in lungs and hair is likely. The estimated mean concentration is somewhat less than currently indicated by tissue measurements. It could be that dietary intake has been underestimated or that there are other sources of intake of chromium. A major factor of uncertainty is in the estimated retention times of chromium in the body.

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

	Air	Diet	Bone	Body
Inhalation pathway				
	$7 \frac{ng}{m^3}$	→	$0.4 \frac{\mu g}{kg}$	
		→		$0.01 \frac{\mu g}{kg}$
Ingestion pathway				
	$4 \frac{ng}{m^3}$	→ $60 \frac{\mu g}{d}$	→ $8 \frac{\mu g}{kg}$	
		→		$0.3 \frac{\mu g}{kg}$
		Total	$8 \frac{\mu g}{kg}$	$0.3 \frac{\mu g}{kg}$

There is only an indication from animal studies and a lack of human data. Additional measurements of tissue concentrations are also required to make valid estimates of the total body content. More precise analyses are needed, particularly recognizing the analytical complications.

It would appear that, except in some occupational settings, exposures to chromium cannot be associated with adverse effects in man. Certain diseases, however, may be related to insufficient or excessive intake of chromium. The more toxic chromate forms have limited availability for transfer to man in most circumstances. Further studies of intake forms of chromium and metabolism are required to make more accurate exposure and health assessments.

References

Anderson, R. A. 1981 Nutritional role of chromium. *Sci. Total Environ.* **17**, 13 – 29.

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

Bennett, B. G. 1981 Exposure commitment concepts and application. In: *Exposure Commitment Assessments of Environmental Pollutants.* Vol. 1, No. 1, MARC Report 23, Monitoring and Assessment Research Centre, Chelsea College, University of London, 1 – 17.

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 – 186.

Bowen, H. J. M. 1975 Residence times of heavy metals in the environment. In: *International Conference on Heavy Metals in the Environment*, Toronto, Canada, Volume **1**, 1 – 19.

Cary, E. E., Allaway, W. H. and Olson, O. E. 1977a Control of chromium concentrations in food plants. 1. Absorption and translocation of chromium by plants. *Agric. and Food Chem.* **25**, 300 – 304.

Cary, E. E., Allaway, W. H. and Olson O. E. 1977b Control of chromium concentrations in food plants. 2. Chemistry of chromium in soils and its availability to plants. *Agric. and Food Chem.* **25**, 305 – 309.

Cawse, P. A. 1980 A survey of atmospheric trace elements in the U.K.: Results for 1978. U.K. Atomic Energy Authority, Report AERE-R9484, Harwell.

Cawse, P. A. 1981 A survey of atmospheric trace elements in the U.K.: Results for 1979 U.K. Atomic Energy Authority, Report AERE-R9886, Harwell.

Clemente, G. F., Cigna Rossi, L., Santaroni, G. P. 1978 Studies of the distribution and related health effects of the trace elements in Italy. In: *Trace Substance in Environmental Health*, D. D. Hemphill (Ed.). University of Missouri, Columbia, 23-30.

Donaldson, D. L. and Rennert, O. M. 1981 Chromium (III) metabolism by the kidney. *Annals of Clin. and Lab. Sci. II(5)* 377 – 385.

Durfor, C. N. and Becker, E. 1964 *Public water supplies of the 100 largest cities in the United States, 1962.* U.S. Geological Survey Water-Supply Paper 1812, U.S. Government Printing Office, Washington D.C.

Elwood, J. W., Beauchamp, J. J. and Allen, C. P. 1980 Chromium levels in fish from a lake chronically contaminated with chromates from cooling towers. *Int. J. Environ. Stud.* **14**, 289 – 298.

Hamilton, E. I., Minski, M. J. and Cleary, J. J. 1973 The concentration and distribution of some stable elements in healthy human tissues for the United Kingdom, *Sci. Total Environ.* **1**, 353.

Hancock, R. G. V. 1979 Neutron activation analysis as a standard reference method for chromium in biological materials. *In: Chromium in Nutrition and Metabolism*, D. Shapcott and J. Hubert (Eds.). Elsevier/North Holland Biomedical Press, Amsterdam, 31 – 42.

Hubert, J. 1979 Techniques for measurement of chromium in biological materials. In: *Chromium in Nutrition and Metabolism*, D. Shapcott and J. Hubert (Eds.). Elsevier/North Holland Biomedical Press, Amsterdam, 15 – 30.

Hyodo, K., Suzuki, S., Furuya, N. and Meshizuka, K. 1980 An analysis of chromium, copper and zinc in organs of a chromate worker. *Int. Arch. of Occup. and Environ. Health.* **46**, 141 – 150.

International Agency for Research on Cancer (IARC) 1973 Chromium and inorganic chromium compounds. In: *IARC Monographs in the evaluation of carcinogenic risk of chemicals to man*, Vol. 2, IARC, Lyon, 100 – 125.

International Commission on Radiological Protection (ICRP) 1980 *Limits for intakes of radionuclides by workers.* ICRP Publication 30, Part 2, Pergamon Press, Oxford.

Kick, H., Bürger, H. and Sommer, K. 1980 Gesamtgehalte an Pb, Zn, Sn, As, Cd, Hg, Cu, Ni, Cr, und Co in landwirtschaftlich und gärtnerish genutzten Boden Nordrhein-Westfalens. *Landwirtsch. Forschung.* **33**(1), 12 – 22.

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.

Lahouti, M. and Peterson, P. J. 1979 Chromium accumulation and distribution in crop plants, *J. Sci. Food Agric.* **30**, 136 – 142.

Langård, S. and Norseth, T. 1979 Chromium. In: *Handbook on the Toxicology of Metals*, L. Friberg, F. Nordberg, V. Vouk (Eds.). Elsevier/North-Holland Biomedical Press, Amsterdam 383 – 397.

Mertz, W. 1969 Chromium occurrence and function in biological systems. *Physiol. Rev.* **49**, 163 – 239.

Mertz, W., Roginski, E. E. and Reba, R. C. 1965 Biological activity and fate of trace quantities of intravenous chromium (III) in the rat. *Am. J. Physiol.* **209**, 489 – 494.

National Academy of Sciences 1974 Chromium. Committee on Biological Effects of Atmospheric Pollutants, Washington D.C.

Norseth, T. 1981 The carcinogenicity of chromium, *Environ. Health Perspect.* **40**, 121 – 130.

Parr, P. D. and Taylor, F. G., Jr. 1980 Incorporation of chromium in vegetation through root uptake and foliar absorption pathways, *Environ. and Exp. Bot.* **20**, 157 – 160.

Peterson, P. J. 1975 Element accumulation by plants and their tolerance of toxic mineral soils. In: *International Conference on Heavy Metals in the Environment*. Toronto, Canada Volume II, Part 1, 39 – 54.

Punsar, S. 1975 Coronary heart disease and drinking water. A search in two Finnish male cohorts for evidence of a water factor. *J. Chronic Dis.* **28**, 259.

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.

Riales, R. 1979 Influence of Brewer's yeast on lipoprotein cholesterol concentrations – A preliminary report. In: *Chromium in Nutrition and Metabolism*, D. Shapcott and J. Hubert (Eds.). Elsevier/North Holland Biomedical Press, Amsterdam, 199 – 212.

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.

Schroeder, H. A., Balassa, J. J. and Tipton, I. H. 1962 Abnormal trace metals in man – chromium. *J. Chronic Dis.* **15**, 941 – 964.

Shapcott, D. and Hubert, J. (Eds.). 1979 *Chromium in Nutrition and Metabolism*. Elsevier/North Holland Biomedical Press, Amsterdam.

Skeffington, R. A., Shewry, P. R. and Peterson, P. J. 1976 Chromium uptake and transport in barley seedlings. *Planta.* **132**, 209 – 214.

Stern, R. M. 1981 Process-dependent risk of delayed health effects for welders, *Environ. Health Perspect.* **41**, 235 – 253.

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

Underwood, E. J. 1977 *Trace Elements in Human and Animal Nutrition*, 4th Edition. Academic Press, New York.

U.S. Environmental Protection Agency 1979 Air Quality data for metals – 1976 – from the National Air Surveillance Networks U.S.E.P.A., Report EPA-600/4-79-054, Environmental Monitoring and Support Laboratory, Research Triangle Park, North Carolina.

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