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PREFACE

The data given in this report are to be used together with the text and dosimetric models described in Part 1 of *ICRP Publication 30*;[1] the chapters referred to in this preface relate to that report.

In order to derive values of the Annual Limit on Intake (ALI) for radioisotopes of scandium, the following assumptions have been made. In the metabolic data for scandium, a fraction 0.4 of the element in the transfer compartment is translocated to the skeleton. It is assumed that this scandium in the skeleton is distributed between cortical bone, trabecular bone and red marrow in proportion to their respective masses. Red marrow is not considered as a source organ in Chapter 7 and for the dosimetry of radioisotopes of scandium in red marrow the same assumptions have been made as for radioisotopes of indium in Part 2, as follows. For photons arising in red marrow, absorbed fractions are taken from Snyder *et al.*[2] For beta particles arising in red marrow the absorbed fraction in red marrow AF(RM ← RM) is assumed to be 1 and the dose equivalent in the whole of bone surfaces is taken to be half of that in red marrow.

In the following pages the relevant metabolic data for each element precede a table of values of ALI and DAC for radioisotopes of that element having radioactive half-lives greater than 10 min. The metabolic models described are for compounds of a stable isotope of the element.

Retention data given in the literature have, where necessary, been corrected for radioactive decay of the radionuclides concerned. Because of the considerable variation of gastrointestinal absorption from individual to individual, values of f_1, the fraction of a stable element reaching body fluids after its entry into the gastrointestinal tract, are given to one significant figure only (Section 6.2, Chapter 6). For inhalation, values of ALI and DAC are given for each different inhalation class (D, W and Y) appropriate for various compounds of the element (Chapter 5).

In the metabolic data, when the symbol t is used for time its unit is always the day unless specified otherwise.

Values of ALI (Bq) are given for the oral and inhalation routes of entry into the body. It is emphasized that the limit for inhalation is the appropriate ALI and that the values of DAC (Bq m^{-3}) for a 40-h working week are given only for convenience and should always be used with caution (Section 3.4, Chapter 3). Values of ALI for inhalation and DAC are for particles with an AMAD of 1 μm. A method of correcting the values for particles of other sizes is described in Section 5.5, Chapter 5 and the required numerical data are given in the Supplement to this Part.

If a value of ALI is determined by the non-stochastic limit on dose equivalent to a particular organ or tissue, the greatest value of the annual intake that satisfies the Commission's recommendation for limiting stochastic effects is shown in parentheses beneath the ALI. The organ or tissue to which the non-stochastic limit applies is shown below these two values. When an ALI is determined by the stochastic limit this value alone is given (Section 4.7, Chapter 4).

All the values of ALI and DAC given here are for occupationally exposed adults and must be used with circumspection for any other purpose (Chapter 9).

References

1. *ICRP Publication 30*, Part 1, *Limits for Intakes of Radionuclides by Workers. Annals of the ICRP*, 2 (3/4), 1979.
2. Snyder, W. S., Ford, Mary R. and Warner, G. G. Estimates of specific absorbed fractions for photon sources uniformly distributed in various organs of a heterogeneous phantom. MIRD Pamphlet No. 5 Revised, Society of Nuclear Medicine (1978).

METABOLIC DATA FOR BERYLLIUM

1. Metabolism

Data from Reference Man (ICRP, 1975)

Beryllium content of the body	36 μg
of soft tissues	27 μg
Daily intake in food and fluids	12 μg

It should be noted that the beryllium content of the tissues of Reference Man and the daily intake of the element in food and fluids are based on a very small amount of data and that the values given may not be very reliable. Indeed the experimental data reviewed below, together with data on the heavier alkaline earths, suggest that the skeleton is likely to contain most of the body's beryllium.

2. Metabolic Model

(a) *Uptake to blood*

The mean fractional absorption of beryllium, administered as the chloride, from the gastrointestinal tract of four different mammalian species has been estimated as 0.006 (Furchner, Richmond and London, 1973). In experiments on rats, Bugryshev *et al.* (1974) have estimated the fractional gastrointestinal absorption of the element, again administered as the chloride, to be between 0.0014 and 0.0021 and a similar value is indicated from experiments on dairy cows (Mullen *et al.*, 1972). The fractional absorption of beryllium, administered as beryllium sulphate, from the gastrointestinal tract of rats is also typically 0.01 or less (Reeves, 1965). In this report f_1 is taken as 0.005 for all compounds of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, halides and nitrates of beryllium to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. However, various experimental studies (Van Cleave and Kaylor, 1955; Reeves and Vorwald, 1967; Suzuki *et al.*, 1972; Sanders, Cannon and Powers, 1978) indicate that beryllium oxide should be assigned to inhalation class Y and beryllium sulphate to inhalation class W. Early experiments by Van Cleave and Kaylor (1955) also indicate a long-term component of beryllium retention in the lung after intratracheal instillation of the citrate.

In this report, oxides, halides and nitrates of beryllium are assigned to inhalation class Y and all other commonly occurring compounds of the element are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	0.005
Y	0.005

(c) *Distribution and retention*

The distribution and retention of beryllium in the body after intravenous injection of the

chloride has been studied in mice, rats, monkeys and dogs (Furchner, Richmond and London, 1973). In each of these species whole body retention is well described by three exponentials with biological half-lives of between 0.2 and 1800 days. As is the case with the heavier alkaline earths, beryllium is predominantly deposited and retained in bone (Furchner, Richmond and London, 1973) although other organs such as liver and spleen contain high concentrations of the element in the first few days after intravenous injection of the sulphate (Van Cleave and Kaylor, 1953) or chloride (Mullen *et al.*, 1972). These high concentrations in liver and spleen may possibly be attributed to the rapid formation of colloids with blood proteins (Van Cleave and Kaylor, 1953). For beryllium entering the systemic circulation from the lung or gastrointestinal tract it is probably appropriate to neglect the liver and spleen as organs of deposition (Van Cleave and Kaylor, 1955; Reeves, 1965; Mullen *et al.*, 1972).

In this report it is assumed that of beryllium leaving the transfer compartment 0.4 is translocated to mineral bone and 0.2 is uniformly distributed throughout all other organs and tissues of the body. The remaining fraction of beryllium leaving the transfer compartment is assumed to go directly to excretion. Beryllium translocated to bone is assumed to be retained there with a biological half-life of 1500 days. Of beryllium translocated to any other organ or tissue fractions of 0.8 and 0.2 are assumed to be retained with biological half-lives of 15 and 1500 days respectively.

3. Classification of Isotopes for Bone Dosimetry

The only radioactive isotopes of beryllium considered in this report are ^{7}Be and ^{10}Be. Both these isotopes have radioactive half-lives of greater than 15 days. Thus, by analogy with the other alkaline earths, these isotopes of beryllium are assumed to be uniformly distributed throughout the volume of mineral bone at all times after their deposition in that tissue.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of beryllium

Radionuclide		Oral	Inhalation	
			Class W	Class Y
		$f_1 = 5 \times 10^{-3}$	$f_1 = 5 \times 10^{-3}$	$f_1 = 5 \times 10^{-3}$
^{7}Be	ALI	2×10^{9}	8×10^{8}	7×10^{8}
	DAC	—	3×10^{5}	3×10^{5}
^{10}Be	ALI	4×10^{7} (4×10^{7}) LLI Wall	6×10^{6}	5×10^{5}
	DAC	—	2×10^{3}	2×10^{2}

METABOLIC DATA FOR CARBON

1. Metabolism

Data from Reference Man (ICRP, 1975)

Carbon content of the body	16 kg
of adipose tissue	9.6 kg
of skeletal muscle	3.0 kg
of bone	0.7 kg
Daily intake in food and fluids	0.3 kg

The concentration of carbon in adipose tissue, including the yellow marrow, is about three times the average whole body concentration. No other organ or tissue of the body concentrates stable carbon to any significant extent.

2. Metabolic Models

In this report ALIs are given only for ^{11}C and ^{14}C labelled organic compounds and for ^{11}C and ^{14}C labelled gases such as carbon monoxide and carbon dioxide. It is emphasized that these ALIs are not appropriate for many other compounds of carbon and that they should be used with circumspection. As a guide to users of this report some discussion of the metabolism of various other compounds of carbon is included. However, an exhaustive review of the extensive literature has not been attempted.

(a) *Uptake to blood*

The fractional absorption of dietary carbon is usually in excess of 0.9. However, some carbon-containing compounds in food, such as cholesterol, fat-soluble vitamins, cellulose and polysaccharides may be less completely absorbed (Bell, Davidson and Emslie-Smith, 1972; ICRP, 1975; Clark and Harries, 1975; Ho *et al.*, 1979).

The fractional absorption of carbon administered in non-dietary forms is very variable. When administered as potassium cyanide (Crawley and Goddard, 1977) or methyl methacrylate (Bratt and Hathway, 1977) absorption is almost complete, whereas polydiethylstilboestrol, octanoic acid and hydrolyzed polyacrylonitrile grafted cellulose are almost completely unabsorbed (ICRP, 1975; Lai *et al.*, 1978).

In this report f_1 is taken to be 1 for organic compounds labelled with radioactive isotopes of carbon.

(b) *Inhalation classes*

There are three main classes of carbon compounds which may be inhaled, organic compounds, gases such as carbon monoxide and carbon dioxide, and aerosols of carbon containing compounds such as carbonates and carbides.

(i) *Organic compounds*

Most organic compounds are not very volatile under normal circumstances and the probability of their being inhaled as vapours is, therefore, small. In circumstances where such

substances are inhaled it would be prudent, as in the case of tritiated organic compounds, to assume that once they enter the respiratory system they are instantaneously and completely translocated to the systemic circulation without changing their chemical form.

(ii) *Gases*

The inhalation of carbon monoxide and its retention in body tissues has been studied extensively. When an individual is exposed to carbon monoxide a small amount of the gas dissolves in tissues. Since the gas has a relatively low solubility in tissue water, doses due to absorbed gas in tissues are insignificant in comparison with doses due to the retention of carbon monoxide bound to haemoglobin or, to a lesser extent, to other iron-haem containing compounds such as cytochrome oxidase (Göthert and Malorny, 1969; Luomanmäki and Coburn, 1969).

Studies of the formation and dissociation of carboxyhaemoglobin (Peterson and Stewart, 1970) indicate that the biological half-life of carbon monoxide in the blood is between 150 and 200 min. In contrast, pulsatile studies using ^{11}CO (Weinreich *et al.*, 1975) indicate that carbon monoxide is retained in blood and with a biological half-life varying from 210 min to more than 1 000 min, with an average of close to 600 min. Further, the data of Peterson and Stewart (1970) can be used in conjunction with the haemoglobin content of the blood of Reference Man (ICRP, 1975) to estimate that about 0.4 of inhaled carbon monoxide becomes bound to haemoglobin. This fraction is in good agreement with the fraction which can be estimated from studies on the pulsatile uptake of the gas (Menkes *et al.*, 1970).

In this report it is assumed that when radioactive carbon monoxide is inhaled 0.4 becomes instantaneously bound to haemoglobin and 0.6 is exhaled. Carbon monoxide bound to haemoglobin is assumed to be uniformly distributed throughout all organs and tissues of the body and retained with a biological half-life of 200 min. This half-life is taken on the basis of Peterson and Stewart's (1970) measurements, since the individuals in these studies were taking gentle exercise, whereas those studied by Weinreich *et al.* (1975) were resting throughout the period of study. It is noted that the degree of exercise may profoundly modify the retention of carbon monoxide in blood.

Although the free diffusion coefficient of carbon dioxide in water is a little less than that of oxygen its solubility coefficient in water is 24 times greater. Thus, carbon dioxide is transferred about 30 times more rapidly than oxygen across the alveolar membrane (Bell, Davidson and Emslie-Smith, 1972). In view of this it is appropriate to assume that all carbon dioxide entering the respiratory system is translocated to blood.

Carbon dioxide in the blood exists mainly as the bicarbonate. Further, since the total anions are insufficient to combine with the sodium ions found in plasma, the combined CO_2 must be present as sodium bicarbonate (Bell, Davidson and Emslie-Smith, 1972). Thus, the retention of inhaled CO_2 in the body can be determined from studies on the retention of intravenously injected sodium bicarbonate.

In studies of 13 normal subjects intravenously injected with ^{14}C-labelled bicarbonate (Winchell *et al.*, 1970) the whole-body retention of bicarbonate in the first 120 min after injection was well described by the function

$$R(t) = 0.175\, e^{-0.693t/5} + 0.825\, e^{-0.693t/60}$$

where t is in minutes.

However, animal studies (Buchanan and Nakao, 1955; Joftes, 1967) indicate that after breathing CO_2 there may be a small component of long-term carbon retention in tissues with a

biological half-life of 10 days or more. This is to be expected since a small fraction of carbon inhaled as CO_2 will be involved in biosynthesis or will exchange with bone carbonate. From the study discussed above (Winchell *et al.*, 1970) it is estimated that no more than about 0.01 of inhaled CO_2 is involved in such reactions.

In this report it is assumed that carbon inhaled as carbon dioxide is uniformly distributed throughout all organs and tissues of the body and that its retention is governed by the retention function.

$$R(t)=0.18\ e^{-0.693t/5}+0.81\ e^{-0.693t/60}+0.01\ e^{-0.693t/60\ 000}$$

where t is in minutes and 60 000 min is the biological half-life for dietary carbon as estimated from Reference Man (ICRP, 1975). This 60 000 min half-life is based on the assumption that the small fraction of carbon incorporated into tissues following inhalation of CO_2 will behave in a similar fashion to carbon entering the systemic circulation following ingestion of dietary carbon.

The uptake and retention of inhaled carbon-containing gases other than carbon monoxide and carbon dioxide is not considered in this report.

(iii) *Carbonates and carbides*

Aerosols containing carbon in the form of a carbonate or carbide in combination with a particular element are not considered in this report. However, some insight on their retention in the lung may be obtained from the metabolic data for the appropriate element or from the report of the Task Group on Lung Dynamics (1966).

(c) *Distribution and retention*

Only two isotopes of carbon are considered in this report, ^{11}C and ^{14}C. Because of their very different radioactive half-lives it is convenient to consider each of these isotopes separately.

^{11}C has a radioactive half-life of 20.38 min. Therefore, little of this isotope will be excreted from the body. This is illustrated by considering the retention of intravenously injected ^{11}C labelled sodium bicarbonate. Even in this case, the retention function given by Winchell *et al.* (1970) indicates that no more than 0.35 of the ^{11}C will escape from the body before decaying. As a result of its short radioactive half-life, it is reasonable to assume that ^{11}C is uniformly distributed throughout all organs and tissues of the body at all times following its entry into the systemic circulation. However, it should be noted that this assumption is invalid for certain ^{11}C labelled substances, e.g. methionine and certain psychoactive drugs (Comar *et al.*, 1976; Mestelan, Crouzel and Comar, 1977; Berger *et al.*, 1978).

In this report it is assumed that inhaled or ingested ^{11}C labelled compounds are instantaneously uniformly distributed throughout all organs and tissues of the body where they are retained indefinitely.

^{14}C has a radioactive half-life of 5 730 years and its radioactive decay can be neglected in the context of this report.

Data from Reference Man (ICRP, 1975) suggest that the biological half-life of dietary carbon in the body is about 40 days. However, the retention of carbon derived from various metabolites in various organs and tissues of the body shows considerable variation. For example, studies on autopsy samples of people exposed to ^{14}C from fallout (Harkness and Walton, 1972; Stenhouse and Baxter, 1977) indicate that bone collagen and bone mineral retain carbon with a biological half-life in excess of 5 years.

Long-term studies on the exhalation of $^{14}CO_2$ after the intravenous injection of glycine-2-^{14}C

into six patients demonstrated four components of ^{14}C retention with biological half-lives of 0.1, 1, 6 and 70 days (Berlin and Tolbert, 1955). Four components of retention with biological half-lives of 0.1, 0.6, 2 and 17 days were found after intravenous injection of glycine or acetate into patients suffering from various diseases (Hellman *et al.*, 1953).

More recently there have been long-term studies on the retention of ^{14}C in the rat after intravenous injection of DTPA (Crawley and Haines, 1979), potassium cyanide (Crawley and Goddard, 1977) and methanol (Crawley, 1977). In these studies much of the ^{14}C is found to be lost very rapidly from the body, but components of retention with biological half-lives of up to 126 days were demonstrated for fat and muscle.

In this report it is assumed that inhaled or ingested ^{14}C labelled compounds are instantaneously uniformly distributed throughout all organs and tissues of the body where they are retained with a biological half-life of 40 days.

It is considered that this assumption will yield realistic whole body doses for ^{14}C labelled metabolites and that it will overestimate whole body doses from most other ^{14}C labelled compounds.

(d) *Microdosimetric considerations*

As with certain tritiated compounds, there are particular compounds of ^{14}C, such as ^{14}C-thymidine, which are specifically incorporated into the DNA of dividing cells. In the case of tritiated compounds it was noted that the appropriate ALI could be as little as 0.02 of the ALI for tritiated water, depending upon the compound. However, the average energy of the β particle from ^{14}C decay is about nine times the average energy of the β particle from ^{3}H decay. Thus, tissues are irradiated much more uniformly from ^{14}C incorporated into DNA than they are from ^{3}H incorporated into DNA. It may be shown (Feinendegen, 1979) that considerations of the absorbed dose in ^{14}C labelled cell nuclei will not result in a more restrictive value of ALI than that derived from consideration of mean dose to body tissues from ^{14}C labelled compounds as discussed in Section (c)). However, it should be noted that β-ray dose is not the only consideration and, as in the case of ^{3}H-5-deoxycytosine, transmutation effects may sometimes be of importance (Feinendegen and Cronkite, 1977). Such effects are considered to be of importance only in case of specific placement of ^{14}C into molecular positions where transmutation effects may arise. The placement of ^{14}C into such positions from ^{14}C labelled compounds has a very low probability.

3. Classification of Isotopes for Bone Dosimetry

Both ^{11}C and ^{14}C are assumed to be uniformly distributed throughout all organs and tissues of the body. Therefore, a classification of isotopes of the element for the purpose of bone dosimetry is not required.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of carbon

Labelled organic compounds

Radionuclide		Oral	Inhalation
^{11}C	ALI	2×10^{10}	2×10^{10}
	DAC	—	6×10^{6}
^{14}C	ALI	9×10^{7}	9×10^{7}
	DAC	—	4×10^{4}

Carbon monoxide

Radionuclide		Inhalation
^{11}C	ALI	4×10^{10}
	DAC	2×10^{7}
^{14}C	ALI	6×10^{10}
	DAC	3×10^{7}

Carbon dioxide

Radionuclide		Inhalation
^{11}C	ALI	2×10^{10}
	DAC	1×10^{7}
^{14}C	ALI	8×10^{9}
	DAC	3×10^{6}

METABOLIC DATA FOR MAGNESIUM

1. Metabolism

Data from Reference Man (ICRP, 1975)

Magnesium content of the body	19 g
of soft tissue	7.8 g
of the skeleton	11 g
Daily intake in food and fluids	0.34 g

2. Metabolic Model

(a) *Uptake to blood*

The fractional gastrointestinal absorption of dietary forms of magnesium is typically 0.2–0.7 (Wacker and Vallee, 1962; Shils, 1973; Schwartz, Spencer and Wentworth, 1978). Recent studies have shown that almost all this variation can be attributed to different levels of magnesium in the diet. Absorption varies from 0.70 ± 0.11 for ingestion of 0.3 mmol of magnesium to 0.14 ± 0.04 for ingestion of 41.7 mmol (Roth and Werner, 1979). In this report f_1 is taken to be 0.5 for all compounds of magnesium.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, carbides, halides and nitrates of magnesium to inhalation class W and all other compounds of the element to inhalation class D. In the absence of any relevant experimental data this classification is adopted here.

Inhalation class	f_1
D	0.5
W	0.5
Y	—

(c) *Distribution and retention*

Stable magnesium is known to be concentrated in mineral bone (Wacker and Vallee, 1962; ICRP, 1975), this tissue being thought to act as a long-term reservoir for soft tissue requirements during deficiency (Wacker and Vallee, 1962).

Studies in man (Silver *et al.*, 1960; Yun *et al.*, 1966; Roessler, 1972) indicate components of retention of magnesium in man with biological half-lives of between 0.25 and 35 days. However, long-term retention probably depends on the level of stable magnesium in the diet (Shils, 1973).

In this report it is assumed that of magnesium leaving the transfer compartment 0.2 goes directly to excretion, 0.4 to mineral bone and 0.4 is distributed uniformly throughout all other organs and tissues of the body. Magnesium translocated to any organ or tissue of the body including mineral bone is assumed to be retained there with a biological half-life of 100 days.

3. Classification of Isotopes for Bone Dosimetry

Since none of the isotopes of magnesium considered in this report has a radioactive half-life of more than 1 day it is appropriate to assume that magnesium is uniformly distributed over the surfaces of mineral bone at all times following its deposition in that tissue.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of magnesium

			Inhalation	
		Oral	Class D	Class W
Radionuclide		$f_1 = 5 \times 10^{-1}$	$f_1 = 5 \times 10^{-1}$	$f_1 = 5 \times 10^{-1}$
^{28}Mg	ALI	2×10^{7}	6×10^{7}	5×10^{7}
	DAC	—	3×10^{4}	2×10^{4}

METABOLIC DATA FOR ALUMINIUM

1. Metabolism

Data from Reference Man (ICRP, 1975)

Aluminium content of the body	61 mg
of soft tissues	40 mg
of the skeleton	21 mg
Daily intake in food and fluids	45 mg

2. Metabolic Model

(a) *Uptake to blood*

There is general agreement that the fractional absorption of aluminium from the gastrointestinal tract is probably not more than 0.01 (ICRP, 1975; Underwood, 1977; Berlyne and Rubin, 1977; Kaehny, Hegg and Alfrey, 1977). In this report f_1 is taken to be 0.01 for all compounds of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, carbides, halides and nitrates of aluminium to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. Data on rats and hamsters (Klosterkotter, 1960; Christie, MacKay and Fisher, 1963) support this classification and it is adopted here. In addition, metallic aluminium is assigned to inhalation class W.

Inhalation class	f_1
D	0.01
W	0.01
Y	—

(c) *Distribution and retention*

The concentration of stable aluminium is greater in the lungs and skeleton than in the remainder of the body (ICRP, 1975). However, the high concentration in the lungs may be due to the inhalation of aluminium in natural dust or industrial fallout (ICRP, 1975; Underwood, 1977). In rats, ^{28}Al intravenously injected as the chloride, was rapidly taken up by the liver (Kushelevsky *et al.*, 1976). This was probably the result of the formation of an insoluble colloid which was sequestered by the reticuloendothelial system, since experiments in which rats were fed a diet containing 1% $Al_2(SO_4)_3$ indicated that the element was not preferentially concentrated in any organ or tissue except the skeleton (Berlyne *et al.*, 1972).

In this report it is assumed that of aluminium leaving the transfer compartment 0.3 is translocated to mineral bone and 0.7 is uniformly distributed throughout all other organs and

tissues of the body. Aluminium deposited in any organ or tissue is assumed to be retained there with a biological half-life of 100 days, a value compatible with the daily intake and total body content of the element given for Reference Man (ICRP, 1975).

3. Classification of Isotopes for Bone Dosimetry

There appear to be no data available on the distribution of aluminium in the skeleton. Because, like the lanthanides, the predominant valence state of aluminium is 3+, isotopes of the element are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of aluminium

			Inhalation	
		Oral	Class D	Class W
Radionuclide		$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$
^{26}Al	ALI	1×10^{7}	2×10^{6}	3×10^{6}
	DAC	—	1×10^{3}	1×10^{3}

METABOLIC DATA FOR SILICON

1. Metabolism

Data from Reference Man (ICRP, 1975)

Silicon content of the body	not given
Silicon content of blood	140 mg
Daily intake in food and fluids	3.5 mg
Daily intake of airborne silicon	~15 mg

Although data on the distribution of stable silicon in Reference Man are limited, data reviewed by King and Belt (1938) indicate that the element is uniformly distributed throughout all organs and tissues except the lung and peribronchial lymph glands. The high concentrations in the lungs and thoracic lymph nodes occur because of the inhalation of silicaceous dusts. Data from Iyengar, Kollmer and Bowen (1978) indicate that the total body content of silicon is between 1 and 3 g.

2. Metabolic Model

(a) *Uptake to blood*

Dietary silicon enters the gastrointestinal tract as monosilicic acid, as solid silica and in organic bound forms such as pectin and mucopolysaccharides (Underwood, 1977). Various studies on guinea-pigs, sheep and cattle (Sauer, Laughland and Davidson, 1959; Nottle, 1966; Nottle and Armstrong, 1966; Bailey, 1967) have shown that the fractional absorption of SiO_2 or dietary silicon from the gastrointestinal tract is typically in the range 3×10^{-3} to 3×10^{-2}, with the larger fractional absorption occurring in studies on animals fed diets low in silicon. It should be noted that recent balance studies on man have indicated that the fractional absorption of dietary silicon can be as much as 0.5 when the silicon is ingested as part of a low fibre diet (Kelsay, Behall and Prather, 1979). In this report f_1 is taken to be 0.01 for all compounds of silicon.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, carbides and nitrates of silicon to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. Data from experiments on rats (ICRP, 1966) tend to support this classification and it is adopted here.

Various studies using fused clay particles (Bailey, Hostford and Strong, 1977; Bailey *et al.*, 1978) indicate that silicon inhaled as an aluminosilicate glass aerosol should be assigned to inhalation class Y.

Inhalation class	f_1
D	0.01
W	0.01
Y	0.01

(c) *Distribution and retention*

Like germanium, silicon appears to be fairly uniformly distributed throughout all organs and tissues of the body at early times after its introduction into the systemic circulation (Mehard and Volcani, 1975). However, studies on mice (Holt, 1950) and guinea-pigs (Sauer, Laughland and Davidson, 1959) indicate that silicon is retained in the body considerably longer than is germanium. In the experiments on guinea-pigs two components of retention were found with biological half-lives of about 5 days and 100 days respectively.

In this report it is assumed that silicon leaving the transfer compartment is uniformly distributed throughout all organs and tissues of the body. Of silicon deposited in any organ or tissue, fractions of 0.4 and 0.6 are assumed to be retained with biological half-lives of 5 and 100 days respectively. This retention function is compatible with the total body content of silicon estimated from Iyengar, Kollmer and Bowen, provided that allowance is made for the translocation of inhaled silicon from the lungs to the systemic circulation.

3. Classification of Isotopes for Bone Dosimetry

Although silicon is known to be involved in the processes of bone mineralization it appears only to be incorporated into specific growth areas and is not present to any great extent in mature apatite (Underwood, 1977). Thus, although silicon is of fundamental importance in bone mineralization and cartilage formation it is probably not concentrated in the skeleton to any significant extent. In this report silicon is assumed to be uniformly distributed throughout all organs and tissues of the body. Therefore, a classification of isotopes of the element for the purposes of bone dosimetry is not required.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of silicon

Radionuclide		Oral	Inhalation		
			Class D	Class W	Class Y
		$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$
^{31}Si	ALI	3×10^{8}	9×10^{8}	1×10^{9}	1×10^{9}
	DAC	—	4×10^{5}	5×10^{5}	4×10^{5}
^{32}Si	ALI	8×10^{7} (1×10^{8}) LLI Wall	9×10^{6}	4×10^{6}	2×10^{5}
	DAC	—	4×10^{3}	2×10^{3}	8×10^{1}

METABOLIC DATA FOR SCANDIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for scandium.

2. Metabolic Model

(a) *Uptake to blood*

There appear to be few data on the absorption of scandium from the gastrointestinal tract. There appears to be little absorption of the element when administered as ^{46}Sc tagged sand (Miller, Byrne and Lyke, 1972) or as scandium chloride (Miller and Byrne, 1970). By analogy with yttrium, f_1 is here taken to be 10^{-4} for all compounds of the element.

(b) *Inhalation classes*

Data from experiments on dogs indicate that $ScCl_3$ and $Sc(OH)_3$ are retained in the lungs with biological half-lives in excess of 700 days (Morrow *et al.*, 1979). In this report all commonly occurring compounds of scandium are assigned to inhalation Class Y.

Inhalation class	f_1
D	—
W	—
Y	10^{-4}

(c) *Distribution and retention*

Various studies have demonstrated that intravenously and intramuscularly injected scandium is preferentially deposited in liver, kidney, spleen, bone and to some extent lung (Durbin, 1960; Rosoff *et al.*, 1963; Rosoff *et al.*, 1965; Hara and Freed, 1972; Byrd *et al.*, 1975; Lachine *et al.*, 1976). However, the details of the distribution vary considerably from experiment to experiment. While colloid formation after injection may be partly responsible for this variation it is not thought to offer a complete explanation (Byrd *et al.*, 1975).

In man the whole body retention of scandium has been studied for 584 days after the intravenous injection of ^{46}Sc nitrilotriacetate (Rosoff *et al.*, 1965). In these studies fractions of 0.1 and 0.9 of the injected scandium were found to be retained in the body with biological half-lives of 5 and 1 500 days respectively. Further, autopsy data on three of the patients studied demonstrated that scandium was retained mainly in the spleen, the liver and the skeleton. Experiments on rats (Byrd *et al.*, 1975) confirm that a large fraction of intravenously injected scandium is avidly retained in the body.

In this report it is assumed that of scandium leaving the transfer compartment fractions of 0.4, 0.3 and 0.1 are translocated to the skeleton, the liver and the spleen respectively. The remaining fraction of scandium leaving the transfer compartment is assumed to be uniformly distributed throughout all other organs and tissues of the body. Of scandium deposited in any organ or

tissue, fractions of 0.1 and 0.9 are assumed to be retained with biological half-lives of 5 and 1 500 days respectively.

(d) *Chelated compounds*

Chelated forms of scandium are not considered in this report. Their metabolism differs considerably from that of other forms of the element (Lachine, Noujaim, Ediss and Wiebe, 1976).

3. Classification of isotopes for bone dosimetry

Autoradiographic studies on a rabbit indicate that, 18 h after intravenous injection of the citrate, scandium in the skeleton is primarily associated with the bone marrow rather than mineral bone (Hara and Freed, 1973). In rats, 11 months after intravenous injection of the citrate, concentrations of scandium in bone and bone marrow are very similar (Byrd, Watson, Cloutier and Hayes, 1975). For the purposes of radiological protection it is assumed that all isotopes of scandium are uniformly distributed throughout bone and bone marrow at all times following their deposition in the skeleton.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of scandium

			Inhalation
		Oral	Class Y
Radionuclide		$f_1 = 1 \times 10^{-4}$	$f_1 = 1 \times 10^{-4}$
^{43}Sc	ALI	3×10^{8}	8×10^{8}
	DAC	—	4×10^{5}
^{44m}Sc	ALI	2×10^{7}	3×10^{7}
	DAC	—	1×10^{4}
^{44}Sc	ALI	1×10^{8}	4×10^{8}
	DAC	—	2×10^{5}
^{46}Sc	ALI	3×10^{7}	9×10^{6}
	DAC	—	4×10^{3}
^{47}Sc	ALI	8×10^{7} (1×10^{8}) LLI Wall	1×10^{8}
	DAC	—	5×10^{4}
^{48}Sc	ALI	3×10^{7}	5×10^{7}
	DAC	—	2×10^{4}
^{49}Sc	ALI	8×10^{8}	2×10^{9}
	DAC	—	8×10^{5}

METABOLIC DATA FOR TITANIUM

1. Metabolism

Data from Reference Man (ICRP, 1975)	
Titanium content of soft tissues	9 mg
of the lungs	2.4 mg
Daily intake in food and fluids	0.85 mg

2. Metabolic Model

(a) *Uptake to blood*

The daily excretion of titanium in the urine of man has been variously reported as 10 μg (Perry and Perry, 1959) and 340 μg (Tipton, Stewart and Martin, 1966). In the latter study both individuals were in negative titanium balance so the titanium in urine may have derived primarily from body stores of the element (Underwood, 1977). If the value of 10 μg is taken as representative this would indicate that the fractional absorption of dietary titanium is probably about 0.01. A fractional absorption of 0.01 was obtained in experiments in which sheep ingested titanium chloride (Miller, Madsen and Hansard, 1976). In this report f_1 is taken to be 0.01 for all compounds of titanium.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, carbides, halides and nitrates of titanium to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. Experiments on the retention of TiO_2 in rats after inhalation (Ferin, 1971; Ferin and Leach, 1973; Ferin and Feldstein, 1978) support this classification. However, data from experiments on $SrTiO_3$ suggest that this compound should be assigned to inhalation class Y (Fish *et al.*, 1964). In this report $SrTiO_3$ is assigned to inhalation class Y, oxides, hydroxides, carbides, halides and nitrates of titanium are assigned to inhalation class W and all other commonly occurring compounds of the element are assigned to inhalation class D.

Inhalation class	f_1
D	0.01
W	0.01
Y	0.01

(c) *Distribution and retention*

Stable titanium is fairly uniformly distributed throughout all organs and tissues of the body except for the lung (ICRP, 1975). The high levels of titanium sometimes found in the lungs are probably due to the inhalation of titanium-containing dusts (Underwood, 1977).

In lambs, injected intravenously with $TiCl_4$ and killed 48 h later, about 0.25 of the titanium

was found in the skeleton, 0.18 in blood, 0.13 in skeletal muscle and 0.11 in internal organs (Miller, Madsen and Hansard, 1976). In mice, chronically exposed to titanium in drinking water, none of the tissues examined seemed to preferentially concentrate the element to any appreciable extent (Schroeder, Balassa and Tipton, 1963).

In lambs, only about 0.04 of intravenously injected titanium is excreted in the first 2 days after injection (Miller, Madsen and Hansard, 1976). Data on the body content and daily intake of titanium of Reference Man (ICRP, 1975) suggest, when taken in conjunction with an f_1 of 0.01, that the biological half-life of titanium in the body is about 600 days.

In this report it is assumed that titanium leaving the transfer compartment is uniformly distributed throughout all organs and tissues of the body and that it is retained in those organs and tissues with a biological half-life of 600 days.

3. Classification of Isotopes for Bone Dosimetry

Titanium is assumed to be uniformly distributed throughout all organs and tissues of the body. Therefore, a classification of isotopes of the element for the purposes of bone dosimetry is not required.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of titanium

			Inhalation		
		Oral	Class D	Class W	Class Y
Radionuclide		$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$
^{44}Ti	ALI	1×10^{7}	4×10^{5}	1×10^{6}	2×10^{5}
	DAC	—	2×10^{2}	4×10^{2}	9×10^{1}
^{45}Ti	ALI	3×10^{8}	9×10^{8}	1×10^{9}	1×10^{9}
	DAC	—	4×10^{5}	5×10^{5}	4×10^{5}

METABOLIC DATA FOR VANADIUM

1. Metabolism

Data from Reference Man (ICRP, 1975)

Vanadium content of adipose tissues	22 mg
Daily intake in food and fluids	2 mg

2. Metabolic Model

(a) *Uptake to blood*

Data from Reference Man (ICRP, 1975) indicate that the fractional absorption of dietary vanadium from the gastrointestinal tract is about 0.01. Further, experiments on rabbits (Curran and Costello, 1956) have shown that the fractional absorption of vanadium, administered as $VOSO_4 \cdot 2H_2O$, is also about 0.01. However, studies on man (Curran, Azarnoff and Bolinger, 1959) indicate that the fractional absorption of vanadium, administered as diammonium oxytartratovanadate, is very variable, ranging from 7×10^{-5} to 2×10^{-3}.

In this report f_1 is taken to be 0.01 for all compounds of vanadium, acknowledging that this is likely to overestimate the gastrointestinal absorption of some compounds of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, carbides and halides of vanadium to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. Experiments on the translocation of vanadium from the lungs of the rat after intratracheal instillation of $VOCl_2$ (Pépin, Bouley and Boudene, 1977) support this classification and it is adopted here.

Inhalation class	f_1
D	0.01
W	0.01
Y	—

(c) *Distribution and retention*

In experiments on mice intravenously injected with $^{48}V_2O_5$ (Söremark and Üllberg, 1961) about 0.7 of the total body content of vanadium was found in the skeleton between 1 and 21 days after injection. In the rabbit about 0.8 of the total body content of vanadium was found in the skeleton after repeated daily intravenous injections of sodium metavanadate monohydrate (Talvitie and Wagner, 1954). However, in experiments in which $^{48}VO_2$ was administered to rats by intravenous injection (Hopkins and Tilton, 1966) the concentration of vanadium was greater in the kidneys, testes, liver and spleen than it was in other organs and tissues of the body in the first 96 hours after injection.

Experiments on mice and rats (Talvitie and Wagner, 1954; Söremark and Üllberg, 1961; Hopkins and Tilton, 1966; Ordzhonikidze *et al.*, 1977; Pépin, Bouley and Boudene, 1977) indicate that between 0.5 and 0.8 of intravenously or intraperitoneally injected vanadium is excreted in the first 24 hours after injection but that the remainder is only excreted very slowly. Data from Reference Man (ICRP, 1975) suggests that this component of long-term retention probably has a biological half-life in excess of 5 000 days.

In this report it is assumed that of vanadium leaving the transfer compartment 0.7 goes directly to excretion, 0.25 is translocated to mineral bone and 0.05 is uniformly distributed throughout all other organs and tissues of the body. Vanadium deposited in any organ or tissue is assumed to be retained there with a biological half-life of 10 000 days.

3. Classification of Isotopes for Bone Dosimetry

There do not appear to be any relevant data available concerning the distribution of vanadium in the skeleton. In this report ^{49}V, which has a radioactive half-life of 330 days, is assumed to be uniformly distributed throughout the volume of mineral bone at all times following its deposition in that tissue. All other, shorter lived, isotopes of vanadium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of vanadium

			Inhalation	
		Oral	Class D	Class W
Radionuclide		$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$
^{47}V	ALI	1×10^9 (1×10^9) ST Wall	3×10^9	4×10^9
	DAC	—	1×10^6	2×10^6
^{48}V	ALI	2×10^7	4×10^7	2×10^7
	DAC	—	2×10^4	9×10^3
^{49}V	ALI	3×10^9 (3×10^9) LLI Wall	1×10^9 (1×10^9) Bone surf.	7×10^8
	DAC	—	5×10^5	3×10^5

METABOLIC DATA FOR NICKEL

1. Metabolism

Data from Reference Man (ICRP, 1975)	
Nickel content of the body	10.0 mg
of soft tissues	5.3 mg
of the skeleton	< 5.0 mg
Daily intake in food and fluids	0.4 mg

2. Metabolic Model

(a) *Uptake to blood*

The fractional uptake of dietary nickel from the gastrointestinal tract is thought to be small (ICRP, 1975; Underwood, 1977) with the data of Horak and Sunderman (1973) indicating an f_1 of 0.01. However, f_1 values of as much as 0.5 have sometimes been reported (ICRP, 1975; Veterans Administration Hospital, 1975). The fractional gastrointestinal absorption of nickel oxide is probably less than 0.01 (Wehner and Craig, 1972). In this report f_1 is taken as 0.05 for all compounds of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, halides, carbides and nitrates of nickel to inhalation class W and all other compounds of the element to inhalation class D. Results of experiments in which hamsters inhaled NiO (Wehner and Craig, 1972) support this classification. However, recent data by Ziemer and Carvalho (1980) suggest that nickel chloride should be reassigned to inhalation class D. In this report oxides, hydroxides and carbides of nickel are assigned to inhalation class W and all other commonly occurring compounds of the element are assigned to inhalation class D.

Inhalation class	f_1
D	0.05
W	0.05
Y	—

When finely divided nickel or its compounds come into contact with carbon monoxide the vapour nickel carbonyl [$Ni(CO)_4$] is formed. Following inhalation, nickel carbonyl is almost completely absorbed from the lungs (Tedeschi and Sunderman, 1957; Sunderman and Selin, 1968; Committee on medical and biologic effects of environmental pollutants, 1975). However, autoradiographic studies on mice indicate that nickel inhaled as nickel carbonyl is at least briefly retained on parenchymal lung surfaces before its entry into the systemic circulation (Oskarsson and Tjälve, 1977). Nickel entering the systemic circulation as nickel carbonyl is broken down in the red cells, and other tissues, to Ni^0 and CO. The Ni^0 is oxidized intracellularly to Ni^{2+} and released to the blood serum, where it behaves similarly to

intravenously injected nickel compounds (Committee on medical and biologic effects of environmental pollutants, 1975). In this report it is assumed that all nickel entering the respiratory system as nickel carbonyl is deposted there and that it is then translocated to the transfer compartment with a biological half-life of 0.1 days. After entry into the transfer compartment the metabolic model for other inorganic compounds of nickel is assumed to apply.

(c) *Distribution and retention*

In mice, rats and rabbits, nickel is preferentially deposited in the kidneys (Smith and Hackley, 1968; Parker and Sunderman, 1974; Jacobsen, Alfheim and Jonsen, 1978; Olsen and Jonsen, 1979). However, data from Reference Man (ICRP, 1975) indicate that the concentration of stable nickel in the kidneys is similar to the average concentration in all soft tissues. It is probably appropriate to attribute the high concentration of nickel in the kidneys at early times after intravenous or intraperitoneal injection to the rapid excretion of the element in the urine (Smith and Hackley, 1968; Onkelinx, Becker and Sunderman, 1973; Olsen and Jonsen, 1979).

In rats a fraction of between 0.5 and 0.8 of a single intravenous injection of nickel, administered as the chloride, is excreted in the first 12 h after injection. After this early phase of rapid excretion the remaining nickel is avidly retained, with the total lost in urine and faeces rising to between 0.6 and 0.9, 72 h after injection (Smith and Hackley, 1968; Sunderman and Selin, 1968).

The experiments of Smith and Hackley (1968) on rats demonstrated an initial phase of nickel clearance from blood with a biological half-life of about 1 h followed by a slower phase with a biological half-life of about 6 h. Other authors (Onkelinx, Becker and Sunderman, 1973) have not observed the first phase of rapid clearance but have observed the slower second phase.

In this report it is assumed that of nickel leaving the transfer compartment a fraction, 0.02, is translocated to the kidneys where it is retained with a biological half-life of 0.2 days. A second fraction, 0.68, is assumed to go directly to excretion without any delay in the kidneys. The remainder of nickel leaving the transfer compartment is assumed to be uniformly distributed throughout all organs and tissues of the body including the kidneys and retained there with a biological half-life of 1200 days. This half-life is compatible with an f_1 value of 0.05 and the daily intake and total body content of nickel given for Reference Man.

3. Classification of Isotopes for Bone Dosimetry

Nickel has not been found to have any affinity for mineralized tissues (Jacobsen, Alfheim and Jonsen, 1978) and, in this report, is assumed to be uniformly distributed throughout all organs and tissues of the body other than the kidneys. Therefore, a classification of isotopes of the element for the purpose of bone dosimetry is not required.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of nickel

Inorganic

			Inhalation	
		Oral	Class D	Class W
Radionuclide		$f_1 = 5 \times 10^{-2}$	$f_1 = 5 \times 10^{-2}$	$f_1 = 5 \times 10^{-2}$
^{56}Ni	ALI	5×10^{7}	7×10^{7}	5×10^{7}
	DAC	—	3×10^{4}	2×10^{4}
^{57}Ni	ALI	6×10^{7}	2×10^{8}	1×10^{8}
	DAC	—	7×10^{4}	5×10^{4}
^{59}Ni	ALI	9×10^{8}	1×10^{8}	3×10^{8}
	DAC	—	6×10^{4}	1×10^{5}
^{63}Ni	ALI	3×10^{8}	6×10^{7}	1×10^{8}
	DAC	—	2×10^{4}	4×10^{4}
^{65}Ni	ALI	3×10^{8}	9×10^{8}	1×10^{9}
	DAC	—	4×10^{5}	5×10^{5}
^{66}Ni	ALI	1×10^{7} (2×10^{7}) LLI Wall	6×10^{7}	2×10^{7}
	DAC	—	2×10^{4}	1×10^{4}

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of nickel

Vapours

Radionuclide		Inhalation
^{56}Ni	ALI	4×10^{7}
	DAC	2×10^{4}
^{57}Ni	ALI	2×10^{8}
	DAC	1×10^{5}
^{59}Ni	ALI	7×10^{7}
	DAC	3×10^{4}
^{63}Ni	ALI	3×10^{7}
	DAC	1×10^{4}
^{65}Ni	ALI	6×10^{8}
	DAC	3×10^{5}
^{66}Ni	ALI	1×10^{8}
	DAC	5×10^{4}

METABOLIC DATA FOR GALLIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for gallium.

2. Metabolic Model

(a) *Uptake to blood*

The work of Dudley and Levine (1949) showed that little or no gallium administered as the chloride is absorbed from the gastrointestinal tract of the rat. In this report, f_1 is taken to be 10^{-3} for all compounds of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, carbides, halides and nitrates of gallium to inhalation class W and all other compounds of the element to inhalation class D. In the absence of any relevant experimental data this classification is adopted here.

Inhalation class	f_1
D	10^{-3}
W	10^{-3}
Y	—

(c) *Distribution and retention*

There is a very large literature concerning the distribution and retention of gallium in various species after intravenous, intramuscular or subcutaneous injection. Experiments on rats have shown that the distribution and retention of radioactive gallium in the body is very much influenced by the total amount of stable gallium administered as carrier (Bruner, Hayes and Perkinson, 1953; Hayes, Carlton and Byrd, 1965; Hayes, 1966). In the presence of stable carrier, radioactive gallium is strongly concentrated in bone, whereas in the case of intravenous injection of carrier free radio-gallium as the citrate the radionuclide is much more diffusely distributed (Hayes, Carlton and Byrd, 1965). In this report a metabolic model appropriate for carrier free gallium is used.

One to 2 h after the intravenous injection of carrier free radio-gallium as the citrate into rats the gallium is fairly uniformly distributed throughout all organs and tissues of the body although the skeleton shows some excess (Hayes, Carlton and Byrd, 1965; Hayes, 1966). By 24 h after injection gallium is predominantly found in the liver, kidney, spleen and skeleton (Bruner, Hayes and Perkinson, 1953).

In man, autopsy data (Brucer *et al.*, 1953; Nelson *et al.*, 1972; Simpkins, Fink and Prasad, 1977) and gamma scanning (Larson, Milder and Johnston, 1973; Newman *et al.*, 1978) identify these same organs together with the adrenal gland and, in the pregnant female, the placenta as

predominant sites of gallium deposition. There is also some evidence that gallium is concentrated in the walls of the gastrointestinal tract (Newstead *et al.*, 1977). However, these data are derived from samples of normal tissue taken close to gastrointestinal tumours.

Studies in rats, rabbits, dogs and man (Munn, Walters and Dudley, 1951; Bruner, Hayes and Perkinson, 1953; Langhammer *et al.*, 1972; Nelson, *et al.*, 1972) indicate that the retention of gallium in the body is adequately described by two exponentials with biological half-lives of about 1 day and 50 days respectively. However, it is noted that in the rat the biological half-life in different organs varies considerably.

In this report it is assumed that of gallium leaving the transfer compartment fractions of 0.3, 0.09 and 0.01 are deposited in mineral bone, liver and spleen respectively. The remaining fraction of gallium leaving the transfer compartment is assumed to be distributed uniformly throughout all other organs and tissues of the body. Of gallium deposited in any organ or tissue, fractions of 0.3 and 0.7 are assumed to be retained with biological half-lives of 1 and 50 days respectively.

(d) *Chelated forms*

Chelated forms of gallium are not considered in this report. Their metabolism differs considerably from that of other compounds of the element (Konikowski, Glenn and Haynie, 1973).

3. Classification of Isotopes for Bone Dosimetry

None of the isotopes of gallium considered in this report has a radioactive half-life of greater than 4 days. For this reason all isotopes of gallium considered here are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of gallium

Radionuclide		Oral	Inhalation	
			Class D	Class W
		$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$
^{65}Ga	ALI	2×10^9 (2×10^9) ST Wall	6×10^9	7×10^9
	DAC	—	3×10^6	3×10^6
^{66}Ga	ALI	4×10^7	1×10^8	1×10^8
	DAC	—	5×10^4	4×10^4
^{67}Ga	ALI	3×10^8	5×10^8	4×10^8
	DAC	—	2×10^5	2×10^5
^{68}Ga	ALI	6×10^8	2×10^9	2×10^9
	DAC	—	6×10^5	8×10^5
^{70}Ga	ALI	2×10^9 (3×10^9) ST Wall	6×10^9	7×10^9
	DAC	—	3×10^6	3×10^6
^{72}Ga	ALI	4×10^7	1×10^8	1×10^8
	DAC	—	5×10^4	5×10^4
^{73}Ga	ALI	2×10^8	6×10^8	6×10^8
	DAC	—	2×10^5	2×10^5

METABOLIC DATA FOR GERMANIUM

1. Metabolism

Although no body content of germanium is given for Reference Man the daily intake of the element in food and fluids is estimated to be 1.5 mg (ICRP, 1975). Data from Iyengar, Kollmer and Bowen (1978) indicate that the body content of germanium is about 2 mg.

2. Metabolic Model

(a) *Uptake to blood*

Data on the germanium content of urine suggest that dietary forms of the element are well absorbed from the gastrointestinal tract of man (Schroeder and Balassa, 1967). In experiments on rats, germanium, orally administered in the form of GeO_2, was almost completely absorbed from the gastrointestinal tract (Rosenfeld, 1954). In this report f_1 is taken as 1 for all compounds of germanium.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, sulphides and halides of germanium to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. In the absence of any relevant experimental data this classification is adopted here.

Inhalation class	f_1
D	1
W	1
Y	—

(c) *Distribution and retention*

Germanium is widely and fairly uniformly distributed throughout all organs and tissues of the rat 12 h to 3 days after both the intraperitoneal injection of sodium germanate (Rosenfeld, 1954), and chronic exposure to the same compound in drinking water from weaning to death (Schoeder *et al.*, 1968). However, during the first 6 h after intraperitoneal injection, the kidney concentration is about five times greater than the concentration in other tissues (Rosenfeld, 1954). High concentrations of germanium in the kidney of the rat are also reported after intravenous injection of $Ge(OH)_4$ (Mehard and Volcani, 1975) and after intramuscular injection of sodium germanate (Durbin, 1960). This excess of germanium in the kidneys at early times after injection is probably attributable to the rapid excretion of the element in the urine (Rosenfeld, 1954; Durbin, 1960).

In this report it is assumed that of germanium leaving the transfer compartment 0.5 goes directly to the kidneys and is retained in them with a biological half-life of 0.02 days before being excreted in the urine. The remaining fraction of germanium leaving the transfer compartment is assumed to be uniformly distributed throughout all organs and tissues of the body where it is retained with a biological half-life of 1 day (Rosenfeld, 1954). Using this half-life, and a 0.25 day

half-life in the transfer compartment, together with an f_1 of 1 and the daily intake of germanium given for Reference Man yields a total body content of 1.6 mg which is comparable with the 2 mg given by Iyengar, Kollmer and Bowen (1978).

3. Classification of Isotopes for Bone Dosimetry

Germanium is assumed to be uniformly distributed throughout all organs and tissues of the body other than the kidneys. Therefore, a classification of isotopes of the element for the purpose of bone dosimetry is not required.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of germanium

			Inhalation	
		Oral	Class D	Class W
Radionuclide		$f_1 = 1$	$f_1 = 1$	$f_1 = 1$
^{66}Ge	ALI	9×10^8	1×10^9	7×10^8
	DAC	—	4×10^5	3×10^5
^{67}Ge	ALI	1×10^9 (2×10^9) ST Wall	3×10^9	4×10^9
	DAC	—	1×10^6	2×10^6
^{68}Ge	ALI	2×10^8	1×10^8	4×10^6
	DAC	—	6×10^4	2×10^3
^{69}Ge	ALI	5×10^8	6×10^8	3×10^8
	DAC	—	2×10^5	1×10^5
^{71}Ge	ALI	2×10^{10}	2×10^{10}	2×10^9
	DAC	—	7×10^6	7×10^5
^{75}Ge	ALI	2×10^9 (3×10^9) ST Wall	3×10^9	3×10^9
	DAC	—	1×10^6	1×10^6
^{77}Ge	ALI	3×10^8	4×10^8	2×10^8
	DAC	—	2×10^5	9×10^4
^{78}Ge	ALI	8×10^8 (9×10^8) ST Wall	8×10^8	8×10^8
	DAC	—	3×10^5	3×10^5

METABOLIC DATA FOR ARSENIC

1. Metabolism

Data from Reference Man (ICRP, 1975)

Arsenic content of soft tissues	~18 mg
of the skeleton	0.1 mg
Daily intake in food and water	1.0 mg

Data from Iyengar, Kollmer and Bowen (1978) indicate that the total body content of arsenic is probably not in excess of 10 mg and could be as little as 0.2 mg.

2. Metabolic Model

(a) *Uptake to blood*

The fractional absorption of arsenic from the gastrointestinal tract is very variable. Data from experiments in which pigs were fed arsanilic acid indicated that the fractional gastrointestinal absorption of this compound was between 0.15 and 0.46. A similar degree of gastrointestinal absorption is indicated by the results of experiments in which cows and dogs were fed sodium or potassium arsenate (Lasko and Peoples, 1975).

In the rat sodium arsenate, arsenic trioxide or naturally occurring arsenic of shrimps appears to be almost completely absorbed from the gastrointestinal tract (Coulson, Remington and Lynch, 1935; Dutkiewicz, 1977). Quantitative absorption of arsenic trioxide is also indicated from experiments on ligated ilecoecal loops of rabbit intestine (Tsutsumi, Nozaki and Maehashi, 1975).

In man, arsenic administered as arsenic trichloride is almost completely absorbed from the gastrointestinal tract (Bettley and O'Shea, 1975) as is dietary arsenic incorporated into fish (Freeman *et al.*, 1979).

In this report f_1 is taken to be 0.5 for all compounds of arsenic.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned all commonly occurring compounds of arsenic to inhalation class W. Experience of the inhalation of arsenic trioxide by man (Holland, McCall and Lanz, 1959) gives some support to this classification and it is adopted here.

Inhalation class	f_1
D	—
W	0.5
Y	—

(c) *Distribution and retention*

In man stable arsenic is fairly uniformly distributed throughout the organs and tissues of the

body with only nails and hair having concentrations of the element substantially greater than the average for the whole body (Smith, 1967).

Studies with radioactive isotopes of arsenic have shown that the metabolism of the element is very dependent upon the chemical form administered and upon the animal species used. Studies in which rats were subcutaneously injected with potassium arsenite (Hunter, Kips and Irvine, 1942), intramuscularily injected with sodium arsenate (Lanz, Wallace and Hamilton, 1950), or intravenously injected with arsenic trichloride (Klassen, 1974) have convincingly demonstrated that arsenic has a marked affinity for the rat erythrocyte. However, this marked affinity for erythrocytes has not been found for other species (Hunter, Kip and Irvine, 1942; Klassen, 1974). In rats fed As_2O_3 the arsenic was found to be concentrated in the liver, spleen and kidneys (Coulson, Remington and Lynch, 1935; Tanaka, 1976). Liver and spleen were also identified as important organs of deposition in experiments in which rats were administered sodium arsenate by various routes (Dutkiewicz, 1977).

In mice, intravenously injected with radioactive arsenilic acid, autoradiographic studies suggest that arsenic is concentrated in bone, kidney cortex, liver and intestinal mucosa (Deak, Csaky and Waddell, 1976). In the pig, liver and kidneys are also identified as important organs of arsenic deposition after the feeding of arsanilic acid (Hanson, *et al.*, 1955; Gitter and Lewis, 1969).

Experiments on guinea pigs and rabbits, together with autopsy data from a patient with lymphoblastic leukaemia indicate that arsenic derived from potassium arsenite is fairly uniformly distributed throughout all organs and tissues of the body (Hunter, Kip and Irvine, 1942). However, autopsy studies on patients with various intracranial neoplasms indicate that arsenic intravenously injected in the trivalent state is deposited preferentially in the liver and kidneys (Mealey, Brownell and Sweet, 1959).

Arsenic, in shrimps or fish, inhaled in cigarette smoke, or administered as arsanilic acid, sodium arsenate and potassium arsenate is rapidly lost from the body (Coulson, Remington and Lynch, 1935; Overby and Frost, 1960; Gitter and Lewis, 1969; Arnold, Kohlhaas and Neiwerth, 1970; Lasko and Peoples, 1975; Freeman *et al.*, 1979). This is in contrast to trivalent compounds of the element which are much more avidly retained (Coulson, Remington and Lynch, 1935; Mealey, Brownell and Sweet, 1959; Klassen, 1974).

The most extensive studies of arsenic metabolism in man (Mealey, Brownell and Sweet, 1959) indicate that the whole body retention of arsenic, intravenously injected as the trivalent arsenite, in man is well represented by:

$$R(t) = 0.35\ e^{-0.693t/0.02} + 0.28\ e^{-0.693t/1.1} + 0.37\ e^{-0.693t/9.6}$$

This retention function is not compatible with the daily intake and total body content of stable arsenic given for Reference Man (ICRP, 1975). However, the content of stable arsenic given for Reference Man is based on a very small amount of human data and other studies (Iyengar, Kollmer and Bowen, 1978) indicate a much smaller body content of the element.

In this report it is assumed that of arsenic leaving the transfer compartment 0.35 goes directly to excretion, 0.07 is translocated to the liver, 0.015 is translocated to the kidneys, 0.005 is translocated to the spleen and 0.56 is uniformly distributed throughout all other organs and tissues of the body. Of arsenic translocated to any organ or tissue fractions of 0.4 and 0.6 are assumed to be retained with biological half-lives of 1 and 10 days respectively.

3. Classification of Isotopes for Bone Dosimetry

Arsenic is assumed to be uniformly distributed throughout all organs and tissues of the body

other than the liver, kidneys and spleen. Therefore, a classification of isotopes of the element for the purpose of bone dosimetry is not required.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of arsenic

Radionuclide		Oral	Inhalation Class W
		$f_1 = 5 \times 10^{-1}$	$f_1 = 5 \times 10^{-1}$
^{69}As	ALI	1×10^9 (2×10^9) ST Wall	4×10^9
	DAC	—	2×10^6
^{70}As	ALI	5×10^8	2×10^9
	DAC	—	8×10^5
^{71}As	ALI	1×10^8	2×10^8
	DAC	—	7×10^4
^{72}As	ALI	3×10^7	5×10^7
	DAC	—	2×10^4
^{73}As	ALI	3×10^8	6×10^7
	DAC	—	3×10^4
^{74}As	ALI	6×10^7	3×10^7
	DAC	—	1×10^4
^{76}As	ALI	4×10^7	5×10^7
	DAC	—	2×10^4
^{77}As	ALI	2×10^8 (2×10^8) LLI Wall	2×10^8
	DAC	—	8×10^4
^{78}As	ALI	3×10^8	8×10^8
	DAC	—	3×10^5

METABOLIC DATA FOR SELENIUM

1. Metabolism

Data from Reference Man (ICRP, 1975)	
Selenium content of soft tissues	13 mg
Daily intake in food and fluids	0.15 mg

2. Metabolic Model

(a) *Uptake to blood*

Selenium incorporated in food is almost completely absorbed from the gastrointestinal tract (ICRP, 1975; Underwood, 1977). Soluble inorganic compounds of the element also have a large fractional absorption from the gastrointestinal tract (Smith, Westfall and Stohlman, 1937; Graham, Veatch and Kaplan, 1971; Thomson and Stewart, 1973; Furchner, London and Wilson, 1975; Underwood, 1977; Pope *et al.*, 1979). However, elemental selenium and selenides appear to be relatively inactive biologically and only a small fraction of these forms of selenium is absorbed during their passage through the gastrointestinal tract (Cerwenka and Cooper, 1961; Underwood, 1977). In this report f_1 is taken to be 0.05 for elemental selenium and for selenides and 0.8 for all other compounds of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, and carbides of selenium to inhalation class W and all other commonly occurring inorganic compounds of the element to inhalation class D. In the absence of any relevant experimental information this classification is adopted for inorganic compounds of selenium. Elemental selenium is assigned to inhalation class W.

Since selenium and selenides are unlikely to remain in the same chemical form indefinitely after their deposition in the lungs it is appropriate to take f_1 as 0.8 for selenium entering the gastrointestinal tract following the deposition of any compound of the element in the lungs.

Inhalation class	f_1
D	0.8
W	0.8
Y	—

(c) *Distribution and retention*

The distribution and retention of various compounds of selenium in the body has been studied in several mammalian species including man (Lathrop, Harper and Malkinson, 1968; Lopez, Preston and Pfander, 1969; Graham, Veatch and Kaplan, 1971; Lathrop *et al.*, 1972; Thomson and Stewart, 1973; Falk and Lindhé, 1974; Evans, 1975; Furchner, London and Wilson, 1975; Johnson, 1977; Kuikka and Nordman; 1978). The most commonly studied

compounds are selenious acid, selenomethionine and sodium selenite. Although it has been claimed that selenomethionine is more avidly retained in the body than is sodium selenite (Cerewenka and Cooper, 1961) the evidence suggests that selenium from both compounds is incorporated into the same metabolic pool within a week of intravenous injection (Thomson and Stewart, 1973). Although the chemical form of selenium injected does not markedly affect its retention in the body, the level of stable selenium in the diet does (Lopez, Preston and Pfander, 1969).

In man, intravenously injected radioactive selenium is concentrated in the liver, kidneys, pancreas and spleen (Lathrop *et al.*, 1972; Kuikka and Nordman, 1978) and this seems also to be the case in other species (Lopez, Preston and Pfander, 1969; Graham, Veatch and Kaplan, 1971; Thomson and Stewart, 1973; Furchner, London and Wilson, 1975). In studies on rats (Thompson and Stewart, 1973; Furchner, London and Wilson, 1975) the biological half-lives for retention in all organs and tissues of the body were found to be very similar. In contrast, data for man (Lathrop *et al.*, 1972) indicate that a large fraction of the selenium deposited in the pancreas is lost from that tissue with a biological half-life of about 1 day.

The whole body retention of selenium in man is well described by three exponentials with biological half-lives of 0.5–7, 20–70 and 120–330 days respectively (Lathrop, Harper and Malkinson, 1968; Lathrop, Johnston, Blau and Rothschild, 1972; Falk and Lindhé, 1974; Johnson, 1977; Toohey, Essling and Huff, 1979). However, it should be noted that animal experiments have demonstrated that selenium retention in the body is very dependent upon the level and form of intake and upon the nature of the rest of the diet (Underwood, 1977).

In this report it is assumed that of selenium leaving the transfer compartment fractions of 0.15, 0.05, 0.01 and 0.005 are translocated to the liver, kidneys, spleen and pancreas respectively. The remaining fraction of selenium leaving the transfer compartment is assumed to be uniformly distributed throughout all other organs and tissues of the body. Of selenium deposited in any organ or tissue fractions 0.1, 0.4 and 0.5 are assumed to be retained with biological half-lives of 3, 30 and 150 days respectively.

This retention function is compatible with the daily intake and total soft tissue content of selenium given for Reference Man (ICRP, 1975) and with the data on the metabolism of the element in man.

3. Classification of Isotopes for Bone Dosimetry

Selenium is assumed to be uniformly distributed throughout all organs and tissues of the body other than the liver, kidneys, spleen and pancreas. Therefore, a classification of isotopes of the element for the purpose of bone dosimetry is not required.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of selenium

Radionuclide		Oral		Inhalation	
				Class D	Class W
		$f_1 = 8 \times 10^{-1}$	$f_1 = 5 \times 10^{-2}$	$f_1 = 8 \times 10^{-1}$	$f_1 = 8 \times 10^{-1}$
^{70}Se	ALI	6×10^{8}	4×10^{8}	1×10^{9}	2×10^{9}
	DAC	—	—	6×10^{5}	7×10^{5}
^{73m}Se	ALI	2×10^{9}	1×10^{9}	6×10^{9}	5×10^{9}
	DAC	—	—	2×10^{6}	2×10^{6}
^{73}Se	ALI	3×10^{8}	1×10^{8}	5×10^{8}	6×10^{8}
	DAC	—	—	2×10^{5}	2×10^{5}
^{75}Se	ALI	2×10^{7}	1×10^{8}	3×10^{7}	2×10^{7}
	DAC	—	—	1×10^{4}	9×10^{3}
^{79}Se	ALI	2×10^{7}	2×10^{8}	3×10^{7}	2×10^{7}
	DAC	—	—	1×10^{4}	9×10^{3}
^{81m}Se	ALI	1×10^{9}	9×10^{8}	3×10^{9}	3×10^{9}
	DAC	—	—	1×10^{6}	1×10^{6}
^{81}Se	ALI	2×10^{9} (3×10^{9}) ST Wall	2×10^{9} (3×10^{9}) ST Wall	8×10^{9}	9×10^{9}
	DAC	—	—	3×10^{6}	4×10^{6}
^{83}Se	ALI	2×10^{9} (2×10^{9}) ST Wall	1×10^{9}	4×10^{9}	5×10^{9}
	DAC	—	—	2×10^{6}	2×10^{6}

METABOLIC DATA FOR PALLADIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for palladium.

2. Metabolic Model

(a) *Uptake to blood*

The fractional absorption of palladium, administered as the chloride, from the gastro-intestinal tract of rats is less than 5×10^{-3} (Moore *et al.*, 1974). Acute toxicity data from experiments on rats indicate that the fractional absorption of palladium administered as PdO or $PdSO_4$ is even smaller (Holbrook *et al.*, 1975). In this report f_1 is taken to be 5×10^{-3} for all compounds of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides and hydroxides of palladium to inhalation class Y, halides and nitrates to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. However, experiments on rats (Moore *et al.*, 1975) indicate that $PdCl_2$ should be assigned to inhalation class D. In this report oxides and hydroxides of palladium are assigned to inhalation class Y, nitrates are assigned to inhalation class W and all other commonly occurring compounds of the element are assigned to inhalation class D.

Inhalation class	f_1
D	5×10^{-3}
W	5×10^{-3}
Y	5×10^{-3}

(c) *Distribution and retention*

In experiments in which rats were injected intravenously with $PdCl_2$ the largest concentrations of palladium were found in the kidney, liver, spleen, lung and bone (Moore *et al.*, 1974; Ando, Hisada and Ando, 1975). In these same experiments it was found that, after an early period of rapid excretion, the retention of palladium in the body was well approximated by a single exponential with a biological half-life of 15 days.

Based on the data reviewed above it is here assumed that of palladium leaving the transfer compartment 0.3 goes directly to excretion, 0.45 is translocated to the liver, 0.15 is translocated to the kidneys, 0.07 is translocated to mineral bone and 0.03 is uniformly distributed throughout all other organs and tissues of the body. Palladium translocated to any organ or tissue is assumed to be retained there with a biological half-life of 15 days.

3. Classification of Isotopes for Bone Dosimetry

There appear to be no relevant data on the distribution of palladium in the skeleton. Only ^{103}Pd and ^{107}Pd have radioactive half-lives of more than 15 days. In this report these two isotopes of palladium are assumed to be uniformly distributed throughout the volume of mineral bone and all other, shorter lived, isotopes of the element are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of palladium

			Inhalation		
		Oral	Class D	Class W	Class Y
Radionuclide		$f_1 = 5 \times 10^{-3}$	$f_1 = 5 \times 10^{-3}$	$f_1 = 5 \times 10^{-3}$	$f_1 = 5 \times 10^{-3}$
^{100}Pd	ALI	5×10^7	5×10^7	5×10^7	5×10^7
	DAC	—	2×10^4	2×10^4	2×10^4
^{101}Pd	ALI	5×10^8	1×10^9	1×10^9	1×10^9
	DAC	—	5×10^5	5×10^5	5×10^5
^{103}Pd	ALI	2×10^8 (3×10^8) LLI Wall	2×10^8	2×10^8	1×10^8
	DAC	—	1×10^5	7×10^4	5×10^4
^{107}Pd	ALI	1×10^9 (1×10^9) LLI Wall	8×10^8 (8×10^8) Kidneys	3×10^8	1×10^7
	DAC	—	3×10^5	1×10^5	6×10^3
^{109}Pd	ALI	9×10^7	2×10^8	2×10^8	2×10^8
	DAC	—	1×10^5	9×10^4	7×10^4

METABOLIC DATA FOR TIN

1. Metabolism

Data from Reference Man (ICRP, 1975)	
Tin content of the body	< 17 mg
of soft tissues	5.8 mg
of the skeleton	< 12 mg
Daily intake in food and fluids	4 mg

Data from Iyengar, Kollmer and Bowen (1978) indicate that the tin content of the skeleton is between 4 and 15 mg.

2. Metabolic Model

(a) *Uptake to blood*

The fractional absorption of dietary or inorganic tin from the gastrointestinal tract is generally small (Barnes and Stoner, 1959; ICRP, 1975; Furchner and Drake, 1976; Underwood, 1977). In the case of stannous chloride the fractional gastrointestinal absorption in mice, rats, monkeys and dogs was always less than 0.05 and was typically about 0.02 (Furchner and Drake, 1976). Data on dietary tin (ICRP, 1975) together with experimental data on whole body retention compared with urinary and faecal losses (Furchner and Drake, 1976) also suggest a fractional gastrointestinal absorption of about this magnitude. A report in which the fractional gastrointestinal absorption of stannous chloride was estimated to be 0.16–0.2 should probably be disregarded since the material appears to have been administered in a solution of pH 0 (Moskalev, 1974). In this report f_1 is taken as 0.02 for all compounds of tin.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned sulphides, oxides, hydroxides, halides and nitrates of tin as well as stannic phosphate to inhalation class W and all other compounds of the element to inhalation class D. The assignment of stannic phosphate to inhalation class W is in agreement with the available experimental data on dogs (Morrow *et al.*, 1968) and the recommendations of the Task Group are adopted here.

Inhalation class	f_1
D	0.02
W	0.02
Y	—

(c) *Distribution and retention*

The distribution and retention of tin after intragastric, intraperitoneal and intravenous administration of the dichloride has been studied in mice, rats, monkeys and dogs (Furchner and Drake, 1976). Whole body retention in each of these species is adequately described by four

exponentials with biological half-lives ranging from 0.2–800 days. Individual organ and tissue retention in rats was similar to whole body retention for all organs and tissues studied.

In the rat, tin intraperitoneally injected as the dichloride is concentrated mainly in bone, although liver, kidney and spleen also exhibit concentrations somewhat above the whole body average in the first 80 days after injection (Furchner and Drake, 1976). Citrates, phosphates, pyrophosphates and tartrates of tin are also concentrated in the skeleton of rats and dogs (Yano, Chu and Anger, 1973; Ando *et al.*, 1973; Grimm *et al.*, 1975).

In this report it is assumed that of tin leaving the transfer compartment 0.5 goes directly to excretion, 0.35 is translocated to mineral bone and 0.15 is uniformly distributed throughout all other organs and tissues of the body. Of tin translocated to any organ or tissue fractions 0.2, 0.2 and 0.6 are assumed to be retained with biological half-lives of 4, 25 and 400 days respectively. This retention function is in good agreement with studies on rats (Furchner and Drake, 1976) and on the concentration of stable tin in the organs and tissues of Reference Man (ICRP, 1975).

3. Classifications of Isotopes for Bone Dosimetry

Of the isotopes of tin considered in this report only ^{113}Sn, ^{119m}Sn ^{121m}Sn, ^{123}Sn and ^{126}Sn have radioactive half-lives in excess of 15 days. These isotopes are assumed to be uniformly distributed throughout the volume of mineral bone at all times following their deposition in that tissue. All other radioactive isotopes of tin are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of tin

Radionuclide		Oral	Inhalation	
			Class D	Class W
		$f_1 = 2 \times 10^{-2}$	$f_1 = 2 \times 10^{-2}$	$f_1 = 2 \times 10^{-2}$
^{110}Sn	ALI	1×10^8	4×10^8	4×10^8
	DAC	—	2×10^5	2×10^5
^{111}Sn	ALI	3×10^9	8×10^9	1×10^{10}
	DAC	—	3×10^6	4×10^6
^{113}Sn	ALI	6×10^7 (7×10^7) LLI Wall	5×10^7	2×10^7
	DAC	—	2×10^4	9×10^3
^{117m}Sn	ALI	6×10^7 (7×10^7) LLI Wall	5×10^7 (8×10^7) Bone surf.	5×10^7
	DAC	—	2×10^4	2×10^4
^{119m}Sn	ALI	1×10^8 (2×10^8) LLI Wall	9×10^7	4×10^7
	DAC	—	4×10^4	2×10^4
^{121m}Sn	ALI	1×10^8 (1×10^8) LLI Wall	3×10^7	2×10^7
	DAC	—	1×10^4	8×10^3
^{121}Sn	ALI	2×10^8 (2×10^8) LLI Wall	6×10^8	4×10^8
	DAC	—	2×10^5	2×10^5
^{123m}Sn	ALI	2×10^9	4×10^9	5×10^9
	DAC	—	2×10^6	2×10^6
^{123}Sn	ALI	2×10^7 (2×10^7) LLI Wall	2×10^7	6×10^6
	DAC	—	1×10^4	3×10^3
^{125}Sn	ALI	1×10^7 (2×10^7) LLI Wall	3×10^7	1×10^7
	DAC	—	1×10^4	5×10^3
^{126}Sn	ALI	1×10^7	2×10^6	2×10^6
	DAC	—	9×10^2	1×10^3
^{127}Sn	ALI	3×10^8	7×10^8	7×10^8
	DAC	—	3×10^5	3×10^5
^{128}Sn	ALI	4×10^8	1×10^9	1×10^9
	DAC	—	4×10^5	6×10^5

METABOLIC DATA FOR ANTIMONY

1. Metabolism

Data from Reference Man (ICRP, 1975)	
Antimony content of the body	~7.9 mg
of soft tissue	~5.9 mg
of the skeleton	2.0 mg
Daily intake in food and fluids	0.05 mg

The antimony content of the tissues of Reference Man are based on neutron activation studies on a single individual and should, therefore, be used with caution.

Recently, Underwood (1977) has reviewed data on the body content and daily intake of antimony. These data indicate that the total body content of antimony is typically about 10 mg but that the daily dietary intake of the element may be as little as 34 μg or as much as 1275 μg. Data from Iyengar, Kollmer and Bowen (1978) suggest that the total body content of antimony is typically in the range 0.5–3.5 mg.

2. Metabolic Model

(a) *Uptake to blood*

Data presented in Reference Man (ICRP, 1975) suggest that the fractional absorption of dietary antimony from the gastrointestinal tract is about 0.8. However, this estimate is based on a rather small value for the daily intake of antimony in food and fluids and a rather large estimate of urinary excretion (ICRP, 1975; Underwood, 1977) and a much smaller value for the fractional absorption of dietary antimony would be more in accord with the available data.

Results from experiments on mice (Waitz *et al.*, 1965) suggest that the fractional absorption of antimony, administered as tartar emetic, from the gastrointestinal tract is about 0.2. However, studies on rats (Moskalev, 1964) indicate a fractional absorption of about 0.05 for this compound. Studies on various trivalent and pentavalent compounds of antimony suggest that the fractional absorption of antimony administered in these forms is not usually more than 0.01 (Rose and Jacobs, 1969; Thomas *et al.*, 1973; Felicetti, Thomas and McClellan, 1974a). In this report f_1 is taken to be 0.1 for antimony ingested as tartar emetic and 0.01 for antimony ingested as any other compound of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, halides, sulphides, sulphates and nitrates of antimony to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. Experiments on the behaviour of the trichloride in rats (Djurić, Thomas and Lie, 1962) the tartrate complex in mice, hamsters and dogs (Thomas *et al.*, 1973; Felicetti, Thomas and McClellan, 1974a, 1974b) and the oxide in mice, dogs and man (Rose and Jacobs, 1969; Thomas *et al.*, 1973; Felicetti, Thomas and McClellan, 1974b) support this classification and it is adopted here.

Inhalation class	f_1
D	0.1
W	0.01
Y	—

(c) *Distribution and retention*

Data on the distribution of stable antimony (Smith, 1967; ICRP, 1975) suggest that the element is only concentrated to a small extent, if at all, in the liver, kidneys and skeleton. Studies in which men were injected either intravenously or intramuscularly with sodium antimony dimercaptosuccinate (Abdallah and Saif, 1962) indicated that the liver concentrated antimony to some extent.

Experiments on mice (Waitz *et al.*, 1965; Molokhia and Smith, 1969; Thomas *et al.*, 1973) indicate that up to half the antimony entering the systemic circulation is deposited in the liver. However, in rats, hamsters and dogs (Durbin, 1960; Djurić, Thomas and Lie, 1962; Moskalev, 1964; Waitz *et al.*, 1965; Felicetti, Thomas and McClellan, 1974b) the fraction of antimony going to the liver is considerably smaller. Also, in the dog, the thyroid is found to accumulate antimony after inhalation (Felicetti, Thomas and McClellan, 1974b) but, in man, no excess of stable antimony is found in this tissue (Smith, 1967).

In man the fractional excretion of intravenously injected antimony is about 0.2 in the first 24 h after injection (Abdallah and Saif, 1962). Much of the remaining antimony is retained with a biological half-life of 5 days but a small component of long-term retention is also indicated. Experiments on the inhalation of antimony by mice, hamsters and dogs (Thomas *et al.*, 1973; Felicetti, Thomas and McClellan, 1974a, 1974b) indicate that about 0.05 of antimony entering the systemic circulation is retained in the body with a biological half-life of between 16 and 100 days.

In this report it is assumed that of antimony leaving the transfer compartment 0.2 goes directly to excretion, 0.2 is translocated to mineral bone, 0.1 is translocated to the liver and the remaining fraction is uniformly distributed throughout all other organs and tissues of the body. Of antimony translocated to any organ or tissue fractions of 0.95 and 0.05 are assumed to be retained with biological half-lives of 5 and 100 days respectively.

3. Classification of Isotopes for Bone Dosimetry

There appear to be no relevant data concerning the distribution of antimony in bone. In this report isotopes of the element are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of antimony

Radionuclide		Oral		Inhalation	
				Class D	Class W
		$f_1 = 1 \times 10^{-1}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-1}$	$f_1 = 1 \times 10^{-2}$
^{115}Sb	ALI	3×10^9	3×10^9	9×10^9	1×10^{10}
	DAC	—	—	4×10^6	5×10^6
^{116m}Sb	ALI	8×10^8	8×10^8	3×10^9	5×10^9
	DAC	—	—	1×10^6	2×10^6
^{116}Sb	ALI	3×10^9 (3×10^9) ST Wall	3×10^9 (3×10^9) ST Wall	1×10^{10}	1×10^{10}
	DAC	—	—	4×10^6	5×10^6
^{117}Sb	ALI	3×10^9	3×10^9	8×10^9	1×10^{10}
	DAC	—	—	3×10^6	4×10^6
^{118m}Sb	ALI	2×10^8	2×10^8	7×10^8	8×10^8
	DAC	—	—	3×10^5	3×10^5
^{119}Sb	ALI	6×10^8	5×10^8	2×10^9	1×10^9
	DAC	—	—	7×10^5	4×10^5
^{120}Sb	ALI	4×10^9 (6×10^9) ST Wall	4×10^9 (6×10^9) ST Wall	2×10^{10}	2×10^{10}
$T\frac{1}{2} = 15.89$ m	DAC	—	—	7×10^6	8×10^6
^{120}Sb	ALI	4×10^7	3×10^7	8×10^7	5×10^7
$T\frac{1}{2} = 5.76$ d	DAC	—	—	3×10^4	2×10^4
^{122}Sb	ALI	3×10^7 (3×10^7) LLI Wall	3×10^7 (3×10^7) LLI Wall	9×10^7	4×10^7
	DAC	—	—	4×10^4	2×10^4
^{124m}Sb	ALI	9×10^9 (1×10^{10}) ST Wall	9×10^9	3×10^{10}	2×10^{10}
	DAC	—	—	1×10^7	9×10^6
^{124}Sb	ALI	2×10^7	2×10^7	3×10^7	9×10^6
	DAC	—	—	1×10^4	4×10^3

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of antimony

Radionuclide		Oral		Inhalation	
				Class D	Class W
		$f_1 = 1 \times 10^{-1}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-1}$	$f_1 = 1 \times 10^{-2}$
^{125}Sb	ALI	8×10^7	7×10^7	9×10^7	2×10^7
	DAC	—	—	4×10^4	8×10^3
^{126}mSb	ALI	2×10^9 (2×10^9) ST Wall	2×10^9 (2×10^9) ST Wall	7×10^9	7×10^9
	DAC	—	—	3×10^6	3×10^6
^{126}Sb	ALI	2×10^7	2×10^7	4×10^7	2×10^7
	DAC	—	—	2×10^4	8×10^3
^{127}Sb	ALI	3×10^7 (3×10^7) LLI Wall	3×10^7 (3×10^7) LLI Wall	8×10^7	3×10^7
	DAC	—	—	3×10^4	1×10^4
^{128}Sb	ALI	5×10^7	4×10^7	2×10^8	1×10^8
$T_{\frac{1}{2}} = 9.01$ h	DAC	—	—	7×10^4	5×10^4
^{128}Sb	ALI	3×10^9 (4×10^9) ST Wall	3×10^9 (4×10^9) ST Wall	1×10^{10}	2×10^{10}
$T_{\frac{1}{2}} = 10.4$ m	DAC	—	—	6×10^6	7×10^6
^{129}Sb	ALI	1×10^8	1×10^8	3×10^8	3×10^8
	DAC	—	—	1×10^5	1×10^5
^{130}Sb	ALI	7×10^8	7×10^8	2×10^9	3×10^9
	DAC	—	—	1×10^6	1×10^6
^{131}Sb	ALI	6×10^8 (6×10^8) Thyroid	6×10^8 (6×10^8) Thyroid	9×10^8 (1×10^9) Thyroid	9×10^8 (2×10^9) Thyroid
	DAC	—	—	4×10^5	4×10^5

METABOLIC DATA FOR LANTHANUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for lanthanum.

2. Metabolic Model

(a) *Uptake to blood*

The fractional absorption of lanthanum from the gastrointestinal tract of rats has been estimated as less than 5×10^{-4} (Hamilton, 1948; Moskalev, Zalikin and Stepanov, 1972). In experiments in which rats ingested the chloride the fractional absorption of the element from the gastrointestinal tract was less than 5×10^{-4} (Moskalev, 1961). However in experiments on dogs the fractional absorption of lanthanum, ingested as the chloride, from the gastrointestinal tract was found to be about 2×10^{-3} (Cuddihy and Boeker, 1970). In this report f_1 is taken to be 10^{-3} for all compounds of lanthanum.

(b) *Inhalation classes*

Experiments on dogs (Cuddihy and Boecker, 1970) and monkeys (Ducousso and Pasquier, 1974) indicate a phase of rapid uptake of lanthanum, inhaled as the chloride, from the lung to the systemic circulation. Following this early phase of rapid uptake the lanthanum which remained in the lungs of the dogs was translocated to the systemic circulation and to the lymph nodes with a biological half-life of about 7 days.

In this report oxides and hydroxides of lanthanum are assigned to inhalation class W and all other commonly occurring compounds of the element to inhalation class D.

Inhalation class	f_1
D	10^{-3}
W	10^{-3}
Y	—

(c) *Distribution and retention*

Studies on mice (Spode and Geniscke, 1958; Rosoff *et al.*, 1963; Higasi *et al.*, 1973; Sullivan *et al.*, 1975), rats (Durbin *et al.*, 1956; Slouka and Králové, 1969; Moskalev, Zalikin and Stepanov, 1972) and dogs (Cuddihy and Boecker, 1970) are in general agreement that the liver is the principal organ of lanthanum deposition. Other important organs of deposition are the skeleton and, to some extent, the kidneys and spleen.

In man, there is very little excretion of lanthanum in the first 4 days after intravenous injection of the chloride (Rosoff *et al.*, 1961) and this is also true in dogs studied for 8 days after intravenous injection (Cuddihy and Boecker, 1970). As in the case of the more extensively studied lanthanides, cerium and promethium, the biological half-life of lanthanum in the body is likely to be in excess of 1 000 days.

Based on the data reviewed above, it is here assumed that of lanthanum leaving the transfer compartment fractions of 0.6 and 0.2 are translocated to the liver and mineral bone respectively. The remaining fraction of lanthanum leaving the transfer compartment is assumed to be uniformly distributed throughout all other organs and tissues of the body. By analogy with the more extensively studied lanthanide cerium, lanthanum deposited in any organ or tissue is assumed to be retained there with a biological half-life of 3 500 days.

(d) *Chelated compounds*

Chelated forms of lanthanum are not considered in this report. Their metabolism differs considerably from that of other compounds of lanthanum.

3. Classification of Isotopes for Bone Dosimetry

There appear to be no relevant data concerning the distribution of lanthanum in mineral bone. By analogy with cerium, promethium and the actinides all isotopes of the element are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of lanthanum

Radionuclide		Oral $f_1 = 1 \times 10^{-3}$	Inhalation Class D $f_1 = 1 \times 10^{-3}$	Inhalation Class W $f_1 = 1 \times 10^{-3}$
^{131}La	ALI	2×10^9	4×10^9	6×10^9
	DAC	—	2×10^6	3×10^6
^{132}La	ALI	1×10^8	4×10^8	4×10^8
	DAC	—	2×10^5	2×10^5
^{135}La	ALI	1×10^9	4×10^9	4×10^9
	DAC	—	2×10^6	1×10^6
^{137}La	ALI	4×10^8	2×10^6 (3×10^6) Liver	1×10^7 (1×10^7) Liver
	DAC	—	1×10^3	4×10^3
^{138}La	ALI	3×10^7	1×10^5	5×10^5
	DAC	—	5×10^1	2×10^2
^{140}La	ALI	2×10^7	5×10^7	4×10^7
	DAC	—	2×10^4	2×10^4
^{141}La	ALI	1×10^8	3×10^8	4×10^8
	DAC	—	1×10^5	2×10^5
^{142}La	ALI	3×10^8	8×10^8	1×10^9
	DAC	—	3×10^5	5×10^5
^{143}La	ALI	1×10^9 (1×10^9) ST Wall	4×10^9	3×10^9
	DAC	—	2×10^6	1×10^6

METABOLIC DATA FOR PRASEODYMIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for praseodymium.

2. Metabolic Model

(a) *Uptake to blood*

The fractional absorption of compounds of praseodymium from the gastrointestinal tract of rats has been variously reported as less than 5×10^{-3} (Hamilton, 1948) and less than 5×10^{-4} (Moskalev, Zalikin and Stepanov, 1972). Experiments on the acute toxicity of the metal and its nitrates in rats (Bruce, Hietbrink and DuBois, 1963) also indicate that the fractional absorption from the gastrointestinal tract is small. By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of praseodymium.

(b) *Inhalation classes*

The limited data available from experiments on rats (Moskalev, Zalikin and Stepanov, 1972) suggest that praseodymium is retained longer in the lungs than are gadolinium, ytterbium, europium and terbium. In view of these results, and by analogy with the more extensively studied lanthanides cerium and promethium, oxides, hydroxides, carbides and fluorides of praseodymium are assigned to inhalation class Y and all other commonly occurring compounds of the element are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	3×10^{-4}

(c) *Distribution and retention*

Studies on mice (Gensicke and Henneberger, 1964) and rats (Durbin, *et al.*, 1956; Moskalev Zalikin and Stepanov, 1972) indicate that intravenously, intraperitoneally or intramuscularly injected praseodymium is deposited mainly in the liver, the skeleton and the kidneys.

Praseodymium deposited in the skeleton of the rat is avidly retained there (Hamilton, 1948; Moskalev, Zalikin and Stepanov, 1972), whereas praseodymium deposited in the liver or kidneys of mice and rats has a biological half-life in those tissues of about 5–10 days (Hamilton, 1948; Gensicke and Henneberger, 1964; Moskalev, Zalikin and Stepanov, 1972). However, experiments have shown that cerium is rapidly lost from the livers of rats but exhibits long-term retention in the livers of cats and dogs (Moskalev, Zalikin and Stepanov, 1970). Chemical similarities between cerium and praseodymium suggest that this effect will also occur for the latter element.

Based on the data reviewed above, it is here assumed that of praseodymium leaving the

transfer compartment fractions of 0.6, 0.25 and 0.05 are translocated to the liver, mineral bone and the kidneys respectively. The remaining fraction of praseodymium leaving the transfer compartment is assumed to go directly to excretion. By analogy with cerium, praseodymium translocated to the liver or to mineral bone is assumed to be retained there with a biological half-life of 3 500 days. Praseodymium translocated to the kidneys is assumed to be retained there with a biological half-life of 10 days.

3. Classification of Isotopes for Bone Dosimetry

By analogy with promethium, and because none of the isotopes of praseodymium considered in this report has a radioactive half-life of more than 15 days, all isotopes of praseodymium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of praseodymium

Radionuclide		Inhalation		
		Oral	Class W	Class Y
		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{136}Pr	ALI	2×10^9 (3×10^9) ST Wall	9×10^9	8×10^9
	DAC	—	4×10^6	3×10^6
^{137}Pr	ALI	1×10^9	6×10^9	5×10^9
	DAC	—	2×10^6	2×10^6
^{138m}Pr	ALI	4×10^8	2×10^9	2×10^9
	DAC	—	8×10^5	7×10^5
^{139}Pr	ALI	1×10^9	4×10^9	4×10^9
	DAC	—	2×10^6	2×10^6
^{142m}Pr	ALI	3×10^9	6×10^9	5×10^9
	DAC	—	3×10^6	2×10^6
^{142}Pr	ALI	4×10^7	8×10^7	7×10^7
	DAC	—	3×10^4	3×10^4
^{143}Pr	ALI	3×10^7 (4×10^7) LLI Wall	3×10^7	2×10^7
	DAC	—	1×10^4	1×10^4
^{144}Pr	ALI	1×10^9 (2×10^9) ST Wall	5×10^9	4×10^9
	DAC	—	2×10^6	2×10^6
^{145}Pr	ALI	1×10^8	3×10^8	3×10^8
	DAC	—	1×10^5	1×10^5
^{147}Pr	ALI	2×10^9 (3×10^9) ST Wall	7×10^9	7×10^9
	DAC	—	3×10^6	3×10^6

METABOLIC DATA FOR NEODYMIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for neodymium.

2. Metabolic Model

(a) *Uptake to blood*

Experiments on the acute toxicity of neodymium to the rat (Bruce, Hietbrink and DuBois, 1963) indicate that the fractional absorption of nitrates or oxides of the element from the gastrointestinal tract is small. By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of neodymium.

(b) *Inhalation classes*

There appear to be no relevant data available concerning the distribution and retention of inhaled compounds of neodymium. By analogy with promethium, which is neodymium's nearest neighbour in the lanthanide series, oxides, hydroxides, carbides and fluorides of neodymium are assigned to inhalation class Y and all other commonly occurring compounds of the element are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	3×10^{-4}

(c) *Distribution and retention*

The limited data available on the distribution of neodymium in the rat after intravenous or intramuscular injection (Durbin *et al.*, 1956, Hisada, Ando and Suzuki, 1976) suggest that the distribution and retention of the element in the body is similar to the distribution and retention of promethium. In this report the metabolic model used for promethium is also used for neodymium.

Of neodymium leaving the transfer compartment it is assumed that equal fractions of 0.45 are translocated to the liver and to mineral bone. The remaining fraction of neodymium leaving the transfer compartment is assumed to go directly to excretion. Neodymium translocated to the liver or to mineral bone is assumed to be retained in those tissues with a biological half-life of 3 500 days.

3. Classification of Isotopes for Bone Dosimetry

By analogy with promethium all isotopes of neodymium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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ICRP Publication 23. Report of the Task Group on Reference Man. Pergamon Press, Oxford, 1975.

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of neodymium

Radionuclide		Oral	Inhalation	
			Class W	Class Y
		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{136}Nd	ALI	6×10^{8}	2×10^{9}	2×10^{9}
	DAC	—	9×10^{5}	8×10^{5}
^{138}Nd	ALI	7×10^{7}	2×10^{8}	2×10^{8}
	DAC	—	1×10^{5}	8×10^{4}
^{139m}Nd	ALI	2×10^{8}	6×10^{8}	5×10^{8}
	DAC	—	3×10^{5}	2×10^{5}
^{139}Nd	ALI	3×10^{9}	1×10^{10}	1×10^{10}
	DAC	—	5×10^{6}	5×10^{6}
^{141}Nd	ALI	6×10^{9}	3×10^{10}	2×10^{10}
	DAC	—	1×10^{7}	9×10^{6}
^{147}Nd	ALI	4×10^{7} (5×10^{7}) LLI Wall	3×10^{7}	3×10^{7}
	DAC	—	1×10^{4}	1×10^{4}
^{149}Nd	ALI	4×10^{8}	1×10^{9}	9×10^{8}
	DAC	—	4×10^{5}	4×10^{5}
^{151}Nd	ALI	3×10^{9}	7×10^{9}	7×10^{9}
	DAC	—	3×10^{6}	3×10^{6}

METABOLIC DATA FOR PROMETHIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for promethium.

2. Metabolic Model

(a) *Uptake to blood*

Absorption of promethium from the gastrointestinal tract is slight. In miniature swine f_1 is about 10^{-5} for promethium administered as the perchlorate (McClellan, Bustad and Keough, 1965). In lactating goats f_1 is about 10^{-3} for promethium administered as the chloride (Ekman and Åberg, 1961). In man f_1 has been estimated as 10^{-5} for promethium administered as the chloride (Palmer, Nelson and Crook, 1970). In conformity with the value for cerium, in this report f_1 is taken to be 3×10^{-4} for all compounds of promethium.

(b) *Inhalation classes*

The ICRP task Group on Lung Dynamics (1966) assigned oxides, hydroxides, carbides and fluorides of promethium to inhalation class Y and all other compounds of the element to inhalation class W. Experiments in which rats and dogs (Shipler *et al.*, 1976) inhaled Pm_2O_3 indicate that promethium inhaled in this form may be somewhat more readily translocated from the lung than would be expected for a class Y compound. Experiments in which dogs inhaled ^{147}Pm perchlorate (Stuart, 1964) indicate that this compound is readily translocated from the lungs as is $PmCl_3$ in the mouse (Gensicke and Nitschke, 1965).

In this report oxides, hydroxides, carbides and fluorides of promethium are assigned to inhalation class Y and all other compounds of the element are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	3×10^{-4}

(c) *Distribution and retention*

Promethium, in common with the other lanthanides is primarily concentrated in liver and bone (Stuart, 1964; Gensicke and Nitschke, 1965; Grigorescu and Weber, 1969; Smith, 1972; Gensicke, 1966). In man it is retained in the body with a biological half-life of greater than 1 000 days (Palmer, Nelson and Crook, 1970). This may be compared with the biological half-life of 3 500 days for the whole body retention of cerium in beagles (Richmond and London, 1966).

In this report it is assumed that of promethium leaving the transfer compartment a fraction, 0.1, goes directly to excreta while fractions 0.45 and 0.45 are translocated to the liver and to the skeleton respectively. Promethium translocated to the liver or to the skeleton is assumed to be retained in those tissues with a biological half-life of 3 500 days.

3. Classification of Isotopes for Bone Dosimetry

Autoradiographic studies (Hamilton, 1948; Kawin, 1958; Hölzer and Gensicke, 1965; Asling *et al.*, 1952) have shown that promethium is deposited principally on the endosteal and periosteal surfaces of bone and also in the vicinity of small blood vessels in compact bone. By analogy with the actinide elements isotopes of promethium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in the skeleton.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of promethium

Radionuclide		Oral	Inhalation	
			Class W	Class Y
		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{141}Pm	ALI	2×10^9 (2×10^9) ST Wall	7×10^9	6×10^9
	DAC	—	3×10^6	3×10^6
^{143}Pm	ALI	2×10^8	2×10^7	3×10^7
	DAC	—	9×10^3	1×10^4
^{144}Pm	ALI	5×10^7	4×10^6	4×10^6
	DAC	—	2×10^3	2×10^3
^{145}Pm	ALI	4×10^8	7×10^6 (8×10^6) Bone surf.	7×10^6
			3×10^3	3×10^3
^{146}Pm	ALI	6×10^7	2×10^6	2×10^6
	DAC	—	8×10^2	7×10^2
^{147}Pm	ALI	2×10^8 (2×10^8) LLI Wall	5×10^6 (7×10^6) Bone surf.	5×10^6
	DAC	—	2×10^3	2×10^3
^{148m}Pm	ALI	3×10^7	1×10^7	1×10^7
	DAC	—	4×10^3	5×10^3
^{148}Pm	ALI	2×10^7 (2×10^7) LLI Wall	2×10^7	2×10^7
	DAC	—	8×10^3	8×10^3
^{149}Pm	ALI	4×10^7 (5×10^7) LLI Wall	7×10^7	7×10^7
	DAC	—	3×10^4	3×10^4
^{150}Pm	ALI	2×10^8	7×10^8	6×10^8
	DAC	—	3×10^5	3×10^5
^{151}Pm	ALI	7×10^7	1×10^8	1×10^8
	DAC	—	6×10^4	5×10^4

METABOLIC DATA FOR SAMARIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for samarium.

2. Metabolic Model

(a) *Uptake to blood*

Experiments on the acute toxicity of samarium to the rat (Bruce, Hietbrink and DuBois, 1963) indicate that the fractional absorption of nitrates and oxides of the element from the gastrointestinal tract is small. By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of samarium.

(b) *Inhalation classes*

Experiments in which rats and dogs inhaled an aerosol of samarium trioxide (Shipler *et al.*, 1976) indicate that this compound should be assigned to inhalation class W. Experiments on mice (Gensicke and Nitschke, 1970) indicate that $SmCl_3$ should also be assigned to inhalation class W. In this report all compounds of samarium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	—

(c) *Distribution and retention*

Studies on mice, rats and dogs (Durbin *et al.*, 1956; Spode and Gensicke, 1960; Rosoff *et al.*, 1963; Gensicke and Nitschke, 1970; Hisada and Ando, 1973; Shipler *et al.*, 1976) indicate that samarium entering the systemic circulation is mainly translocated to the liver and the skeleton with smaller amounts going to the kidneys, the spleen and the pancreas. Further, data on rats and dogs (Shipler *et al.*, 1976) indicate that the distribution and retention of samarium in the body is very similar to that of promethium. Therefore, in this report, the metabolic model used for promethium is also used for samarium.

It is assumed that of samarium leaving the transfer compartment equal fractions of 0.45 are translocated to the liver and to mineral bone. The remaining fraction of samarium leaving the transfer compartment is assumed to go directly to excretion. Samarium translocated to the liver or to mineral bone is assumed to be retained in those tissues with a biological half-life of 3 500 days.

(d) *Chelated compounds*

Chelated forms of samarium are not considered in this report. Their metabolism differs considerably from that of other compounds of the element (Rosoff, Siegel, Williams and Spencer, 1963).

3. Classification of Isotopes for Bone Dosimetry

By analogy with promethium, all isotopes of samarium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of samarium

			Inhalation
		Oral	Class W
Radionuclide		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{141m}Sm	ALI	1×10^9	4×10^9
	DAC	—	2×10^6
^{141}Sm	ALI	2×10^9 (2×10^9) ST Wall	7×10^9
	DAC	—	3×10^6
^{142}Sm	ALI	3×10^8	1×10^9
	DAC	—	4×10^5
^{145}Sm	ALI	2×10^8	2×10^7
	DAC	—	8×10^3
^{146}Sm	ALI	5×10^5 (9×10^5) Bone surf.	1×10^3 (2×10^3) Bone surf.
	DAC	—	6×10^{-1}
^{147}Sm	ALI	6×10^5 (1×10^6) Bone surf.	1×10^3 (3×10^3) Bone surf.
	DAC	—	6×10^{-1}
^{151}Sm	ALI	5×10^8 (5×10^8) LLI Wall	4×10^6 (7×10^6) Bone surf.
	DAC	—	2×10^3
^{153}Sm	ALI	6×10^7 (7×10^7) LLI Wall	1×10^8
	DAC	—	4×10^4
^{155}Sm	ALI	2×10^9 (3×10^9) ST Wall	8×10^9
	DAC	—	3×10^6
^{156}Sm	ALI	2×10^8	3×10^8
	DAC	—	1×10^5

METABOLIC DATA FOR EUROPIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for europium.

2. Metabolic Model

(a) *Uptake to blood*

The fractional absorption of europium, administered as the chloride from the gastrointestinal tract of the rat, is typically in the range 2×10^{-4} to 3×10^{-3} (Berke, 1970). Other experiments on rats (Durbin *et al.*, 1956; Moskalev, Zalikin and Stepanov, 1972) also indicate that the gastrointestinal absorption of various compounds of the element is typically of this order of magnitude or less. In this report f_1 is taken to be 10^{-3} for all compounds of europium.

(b) *Inhalation classes*

Experiments in which mice and rats inhaled nitrates, chlorides and oxides of europium (Berke, 1964; Berke, Wilson and Berke, 1968; Johnson and Ziemer, 1971) suggest that all these compounds should be assigned to inhalation class W. However, other experiments on rats (Suzuki *et al.*, 1969) indicate that europium nitrate should be assigned to inhalation class Y.

Data from studies on two healthy adult males who accidentally inhaled europium oxide (Ziemer, George and Kessler, 1968) indicate that this compound should be assigned to inhalation class W.

In this report all commonly occurring compounds of europium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	10^{-3}
Y	—

(c) *Distribution and retention*

The distribution and retention of europium in the rat after intravenous injection of the chloride has been extensively studied by Berke (1968). In common with other studies (Durbin *et al.*, 1956a; Durbin *et al.*, 1956b; Moskalev, Zalikin and Stepanov, 1972) similar fractions of europium were found to be deposited in the skeleton and the liver. Also, about 0.06 of intravenously injected europium was found to be deposited in the kidneys.

There is very long-term retention of europium deposited in the skeleton of rats whereas europium deposited in the liver and kidneys is lost from the body with a biological half-life of about 10 days (Berke, 1968; Moskalev, Zalikin and Stepanov, 1972). However, studies with cerium have shown that although that element is rapidly lost from the livers of rats it exhibits long-term retention in the livers of cats and dogs (Moskalev, Zalikin and Stepanov, 1970).

Chemical similarities between cerium and europium suggest that this effect will also occur for the latter element.

Based on the data reviewed above, it is here assumed that of europium leaving the transfer compartment fractions of 0.4, 0.4 and 0.06 are translocated to the liver, to mineral bone and to the kidneys respectively. The remaining fraction of europium leaving the transfer compartment is assumed to go directly to excretion. By analogy with cerium, europium deposited in the liver or in mineral bone is assumed to be retained there with a biological half-life of 3 500 days. Europium deposited in the kidneys is assumed to be retained there with a biological half-life of 10 days.

3. Classification of Isotopes for Bone Dosimetry

Data on the distribution of europium between various bones of the rat skeleton (Berke, 1968) indicate that the element is mainly associated with bone surface rather than bone volume. In this respect it is analogous to promethium. In this report all isotopes of europium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of europium

Radionuclide		Oral	Inhalation Class W
		$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$
^{145}Eu	ALI	6×10^7	7×10^7
	DAC	—	3×10^4
^{146}Eu	ALI	4×10^7	5×10^7
	DAC	—	2×10^4
^{147}Eu	ALI	1×10^8	6×10^7
	DAC	—	3×10^4
^{148}Eu	ALI	4×10^7	1×10^7
	DAC	—	5×10^3
^{149}Eu	ALI	4×10^8	1×10^8
	DAC	—	5×10^4
^{150}Eu	ALI	1×10^8	3×10^8
($T_{\frac{1}{2}} = 12.62$ h)	DAC	—	1×10^5
^{150}Eu	ALI	3×10^7	7×10^5
($T_{\frac{1}{2}} = 34.2$ y)	DAC	—	3×10^2
^{152m}Eu	ALI	1×10^8	2×10^8
	DAC	—	1×10^5
^{152}Eu	ALI	3×10^7	9×10^5
	DAC	—	4×10^2
^{154}Eu	ALI	2×10^7	7×10^5
	DAC	—	3×10^2
^{155}Eu	ALI	1×10^8	3×10^6 (5×10^6) Bone surf.
	DAC	—	1×10^3
^{156}Eu	ALI	2×10^7	2×10^7
	DAC	—	7×10^3
^{157}Eu	ALI	8×10^7	2×10^8
	DAC	—	8×10^4
^{158}Eu	ALI	7×10^8	2×10^9
	DAC	—	9×10^5

METABOLIC DATA FOR GADOLINIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for gadolinium.

2. Metabolic Model

(a) *Uptake to blood*

Data from experiments on the acute toxicity of nitrates and oxides of gadolinium to the rat (Bruce, Hietbrink and DuBois, 1963) indicate that the fractional absorption of compounds of gadolinium from the gastrointestinal tract is small. By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of gadolinium.

(b) *Inhalation classes*

Experiments in which gadolinium chloride was intratracheally instilled into rats (Zalikin, 1972) indicate that gadolinium administered in this form is rapidly and almost completely translocated from the lungs to the systemic circulation.

In this report, the more insoluble compounds of gadolinium, oxides, hydroxides and fluorides are assigned to inhalation class W and all other commonly occurring compounds of the element to inhalation class D.

Inhalation class	f_1
D	3×10^{-4}
W	3×10^{-4}
Y	—

(c) *Distribution and retention*

The distribution and retention of gadolinium in rats after intravenous injection of the chloride has been studied by Zalikin (1972). The element is cleared rapidly from the blood depositing in the liver, skeleton and kidneys. These three tissues are also identified as important sites of deposition in studies on rats bearing Yoshida sarcomas (Hisada and Ando, 1973). Liver and skeleton were also identified as the main sites of deposition in rats injected intramuscularly with ^{159}Gd (Durbin *et al.*, 1956).

Gadolinium is lost quite rapidly from the liver and kidneys of the rat, but exhibits long-term retention in the skeleton (Zalikin, 1972). However, studies with cerium have shown that although that element is rapidly lost from the livers of rats it exhibits long-term retention in the livers of cats and dogs (Moskalev, Zalikin and Stepanov, 1970). Chemical similarities between cerium and gadolinium suggest that this effect will also occur for the latter element.

Based on the data reviewed above, it is here assumed that of gadolinium leaving the transfer compartment fractions of 0.45, 0.3 and 0.03 are translocated to mineral bone, liver and kidneys

respectively. The remaining fraction of gadolinium leaving the transfer compartment is assumed to go directly to excretion. Gadolinium translocated to the kidneys is assumed to be retained there with a biological half-life of 10 days. By analogy with cerium, the biological half-life of gadolinium deposited in the liver or in mineral bone is assumed to be 3 500 days.

3. Classification of Isotopes for Bone Dosimetry

By analogy with promethium all isotopes of gadolinium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of gadolinium

Radionuclide		Oral	Inhalation	
			Class D	Class W
		$f_1 = 3 \times 10^{-4}$	$f_1 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{145}Gd	ALI	2×10^9 (2×10^9) ST Wall	6×10^9	6×10^9
	DAC	—	2×10^6	3×10^6
^{146}Gd	ALI	5×10^7	5×10^6	1×10^7
	DAC	—	2×10^3	4×10^3
^{147}Gd	ALI	7×10^7	2×10^8	1×10^8
	DAC	—	6×10^4	5×10^4
^{148}Gd	ALI	4×10^5 (9×10^5) Bone surf.	3×10^2 (6×10^2) Bone surf.	1×10^3 (2×10^3) Bone surf.
	DAC	—	1×10^{-1}	5×10^{-1}
^{149}Gd	ALI	1×10^8	8×10^7	9×10^7
	DAC	—	3×10^4	4×10^4
^{151}Gd	ALI	2×10^8	1×10^7 (2×10^7) Bone surf.	4×10^7
	DAC	—	6×10^3	2×10^4
^{152}Gd	ALI	6×10^5 (1×10^6) Bone surf.	4×10^2 (8×10^2) Bone surf.	2×10^3 (3×10^3) Bone surf.
	DAC	—	2×10^{-1}	6×10^{-1}
^{153}Gd	ALI	2×10^8	5×10^6 (9×10^6) Bone surf.	2×10^7
	DAC	—	2×10^3	9×10^3
^{159}Gd	ALI	1×10^8	3×10^8	2×10^8
	DAC	—	1×10^5	9×10^4

METABOLIC DATA FOR TERBIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for terbium.

2. Metabolic Model

(a) *Uptake to blood*

The fractional absorption of terbium from the gastrointestinal tract of the rat has been variously reported to be less than 10^{-3} (Durbin *et al.*, 1956) and less than 5×10^{-4} (Moskalev, Zalikin and Stepanov, 1972). By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of terbium.

(b) *Inhalation classes*

The small amount of data available from experiments on rats (Moskalev, Zalikin and Stepanov, 1972) indicate that the retention of compounds of terbium in the lung is similar to the retention of compounds of the more extensively studied lanthanide europium. For this reason all commonly occurring compounds of terbium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	—

(c) *Distribution and retention*

In experiments on rats, intramuscularly or intravenously injected terbium is deposited mainly in the skeleton, liver and kidneys (Durbin *et al.*, 1956a; Durbin *et al.*, 1956b, Magnusson, 1963; Zalikin and Tronova, 1969; Beyer *et al.*, 1978). Usually rather more terbium is deposited in the skeleton than in the liver. However, this is not always the case (Magnusson, 1963).

There is long-term retention of terbium deposited in the skeleton (Durbin *et al.*, 1956b; Zalikin and Tronova, 1969), whereas most of the terbium deposited in the liver or the kidneys of the rat is lost from those tissues with a biological half-life of a few days (Durbin *et al.*, 1956b; Zalikin and Tronova, 1969; Beyer *et al.*, 1978). However, studies with cerium have shown that although that element is rapidly lost from the livers of rats it exhibits long-term retention in the livers of cats and dogs (Moskalev, Zalikin and Stepanov, 1970). Chemical similarities between cerium and terbium suggest that this effect will also occur for the latter element.

Based on the data reviewed above, it is here assumed that of terbium leaving the transfer compartment fractions of 0.5, 0.25 and 0.05 are translocated to mineral bone, the liver and the kidneys respectively. The remaining fraction of terbium leaving the transfer compartment is assumed to go directly to excretion. By analogy with cerium, terbium deposited in mineral bone or in the liver is assumed to be retained there with a biological half-life of 3 500 days. Terbium deposited in the kidneys is assumed to be retained there with a biological half-life of 10 days.

3. Classification of Isotopes for Bone Dosimetry

By analogy with promethium, all isotopes of terbium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of terbium

			Inhalation
		Oral	Class W
Radionuclide		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{147}Tb	ALI	3×10^8	1×10^9
	DAC	—	5×10^5
^{149}Tb	ALI	2×10^8	3×10^7
	DAC	—	1×10^4
^{150}Tb	ALI	2×10^8	8×10^8
	DAC	—	3×10^5
^{151}Tb	ALI	1×10^8	3×10^8
	DAC	—	1×10^5
^{153}Tb	ALI	2×10^8	3×10^8
	DAC	—	1×10^5
^{154}Tb	ALI	6×10^7	2×10^8
	DAC	—	7×10^4
^{155}Tb	ALI	2×10^8	3×10^8
	DAC	—	1×10^5
^{156m}Tb	ALI	3×10^8	3×10^8
(Tr = 24.4 h)	DAC	—	1×10^5
^{156m}Tb	ALI	6×10^8	1×10^9
(Tr = 5.0 h)	DAC	—	4×10^5
^{156}Tb	ALI	4×10^7	5×10^7
	DAC	—	2×10^4
^{157}Tb	ALI	2×10^9 (2×10^9) LLI Wall	1×10^7 (2×10^7) Bone surf.
	DAC	—	5×10^3
^{158}Tb	ALI	5×10^7	7×10^5
	DAC	—	3×10^2
^{160}Tb	ALI	3×10^7	8×10^6
	DAC	—	4×10^3
^{161}Tb	ALI	6×10^7 (7×10^7) LLI Wall	6×10^7
	DAC	—	2×10^4

METABOLIC DATA FOR DYSPROSIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for dysprosium.

2. Metabolic Model

(a) *Uptake to blood*

Experiments on the acute toxicity of nitrates and oxides of dysprosium to rats (Bruce, Hietbrink and DuBois, 1963) indicate that the fractional absorption of the element from the gastrointestinal tract is small. By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of dysprosium.

(b) *Inhalation classes*

There appear to be no relevant data available concerning the inhalation of compounds of dysprosium. By analogy with the more extensively studied lanthanides samarium and europium, all commonly occurring compounds of dysprosium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	—

(c) *Distribution and retention*

The small amount of data available from studies on mice and rats (Durbin *et al.*, 1956; Hayes, Byrd and Carlton, 1968; Hisada, Ando and Suzuki, 1976) indicate that of dysprosium entering the systemic circulation fractions of about 0.6, 0.1 and 0.02 are deposited in the skeleton, the liver and the kidneys respectively.

In this report it is assumed that of dysprosium leaving the transfer compartment fractions of 0.6, 0.1 and 0.02 are translocated to mineral bone, the liver and the kidneys respectively. The remaining fraction of dysprosium leaving the transfer compartment is assumed to go directly to excretion. By analogy with cerium, dysprosium deposited in mineral bone or the liver is assumed to be retained there with a biological half-life of 3 500 days. By analogy with terbium, which is dysprosium's nearest neighbour in the lanthanide series, dysprosium translocated to the kidneys is assumed to be retained there with a biological half-life of 10 days.

(d) *Chelated compounds*

Chelated forms of dysprosium are not considered in this report. Their metabolism differs considerably from that of other compounds of the element (Hayes, Byrd and Carlton, 1968).

3. Classification of Isotopes for Bone Dosimetry

By analogy with promethium, all isotopes of dysprosium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of dysprosium

			Inhalation
		Oral	Class W
Radionuclide		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{155}Dy	ALI	3×10^8	9×10^8
	DAC	—	4×10^5
^{157}Dy	ALI	7×10^8	2×10^9
	DAC	—	1×10^6
^{159}Dy	ALI	5×10^8	9×10^7
	DAC	—	4×10^4
^{165}Dy	ALI	5×10^8	2×10^9
	DAC	—	7×10^5
^{166}Dy	ALI	2×10^7 (3×10^7) LLI Wall	3×10^7
	DAC	—	1×10^4

METABOLIC DATA FOR HOLMIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for holmium.

2. Metabolic Model

(a) *Uptake to blood*

Experiments on the acute toxicity of holmium to rats (Bruce, Hietbrink and DuBois, 1963) indicate that the fractional absorption of nitrates and oxides of the element from the gastrointestinal tract is small. In this report f_1 is taken to be 3×10^{-4} for all compounds of holmium.

(b) *Inhalation classes*

There do not appear to be any relevant data available concerning the inhalation of holmium. By analogy with the more extensively studied lanthanide thulium, all compounds of holmium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	—

(c) *Distribution and retention*

Although it is generally agreed that the liver and the skeleton are the two main tissues of deposition for holmium their relative importance is not well established. Studies on the rat after intramuscular injection (Durbin *et al.*, 1956) indicate that the fractions of holmium entering the systemic circulation which are deposited in the skeleton and the liver are about 0.6 and 0.05 respectively. However, other experiments on rats (Magnusson, 1963) indicate that about 0.6 of intravenously injected holmium is deposited in the liver. This latter result is in good agreement with studies on mice in which 0.4 of intraperitoneally injected holmium was found in the liver and only about 0.05 in the skeleton (Spode, Gensicke and Glaser, 1959).

Studies on mice (Spode, Gensicke and Glaser, 1959) also indicate that about 0.05 of holmium entering the systemic circulation is deposited in the pancreas.

Based on the data reviewed above, it is here assumed that of holmium leaving the transfer compartment fractions of 0.4, 0.4 and 0.05 are translocated to mineral bone, the liver and the pancreas respectively. The remaining fraction of holmium leaving the transfer compartment is assumed to go directly to excretion. By analogy with cerium, holmium translocated to any organ or tissue is assumed to be retained there with a biological half-life of 3 500 days.

3. Classification of Isotopes for Bone Dosimetry

By analogy with promethium, all isotopes of holmium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of holmium

		Oral	Inhalation Class W
Radionuclide		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{155}Ho	ALI	2×10^{9}	6×10^{9}
	DAC	—	2×10^{6}
^{157}Ho	ALI	1×10^{10}	5×10^{10}
	DAC	—	2×10^{7}
^{159}Ho	ALI	8×10^{9}	4×10^{10}
	DAC	—	2×10^{7}
^{161}Ho	ALI	4×10^{9}	2×10^{10}
	DAC	—	6×10^{6}
^{162m}Ho	ALI	2×10^{9}	1×10^{10}
	DAC	—	4×10^{6}
^{162}Ho	ALI	2×10^{10} (3×10^{10}) ST Wall	9×10^{10}
	DAC	—	4×10^{7}
^{164m}Ho	ALI	4×10^{9}	1×10^{10}
	DAC	—	5×10^{6}
^{164}Ho	ALI	7×10^{9} (8×10^{9}) ST Wall	2×10^{10}
	DAC	—	1×10^{7}
^{166m}Ho	ALI	2×10^{7}	3×10^{5}
	DAC	—	1×10^{2}
^{166}Ho	ALI	3×10^{7} (3×10^{7}) LLI Wall	7×10^{7}
	DAC	—	3×10^{4}
^{167}Ho	ALI	6×10^{8}	2×10^{9}
	DAC	—	9×10^{5}

METABOLIC DATA FOR ERBIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for erbium.

2. Metabolic Model

(a) *Uptake to blood*

There do not appear to be any relevant data available concerning the absorption of erbium from the gastrointestinal tract. By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of erbium.

(b) *Inhalation classes*

There do not appear to be any relevant data available on the inhalation of compounds of erbium. By analogy with the more extensively studied lanthanide thulium, its nearest neighbour in the lanthanide series, all compounds of erbium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	—

(c) *Distribution and retention*

Data from experiments on mice, rats and rabbits (Durbin *et al.*, 1956; O'Mara, McAfee and Subramanian, 1969; Rao, Goodwin and Khalil, 1974; Hisanda, Ando and Suzuki, 1976) indicate that erbium is mainly deposited in the skeleton with the liver and, to some extent, the kidneys as secondary sites of deposition.

Based on the data reviewed above, it is here assumed that of erbium leaving the transfer compartment fractions of 0.6 and 0.05 are translocated to mineral bone and the liver respectively. A further fraction, 0.1, is assumed to be uniformly distributed throughout all organs and tissues of the body other than the liver and the skeleton. The remaining fraction of erbium leaving the transfer compartment is assumed to go directly to excretion. By analogy with cerium, erbium translocated to any organ or tissue is assumed to be retained there with a biological half-life of 3 500 days.

3. Classification of Isotopes for Bone Dosimetry

Since none of the isotopes of erbium considered in this report have radioactive half-lives in excess of 10 days, and by analogy with promethium, all isotopes of erbium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of erbium

			Inhalation
		Oral	Class W
Radionuclide		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{161}Er	ALI	6×10^8	2×10^9
	DAC	—	1×10^6
^{165}Er	ALI	2×10^9	7×10^9
	DAC	—	3×10^6
^{169}Er	ALI	1×10^8 (1×10^8) LLI Wall	9×10^7
	DAC	—	4×10^4
^{171}Er	ALI	1×10^8	4×10^8
	DAC	—	2×10^5
^{172}Er	ALI	4×10^7 (5×10^7) LLI Wall	5×10^7
	DAC	—	2×10^4

METABOLIC DATA FOR THULIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for thulium.

2. Metabolic Model

(a) *Uptake to blood*

There do not appear to be any relevant data available on the gastrointestinal absorption of thulium. By analogy with the other lanthanides the fractional absorption of the element from the gastrointestinal tract is expected to be very small. By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of thulium.

(b) *Inhalation classes*

Data from experiments in which dogs inhaled an aerosol of thulium oxide (Thomas and Kingsley, 1970) and studies on a man who had accidentally inhaled sub-micron particles of thulium oxide (Yabe *et al.*, 1974) indicate that this compound should be assigned to inhalation class W. In this report all commonly occurring compounds of thulium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	—

(c) *Distribution and retention*

Experiments on mice (Steinberg *et al.*, 1973; Beyer *et al.*, 1978), rats (Durbin *et al.*, 1956a; Durbin *et al.*, 1956b; Thomas and Kingsley, 1969; Beyer *et al.*, 1978; Hiraki *et al.*, 1978), rabbits (Chandra *et al.*, 1971; Beyer *et al.*, 1978) dogs (Thomas and Kingsley, 1970) and man (Steinberg *et al.*, 1973) demonstrate that thulium is mainly deposited in the skeleton with the liver and to some extent kidneys as secondary organs of deposition.

Studies on rats (Durbin *et al.*, 1956b; Thomas and Kingsley, 1969) give a biological half-life for thulium retention in the skeleton in excess of 1 000 days. Other experiments on rats (Hiraki *et al.*, 1978) also indicate a very long biological half-life for thulium in the skeleton and this is confirmed by measurements on the distribution of thulium in dogs after inhalation of the oxide (Thomas and Kingsley, 1970).

In man, the whole body retention of thulium has been studied for 15 days after intravenous injection of the citrate into four patients (Steinberg *et al.*, 1973). About 0.2 of the injected thulium was lost from the body in the first 3 days after injection. Subsequently only about 0.002 of the systemic thulium was excreted in the urine each day.

Based on the data reviewed above, it is here assumed that of thulium leaving the transfer compartment fractions of 0.65 and 0.04 are translocated to mineral bone and the liver respectively. A further fraction, 0.1, is assumed to be uniformly distributed throughout all organs and tissues of the body other than the liver and mineral bone. The remaining fraction of thulium leaving the transfer compartment is assumed to go directly to excretion. By analogy with the more extensively studied lanthanide cerium, thulium translocated to any organ or tissue is assumed to be retained there with a biological half-life of 3 500 days.

3. Classification of Isotopes for Bone Dosimetry

Autoradiographic studies on 6 month old puppies (Jowsey, Rowland and Marshall, 1958) have shown that ^{91}Y, ^{144}Ce and ^{170}Tm are deposited on highly calcified, nongrowing bone surfaces. This indicates that the distribution of thulium in bone more nearly resembles the distribution of the actinides than it does the distribution of the alkaline earths. For this reason all isotopes of thulium are assumed to be uniformly distributed over bone surfaces at all times following the deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of thulium

Radionuclide		Oral	Inhalation Class W
		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{162}Tm	ALI	2×10^9 (3×10^9) ST Wall	1×10^{10}
	DAC	—	4×10^6
^{166}Tm	ALI	2×10^8	5×10^8
	DAC	—	2×10^5
^{167}Tm	ALI	8×10^7 (9×10^7) LLI Wall	7×10^7
	DAC	—	3×10^4
^{170}Tm	ALI	3×10^7 (4×10^7) LLI Wall	8×10^6
	DAC	—	3×10^3
^{171}Tm	ALI	4×10^8 (5×10^8) LLI Wall	1×10^7 (2×10^7) Bone surf.
	DAC	—	4×10^3
^{172}Tm	ALI	3×10^7 (3×10^7) LLI Wall	4×10^7
	DAC	—	2×10^4
^{173}Tm	ALI	2×10^8	4×10^8
	DAC	—	2×10^5
^{175}Tm	ALI	2×10^9 (3×10^9) ST Wall	1×10^{10}
	DAC	—	4×10^6

METABOLIC DATA FOR YTTERBIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for ytterbium.

2. Metabolic Model

(a) *Uptake to blood*

The fractional absorption of ytterbium from the gastrointestinal tract of the rat has been reported as less than 5×10^{-4} (Moskalev, Zalikin and Stepanov, 1972). By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of ytterbium.

(b) *Inhalation classes*

The small amount of data available on the inhalation or intratracheal administration of ytterbium (Moskalev, Zalikin and Stepanov, 1972) indicates that the element is only slowly cleared from the lung. Thus the behaviour of ytterbium in the lung appears to resemble that of the lighter lanthanides, cerium and promethium, rather than that of europium which is closer to it in mass. Therefore, by analogy with cerium, oxides, hydroxides, and fluorides of ytterbium are assigned to inhalation class Y and all other commonly occurring compounds of the element are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	3×10^{-4}

(c) *Distribution and retention*

Data from studies on mice (Higasi *et al.*, 1973; Francke *et al.*, 1976), rats (Moskalev, Zalikin and Stepanov, 1972; Hisada and Ando, 1973; Baltrukiewicz, Burakowski and Pogorzelska-Lis, 1975; Beyer *et al.*, 1978) and man (Tatsuno, Bunko and Kato, 1974) demonstrate that the skeleton is the main organ of deposition for ytterbium, although deposition also occurs in the liver, in the kidneys and, to some extent, in the spleen.

In the mouse the whole body retention of ytterbium, intravenously injected as the citrate, was found to be well described by two exponentials with biological half-lives of 0.3 and 800 days (Anzai *et al.*, 1974). In the rat three exponentials were found with biological half-lives of 0.15, 8 and 850 days (Ando *et al.*, 1977).

Data from studies on mice (Francke *et al.*, 1976) and rats (Baltrukiewicz, Burakowski and Pogorzelska-Lis, 1975; Beyer *et al.*, 1978) indicate that ytterbium is more rapidly lost from the kidneys than it is from other tissues.

Based on the data reviewed above, it is here assumed that of ytterbium leaving the transfer compartment fractions of 0.5, 0.03, 0.02 and 0.005 are translocated to mineral bone, liver,

kidneys and spleen respectively. The remaining fraction of ytterbium leaving the transfer compartment is assumed to go directly to excretion. By analogy with europium and terbium, and because of the experimental data reviewed above, ytterbium translocated to the kidneys is assumed to be retained there with a biological half-life of 10 days. Ytterbium translocated to any other organ or tissue of the body is assumed to be retained there with a biological half-life of 3 500 days. This half-life, corresponding to the biological half-life used for cerium, is adopted because the shorter whole-body half-life of 800 days found for rodents is thought to be due in part to the relatively rapid turnover of the rodent skeleton.

(d) *Chelated compounds*

Chelated compounds of ytterbium are not considered in this report. Their metabolic behaviour differs considerably from that of other compounds of the element (Baltrukiewicz, Burakowski and Pogorzelska-Lis, 1975).

3. Classification of Isotopes for Bone Dosimetry

By analogy with promethium, and because no isotope of ytterbium considered in this report has a radioactive half-life of more than 32 days, all isotopes of the element are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of ytterbium

Radionuclide		Oral	Inhalation	
			Class W	Class Y
		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{162}Yb	ALI	3×10^{9}	1×10^{10}	1×10^{10}
	DAC	—	5×10^{6}	4×10^{6}
^{166}Yb	ALI	5×10^{7}	7×10^{7}	7×10^{7}
	DAC	—	3×10^{4}	3×10^{4}
^{167}Yb	ALI	1×10^{10}	3×10^{10}	3×10^{10}
	DAC	—	1×10^{7}	1×10^{7}
^{169}Yb	ALI	7×10^{7}	3×10^{7}	3×10^{7}
	DAC	—	1×10^{4}	1×10^{4}
^{175}Yb	ALI	1×10^{8} (1×10^{8}) LLI Wall	1×10^{8}	1×10^{8}
	DAC	—	5×10^{4}	5×10^{4}
^{177}Yb	ALI	6×10^{8}	2×10^{9}	2×10^{9}
	DAC	—	8×10^{5}	7×10^{5}
^{178}Yb	ALI	5×10^{8}	1×10^{9}	1×10^{9}
	DAC	—	6×10^{5}	6×10^{5}

METABOLIC DATA FOR LUTETIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for lutetium.

2. Metabolic Model

(a) *Uptake to blood*

Data on the acute toxicity of lutetium chloride in mice (Haley *et al.*, 1964) and lutetium as the nitrate or the oxide in rats (Bruce, Hietbrink and DuBois, 1963) indicate that the fractional absorption of lutetium from the gastrointestinal tract is typically less than 5×10^{-3}. By analogy with the more extensively studied lanthanide cerium, f_1 is taken to be 3×10^{-4} for all compounds of lutetium.

(b) *Inhalation classes*

There do not appear to be any relevant data available concerning the behaviour of lutetium after inhalation. As in the case of ytterbium, in its lung retention lutetium probably more closely resembles cerium and promethium than it does europium. Therefore, by analogy with cerium, oxides, hydroxides and fluorides of lutetium are assigned to inhalation class Y and all other commonly occurring compounds of the element are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	3×10^{-4}
Y	3×10^{-4}

(c) *Distribution and retention*

Data from experiments on mice (Spode, Gensicke and Glaser, 1958; Müller, Linzner and Schäffer, 1978), rats (Durbin *et al.*, 1956; Marx, 1972; Beyer *et al.*, 1978) and rabbits (O'Mara, McAfee and Subramanian, 1969) indicate that more than half the lutetium entering the systemic circulation is deposited in the skeleton with only small amounts going to the liver and kidneys.

In experiments on mice (Müller, Linzner and Schäffer, 1978) about half the lutetium entering the systemic circulation was excreted in the first 24 h after injection. However, in experiments on rats the fractional excretion in the first 24 h was between 0.1 and 0.2 (Durbin *et al.*, 1956; Marx, 1972; Beyer *et al.*, 1978).

In common with other, more extensively studied lanthanides, lutetium has a biological half-life of between 10 and 40 days in the soft tissues of mice and rats but has a very long biological half-life in the skeleton (Marx, 1972; Müller, Linzner and Schäffer, 1978). However, studies with cerium have shown that although that lanthanide is rapidly lost from the livers of mice and rats it exhibits long-term retention in the livers of cats and dogs (Moskalev, Zalikin and Stepanov,

1970). Chemical similarities between cerium and lutetium suggest that this effect will also occur for the latter element.

Based on the data reviewed above, it is here assumed that of lutetium leaving the transfer compartment fractions of 0.6, 0.02 and 0.005 are translocated to mineral bone, the liver and the kidneys respectively. The remaining fraction of lutetium leaving the transfer compartment is assumed to go directly to excretion.

By analogy with the more extensively studied lanthanide cerium, lutetium deposited in mineral bone or the liver is assumed to be retained there with a biological half-life of 3 500 days. By analogy with europium and terbium, lutetium deposited in the kidneys is assumed to be retained there with a biological half-life of 10 days.

3. Classification of Isotopes for Bone Dosimetry

The initial deposition of lutetium in mineral bone is very similar to that of promethium (Müller, Linzner and Schäffer, 1978). Thus, by analogy, in this report it is assumed that all isotopes of lutetium are uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of lutetium

Radionuclide		Oral	Inhalation	
			Class W	Class Y
		$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$	$f_1 = 3 \times 10^{-4}$
^{169}Lu	ALI	9×10^7	2×10^8	2×10^8
	DAC	—	7×10^4	6×10^4
^{170}Lu	ALI	4×10^7	8×10^7	7×10^7
	DAC	—	3×10^4	3×10^4
^{171}Lu	ALI	7×10^7	7×10^7	7×10^7
	DAC	—	3×10^4	3×10^4
^{172}Lu	ALI	4×10^7	4×10^7	4×10^7
	DAC	—	2×10^4	2×10^4
^{173}Lu	ALI	2×10^8	1×10^7 (2×10^7) Bone surf.	1×10^7
	DAC	—	4×10^3	4×10^3
^{174m}Lu	ALI	8×10^7 (1×10^8) LLI Wall	9×10^6 (1×10^7) Bone surf.	8×10^6
	DAC	—	4×10^3	3×10^3
^{174}Lu	ALI	2×10^8	4×10^6 (8×10^6) Bone surf.	6×10^6
	DAC	—	2×10^3	2×10^3
^{176m}Lu	ALI	3×10^8	9×10^8	8×10^8
	DAC	—	4×10^5	4×10^5
^{176}Lu	ALI	3×10^7	2×10^5 (4×10^5) Bone surf.	3×10^5
	DAC	—	7×10^1	1×10^2
^{177m}Lu	ALI	3×10^7	4×10^6 (5×10^6) Bone surf.	3×10^6
	DAC	—	2×10^3	1×10^3
^{177}Lu	ALI	8×10^7 (9×10^7) LLI Wall	8×10^7	8×10^7
	DAC	—	3×10^4	3×10^4
^{178m}Lu	ALI	2×10^9 (2×10^9) ST Wall	7×10^9	6×10^9
	DAC	—	3×10^6	3×10^6
^{178}Lu	ALI	1×10^9 (2×10^9) ST Wall	5×10^9	4×10^9
	DAC	—	2×10^6	2×10^6
^{179}Lu	ALI	2×10^8	7×10^8	6×10^8
	DAC	—	3×10^5	2×10^5

METABOLIC DATA FOR HAFNIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for hafnium.

2. Metabolic Model

(a) *Uptake to blood*

There do not appear to be any relevant data available on the absorption of compounds of hafnium from the gastrointestinal tract. By analogy with the chemically similar and more extensively studied element zirconium, f_1 is taken to be 0.002 for all compounds of hafnium.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, halides, carbides and nitrates of hafnium to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. In the absence of any relevant experimental data this classification is adopted here.

Inhalation class	f_1
D	0.002
W	0.002
Y	—

(c) *Distribution and retention*

The chemical properties of hafnium are very similar to those of zirconium. In the absence of any relevant data on the metabolism of hafnium, the metabolic model for zirconium is used.

Of hafnium leaving the transfer compartment 0.5 is assumed to be translocated to mineral bone and 0.5 is assumed to be uniformly distributed throughout all other organs and tissues of the body. Hafnium deposited in mineral bone is assumed to be retained there with a biological half-life of 8 000 days whereas hafnium translocated to any other organ or tissue is assumed to be retained there with a biological half-life of 7 days.

3. Classification of Isotopes for Bone Dosimetry

By analogy with zirconium all isotopes of hafnium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of hafnium

Radionuclide		Oral	Inhalation	
			Class D	Class W
		$f_1 = 2 \times 10^{-3}$	$f_1 = 2 \times 10^{-3}$	$f_1 = 2 \times 10^{-3}$
^{170}Hf	ALI	1×10^8	2×10^8	2×10^8
	DAC	—	9×10^4	7×10^4
^{172}Hf	ALI	5×10^7	3×10^5	1×10^6
			(7×10^5)	(2×10^6)
			Bone surf.	Bone surf.
	DAC	—	1×10^2	6×10^2
^{173}Hf	ALI	2×10^8	5×10^8	4×10^8
	DAC	—	2×10^5	2×10^5
^{175}Hf	ALI	1×10^8	4×10^7	4×10^7
			(4×10^7)	
			Bone surf.	
	DAC	—	1×10^4	2×10^4
^{177m}Hf	ALI	7×10^8	2×10^9	3×10^9
	DAC	—	9×10^5	1×10^6
^{178m}Hf	ALI	9×10^6	5×10^4	2×10^5
			(9×10^4)	(3×10^5)
			Bone surf.	Bone surf.
	DAC	—	2×10^1	8×10^1
^{179m}Hf	ALI	4×10^7	1×10^7	2×10^7
			(2×10^7)	
			Bone surf.	
	DAC	—	5×10^3	9×10^3
^{180m}Hf	ALI	3×10^8	8×10^8	9×10^8
	DAC	—	3×10^5	4×10^5
^{181}Hf	ALI	4×10^7	6×10^6	2×10^7
			(1×10^7)	
			Bone surf.	
	DAC	—	3×10^3	7×10^3
^{182m}Hf	ALI	1×10^9	3×10^9	5×10^9
	DAC	—	1×10^6	2×10^6
^{182}Hf	ALI	7×10^6	3×10^4	1×10^5
		(1×10^7)	(7×10^4)	(3×10^5)
		Bone surf.	Bone surf.	Bone surf.
	DAC	—	1×10^1	5×10^1
^{183}Hf	ALI	8×10^8	2×10^9	2×10^9
	DAC	—	7×10^5	9×10^5
^{184}Hf	ALI	9×10^7	3×10^8	2×10^8
	DAC	—	1×10^5	1×10^5

METABOLIC DATA FOR TANTALUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for tantalum.

2. Metabolic Model

(a) *Uptake to blood*

Data from experiments on rats (Fleshman, Silva and Shore, 1971) suggest that the fractional absorption of tantalum, administered as potassium tantalate, from the gastrointestinal tract of the rat is about 10^{-3}. Other studies on rats (Doull and DuBois, 1949) indicate that the fractional absorption of tantalum, administered as the oxide, is also small. In this report f_1 is taken as 10^{-3} for all compounds of tantalum.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, halides, carbides and nitrates of tantalum to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. However, experience in man indicates that tantalum oxide behaves as a class Y material (Sill *et al.*, 1969; Newton, 1977).

There is a considerable amount of information on the inhalation of metallic tantalum powders (Upham *et al.*, 1971; Bianco *et al.*, 1974; Causse, 1975; Morrow *et al.*, 1976) and this suggests that elemental tantalum should be assigned to inhalation class Y.

In this report elemental tantalum together with oxides, hydroxides, halides, carbides, nitrates and nitrides of the element are assigned to inhalation class Y. All other commonly occurring compounds of tantalum are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	10^{-3}
Y	10^{-3}

(c) *Distribution and retention*

Experiments on rats (Fleshman, Silva and Shore, 1971) show that tantalum absorbed from the gastrointestinal tract is concentrated in the kidneys and skeleton and that it is more avidly retained in the skeleton than it is in soft tissues. Studies on the distribution of tantalum in rats after intramuscular injection of Ta_2O_5 indicate that the liver, kidneys and skeleton are major organs of deposition (Durbin, 1960). However, the implication of the liver as a site of deposition in these latter studies may reflect the tendency of tantalum to form colloids *in vivo* (Durbin, 1960).

In experiments in which rats ingested potassium tantalate, two components of retention were found with biological half-lives of 1.8–4.7 days and 62–119 days respectively (Fleshman, Silva and Shore, 1971).

In this report it is assumed that of tantalum leaving the transfer compartment fractions of 0.06 and 0.3 are translocated to kidneys and mineral bone respectively. The remaining fraction of tantalum leaving the transfer compartment is assumed to be uniformly distributed throughout all other organs and tissues of the body. Tantalum deposited in mineral bone is assumed to be retained there with a biological half-life of 100 days. Of tantalum deposited in any other organ or tissue equal fractions of 0.5 are assumed to be retained with biological half-lives of 4 and 100 days respectively.

3. Classification of Isotopes for Bone Dosimetry

Of the isotopes of tantalum considered in this report only ^{179}Ta, ^{180}Ta and ^{182}Ta have radioactive half-lives in excess of 15 days. These isotopes are assumed to be uniformly distributed throughout the volume of mineral bone at all times following their deposition in that tissue. All other radioactive isotopes of tantalum are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of tantalum

Radionuclide		Oral	Inhalation	
			Class W	Class Y
		$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$
^{172}Ta	ALI	1×10^9	5×10^9	4×10^9
	DAC	—	2×10^6	2×10^6
^{173}Ta	ALI	2×10^8	7×10^8	6×10^8
	DAC	—	3×10^5	3×10^5
^{174}Ta	ALI	1×10^9	4×10^9	3×10^9
	DAC	—	2×10^6	1×10^6
^{175}Ta	ALI	2×10^8	6×10^8	5×10^8
	DAC	—	2×10^5	2×10^5
^{176}Ta	ALI	1×10^8	5×10^8	4×10^8
	DAC	—	2×10^5	2×10^5
^{177}Ta	ALI	4×10^8	7×10^8	7×10^8
	DAC	—	3×10^5	3×10^5
^{178}Ta	ALI	6×10^8	3×10^9	3×10^9
	DAC	—	1×10^6	1×10^6
^{179}Ta	ALI	8×10^8	2×10^8	3×10^7
	DAC	—	8×10^4	1×10^4
^{180m}Ta	ALI	9×10^8	2×10^9	2×10^9
	DAC	—	1×10^6	9×10^5
^{180}Ta	ALI	6×10^7	2×10^7	9×10^5
	DAC	—	7×10^3	4×10^2
^{182m}Ta	ALI	6×10^9 (8×10^9) ST Wall	2×10^{10}	2×10^{10}
	DAC	—	8×10^6	6×10^6
^{182}Ta	ALI	3×10^7	1×10^7	5×10^6
	DAC	—	5×10^3	2×10^3
^{183}Ta	ALI	3×10^7 (4×10^7) LLI Wall	4×10^7	4×10^7
	DAC	—	2×10^4	2×10^4
^{184}Ta	ALI	7×10^7	2×10^8	2×10^8
	DAC	—	8×10^4	7×10^4
^{185}Ta	ALI	1×10^9	3×10^9	2×10^9
	DAC	—	1×10^6	1×10^6
^{186}Ta	ALI	2×10^9 (3×10^9) ST Wall	9×10^9	8×10^9
	DAC	—	4×10^6	3×10^6

METABOLIC DATA FOR TUNGSTEN

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for tungsten.

2. Metabolic Model

(a) *Uptake to blood*

In experiments in which W-181 labelled tungstate was administered orally to dairy cows the fractional gastrointestinal absorption of tungsten was 0.2 (Mullen, Bretthauer and Stanley, 1976). In sheep and pigs the fractional absorption of tungsten administered as tungstate was typically more than 0.4. However, absorption decreased markedly when the animals were fed on a diet high in roughage (Bell and Sneed, 1970). This suggests that tungsten absorption may be inhibited by adsorption of the element to food particles, especially those high in cellulose. This may explain why in experiments on goats the fractional gastrointestinal absorption of tungsten, administered as tungstate, has been reported as about 0.05 (Ekman *et al.*, 1977). In experiments on rats the fractional absorption of tungsten administered as tungstate was found to be more than 0.4 (Ballou, 1960; Kaye, 1968) whereas the fractional absorption of tungsten administered as tungstic acid was about 0.01 (Ballou, 1960). In dogs, the fractional absorption of tungsten, administered as the oxide, is about 0.25 (Aamodt, 1971). Experiments in which peccaries ingested debris from a nuclear explosion gave a fractional absorption of between 0.1 and 0.2 for tungsten (Chertok and Lake, 1971b and 1971c). In this report f_1 is taken as 0.01 for tungstic acid and 0.3 for all other compounds of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides and hydroxides of tungsten to inhalation class Y, sulphides and halides to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. However, experiments on dogs (Aamodt, 1975) suggest that tungstic oxide behaves as a class D compound. In this report all compounds of tungsten are assigned to inhalation class D.

Inhalation class	f_1
D	0.3
W	—
Y	—

(c) *Distribution and retention*

Various studies have demonstrated that after ingestion, inhalation, intramuscular or intravenous injection high concentrations of tungsten are found in the skeleton, the spleen, the kidneys and the liver (Scott, 1952; Wase, 1956; Ballou, 1960; Durbin, 1960; Fleishman, Krotz and Silva, 1966; Kaye, 1968; Chertok and Lake, 1971a; Aamodt, 1971; Ekman *et al.*, 1977).

Results from studies on the distribution of tungsten in rodents are very erratic. For example

Wase (1956) and Kaye (1968) found that by 3 days after administration the concentration of tungsten in the skeleton was much larger than the concentration in any other organ or tissue, whereas Ballou (1960) found that for the first month after ingestion of sodium tungstate the concentration of tungsten in the spleen was an order of magnitude larger than the concentration of tungsten in the skeleton.

In studies on goats (Ekman *et al.*, 1977) it was found that 8 days after intravenous injection of sodium tungstate the concentrations of tungsten in the liver, the kidneys and the skeleton were all very similar and about a factor of three larger than the concentrations of tungsten in the spleen, the lung, and the adrenal gland. In studies on dogs which inhaled tungstic oxide and were killed 165 days later (Aamodt, 1971) the fractions of the terminal body content of tungsten in the skeleton, the lungs, the kidneys, the liver and skeletal muscle were 0.37, 0.31, 0.15, 0.097 and 0.057 respectively.

The whole-body retention of tungsten in rats in the first 100 days after ingestion of tungstate (Ballou, 1960) was found to be well described by three exponentials with biological half-lives of 1.2, 5 and 70 days. However, other experiments in which tungstate was administered orally to rats (Kaye, 1968) showed that whole-body retention during the first 254 days after administration was well described by three exponentials with biological half-lives of 1, 14, and 1 000 days.

The whole-body retention of tungsten in goats intravenously injected with sodium tungstate (Ekman *et al.*, 1977) in the first 8 days after injection was well described by a retention function of the form

$$R(t)=0.6\ e^{-0.693t/0.15}+0.3\ e^{-0.693t/0.8}+0.1\ e^{-0.693t/6.4}$$

In experiments in which dogs were intravenously injected with sodium tungstate (Aamodt, 1973) whole body retention in the first 131 days after injection was well described by a retention function of the form

$$R(t)=0.815\ e^{-0.693t/0.06}+0.15\ e^{-0.693t/0.4}+0.023\ e^{-0.693t/3.6}+0.011\ e^{-0.693t/100}$$

In this report the model for the systemic distribution and retention of tungsten is mainly based on data from studies on dogs and goats. However, it is considered that the data from experiments on rats which indicate a very long-term component of retention in the skeleton cannot be disregarded. It is, therefore, assumed that of tungsten leaving the transfer compartment 0.95 goes directly to excretion, 0.025 is translocated to mineral bone, 0.01 is translocated to the kidneys, 0.01 is translocated to the liver and 0.005 is translocated to the spleen. Of tungsten translocated to mineral bone, fractions of 0.2, 0.1 and 0.7 are assumed to be retained there with biological half-lives of 5, 100 and 1 000 days respectively. Of tungsten translocated to any other organ or tissue, fractions of 0.7 and 0.3 are assumed to be retained with biological half-lives of 5 and 100 days respectively.

3. Classification of Isotopes for Bone Dosimetry

Of the isotopes of tungsten considered in this report only ^{178}W, ^{181}W, ^{185}W and ^{188}W have radioactive half-lives in excess of 15 days. These isotopes are assumed to be uniformly distributed throughout the volume of mineral bone at all times following their deposition in that tissue. All other radioactive isotopes of tungsten are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of tungsten

Radionuclide		Oral		Inhalation
				Class D
		$f_1 = 1 \times 10^{-2}$	$f_1 = 3 \times 10^{-1}$	$f_1 = 3 \times 10^{-1}$
^{176}W	ALI	4×10^{8}	5×10^{8}	2×10^{9}
	DAC	—	—	8×10^{5}
^{177}W	ALI	8×10^{8}	9×10^{8}	3×10^{9}
	DAC	—	—	1×10^{6}
^{178}W	ALI	2×10^{8}	3×10^{8}	7×10^{8}
	DAC	—	—	3×10^{5}
^{179}W	ALI	2×10^{10}	2×10^{10}	6×10^{10}
	DAC	—	—	3×10^{7}
^{181}W	ALI	6×10^{8}	7×10^{8}	1×10^{9}
	DAC	—	—	5×10^{5}
^{185}W	ALI	8×10^{7} (1×10^{8}) LLI Wall	1×10^{8} (1×10^{8}) LLI Wall	2×10^{8}
	DAC	—	—	1×10^{5}
^{187}W	ALI	7×10^{7}	1×10^{8}	3×10^{8}
	DAC	—	—	1×10^{5}
^{188}W	ALI	1×10^{7} (2×10^{7}) LLI Wall	2×10^{7} (3×10^{7}) LLI Wall	5×10^{7}
	DAC	—	—	2×10^{4}

METABOLIC DATA FOR PLATINUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for platinum.

2. Metabolic Model

(a) *Uptake to blood*

The fractional absorption of platinum, administered as the chloride, from the gastrointestinal tract of the rat has been measured to be about 0.01 (Moore *et al.*, 1975a). This small fractional absorption is in agreement with results from experiments on the acute toxicity of various salts of platinum to rats (Holbrook *et al.*, 1975). In this report f_1 is taken to be 0.01 for all compounds of platinum.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides and hydroxides of platinum to inhalation class Y, halides and nitrates to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. However, experiments on rats (Moore *et al.*, 1975b) suggest that platinum whether inhaled as the metal, the oxide or the sulphate is rapidly translocated from the lungs. In this report all compounds of platinum are assigned to inhalation class D.

Inhalation class	f_1
D	0.01
W	—
Y	—

(c) *Distribution and retention*

Experiments on rats have shown that, after intravenous injection, platinum is concentrated in the kidneys and to some extent also in the liver, the spleen and the adrenals (Moore *et al.*, 1975a). In studies on the distribution of platinum in the rat, after inhalation of the metal, high concentrations of the element were found in the kidneys and, to a lesser extent, in bone (Moore *et al.*, 1975b). Analysis of the concentration of stable platinum in various tissues of rats fed a diet supplemented with $Pt(SO_4)_2 . 4H_2O$ confirm that the kidneys take up platinum to a considerable extent (Yoakum, Stewart and Sterrett, 1975).

Studies on the distribution and retention of platinum in rats after intravenous injection of the chloride (Moore *et al.*, 1975a) indicate that of platinum entering the systemic circulation about 0.1 is rapidly excreted and that most of the remainder is retained in the body with a biological half-life of 8 days. However, a small long-term component of platinum retention is also indicated by these experiments.

Studies on mice, rabbits and dogs indicate that the distribution of platinum after intravenous administration of the antineoplastic drug *cis*-diamminedichloroplatinum (II) is very similar to the distribution found after intravenous administration of other chemical forms of the element (Lange, Spencer and Harder, 1972; Litterst *et al.*, 1976; Schlesinger, Manaka and Wolf, 1977). In studies on man about 0.25– 0.3 of the platinum injected as *cis*-$Pt(NH_2)_2Cl_2$ was excreted during the first 24 h and much of the remainder was retained with a biological half-life of 8–10 days (Smith and Taylor, 1974).

An estimate of the biological half-life of the long-term component of platinum retention in the body can be obtained from experiments in which various species were injected intravenously with compounds of the chemically similar element iridium (Furchner, Richmond and Drake, 1971). In these experiments three components of whole body retention were found with biological half-lives of 0.3, 8 and 200 days respectively.

In this report it is assumed that of platinum leaving the transfer compartment a fraction of 0.2 goes directly to excretion. Further fractions of 0.1, 0.1, 0.01 and 0.001 are assumed to be translocated to the kidneys, the liver, the spleen and the adrenal glands respectively. The remaining fraction of platinum leaving the transfer compartment is assumed to be uniformly distributed throughout all other organs and tissues of the body. Of platinum deposited in any organ or tissue, fractions of 0.95 and 0.05 are assumed to be retained with biological half-lives of 8 and 200 days respectively.

3. Classification of Isotopes for Bone Dosimetry

Platinum is assumed to be uniformly distributed throughout all organs and tissues of the body other than the kidneys, liver, spleen and adrenals. Therefore, a classification of isotopes of the element for the purposes of bone dosimetry is not required.

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of platinum

			Inhalation
		Oral	Class D
Radionuclide		$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$
^{186}Pt	ALI	5×10^{8}	1×10^{9}
	DAC	—	6×10^{5}
^{188}Pt	ALI	6×10^{7}	6×10^{7}
	DAC	—	3×10^{4}
^{189}Pt	ALI	4×10^{8}	1×10^{9}
	DAC	—	4×10^{5}
^{191}Pt	ALI	1×10^{8}	3×10^{8}
	DAC	—	1×10^{5}
^{193m}Pt	ALI	9×10^{7} (1×10^{8}) LLI Wall	2×10^{8}
	DAC	—	9×10^{4}
^{193}Pt	ALI	1×10^{9} (2×10^{9}) LLI Wall	9×10^{8}
	DAC	—	4×10^{5}
^{195m}Pt	ALI	7×10^{7} (8×10^{7}) LLI Wall	2×10^{8}
	DAC	—	7×10^{4}
^{197m}Pt	ALI	6×10^{8}	2×10^{9}
	DAC	—	7×10^{5}
^{197}Pt	ALI	1×10^{8}	4×10^{8}
	DAC	—	1×10^{5}
^{199}Pt	ALI	2×10^{9}	5×10^{9}
	DAC	—	2×10^{6}
^{200}Pt	ALI	4×10^{7}	1×10^{8}
	DAC	—	5×10^{4}

METABOLIC DATA FOR THALLIUM

1. Metabolism

No body content of thallium is given for Reference Man (ICRP, 1975). However, the daily intake of the element in food and fluids is estimated to be 1.5 μg.

2. Metabolic Model

(a) *Uptake to blood*

Thallium is readily absorbed from the gastrointestinal tract whether administered as the sulphate (Lund, 1956) or the nitrate (Lie, Thomas and Scott, 1960). In this report f_1 is taken as 1 for all compounds of the element.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides, hydroxides, halides and nitrates of thallium to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. However, most of the compounds which the Task Group assigned to inhalation class W are highly soluble in aqueous media. Further, experiments in which thallium nitrate was administered to rats by intratreacheal instillation (Lie, Thomas and Scott, 1960) suggest that this compound should be assigned to inhalation class D. In this report all compounds of thallium are assigned to inhalation class D.

Inhalation class	f_1
D	1
W	—
Y	—

(c) *Distribution and retention*

The metabolism of intravenously injected thallium has been studied extensively in various species including man (Barclay, Peacock and Karnofsky, 1953; Lund, 1956; Lie, Thomas and Scott, 1960; Bradley-Moore *et al.*, 1975; Atkins *et al.*, 1977; Strauss, Harrison and Pitt, 1977; Suzuki *et al.*, 1978). There is general agreement that the element is rapidly lost from blood and is fairly uniformly distributed throughout all organs and tissues of the body except for the kidneys which contain about 0.03 of the injected activity (Bradley-Moore *et al.*, 1975; Atkins *et al.*, 1977; Strauss, Harrison and Pitt, 1977; Suzuki, *et al.*, 1978). Studies on goats and men show that all organs and tissues of the body, including the kidneys, lose thallium at approximately the same rate (Bradley-Moore *et al.*, 1975; Atkins *et al.*, 1977).

In normal man the biological half-life of thallium in the body is about 10 days (Atkins *et al.*, 1977).

In this report it is assumed that thallium entering the systemic circulation is instantaneously translocated to the various organs and tissues of the body. Of this thallium 0.03 is assumed to be

deposited in the kidneys and 0.97 is assumed to be uniformly distributed throughout all other organs and tissues of the body. Thallium deposited in any organ or tissue is assumed to be retained there with a biological half-life of 10 days.

3. Classification of Isotopes for Bone Dosimetry

Thallium is assumed to be uniformly distributed throughout all organs and tissues of the body other than the kidneys. Therefore, a classification of isotopes of the element for the purpose of bone dosimetry is not required.

References

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Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of thallium

			Inhalation
		Oral	Class D
Radionuclide		$f_1 = 1$	$f_1 = 1$
^{194m}Tl	ALI	2×10^9 (3×10^9) ST Wall	6×10^9
	DAC	—	2×10^6
^{194}Tl	ALI	9×10^9 (1×10^{10}) ST Wall	2×10^{10}
	DAC	—	9×10^6
^{195}Tl	ALI	2×10^9	5×10^9
	DAC	—	2×10^6
^{197}Tl	ALI	3×10^9	4×10^9
	DAC	—	2×10^6
^{198m}Tl	ALI	1×10^9	2×10^9
	DAC	—	8×10^5
^{198}Tl	ALI	7×10^8	1×10^9
	DAC	—	5×10^5
^{199}Tl	ALI	2×10^9	3×10^9
	DAC	—	1×10^6
^{200}Tl	ALI	3×10^8	4×10^8
	DAC	—	2×10^5
^{201}Tl	ALI	6×10^8	8×10^8
	DAC	—	3×10^5
^{202}Tl	ALI	1×10^8	2×10^8
	DAC	—	8×10^4
^{204}Tl	ALI	6×10^7	8×10^7
	DAC	—	3×10^4

METABOLIC DATA FOR ASTATINE

1. Metabolism

There are no isotopes of astatine with radioactive half-lives in excess of 9 h and such isotopes exist in nature only in conjunction with their parent isotopes of uranium and thorium.

2. Metabolic Model

(a) *Uptake to blood*

By analogy with the lighter halides, chlorine, bromine and iodine, f_1 is taken to be 1 for all compounds of astatine.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned the heavier halides of all elements either to inhalation class D or to inhalation class W. For information concerning the classification of halides of a particular element the metabolic data for that element, or the Task Group Report, should be consulted.

Inhalation class	f_1
D	1
W	1
Y	—

(c) *Distribution and retention*

By analogy with chlorine and bromine, astatine leaving the transfer compartment is assumed to be uniformly distributed throughout all organs and tissues of the body where it is retained with a biological half-life of 10 days.

3. Classification of Isotopes for Bone Dosimetry

Astatine is assumed to be uniformly distributed throughout the body. Therefore, a classification of isotopes of the element for the purposes of bone dosimetry is not required.

Reference

ICRP Task Group on Lung Dynamics (1966). Deposition and retention models for internal dosimetry of the human respiratory tract. *Health Phys.* **12**, 173–207.

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of astatine

Radionuclide		Oral	Inhalation	
			Class D	Class W
		$f_1 = 1$	$f_1 = 1$	$f_1 = 1$
^{207}At	ALI	2×10^8	1×10^8	8×10^7
	DAC	—	4×10^4	3×10^4
^{211}At	ALI	5×10^6	3×10^6	2×10^6
	DAC	—	1×10^3	8×10^2

METABOLIC DATA FOR FRANCIUM

1. Metabolism

There are no isotopes of francium with radioactive half-lives in excess of 22 min and the one isotope of the element present in nature, ^{223}Fr, only occurs in conjunction with its parent ^{227}Ac.

2. Metabolic Model

(a) *Uptake to blood*

By analogy with potassium, rubidium and caesium, f_1 is taken to be 1 for all compounds of francium.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned all compounds of francium to inhalation class D. In the absence of any relevant experimental data and by analogy with the more extensively studied element caesium, this classification is adopted here.

Inhalation class	f_1
D	1
W	—
Y	—

(c) *Distribution and retention*

In view of the chemical similarities between francium and caesium, it is assumed that francium leaving the transfer compartment is uniformly distributed throughout all organs and tissues of the body. Since none of the isotopes of francium considered in this report has a radioactive half-life in excess of 22 min, francium translocated to any organ or tissue may be assumed to be retained there indefinitely.

3. Classification of Isotopes for Bone Dosimetry

Francium is assumed to be uniformly distributed throughout all organs and tissues of the body. Therefore, a classification of isotopes of the element for the purposes of bone dosimetry is not required.

Reference

ICRP Task Group on Lung Dynamics (1966). Deposition and retention models for internal dosimetry of the human respiratory tract. *Health Phys.* **12**, 173–207.

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of francium

			Inhalation
		Oral	Class D
Radionuclide		$f_1 = 1$	$f_1 = 1$
^{222}Fr	ALI	8×10^7	2×10^7
	DAC	—	7×10^3
^{223}Fr	ALI	2×10^7	3×10^7
	DAC	—	1×10^4

METABOLIC DATA FOR ACTINIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for actinium.

2. Metabolic Model

(a) *Uptake to blood*

Early studies by Hamilton (1948) indicated that the fractional absorption of actinium from the gastrointestinal tract was less than 0.05. A later study (Campbell, Robajdek and Anthony, 1956) indicates that the fractional absorption of actinium from the gastrointestinal tract of rats is considerably less than 0.01 when the element is administered as the chloride. In this report f_1 is taken to be 10^{-3} for all compounds of actinium.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides and hydroxides of actinium to inhalation class Y, halides and nitrates to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. Data from accidental exposures of man (Newton, 1966, 1968) demonstrate that compounds of actinium can behave as inhalation class Y materials. In the absence of more detailed experimental data the Task Group's classification of compounds of actinium is adopted here.

Inhalation class	f_1
D	10^{-3}
W	10^{-3}
Y	10^{-3}

(c) *Distribution and retention*

Like the other actinides, intravenously or intramuscularly injected actinium is concentrated in the liver, skeleton and, to some extent, the kidneys (Hamilton, 1948; Campbell, Robajdek and Anthony, 1956; Taylor, 1967; Newton, Rundo and Sandalls, 1968; Taylor, 1970; Newton and Brown, 1974). These data are generally in accord with the ICRP (1972) recommendation that the metabolic model used for plutonium should also be used for the other actinides.

In this report it is assumed that of actinium leaving the transfer compartment fractions of 0.45 and 0.45 are translocated to mineral bone and the liver respectively. The fraction of actinium translocated to the gonads is assumed to be 3.5×10^{-4} for the testes and 1.1×10^{-4} for the ovaries, these values corresponding to a fractional translocation to the gonads of 10^{-5} per g of gonadal tissues. The remainder of actinium leaving the transfer compartment is assumed to go directly to excretion.

Actinium translocated to mineral bone is assumed to be retained with a biological half-life of 100 years, whereas actinium translocated to the liver is assumed to be retained with a biological half-life of 40 years. Actinium translocated to the gonads is assumed to be retained there indefinitely.

3. Classification of Isotopes for Bone Dosimetry

The distribution of actinium in the skeleton of the rat is similar to the distribution of americium and curium (Hamilton, 1948). By analogy with these other actinides, all isotopes of actinium considered in this report are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

References

Campbell, J. E., Robajdek, E. S. and Anthony, D. S. (1956). The metabolism of ^{227}Ac and its daughters ^{227}Th and ^{223}Ra by rats. *Rad. Res.* **4**, 294–302.

Hamilton, J. G. (1948). The metabolic properties of the fission products and actinide elements. *Rev. Mod. Phys.* **20**, 718–728.

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ICRP Publication 23. Report of the Task Group on Reference Man. Pergamon Press, Oxford, 1975.

Newton, D. (1966). A case of accidental inhalation of actinium-227. *Health Phys.* **12**, 1129–1138.

Newton, D. (1968). A case of accidental inhalation of protactinium-231 and actinium-227. *Health Phys.* **15**, 11–17.

Newton, D. and Brown, D. A. (1974). The long-term retention of systemic protactinium-231 and actinium-227. *Health Phys.* **27**, 459–467.

Newton, D., Rundo, J. and Sandalls, F. J. (1968). A case of internal contamination with ^{231}Pa and ^{227}Ac via a puncture wound. *In: Diagnosis and Treatment of Deposited Radionuclides*, pp. 521–533 (eds. Kornberg, H. A. and Norwood, W. D.). Excerpta Medica Foundation, Amsterdam.

Taylor, D. M. (1967). The effects of desferrioxamine on the retention of actinide elements in the rat. *Health Phys.* **13**, 135–140.

Taylor, D. M. (1970). The metabolism of actinium in the rat. *Health Phys.* **19**, 411–418.

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of actinium

			Inhalation		
		Oral	Class D	Class W	Class Y
Radionuclide		$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$
^{224}Ac	ALI	7×10^7 (7×10^7) LLI Wall	1×10^6 (1×10^6) Bone surf.	2×10^6	2×10^6
	DAC	—	4×10^2	8×10^2	7×10^2
^{225}Ac	ALI	2×10^6 (2×10^6) LLI Wall	1×10^4 (2×10^4) Bone surf.	2×10^4	2×10^4
	DAC	—	4	1×10^1	1×10^1
^{226}Ac	ALI	5×10^6 (5×10^6) LLI Wall	1×10^5 (1×10^5) Bone surf.	2×10^5	2×10^5
	DAC	—	5×10^1	8×10^1	7×10^1
^{227}Ac	ALI	7×10^3 (1×10^4) Bone surf.	2×10^1 (3×10^1) Bone surf.	6×10^1 (1×10^2) Bone surf.	1×10^2
	DAC	—	6×10^{-3}	3×10^{-2}	6×10^{-2}
^{228}Ac	ALI	9×10^7	4×10^5 (6×10^5) Bone surf.	1×10^6 (2×10^6) Bone surf.	2×10^6
	DAC	—	1×10^2	6×10^2	7×10^2

METABOLIC DATA FOR PROTACTINIUM

1. Metabolism

No data are given in Reference Man (1975) for protactinium.

2. Metabolic Model

(a) *Uptake to blood*

The experiments of Hamilton (1948) indicate that the fractional absorption of protactinium from the gastrointestinal tract of the rat is less than 5×10^{-4}. More recent experiments have indicated that the fractional absorption of protactinium, administered as the citrate, from the gastrointestinal tract of the rat does not exceed 0.01–0.02 (Zalikin, 1966a) and may be as low as 6×10^{-5} (Zalikin, 1966b). Other experiments by Zalikin (1969) on an unidentified compound of the element indicate a fractional absorption of about 10^{-3}. In this report f_1 is taken to be 10^{-3} for all compounds of protactinium.

(b) *Inhalation classes*

The ICRP Task Group on Lung Dynamics (1966) assigned oxides and hydroxides of protactinium to inhalation class Y, halides and nitrates to inhalation class W and all other commonly occurring compounds of the element to inhalation class D. Studies on a man who accidentally inhaled Pa_2O_5 or $KPaO_3$ (Newton, 1968) are in agreement with this classification. However, in studies in which ^{233}Pa citrate was administered intratracheally to rats (Zalikin, 1966b), protactinium was found to be retained in the lungs with a biological half-life of about 70 days.

In this report oxides and hydroxides of protactinium are assigned to inhalation class Y and all other commonly occurring compounds of the element are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	10^{-3}
Y	10^{-3}

(c) *Distribution and retention*

Studies on rats (Hamilton, 1948; Zalikin, 1966a; Zalikin, 1969) have shown that protactinium entering the systemic circulation is mainly deposited in the skeleton with the liver and the kidneys as secondary sites of deposition. Protactinium deposited in the skeleton is retained there with a biological half-life in excess of 100 days (Hamilton, 1948; Zalikin, 1969) whereas protactinium deposited in the liver or the kidneys has a biphasic retention, the two components having biological half-lives of about 10 and 60 days respectively (Zalikin, 1969).

Data from studies on a man accidentally contaminated with ^{231}Pa through a puncture wound in his hand (Newton, Rundo and Sandalls, 1968; Newton and Brown, 1974) indicate

that after an early phase of excretion the remaining fraction of protactinium is retained in the body almost indefinitely, probably mainly in the skeleton.

In this report it is assumed that of protactinium leaving the transfer compartment fractions of 0.4, 0.15 and 0.02 are translocated to mineral bone, the liver and the kidneys respectively. The remaining fraction of protactinium leaving the transfer compartment is assumed to go directly to excretion. By analogy with plutonium and the transplutonic actinides, protactinium deposited in mineral bone is assumed to be retained with a biological half-life of 100 years. Of protactinium deposited in the liver, fractions 0.7 and 0.3 are assumed to be retained with biological half-lives of 10 and 60 days respectively. Of protactinium deposited in the kidneys fractions of 0.2 and 0.8 are assumed to be retained with biological half-lives of 10 and 60 days respectively.

3. Classification of Isotopes for Bone Dosimetry

There appear to be no relevant data concerning the distribution of protactinium in the skeleton. By analogy with the other actinides all isotopes of protactinium are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

References

Hamilton, J. G. (1948). The metabolic properties of the fission products and actinide elements. *Rev. Mod. Phys.* **20**, 718–728.

ICRP Task Group on Lung Dynamics (1966). Deposition and retention models for internal dosimetry of the human respiratory tract. *Health Phys.* **12**, 173–207.

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Newton, D. and Brown, D. A. (1974). The long-term retention of systemic protactinium-231 and actinium-227. *Health Phys.* **27**, 459–467.

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Zalikin, G. A. (1966a). Distribution of ^{233}Pa in the organism of the rat. In: *Raspredelenie i biologicheskoe deistvie radioaktivnykh izotopov*, pp. 30–36 (ed. Moskalev, Yu. I.). Moscow. Translated in Distribution and biological effects of radioactive isotopes AEC-tr-6944 (Rev.), pp. 35–43.

Zalikin, G. A. (1966b). The distribution of ^{233}Pa in the organism of rats in endotracheal, subcutaneous and peroral introduction. *Med. Radiol.* **11**, 67–70.

Zalikin, G. A. (1969). On the distribution of ^{233}Pa in the body of rats. In: *Radioaktivnye izotopy i organizm, izdatel'stvo* (ed. Moskalev, Yu. I.). Moscow. Translated in Radioactive isotopes and the body, AEC-tr-7195, pp. 136–146.

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of protactinium

Radionuclide		Oral	Inhalation	
			Class W	Class Y
		$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$	$f_1 = 1 \times 10^{-3}$
^{227}Pa	ALI	1×10^{8}	4×10^{6}	4×10^{6}
	DAC	—	2×10^{3}	2×10^{3}
^{228}Pa	ALI	5×10^{7}	5×10^{5} (8×10^{5}) Bone surf.	4×10^{5}
	DAC	—	2×10^{2}	2×10^{2}
^{230}Pa	ALI	2×10^{7} (3×10^{7}) Bone surf.	2×10^{5}	1×10^{5}
	DAC	—	7×10^{1}	5×10^{1}
^{231}Pa	ALI	7×10^{3} (2×10^{4}) Bone surf.	6×10^{1} (1×10^{2}) Bone surf.	1×10^{2} (2×10^{2}) Bone surf.
	DAC	—	2×10^{-2}	6×10^{-2}
^{232}Pa	ALI	5×10^{7}	8×10^{5} (2×10^{6}) Bone surf.	2×10^{6} (3×10^{6}) Bone surf.
	DAC	—	3×10^{2}	9×10^{2}
^{233}Pa	ALI	5×10^{7} (6×10^{7}) LLI Wall	3×10^{7}	2×10^{7}
	DAC	—	1×10^{4}	9×10^{3}
^{234}Pa	ALI	9×10^{7}	3×10^{8}	2×10^{8}
	DAC	—	1×10^{5}	1×10^{5}

METABOLIC DATA FOR BERKELIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for berkelium.

2. Metabolic Model

(a) *Uptake to blood*

The fractional gastrointestinal absorption of intragastrically administered $^{248}BkCl_3$ in the rat can be estimated to be about 10^{-4} (Hungate *et al.*, 1972). By analogy with americium f_1 is taken as 5×10^{-4} for all compounds of berkelium.

(b) *Inhalation classes*

There is a limited amount of data available on the inhalation (Rundo and Sedlet, 1973) and intratracheal administration (Hungate *et al.*, 1972) of berkelium, but these data are insufficient for the assignment of an inhalation class to the element. By analogy with americium all compounds of berkelium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	5×10^{-4}
Y	—

(c) *Distribution and retention*

Data from experiments on rats (Hungate *et al.*, 1972) indicate that of berkelium entering the systemic circulation most is translocated to the liver and the skeleton, although small fractions may be translocated to the kidney and the spleen. In this report the ICRP (1972) recommendation that the metabolic model used for plutonium should also be used for the other actinides has been adopted for berkelium.

Of berkelium leaving the transfer compartment, it is assumed that 0.45 is translocated to mineral bone and 0.45 to the liver. The fraction of the element translocated to the gonads is assumed to be 3.5×10^{-4} for the testes and 1.1×10^{-4} for the ovaries, these values corresponding to a fractional translocation to the gonads of 10^{-5} per g of gonadal tissue. The remainder of berkelium leaving the transfer compartment is assumed to go directly to excretion.

Berkelium deposited in mineral bone is assumed to be retained with a biological half-life of 100 years, whereas berkelium deposited in the liver is assumed to be retained with a biological half-life of 40 years. Berkelium deposited in gonadal tissue is assumed to be retained there indefinitely.

(d) *Chelated compounds*

Chelated forms of berkelium are not considered in this report. They may have greater biological mobility than other compounds of berkelium.

3. Classification of Isotopes for Bone Dosimetry

By analogy with other actinides, all isotopes of berkelium considered in this report are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

References

Hungate, F. P., Ballou, J. E., Mahlum, D. D., Kashima, M., Smith, V. H., Sanders, C. L., Baxter, D. W., Sikov, M. R. and Thompson, R. C. (1972). Preliminary data on ^{253}Es and ^{249}Bk metabolism in rats. *Health Phys.* **22**, 653–656.

ICRP Publication 19. The Metabolism of Compounds of Plutonium and other Actinides. Pergamon Press, Oxford, 1972.

ICRP Publication 23. Report of the Task Group on Reference Man. Pergamon Press, Oxford, 1975.

Rundo, J. and Sedlet, J. (1973). Retention and elimination of berkelium-249-californium-249 following acute accidental inhalation. In: *Proc. 3rd Int. Cong. Radiat. Prot. Assoc.* CONF 730901, Vol. 2, pp. 731–735 (ed. Snyder, W. S.).

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of berkelium

		Oral	Inhalation Class W
Radionuclide		$f_1 = 5 \times 10^{-4}$	$f_1 = 5 \times 10^{-4}$
^{245}Bk	ALI	8×10^{7}	5×10^{7}
	DAC	—	2×10^{4}
^{246}Bk	ALI	1×10^{8}	1×10^{8}
	DAC	—	5×10^{4}
^{247}Bk	ALI	4×10^{4}	2×10^{2}
		(8×10^{4})	(3×10^{2})
		Bone surf.	Bone surf.
	DAC	—	8×10^{-2}
^{249}Bk	ALI	2×10^{7}	8×10^{4}
		(3×10^{7})	(1×10^{5})
		Bone surf.	Bone surf.
	DAC	—	3×10^{1}
^{250}Bk	ALI	4×10^{8}	2×10^{7}
			(3×10^{7})
			Bone surf.
	DAC	—	7×10^{3}

METABOLIC DATA FOR EINSTEINIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for einsteinium.

2. Metabolic Model

(a) *Uptake to blood*

Experiments on rats (Hungate *et al.*, 1972) indicate that einsteinium and americium are both absorbed from the gastrointestinal tract to a similar extent. Therefore, by analogy with americium, f_1 is here taken to be 5×10^{-4} for all compounds of einsteinium.

(b) *Inhalation classes*

Data from experiments on rats (Hungate *et al.*, 1972; Ballou, Dagle and Morrow, 1975; Ballou *et al.*, 1979) suggest that chlorides, nitrates and hydroxides of einsteinium should be assigned to inhalation class W. Thus, from these data and by analogy with the more extensively studied americium, all compounds of einsteinium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	5×10^{-4}
Y	—

(c) *Distribution and retention*

Like the other actinides, einsteinium entering the systemic circulation is preferentially deposited in the skeleton, liver and, to some extent, the kidneys (Hungate *et al.*, 1972; Parker *et al.*, 1972; Ballou, Dagle and Morrow, 1975; Lloyd *et al.*, 1975). These data are generally in accord with the ICRP (1972) recommendation that the metabolic model used for plutonium should also be used for the other actinides.

In this report it is assumed that of einsteinium leaving the transfer compartment fractions of 0.45 and 0.45 are translocated to mineral bone and the liver respectively. The fraction of einsteinium translocated to the gonads is assumed to be 3.5×10^{-4} for the testes and 1.1×10^{-4} for the ovaries, these values corresponding to a fractional translocation to the gonads of 10^{-5} per g of gonadal tissue. The remainder of einsteinium leaving the transfer compartment is assumed to go directly to excretion.

Einsteinium translocated to mineral bone is assumed to be retained with a biological half-life of 100 years, whereas einsteinium translocated to the liver is assumed to be retained with a biological half-life of 40 years. Einsteinium translocated to the gonads is assumed to be retained there indefinitely.

(d) *Chelated compounds*

Chelated forms of einsteinium are not considered in this report. It is known that their biological behaviour differs considerably from that of other compounds of the element (Hayes *et al.*, 1973).

3. Classification of Isotopes for Bone Dosimetry

Autoradiographic studies (Ballou, Dagle, Gies and Smith, 1979) have shown that the distribution of einsteinium in rat bone resembles the distribution of plutonium. Thus, by analogy with the other actinides all isotopes of einsteinium considered in this report are assumed to be uniformly distributed over the surfaces of mineral bone at all times following their deposition in that tissue.

References

Ballou, J. E., Dagle, G. E. and Morrow, W. G. (1975). The long-term effects of intratracheally instilled $^{253}EsCl_3$ in rats. *Health Phys.* **29**, 267–272.

Ballou, J. E., Dagle, G. E., Gies, R. A. and Smith, L. G. (1979). Late effects of inhaled $^{253}Es(NO_3)_3$ in rats. *Health Phys.* **37**, 301–310.

Hayes, R. L., Rafter, J. J., Washburn, L. C. and Byrd, B. L. (1973). Affinity of ^{253}einsteinium for tumour tissue. *Nature (New Biology)* **246**, 23–25.

Hungate, F. P., Ballou, J. E., Mahlum, D. D., Kashima, M., Smith, V. H., Sanders, C. L., Baxter, D. W., Sikov, M. R. and Thompson, R. C. (1972). Preliminary data on ^{253}Es and ^{249}Bk metabolism in rats. *Health Phys.* **22**, 653–656.

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ICRP Publication 23. Report of the Task Group on Reference Man. Pergamon Press, Oxford, 1975.

Lloyd, R. D., Dockum, J. G., Atherton, D. R., Mays, C. W. and Williams, J. L. (1975). The early retention, excretion and distribution of injected einsteinium citrate in beagles. *Health Phys.* **28**, 585–589.

Parker, H. G., Wright, S. R., Low-Beer, A. de G. and Yaeger, D. J. (1972). The metabolism of ^{253}Es in mice. *Health Phys.* **22**, 647–651.

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of einsteinium

Radionuclide		Oral	Inhalation Class W
		$f_1 = 5 \times 10^{-4}$	$f_1 = 5 \times 10^{-4}$
^{250}Es	ALI	2×10^9	2×10^7 (4×10^7) Bone surf.
	DAC	—	1×10^4
^{251}Es	ALI	3×10^8	4×10^7 (4×10^7) Bone surf.
	DAC	—	2×10^4
^{253}Es	ALI	8×10^6 (8×10^6) LLI Wall	6×10^4
	DAC	—	2×10^1
^{254m}Es	ALI	1×10^7 (1×10^7) LLI Wall	4×10^5
	DAC	—	2×10^2
^{254}Es	ALI	8×10^5 (1×10^6) Bone surf.	4×10^3 (5×10^3) Bone surf.
	DAC	—	2

METABOLIC DATA FOR FERMIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for fermium.

2. Metabolic Model

(a) *Uptake to blood*

There are no data available on the uptake of fermium from the gastrointestinal tract. By analogy with americium, f_1 is taken as 5×10^{-4} for all compounds of the element.

(b) *Inhalation classes*

By analogy with americium and curium, all compounds of fermium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	5×10^{-4}
Y	—

(c) *Distribution and retention*

The ICRP (1972) recommended that the metabolic model used for plutonium should also be used for the other actinides. This recommendation is generally in accord with the results from studies on americium, curium, californium and einsteinium, and has been adopted here for fermium.

Of fermium leaving the transfer compartment, it is assumed that 0.45 is translocated to mineral bone and 0.45 to the liver. The fraction of fermium translocated to the gonads is assumed to be 3.5×10^{-4} for the testes and 1.1×10^{-4} for the ovaries; these values correspond to a fractional translocation to the gonads of 10^{-5} per g of gonadal tissue. The remainder of fermium leaving the transfer compartment is assumed to go directly to excretion.

Fermium deposited in mineral bone is assumed to be retained there with a biological half-life of 100 years, whereas fermium deposited in the liver is assumed to be retained there with a biological half-life of 40 years. Fermium deposited in gonadal tissue is assumed to be retained there indefinitely.

(d) *Chelated compounds*

Chelated forms of fermium are not considered in this report. They may have greater biological mobility than other compounds of the element.

3. Classification of Isotopes for Bone Dosimetry

By analogy with other actinides, and because no isotope of fermium has a radioactive half-life much in excess of 100 days, all isotopes of the element considered in this report are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

References

ICRP Publication 19. The Metabolism of Compounds of Plutonium and other Actinides. Pergamon Press, Oxford, 1972.
ICRP Publication 23. Report of the Task Group on Reference Man. Pergamon Press, Oxford, 1975.

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of fermium

			Inhalation
		Oral	Class W
Radionuclide		$f_1 = 5 \times 10^{-4}$	$f_1 = 5 \times 10^{-4}$
^{252}Fm	ALI	2×10^7	5×10^5
	DAC	—	2×10^2
^{253}Fm	ALI	5×10^7 (5×10^7) LLI Wall	4×10^5
	DAC	—	2×10^2
^{254}Fm	ALI	1×10^8	4×10^6
	DAC	—	2×10^3
^{255}Fm	ALI	2×10^7	8×10^5
	DAC	—	3×10^2
^{257}Fm	ALI	2×10^6 (3×10^6) Bone surf.	9×10^3
	DAC	—	4

METABOLIC DATA FOR MENDELEVIUM

1. Metabolism

No data are given in Reference Man (ICRP, 1975) for mendelevium.

2. Metabolic Model

(a) *Uptake to blood*

There are no data available on the uptake of mendelevium from the gastrointestinal tract. By analogy with americium, f_1 is taken as 5×10^{-4} for all compounds of the element.

(b) *Inhalation classes*

By analogy with americium and curium all compounds of mendelevium are assigned to inhalation class W.

Inhalation class	f_1
D	—
W	5×10^{-4}
Y	—

(c) *Distribution and retention*

The ICRP (1972) recommended that the metabolic model used for plutonium should also be used for the other actinides. This recommendation is generally in accord with the results from studies on americium, curium, californium and einsteinium and has been adopted here.

Of mendelevium leaving the transfer compartment it is assumed that 0.45 is translocated to mineral bone and 0.45 to the liver. The fraction of mendelevium translocated to the gonads is assumed to be 3.5×10^{-4} for the testes and 1.1×10^{-4} for the ovaries; these values correspond to a fractional translocation to the gonads of 10^{-5} per g of gonadal tissue. The remainder of mendelevium leaving the transfer compartment is assumed to go directly to excretion.

Mendelevium deposited in mineral bone is assumed to be retained with a biological half-life of 100 years, whereas mendelevium deposited in the liver is assumed to be retained with a biological half-life of 40 years. Mendelevium deposited in gonadal tissue is assumed to be retained there indefinitely.

(d) *Chelated compounds*

Chelated forms of mendelevium are not considered in this report; they may have greater biological mobility than other compounds of the element.

3. Classification of Isotopes for Bone Dosimetry

By analogy with other actinides and because no isotope of mendelevium has a half-life longer than 55 days, all isotopes of the element considered in this report are assumed to be uniformly distributed over bone surfaces at all times following their deposition in mineral bone.

References

ICRP Publication 19. The Metabolism of Compounds of Plutonium and other Actinides. Pergamon Press, Oxford, 1972.
ICRP Publication 23. Report of the Task Group on Reference Man. Pergamon Press, Oxford, 1975.

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of mendelevium

			Inhalation
		Oral	Class W
Radionuclide		$f_1 = 5 \times 10^{-4}$	$f_1 = 5 \times 10^{-4}$
^{257}Md	ALI	3×10^{8}	4×10^{6}
	DAC	—	1×10^{3}
^{258}Md	ALI	3×10^{6}	1×10^{4}
		(3×10^{6})	(1×10^{4})
		Bone surf.	Bone surf.
	DAC	—	5

ADDENDUM

ICRP Publication 30, Part 1, 1979

The following lines of text are amended as shown:

Page 37, line 4 from the bottom: >0.006 (not $\geqslant$)

Page 38, line 3: $0.006 < AF(BS$ (not $\leqslant$)

Page 50, line 11 from the bottom and the last line: (Sv m^3 Bq^{-1} h^{-1}) (not m^{-3})

The figures 5.1 and 5.2 published on pages 24 and 25 of ICRP Publication 30 are amended as follows:

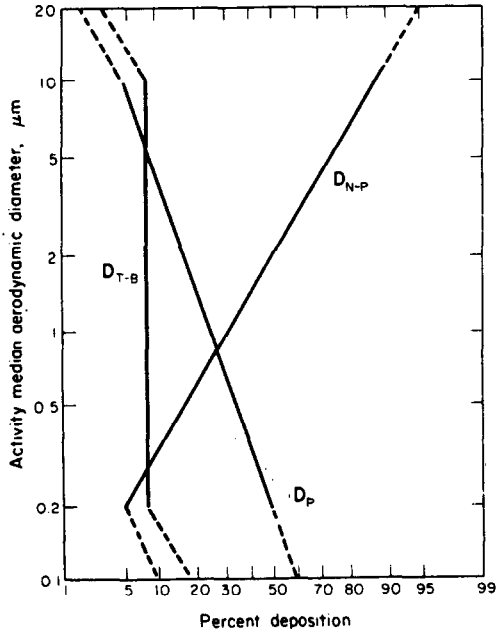

Fig. 5.1. Deposition of dust in the respiratory system. The percentage of activity or mass of an aerosol which is deposited in the N-P, T-B and P regions is given in relation to the Activity Median Aerodynamic Diameter (AMAD) of the aerosol distribution. The model is intended for use with aerosol distributions with AMADs between 0.2 and 10 μm and with geometric standard deviations of less than 4.5. Provisional estimates of deposition further extending the size range are given by the dashed lines. For an unusual distribution with an AMAD of greater than 20 μm, complete deposition in N-P can be assumed. The model does not apply to aerosols with AMADs of less than 0.1 μm.

Region	Compartment	Class D T day	D F	W T day	W F	Y T day	Y F
N-P	a	0.01	0.5	0.01	0.1	0.01	0.01
(D_{N-P} = 0.30)	b	0.01	0.5	0.40	0.9	0.40	0.99
T-B	c	0.01	0.95	0.01	0.5	0.01	0.01
(D_{T-B} = 0.08)	d	0.2	0.05	0.2	0.5	0.2	0.99
	e	0.5	0.8	50	0.15	500	0.05
P	f	n.a.	n.a.	1.0	0.4	1.0	0.4
(D_P = 0.25)	g	n.a.	n.a.	50	0.4	500	0.4
	h	0.5	0.2	50	0.05	500	0.15
L	i	0.5	1.0	50	1.0	1 000	0.9
	j	n.a.	n.a.	n.a.	n.a.	∞	0.1

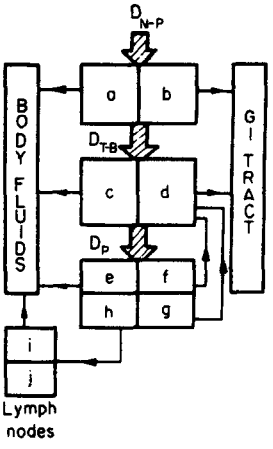

Fig. 5.2. Mathematical model used to describe clearance from the respiratory system. The values for the removal half-times, T_{a-i} and compartmental fractions, F_{a-i} are given in the tabular portion of the figure for each of the three classes of retained materials. The values given for D_{N-P}, D_{T-B} and D_P (left column) are the regional depositions for an aerosol with an AMAD of 1 μm. The schematic drawing identifies the various clearance pathways from compartments a-i in the four respiratory regions, N-P, T-B, P and L.
n.a. = not applicable.

Page 76 of Part 1 and pages 54 and 55 of its Supplement: As a consequence of the Commission's decision to reduce its recommended dose equivalent limit for the lens of the eye from 0.3 Sv to 0.15 Sv in a year, values of DAC for ^{81}Kr and ^{83m}Kr are amended as follows.

Derived air concentrations DAC(Bq m^{-3}) (40 h wk) for isotopes of krypton

Radionuclide	Semi-infinite cloud	1 000 m^3 room	500 m^3 room	100 m^3 room
^{81}Kr	2×10^7	1×10^8 (5×10^8) Lens	1×10^8 (6×10^8) Lens	1×10^8 (9×10^8) Lens
^{83m}Kr	4×10^8 (7×10^9) Lens	4×10^8 (7×10^9) Lens	4×10^8 (7×10^9) Lens	4×10^8 (8×10^9) Lens

Page 78, The values of ALI and DAC for ^{80}Sr are incorrect because no allowance was made for the contribution to committed dose equivalent of ^{80}Rb, the daughter of ^{80}Sr. The corrected dosimetric data are given in Supplement B of Part 3. The correct values of ALI and DAC are given below:

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of strontium

				Inhalation	
		Oral		Class D	Class Y
Radionuclide		$f_1 = 3 \times 10^{-1}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 3 \times 10^{-1}$	$f_1 = 1 \times 10^{-2}$
^{80}Sr	ALI	2×10^8	2×10^8	4×10^8	5×10^8
	DAC	—	—	2×10^5	2×10^5

Page 82, The values of ALI and DAC given for ^{93m}Nb were calculated using the incorrect assumption that the radionuclide would be distributed uniformly to bone surface rather than throughout mineral bone as stated in the metabolic model. The correct values of ALI and DAC are given below. Revised tables of dosimetric data are given in Supplement B to Part 3.

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of niobium

			Inhalation	
		Oral	Class W	Class Y
Radionuclide		$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$	$f_1 = 1 \times 10^{-2}$
^{93m}Nb	ALI	3×10^8 (4×10^8) LLI Wall	7×10^7	6×10^6
	DAC	—	3×10^4	3×10^3

Page 84, In Section 3 it is incorrect to state that none of the radioisotopes of molybdenum considered has a radioactive half-life greater than 3 days. ^{93}Mo has a half-life of 3.5×10^3 years and should perhaps be considered to be distributed throughout the volume of mineral bone rather than on its surface following its deposition in the skeleton. Such an assumption will make only a marginal difference to the values of ALI and DAC quoted.

Page 86, In Section 3 it is incorrect to state that none of the radioisotopes of tellerium considered has a radioactive half-life greater than 200 days. ^{123}Te has a radioactive half-life of 10^{13} years and should perhaps be considered as a volume rather than a surface seeker of mineral bone. This would have the effect of increasing the values of ALI shown. However, the mass of the ALI is so great that ^{123}Te will not present a radiation hazard in any practical circumstance.

Page 87, Some of the values of ALI and DAC shown for radioisotopes of tellurium are incorrect due to a transcription error. The correct values are as follows:

Annual limits on intake ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of tellurium

Radionuclide		Oral	Inhalation	
			Class D	Class W
		$f_1 = 2 \times 10^{-1}$	$f_1 = 2 \times 10^{-1}$	$f_1 = 2 \times 10^{-1}$
^{131}Te	ALI	1×10^8	2×10^8	2×10^8
		(2×10^8)	(5×10^8)	(4×10^8)
		Thyroid	Thyroid	Thyroid
	DAC	–	8×10^4	8×10^4
^{131m}Te	ALI	1×10^7	2×10^7	1×10^7
		(2×10^7)	(5×10^7)	(3×10^7)
		Thyroid	Thyroid	Thyroid
	DAC	–	6×10^3	6×10^3
^{132}Te	ALI	8×10^6	9×10^6	8×10^6
		(2×10^7)	(3×10^7)	(2×10^7)
		Thyroid	Thyroid	Thyroid
	DAC	–	4×10^3	3×10^3
^{133}Te	ALI	5×10^8	8×10^8	8×10^8
		(1×10^9)	(2×10^9)	(2×10^9)
		Thyroid	Thyroid	Thyroid
	DAC	–	4×10^5	4×10^5
^{133m}Te	ALI	1×10^8	2×10^8	2×10^8
		(2×10^8)	(5×10^8)	(5×10^8)
		Thyroid	Thyroid	Thyroid
	DAC	–	8×10^4	8×10^4
^{134}Te	ALI	6×10^8	9×10^8	9×10^8
		(9×10^8)	(2×10^9)	(2×10^9)
		Thyroid	Thyroid	Thyroid
	DAC	–	4×10^5	4×10^5

Page 88, The second paragraph of Section (c) *Distribution and Retention* should read as follows:

"Of iodine entering the transfer compartment a fraction, 0.3, is assumed to be translocated to the thyroid while the remainder is assumed to go directly to excretion. Iodine in the thyroid is assumed to be retained with a biological half-life of 80 days and to be lost from the gland in the form of organic iodine. Organic iodine is assumed to be uniformly distributed among all organs and tissues of the body other than the thyroid and to be retained there with a biological half-life of 12 days. One-tenth of this organic iodine is assumed to go directly to faecal excretion and the rest is assumed to be returned to the transfer compartment as inorganic iodine so that the effective half-life of iodine in the thyroid is 120 days."

Values of ALI and DAC for radioisotopes of iodine are not changed.

ICRP Publication 30, Part 2, 1980

Page 51, Values of ALI and DAC for ^{178}Re are incorrect because ^{178}Ta (2.2 h) was incorrectly included in the decay chain instead of ^{178}Ta (9.31 min). A revised drawing of the decay scheme and tables of dosimetric data will be given in Supplement B to Part 3. The revised values of ALI and DAC are as follows:

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of rhenium

Radionuclide		Oral	Inhalation	
			Class D	Class W
		$f_1 = 8 \times 10^{-1}$	$f_1 = 8 \times 10^{-1}$	$f_1 = 8 \times 10^{-1}$
^{178}Re	ALI	3×10^9 (4×10^9) ST Wall	1×10^{10}	1×10^{10}
	DAC	—	4×10^6	5×10^6

Page 57, Due to wrong transcription of metabolic data into the computer, small errors have occurred in the values of ALI and DAC for isotopes of gold. The correct dosimetric data are shown in Supplement B to Part 3 and the correct values of ALI and DAC are shown below:

Annual limits on intake, ALI(Bq) and derived air concentrations, DAC(Bq m^{-3}) (40 h wk) for isotopes of gold

Radionuclide		Oral	Inhalation		
			Class D	Class W	Class Y
		$f_1 = 1 \times 10^{-1}$	$f_1 = 1 \times 10^{-1}$	$f_1 = 1 \times 10^{-1}$	$f_1 = 1 \times 10^{-1}$
^{193}Au	ALI	3×10^8	1×10^9	8×10^8	7×10^8
	DAC	—	4×10^5	3×10^5	3×10^5
^{194}Au	ALI	1×10^8	3×10^8	2×10^8	2×10^8
	DAC	—	1×10^5	8×10^4	8×10^4
^{195}Au	ALI	2×10^8	4×10^8	5×10^7	2×10^7
	DAC	—	2×10^5	2×10^4	7×10^3
^{198m}Au	ALI	4×10^7	1×10^8	4×10^7	4×10^7
	DAC	—	4×10^4	2×10^4	2×10^4
^{198}Au	ALI	5×10^7	1×10^8	7×10^7	6×10^7
	DAC	—	6×10^4	3×10^4	3×10^4
^{199}Au	ALI	1×10^8 (1×10^8) LLI Wall	3×10^8	1×10^8	1×10^8
	DAC	—	1×10^5	6×10^4	6×10^4
^{200m}Au	ALI	4×10^7	1×10^8	1×10^8	9×10^7
	DAC	—	5×10^4	4×10^4	4×10^4
^{200}Au	ALI	1×10^9	2×10^9	3×10^9	3×10^9
	DAC	—	1×10^6	1×10^6	1×10^6
^{201}Au	ALI	3×10^9 (3×10^9) ST Wall	8×10^9	9×10^9	8×10^9
	DAC	—	3×10^6	4×10^6	3×10^6