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Depleted uranium

Sources, Exposure and Health Effects

Department of Protection of the Human Environment
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Preface

Depleted uranium (DU) has been used in medical and industrial applications for decades but only since its use in military conflicts in the Gulf and the Balkans has public concern been raised about potential health consequences from exposure to it. Concerns have been particularly for peacekeeping forces, humanitarian workers and local populations living and working in areas contaminated by DU following conflict.

There has been a large amount of research on the health consequences to workers in the mining and milling of uranium, and on its use in nuclear power, that enables a reasonable assessment of its impact on human health[1]. Since DU acts chemically in the same way as uranium, and the radiological toxicity is somewhat less than uranium, this research can be used to evaluate health risks from ingestion, inhalation and contact with DU.

In late 1999, the WHO Department on the Protection of the Human Environment (PHE) recognized the need for an independent review of the scientific literature from which health risks could be assessed from various DU exposure situations. Professor Barry Smith from the British Geological Survey, UK, was contracted to prepare the draft from the literature that would be subject to rigorous scientific review. The format of the review was to be modelled on monographs in the WHO environmental health criteria series.

An ad hoc review and oversight group of WHO staff members was formed and coordinated by Dr Michael Repacholi. Participants and contributors to the review included: Drs Ala Alwan, Antero Aitio, Jamie Bartram, Keith Baverstock, Elisabeth Cardis, Carlos Corvalan, Marilyn Fingerhut, Yoshikazu Hayashi, Richard Helmer, Jenny Pronczuk, Colin Roy, Dieter Schwela, Gennadi Souchkevich and Maged Younes.

The National Radiological Protection Board (NRPB) in the United Kingdom, a WHO Collaborating Centre on ionizing and non-ionizing radiation, provided many contributions relating to the radiological toxicity of DU. These contributions were provided by Dr Neil Stradling and other staff identified below.

The National Institute of Occupational Safety and Health (NIOSH) of the Center for Disease Control (CDC) in the USA, a WHO Collaborating Centre on occupational health, provided contributions mainly related to DU occupational health and safety requirements, protective measures and health monitoring. These contributions were provided by Dr Jim Neton and other staff identified below. The Centre for Health Promotion and Preventative Medicine (CHPPM) in the USA, provided contributions relating mainly to DU applications, radiological toxicity and medical care of people exposed to DU. These contributions were provided by Dr Mark Melanson and other staff identified below.

The International Atomic Energy Agency (IAEA) provided contributions on the effects of ionizing radiation and internationally recognized standards. These contributions were provided by Dr Carol Robinson and Dr Tiberio Cabianca.

Included in these reviewers and contributors are members of the International Commission on Radiological Protection (ICRP) an NGO in formal relations with WHO.

[1] The Government of Iraq has reported increases in cancers, congenital abnormalities and other diseases following the Gulf war in 1991, but there are no published results for review. WHO is working with the Government of Iraq to prepare studies to investigate this situation.

There have been a large number of contributors to this monograph, and it has been reviewed widely. In addition to the internal WHO review group, contributors and reviewers are listed below in alphabetical order:

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- ATSDR, USA: Dr Sam Keith
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The monograph was technically edited by Professor Barry Smith and language edited by Audrey Jackson, both from the British Geological Survey, UK. WHO acknowledges, with sincere gratitude, the contributions of all the authors and reviewers of this important monograph. In addition, WHO also acknowledges with thanks the photographs related to DU provided by IAEA, NIOSH and ATSDR.

WHO has and will continue to work with the United Nations Environment Programme (UNEP), the International Atomic Energy Agency (IAEA) and other UN agencies, Collaborating Centres and NGOs to advance our knowledge about exposure to DU and other environmental risk factors that could have consequences for health. Such programmes are aimed at providing essential information to member states and assisting national health services to deal with chemical, physical and biological risk factors in their environment.

Executive Summary

This scientific review on depleted uranium is part of the World Health Organization's (WHO's) ongoing process of assessment of possible health effects of exposure to chemical, physical and biological agents. Concerns about possible health consequences to populations residing in conflict areas where depleted uranium munitions were used have raised many important environmental health questions that are addressed in this monograph.

Purpose and scope

The main purpose of the monograph is to examine health risks that could arise from exposure to depleted uranium. The monograph is intended to be a desk reference providing useful information and recommendations to WHO Member States so that they may deal appropriately with the issue of depleted uranium and human health.

Information is given on sources of depleted uranium exposure, the likely routes of acute and chronic intake, the potential health risks from both the radiological and chemical toxicity standpoints and future research needs. Several ways of uptake of compounds with widely different solubility characteristics are also considered.

Information about uranium is used extensively because it behaves in the body the same way as depleted uranium.

Uranium and depleted uranium

Uranium is a naturally occurring, ubiquitous, heavy metal found in various chemical forms in all soils, rocks, seas and oceans. It is also present in drinking water and food. On average, approximately 90 µg (micrograms) of uranium exist in the human body from normal intakes of water, food and air; approximately 66% is found in the skeleton, 16% in the liver, 8% in the kidneys and 10% in other tissues.

Natural uranium consists of a mixture of three radioactive isotopes which are identified by the mass numbers ^{238}U(99.27% by mass), ^{235}U(0.72%) and ^{234}U(0.0054%).

Uranium is used primarily in nuclear power plants; most reactors require uranium in which the ^{235}U content is enriched from 0.72% to about 3%. The uranium remaining after removal of the enriched fraction is referred to as depleted uranium. Depleted uranium typically contains about 99.8% ^{238}U, 0.2% ^{235}U and 0.0006% ^{234}U by mass.

For the same mass, depleted uranium has about 60% of the radioactivity of uranium.

Depleted uranium may also result from the reprocessing of spent nuclear reactor fuel. Under these conditions another uranium isotope, ^{236}U may be present together with very small amounts of the transuranic elements plutonium, americium and neptunium and the fission product technetium-99. The increase in the radiation dose from the trace amounts of these additional elements is less than 1%. This is insignificant with respect to both chemical and radiological toxicity.

Uses of depleted uranium

Depleted uranium has a number of peaceful applications: counterweights or ballast in aircraft, radiation shields in medical equipment used for radiation therapy and containers for the transport of radioactive materials.

Due to its high density, which is about twice that of lead, and other physical properties, depleted uranium is used in munitions designed to penetrate armour plate. It also reinforces military vehicles, such as tanks.

Exposure and exposure pathways

Individuals can be exposed to depleted uranium in the same way they are routinely exposed to natural uranium, i.e. by inhalation, ingestion and dermal contact (including injury by embedded fragments).

Inhalation is the most likely route of intake during or following the use of depleted uranium munitions in conflict or when depleted uranium in the environment is re-suspended in the atmosphere by wind or other forms of disturbance. Accidental inhalation may also occur as a consequence of a fire in a depleted uranium storage facility, an aircraft crash or the decontamination of vehicles from within or near conflict areas.

Ingestion could occur in large sections of the population if their drinking water or food became contaminated with depleted uranium. In addition, the ingestion of soil by children is also considered a potentially important pathway.

Dermal contact is considered a relatively unimportant type of exposure since little of the depleted uranium will pass across the skin into the blood. However, depleted uranium could enter the systemic circulation through open wounds or from embedded depleted uranium fragments.

Body retention

Most (>95%) uranium entering the body is not absorbed, but is eliminated via the faeces. Of the uranium that is absorbed into the blood, approximately 67% will be filtered by the kidney and excreted in the urine in 24 hours.

Typically between 0.2 and 2% of the uranium in food and water is absorbed by the gastrointestinal tract. Soluble uranium compounds are more readily absorbed than those which are insoluble.

Health effects

Potentially depleted uranium has both chemical and radiological toxicity with the two important target organs being the kidneys and the lungs. Health consequences are determined by the physical and chemical nature of the depleted uranium to which an individual is exposed, and to the level and duration of exposure.

Long-term studies of workers exposed to uranium have reported some impairment of kidney function depending on the level of exposure. However, there is also some evidence that this impairment may be transient and that kidney function returns to normal once the source of excessive uranium exposure has been removed.

Insoluble inhaled uranium particles, 1-10 µm in size, tend to be retained in the lung and may lead to irradiation damage of the lung and even lung cancer if a high enough radiation dose results over a prolonged period.

Direct contact of depleted uranium metal with the skin, even for several weeks, is unlikely to produce radiation-induced erythema (superficial inflammation of the skin) or other short term effects. Follow-up studies of veterans with embedded fragments in the tissue have shown detectable levels of depleted uranium in the urine, but without apparent health consequences. The radiation dose to military personnel within an armoured vehicle is very unlikely to exceed the average annual external dose from natural background radiation from all sources.

Guidance on chemical toxicity and radiological dose

The monograph gives for the different types of exposure the tolerable intake, an estimate of the intake of a substance that can occur over a lifetime without appreciable health risk. These tolerable intakes are applicable to long term exposure. Single and short term exposures to higher levels may be tolerated without adverse effects but quantitative information is not available to assess how much the long term tolerable intake values may be temporarily exceeded without risk.

The general public's ingestion of soluble uranium compounds should not exceed the tolerable intake of 0.5 µg per kg of body weight per day. Insoluble uranium compounds are markedly less toxic to the kidneys, and a tolerable intake of 5 µg per kg of body weight per day is applicable.

Inhalation of soluble or insoluble depleted uranium compounds by the public should not exceed 1 $\mu g/m^3$ in the respirable fraction. This limit is derived from renal toxicity for soluble uranium compounds, and from radiation exposure for insoluble uranium compounds.

Excessive worker exposure to depleted uranium via ingestion is unlikely in workplaces where occupational health measures are in place.

Occupational exposure to soluble and insoluble uranium compounds, as an 8-hour time weighted average should not exceed 0.05 mg/m^3. This limit is also based both on chemical effects and radiation exposure.

Radiation dose limits

Radiation dose limits are prescribed for exposures above natural background levels.

For occupational exposure, the effective dose should not exceed 20 millisieverts (mSv) per year averaged over five consecutive years, or an effective dose of 50 mSv in any single year. The equivalent dose to the extremities (hands and feet) or the skin should not exceed 500 mSv in a year.

For exposure of the general public the effective dose should not exceed 1 mSv in a year; in special circumstances, the effective dose can be limited to 5 mSv in a single year provided that the average dose over five consecutive years does not exceed 1 mSv per year. The equivalent dose to the skin should not exceed 50 mSv in a year.

Assessment of intake and treatment

For the general population it is unlikely that the exposure to depleted uranium will significantly exceed the normal background uranium levels. When there is a good reason to believe that an exceptional exposure has taken place, the best way to verify this is to measure uranium in the urine.

The intake of depleted uranium can be determined from the amounts excreted daily in urine. depleted uranium levels are determined using sensitive mass spectrometric techniques; in such circumstances it should be possible to assess doses at the mSv level.

Faecal monitoring can give useful information on intake if samples are collected soon after exposure.

External radiation monitoring of the chest is of limited application because it requires the use of specialist facilities, and measurements need to be made soon after exposure for the purpose of dose assessment. Even under optimal conditions the minimum doses that can be assessed are in the tens of mSv.

There is no suitable treatment for highly exposed individuals that can be used to appreciably reduce the systemic content of depleted uranium when the time between exposure and treatment exceeds a few hours. Patients should be treated based on the symptoms observed.

Conclusions: Environment

Only military use of depleted uranium is likely to have any significant impact on environmental levels. Measurements of depleted uranium at sites where depleted uranium munitions were used indicate only localized (within a few tens of metres of the impact site) contamination at the ground surface. However, in some instances the levels of contamination in food and ground water could rise after some years and should be monitored and appropriate measures taken where there is a reasonable possibility of significant quantities of depleted uranium entering the food chain. The WHO guidelines for drinking-water quality, 2 µg of uranium per litre, would apply to depleted uranium.

Where possible clean-up operations in conflict impact zones should be undertaken where there are substantial numbers of radioactive particles remaining and depleted uranium contamination levels are deemed unacceptable by qualified experts. Areas with very high concentrations of depleted uranium may need to be cordoned off until they are cleaned up

Since depleted uranium is a mildly radioactive metal, restrictions are needed on the disposal of depleted uranium. There is the possibility that depleted uranium scrap metal could be added to other scrap metals for use in refabricated products. Disposal should conform to appropriate recommendations for use of radioactive materials.

Conclusions: Exposed populations

Limitation on human intake of soluble depleted uranium compounds should be based on a tolerable intake value of 0.5 µg per kg of body weight per day, and that the intake of insoluble depleted uranium compounds should be based on both chemical effects and the radiation dose limits prescribed in the International Basic Safety Standards (BSS) on

radiation protection. Exposure to depleted uranium should be controlled to the levels recommended for protection against radiological and chemical toxicity outlined in the monograph for both soluble and insoluble depleted uranium compounds.

General screening or monitoring for possible depleted uranium-related health effects in populations living in conflict areas where depleted uranium has been used is not necessary. Individuals who believe they have been exposed to excessive amounts of depleted uranium should consult their medical practitioner for examination, appropriate treatment of any symptoms and follow-up.

Young children could receive greater depleted uranium exposure when playing within a conflict zone because of hand-to-mouth activity that could result in high depleted uranium ingestion from contaminated soil. This type of exposure needs to be monitored and necessary preventative measures taken.

Conclusions: Research

Gaps in knowledge exist and further research is recommended in key areas that would allow better health risk assessments to be made. In particular, studies are needed to clarify our understanding of the extent, reversibility and possible existence of thresholds for kidney damage in people exposed to depleted uranium. Important information could come from studies of populations exposed to naturally elevated concentrations of uranium in drinking water.

Depleted uranium:

Sources, Exposure and Health Effects

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1 Introduction

WHO commissions reviews and undertakes health risk assessments associated with exposure to potentially hazardous physical, chemical and biological agents in the home, work place and environment. This monograph on the chemical and radiological hazards associated with exposure to depleted uranium is one such assessment.

The purpose of this monograph is to provide generic information on any risks to health from depleted uranium from all avenues of exposure to the body and from any activity where human exposure could likely occur. Such activities include those involved with fabrication and use of DU products in industrial, commercial and military settings.

While this monograph is primarily on DU, reference is also made to the health effects and behaviour of uranium, since uranium acts on body organs and tissues in the same way as DU and the results and conclusions from uranium studies are considered to be broadly applicable to DU. However, in the case of effects due to ionizing radiation, DU is less radioactive than uranium.

This review is structured as broadly indicated in Figure 1.1, with individual chapters focussing on the identification of environmental and man-made sources of uranium and DU, exposure pathways and scenarios, likely chemical and radiological hazards and where data is available commenting on exposure-response relationships.

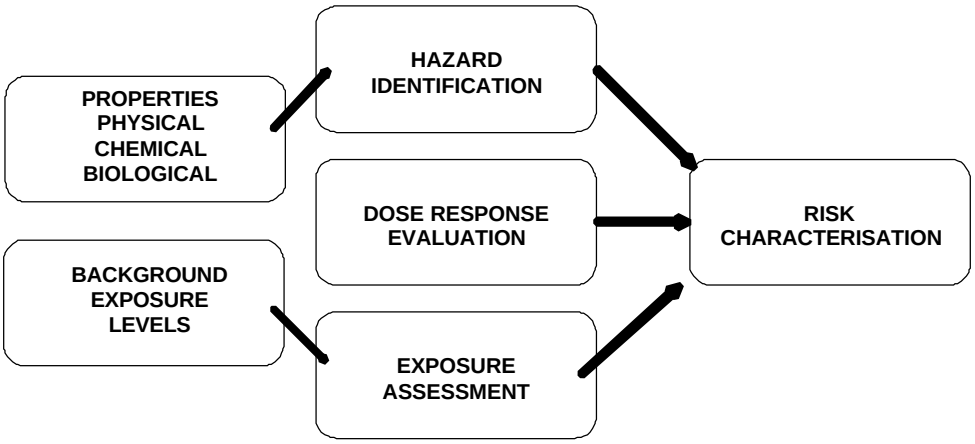

Figure 1.1 Schematic diagram, depicting areas covered by this monograph.

It is expected that the monograph could be used as a reference for health risk assessments in any application where DU is used and human exposure or contact could result. Information is provided that should also be applicable to exposure of people during or following military conflict. Details are provided on the characteristics of uranium and DU, historical and current applications, behaviour in the environment, factors that influence human exposure, how it acts on the body, chemical and radiological toxicity, health and medical monitoring, protective measures and public health standards.

Gaps in knowledge have been identified that require further research which will improve future health risk assessments. Scientists world wide are encouraged to take up the challenge to undertake this research in order that a more complete picture of the health effects of DU will result.

Readers who will benefit most from this monograph are:

- scientists involved in the assessment of health risks and environmental protection.
- clinicians needing to assess situations where people may be exposed excessively to DU and may be considering medical monitoring or follow-up.
- national authorities developing programmes to deal with human exposure and environmental contamination following conflicts where DU weapons were used.

A glossary of key terms and units is provided.

2 Properties of uranium and depleted uranium

2.1 Uranium

Uranium is a silver-white, lustrous, dense, natural and commonly occurring weakly radioactive element. It is ubiquitous throughout the natural environment, being found in varying but small amounts in rocks, soils, water, air, plants, animals and in all human beings.

Elemental uranium has an atomic number of 92 and an atomic weight of 238.0289 g/mol and is a member of the actinide series of the periodic table. Metallic uranium has a high density of 19 g/cm^3, slightly less than tungsten but significantly greater than lead with a density of 11.3 g/cm^3. The metal tarnishes in air and is ductile, malleable and capable of being highly polished.

Natural uranium contains three radioactive isotopes (or radio-isotopes) ^{234}U, ^{235}U and ^{238}U. The percentage of each radio-isotope by weight is about 0.0054% ^{234}U, 0.72% ^{235}U and 99.27% ^{238}U. About 48.9% of the radioactivity is associated with ^{234}U, 2.2% with ^{235}U and 48.9% with ^{238}U. The half-lives (time for the radioactivity to decay to half its original value) of the uranium radio-isotopes are very long, 244 000 years for ^{234}U, 710 million years for ^{235}U and 4500 million years for ^{238}U. The longer the half-life the less radioactive is a given mass of material. Uranium decays into many other radio-isotopes, called progeny, until it finally ends up as stable (nonradioactive) isotopes of lead. Due to its long radioactive half-life in comparison to the age of the solar system, uranium is considered to be a naturally occurring primordial radio-element.

When finely divided in air, uranium metal is combustible and ignites readily, a property known as pyrophoricity which is also typical of other metals such as aluminium (Al) and iron (Fe). Thus when used in military armour or projectiles, or when present in an air crash or fire in which significant heat is generated, uranium may form fine dust containing a mixture of uranium oxides.

All isotopes of uranium undergo the same chemical reactions in nature and possess almost identical physical characteristics, such as melting point, boiling point and volatility. The radioactive properties (half-life, specific activity, decay mode etc.) of all uranium isotopes are however different.

In their pure form, natural uranium, enriched uranium and DU vary only according to their isotopic composition and are therefore almost chemically identical, undergoing identical chemical reactions in the environment and exerting the same chemical, biochemical and biological effects on the human body. Where small differences in chemical behaviour exist these will be due to the small mass difference between various uranium isotopes.

Uranium compounds These differ substantially in their chemical and physiological properties and in the toxicological effects they exert. For example, compounds such as uranium trioxide (UO_3), uranyl chloride, uranyl nitrate and uranyl ethanoate are relatively soluble, whereas uranium dioxide (UO_2) and triuranium octaoxide (U_3O_8) are considered to be relatively insoluble. Many publications have been devoted to the aqueous chemistry, mineralogy and properties of uranium, and numerous reviews are available (e.g. Burns and Finch, 1999; ATSDR, 1999, DeVivo et al., 1984).

Units Units of concentration used to describe the abundance of uranium are diverse, and often complicate, rather than facilitate a comparison of data from differing sources. The concentrations of individual isotopes of uranium are generally recorded as the radiochemical activity present in a unit volume of a substance, e.g. picocuries per litre (pCi/l) or bequerels per litre (Bq/l), picocuries per kilogram (pCi/kg) or bequerels per kilogram (Bq/kg). The use of picocuries is still common in the literature even though this has been superseded by the bequerel as the SI unit for radioactivity. One Bq is defined as one nuclear transformation per second. One Bq is equal to approximately 27 pCi.

It is equally common practice to measure and report natural uranium content or concentration in mass units (i.e. µg/l, µg/kg or moles/kg). These units are used throughout this work for consistency. Concentrations of uranium may also be quoted in terms of molar concentrations, 1 µmole of uranium being equivalent to 0.000 238 g of ^{238}U and correspondingly 1 µg of ^{238}U = 0.0042 µmole.

Radio-isotope properties In most situations the natural uranium isotopes occur with the relative mass abundances given in Table 2.1. However, in some circumstances such as in the natural nuclear reactor at Oklo in Gabon (e.g. Burns and Finch, 1999) and in natural waters (e.g. Ivanovich and Harmon, 1982), soils and atmospheric dusts (US EPA, 1994), these ratios may be influenced by natural nuclear and chemical processes that have lead to the enrichment or depletion of ^{235}U and/or ^{234}U relative to ^{238}U. It is therefore important when using isotopic ratios to forensically identify exposure to DU that the potential for changes in natural isotopic ratios are taken into account.

Table 2.1 Relative mass abundances and isotopic ratios for natural uranium isotopes (Steiger and Jager, 1977; Kaye and Laby, 1993).

Isotope	Abundance
^{238}U -	99.2745%
^{235}U -	0.7200%
^{234}U -	0.0054%
$^{235}U/^{238}U$	0.00725
$^{234}U/^{238}U$*	0.0000554

* Range from 0.000 05 to 0.0004 in atmospheric dusts (US EPA, 1994) and 0.000 03 to 0.0014 in natural waters (Ivanovich and Harmon, 1982).

In nature, uranium occurs in conjunction with its radioactive decay products (Tables 2.2 and 2.3). However, when purified for chemical or nuclear purposes, uranium is generally separated from its decay products and is therefore less radioactive than original impure ore containing a similar weight of uranium.

Table 2.2 ^{238}U decay series (Kaye and Laby, 1993).

Isotope	Half-life	Principal decay mode
^{238}U	4.5×10^{9} y	alpha
^{234}Th	24 d	beta
$^{234}Pa^{m}$	1.17m	beta
^{234}Pa	6.8 h	beta
^{234}U	2.4×10^{5} y	alpha
^{230}Th	7.3×10^{3} y	alpha
^{226}Ra	1.6×10^{3} y	alpha
^{222}Rn	3.8 d	alpha
^{218}Po	3.1 m	alpha
^{218}At	2 s	alpha
^{214}Pb	27 m	beta
^{214}Bi	20 m	beta
^{214}Po	160 µs	alpha
^{210}Tl	1.3 m	beta
^{210}Pb	22 y	beta
^{210}Bi	5.0 d	beta
^{210}Po	138 d	alpha
^{206}Tl	4.2 m	beta
^{206}Pb	Stable	—

Table 2.3 ^{235}U decay series (Kaye and Laby, 1993).

Isotope	Half-life	Principal decay mode
^{235}U	7.0×10^{8} y	alpha
^{231}Th	26 h	beta
^{231}Pa	3.3×10^{4} y	alpha
^{227}Ac	22 y	beta
^{227}Th	19 d	alpha
^{223}Fr	21.8 m	alpha
^{223}Ra	11.4 d	alpha
^{219}Rn	4.0 s	alpha
^{215}Po	1.8 ms	alpha
^{215}At	1×10^{-4} s	alpha
^{211}Pb	36.1 m	beta
^{211}Bi	2.2 m	alpha
^{211}Po	0.5 s	alpha
^{207}Tl	4.8 m	beta
^{207}Pb	Stable	—

2.2 Depleted uranium

DU, as a by-product of uranium enrichment (see Annex 1) required for the nuclear industry, has only been available since about 1940. As such it can only contain very low levels of many of the naturally occurring radioactive decay products of uranium listed in Tables 2.2 and 2.3.

Uranium is classed as DU when the abundances of ^{235}U and ^{234}U are reduced relative to ^{238}U. Depleted uranium typically has around 0.3% to 0.2% ^{235}U by mass, although the Nuclear Regulatory Commission in the US defines DU as uranium in which the percentage of ^{235}U is less than 0.711% (NRC, 2000). Consequently, DU has a marginally higher percentage of ^{238}U (99.8%) than naturally occurring uranium (99.3%). The isotopic composition of DU typically used by the US Department of Defence as quoted in CHPPM (2000) is ^{234}U = 0.0006%, ^{235}U = 0.2%, ^{236}U = 0.0003% and ^{238}U = 99.8%.

Some preparations of DU may also contain transuranic elements and fission products (e.g. Rich, 1988; CHPPM, 2000). This occurs because the same enrichment plants are used for both natural uranium and for reprocessing spent uranium nuclear fuel rods. The latter can cause contamination of the enrichment plant with reactor-made isotopes that will subsequently contaminate the DU from natural uranium enrichment. Such DU may consequently be very slightly more radioactive than DU derived from mined uranium ore. Typical trace isotopes identified as being present in DU used in munitions and armour manufacture include ^{238}Pu, ^{239}Pu, ^{240}Pu, ^{241}Am, ^{237}Np and ^{99}Tc. These impurities typically add less than 1% to the dose from DU and are therefore inconsequential from a radiological or chemical toxicity standpoint (CHPPM, 2000). This has been verified from a radiological standpoint based on available data regarding the concentrations of these isotopes in DU stocks from the USA (see Annex 2).

A recent survey of DU in Kosovo by the United National Environment Programme provided an analysis of penetrators found in conflict areas (UNEP, 2001). The activity concentration of transuranic elements in these penetrators indicated that there was up to 12 Bq/kg of plutonium isotopes and for ^{236}U the activity was up to 61 kBq/kg. This compared to an activity concentration of 12 700 kBq/kg for ^{238}U.

^{238}U has a longer half-life than either ^{235}U or ^{234}U and it is present in a much greater abundance in natural and DU than ^{235}U or ^{234}U. The number of alpha particles produced per year in one milligram of natural uranium from the decay of ^{238}U, ^{235}U and ^{234}U may be calculated to be 3.9×10^{11}, 1.7×10^{10} and 3.9×10^{11}, respectively.

Specific activities and data related to isotopes commonly found in DU are given in Table 2.4.

As well as decay through the emission of alpha particles, atoms of ^{238}U may also decay through spontaneous fission, an energetic process that releases approximately 40 times more energy per nuclear decay. The spontaneous fission half-life for ^{238}U is estimated to be 8.5×10^{17} years (De Carvalho et al., 1982) which, although much longer than its alpha decay half-life, results in approximately two atoms of ^{238}U in every milligram of uranium decaying by this process each year. Similarly rates of spontaneous fission from other natural and anthropogenic (human produced) isotopes of actinides associated with DU are low compared to the rates of other decay modes (e.g. alpha).

DU has a specific activity of 14.8 Bq/mg which is approximately 60% that of natural uranium (25.4 Bq/mg) due to the partial removal of ^{234}U. In practice, not all ^{234}U is removed in the separation process and the exact concentration remaining in DU depends on separation plant characteristics and required yields.

Following chemical separation of uranium, radioactive progeny (or 'daughter products') are produced by the radioactive decay of uranium 'ingrow' (for an extensive discussion of radioactive ingrowth, radioactive equilibrium, secular equilibrium, radioactive decay series and their effect on isotopic composition the reader is referred to Faure, 1986). During the first year, the activity of immediate progeny (the beta decaying isotopes ^{234}Th, ^{234m}Pa and ^{231}Th) reach secular equilibrium. Following this initial ingrowth period the activity of isotopes remains approximately constant for over 1 000 years (Figure 2.2a and 2.2b), although ^{231}Pa (^{235}U decay chain) will begin to ingrow during this period (Figure 2.2b)

Table 2.4 Specific activities of uranium and other radionuclides associated with DU (Lederer et al., 1978; Kaye and Laby, 1993).

Radionuclide (Decay Modes)	Half-life (million years)	l (per second)	Atomic weight (MW)	Specific activity as Bq/mg
Natural U	-	-	-	25.4
^{238}U (α) + (sf_{rare})	4470	4.91×10^{-18}	238	12.4
^{236}U (α)	23.42	9.38×10^{-16}	236	2390
^{235}U (α and γ)	704	3.12×10^{-17}	235	80
^{234}U (α)	0.245	8.96×10^{-14}	234	2.31×10^{5}
^{232}U (α)	0.000072	3.05×10^{-10}	232	7.92×10^{8}
^{231}Pa (α and γ)	3.28×10^{-2}	6.70×10^{-13}	231	1.75×10^{6}
$^{234}Pa^{m}$ (β)	2.29×10^{-12}	9.59×10^{-3}	234	2.47×10^{16}
^{231}Th (β and γ)	2.91×10^{-9}	7.54×10^{-6}	231	1.97×10^{13}
^{234Th} (β and γ)	6.06×10^{-8}	3.33×10^{-7}	234	8.57×10^{11}

sf_{rare} indicates decay through **s**pontaneous **f**ission with a half-life much greater than that of other major decay modes.

Note: ^{236}U does not occur naturally although it may be present in DU derived from the nuclear industry.

The short half-lives, chemical nature and decay mode of many of the decay products of ^{238}U and ^{235}U significantly increase the radiological hazard of natural uranium mineralisation and uranium ore. These radioactive progeny are removed during the preliminary stages of uranium ore purification, making the processed chemically pure uranium significantly less radiologically toxic than equivalent amounts of the original ore.

On a weight-by-weight basis the reduced proportion of ^{235}U and ^{234}U, and the absence of radioactive progeny such as radium in DU, means that DU poses less of a radiological hazard than either pure processed uranium or naturally occurring uranium ores, respectively. Figures 2.2a and 2.2b show the ingrowth of ^{235}U and ^{238}U progeny,

and consequent increase in total activity over time in freshly prepared DU. One result is a marked increase in beta and gamma radiation levels as times passes, although the level of alpha decay continues to be the principal concern in respect of internal dose estimation.

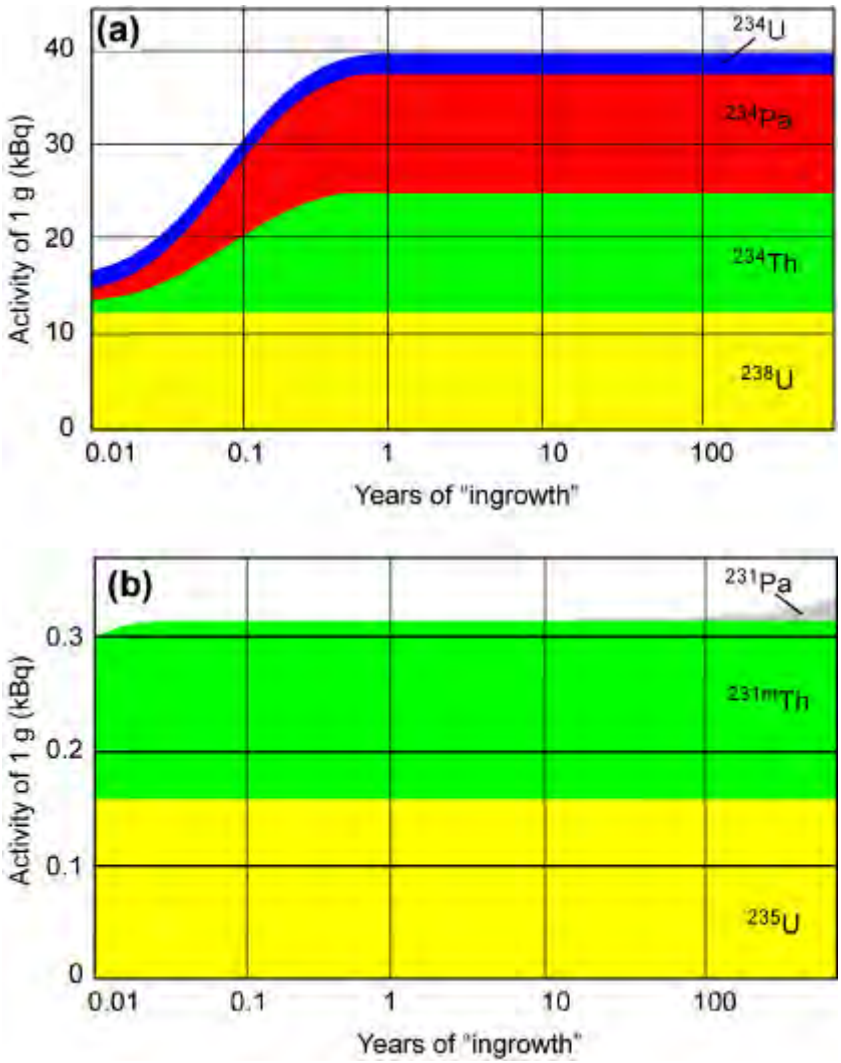

Figure 2.2a ^{238}U decay and **Figure 2.2b** ^{235}U decay**.** Variation in activity (kBq/g) of uranium and uranium decay products for the first 1000 years following chemical separation of a sample of DU containing 0.2% ^{235}U and a typical amount of ^{234}U. Note the numerical dominance in activity due to the ^{238}U decay series and the beginning 'ingrowth' of ^{231}Pa (^{235}U decay series) after approximately 500 years.

2.3 Summary

Natural uranium consists of three different isotopes: by mass the composition is ^{234}U (0.0054%), ^{235}U (0.72%) and ^{238}U (99.2746%). It is present to some extent in all environmental materials (e.g. soils, surface waters, groundwaters, foodstuffs, air, etc.).

Uranium is classed as depleted uranium (DU) when the abundance of its isotopes ^{235}U and ^{234}U are reduced relative to ^{238}U. Depleted uranium has typically about 60% of the radioactivity found in natural uranium with only an infinitesimal change in mass.

Both uranium and DU and their immediate decay products (e.g. ^{234}Th, ^{234m}Pa and ^{231}Th) emit alpha and beta particles with a very small amount of gamma radiation. Alpha and

beta radiations are not very penetrating and are easily absorbed in the air and the skin. The half-life of DU, or the time for the radiation dose to decay to half its original value is many millions of years.

Natural uranium and DU are considered to be weakly radioactive. In the event that DU is produced by enrichment of spent reactor fuel rods, the additional radio-isotopes in the DU increase the overall radiation dose of the DU by less than 1%. This additional radiation dose is considered to produce no additional radiological consequence.

3 Uranium in the environment

Uranium occurs in rocks, sea and fresh water and in the human body (see Table 3.1). Uranium (U) is a naturally occurring element with an average abundance in the Earth's crust of about 2 mg per kg (range 0.1 to 20 mg per kg). It is more abundant than silver or gold. For example Alloway, (1995) has quoted an average concentration and range in typical crustal rocks of 2.5 mg/kg and 0.05 mg/kg to 5 mg/kg, respectively, although higher concentrations commonly exist in some mineralized environments.

Table 3.1 Concentrations of uranium in various environmental systems and materials (Kaye and Laby, 1993).

Physical Entity	Abundance (mg/kg)
Crustal rocks	1.800*
Sea water	0.0033
Stream water	0.00004
Human	0.001

* Note different value from that quoted by Alloway (1995) emphasising the variability of such gross compositional estimates, which is also the case for many other elements in crustal rocks.

During natural geological processes such as the partial melting and fractional crystallisation of the Earth's mantle, uranium becomes preferentially concentrated in the liquid phase and consequently becomes incorporated into the more silica-rich products. Because of this, igneous rocks such as granites are typically enhanced in uranium compared with rocks of basaltic or ultramafic composition. Uranium released into the terrestrial environment from the weathering of igneous rocks has also become redistributed and concentrated over geological time into many sedimentary rocks such as siltstones and mudstones. In these rocks it is commonly associated with organic matter and phosphates (particularly in marine environments due to the relatively high concentration of uranium in seawater). Further enrichment due to geochemical processes leads to the formation of primary and secondary ores (i.e. 0.1% to 5 % U; 1000 to 50 000 mg/kg) and an elevated abundance in certain geological materials such as phosphorite (i.e. 0.01% to 1% U; 100 to 10 000 mg/kg). Uranium is also present at relatively high concentrations in sea water and has a wide abundance in natural waters, often being more abundant in groundwater than surface water due to contact with underlying rock structures and weathering of surface rocks.

Uranium mineralisation occurs in many parts of the world (see Annex 3) and in a variety of mineral deposits (Plant et al., 1999; De Vivo et al., 1984). Relatively high-grade ores found in Canada and West Africa contain percentage levels of uranium in the form of pitchblende (uranium dioxide) and uraninite (mixed UO_2-U_3O_8). Low-grade sources such as uranium-bearing phosphorites, lignites, and shales are widespread but contain less than 0.01% U. However, the concentration of uranium in such low-grade sources still often exceeds the concentrations observed in granites by up to two orders of magnitude. Similarly uranium may also be found in association with other metalliferrous and non-metalliferrous mineralisation. Examples of this include

phosphates and other commercial sources of phosphorus such as some deposits of clays, guano, sedimentary molybdenum deposits and coal. In nature, uranium forms chemical compounds in three ionic states, U^{4+}, U^{5+} and U^{6+}, although U^{6+} exists only under oxidising conditions.

Table 3.2 Concentration of uranium in rocks (De Vivo et al., 1984).

Rock Type	Uranium Concentration (mg/kg)
Basalts (tholeitic and plateau, andesitic and alkali)	0.1-1.0
Carbonate rocks (of North America and the Russian platform)	2
Tertiary rocks (of the Texas Gulf Coast)	2-4
North American and Russian platform (average)	3.7
Granites (of USA. Russia and France)	2-15
Alkaline intrusives (Russia)	3-20

This chapter discusses the abundance and spatial variability of uranium in the environment. In the context of studies of DU, such data are also essential in order to compare exposure due to natural baseline concentrations of uranium with that due to anthropogenically introduced DU.

Without the presence of uranium, Earth would be a rather different planet as heat produced by the radioactive decay of uranium is partly responsible, in addition to other naturally occurring radionuclides such as ^{232}Th and ^{40}K, for keeping the Earth's core and mantle hot enough for convective flow to occur (Plant et al., 1999). Similarly, it is also important to realize that uranium may become depleted (or enriched) in some of its isotopes due to purely naturally occurring processes such as chemical weathering.

When considering radiation toxicity of DU in a given area, it is also important to know the levels of radioactivity existing in the environment prior to the DU being deposited. In this context, radioactivity due to previous man-made events such as the Chernobyl reactor accident and weapons testing need to be taken into account to determine the true background levels.

3.1 Air

Reported background levels of uranium in air vary widely. For example, WHO (1998b) quotes values in ambient air from 0.02 ng/m^3 to 0.076 ng/m^3, while in the USA, the NCRP quotes a background concentration of 0.30 ng/m^3 (NCRP, 1975) and the US EPA a range of 0.15 to 0.40 ng/m^3 in 51 urban and rural areas across the USA (US EPA, 1986). During these surveys it has also been established that $^{234}U/^{238}U$ ratios vary widely in dust samples (range 0.000 05 to 0.000 40 as mass abundance or 1 to 7 as an activity ratio, indicating the presence of excess ^{234}U). It was considered that this enrichment was due to natural processes such as alpha recoil (e.g. Faure, 1986) and that atmospheric levels of uranium were principally derived from suspension of soils

(ATSDR, 1999). This observation is also consistent with such dusts containing absorbed weathered uranium, as alpha recoil would lead to depletion of primary uranium phases in soils, due to the preferential leaching of ^{234}U. Bou-Rabee (1995) measured uranium concentrations and isotopic ratios in 8 air samples collected following the Gulf-War (sampled in 1993–1994). The observed concentrations varied between 0.22 and 0.42 ng/m^3 with $^{235}U/^{238}U$ ratios ranging between 0.005 and 0.007.

When undertaking local measurements of DU in air, it is essential to take into account the levels of natural uranium in that air, especially in dusty environments. Also, it may be necessary in some cases to take into consideration the short and long range transport of natural and anthropogenically produced atmospheric particulates. Particularly as the transport of other airborne particulates has been observed to occur over large distances (UN, 1991).

In addition to other carcinogens, tobacco smoke contains significant quantities of uranium and ^{210}Po. Smoking two packs of cigarettes produces in the region of 50 ng of uranium in a form that may subsequently be inhaled (WHO, 1998b). Elevated levels of uranium in air (e.g. 3 ng/m^3) have also been found down wind of coal fired power stations associated with their discharges (NCRP, 1975).

Many of the data relating to uranium concentrations in air come from the mining of uranium and from industries such as the fertilizer industry in which uranium rich dusts may be produced. These data have not been collated in this chapter because these levels represent extremes of conditions unlikely to be replicated outside the mining and chemical industries.

Concentrations of various dusts in uranium mining have progressively decreased over the years complicating the use of such data in epidemiological studies. For example, potential dust exposure in the underground mines of Wismut Ltd during the late 1940s and early 1950s were estimated (Bauer, 1997) to be in the order of 33.7 mg/m^3 (total weight of airborne particulates). However, the same author noted that exposure to dust was reduced to less than 3% of this level after the adoption, of water rather than air flush, drilling in the 1960s.

Levels of uranium in air surrounding nuclear facilities in which uranium is handled in the preparation and fabrication of fuel assemblies are commonly sampled to monitor discharge released from such sites. Data from measurements in the United Kingdom indicate annual atmospheric discharges from such sites to be in the range of less than 0.005 to 130 kg (MAFF, 1999). Similar releases are documented elsewhere. For example, it has been estimated that airborne releases of uranium at one U.S. Department of Energy facility amounted to 310 000 kg between 1951 and 1988 (equivalent to a rate of approximately 8000 kg per annum). This produced an estimated offsite inventory of 2130–6140 kg of excess uranium in the top 5 cm of soil in the vicinity of the facility (Meyer et al. 1996).

Other data from the USA and Canada have also shown elevated uranium levels in and around milling and processing facilities, measured values ranging from 3 to 200 ng/m^3 at distances of up to 2 km from site boundaries (ATSDR, 1990; 1999).

3.2 Water

Uranium is always present in surface water and groundwater. There is an extremely wide range of concentrations from below 0.01 µg/l to in excess of 1500 µg/l water. Its

natural abundance in water is variable and reflects the concentration of uranium in surrounding rocks and soils through and/or over which water may pass (e.g. see Figure 3.1), and the mobility of various forms of uranium in the prevailing aqueous environment (Figure 3.2). Concentrations of uranium in rainfall are low and variable (e.g. range 0.018 to 0.17 µg/l for the USA during March–May 1993; ASTDR, 1999)

Various anthropogenic activities involving the processing or use of materials rich in uranium may modify the natural abundance of uranium in water. These activities include the use of phosphate fertilizers, various mining activities (uranium, silver, and other mineral mines) and the industrial processing of uranium for the manufacture of nuclear fuel and other products, including DU for various uses. Although the isotopic ratio of ^{235}U to ^{238}U generally remains constant in waters some fractionation occurs in the relative isotopic abundance of ^{234}U compared to ^{238}U and ^{235}U due to disequilibrium phenomena. Additionally, variations in ^{235}U to ^{238}U ratios have been measured in the vicinity of Oklo, Gabon in a natural uranium deposit which has been shown to have reached nuclear criticality approximately 2000 million years ago as a result of natural processes (e.g. Burns and Finch, 1999).

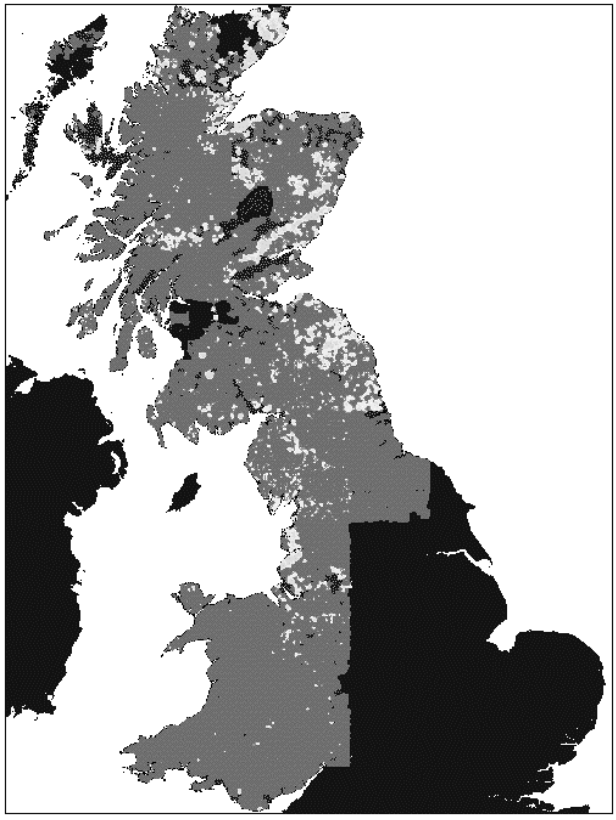

Figure 3.1 Map showing areas (in light grey) of the United Kingdom in which the measured concentration of uranium in surface stream waters exceeds 2 µg/l. The total number of samples in the sampled area (dark grey) exceeds 100 000. The mean uranium concentration in these surface waters has been determined to be 0.65 µg/lwith a maximum observed concentration of 233 µg/l. The spatial distribution of elevated concentrations of uranium in stream water were observed to reflect areas of high uranium mobility rather than the abundance of uranium in bedrock, and also to reflect areas of anthropogenic input such as nuclear facilities handling the purification and production of fissile uranium (British Geological Survey, 1992; 1997). This figure has been compiled from data collected during the Geochemical Baseline Survey of the Environment (G-BASE), British Geological Survey.

Uranium acetates, sulphates, carbonates, chlorides and nitrates readily dissolve in water and their chemical form in typical groundwaters and surface waters is generally dominated by the presence of carbonate species, although sulphate and phosphate may also form important species in other circumstances (e.g. Bernhard et al., 1998; Smith et al., 2000). These carbonate complexes may be either negatively charged or neutral and, as such, are highly mobile in most soils despite the presence of cation exchangers such as clays (Abdelouas et al., 1998; Duff and Amrhein, 1996; Elless and Lee, 1998). This makes uranium particularly mobile in soils and infiltrating groundwater in arid and semi-arid regions, such as those typified by Mediterranean environments. Under weakly acidic conditions typical of soils and infiltrating groundwaters found in wetter climes, the chemical speciation of uranium may become dominated by the formation of stable complexes with soil organic matter. This commonly results in the retention and accumulation of uranium in peat deposits, although in situations where a significant proportion of the organic matter is in the dissolved form this may assist in the dissolution and mobilisation of soil-bound uranium (Ebbs et al., 1998; Benes et al., 1998). In addition to transport in the dissolved phase, labile uranium may also be redistributed from soils and sediments into watercourses and surface water reservoirs as, or sorbed onto, particulate matter during storms and other modes of physical erosion (Batson et al., 1996; Zielinski et al., 1997; Porcelli et al., 1997).

Drinking water: Many studies have been undertaken on the concentrations of uranium in drinking water. These have included surveys of treated water, surface water, groundwater and bottled water. However, significant improvements in the precision and accuracy of analytical methods used to determine uranium content over the past twenty years raises doubts as to the reliability of some of the data collected prior to the 1980s. A summary of typical studies and surveys performed prior to 1980 on the concentration and isotopic signature of uranium in water is given in Ivanovich and Harmon, (1982). A selection of data from studies performed since 1980 are given in Annex 3

From this selection of studies and those quoted elsewhere (i.e. WHO, 1998a, 1998b) it can be concluded that the concentration of uranium determined in drinking water covers a very wide range. It is generally affected not only by the presence of significant concentrations of uranium in the local geological or surface environment, but also by the weatherability of the media containing uranium and its mobility in solution. This wide range of concentration, together with significant regional and local variations (due to local scale variability in geological structure), makes it impossible to estimate typical exposures to the presence of natural uranium from drinking water and by inference the likely importance of additional sources such as DU. However, it is clear from these studies that the probability of a drinking water source containing uranium at a concentration of about 2 µg/l is relatively high, getting progressively smaller over the range 5 to 10 µg/l and becoming very small above 10 µg/l. Such data indicates that many countries are likely to have drinking water supplies in which the current WHO guideline for uranium in drinking water (2 µg/l; WHO, 1998a) is likely to be exceeded.

3.3 Soil

There are few systematic studies in which uranium has been measured over larger areas with high resolution. A selection of more recent generalized studies in which significant attention has been paid to define baseline or benchmark data are presented in Annex 3. Additionally the advent of high sensitivity, high resolution airborne and seabed gamma spectrometry has enabled uranium to be routinely determined at baseline levels across relatively large areas.

The concentration of uranium in soil varies widely. This reflects the abundance of uranium in the parent geological materials from which the soils were formed, the soil development processes (for example uranium may become concentrated in organic-rich horizons) or leaching, and the addition of uranium from anthropogenic activities such as fertilizer application or military conflicts/training.

Levels of uranium in soils generally not associated with known sources of anthropogenic contamination or obvious areas of mineralisation indicate median concentrations in the order of 1 to 2 mg/kg. However, variations can be very spatially dependent and may reflect not only geologically derived sources of elevated uranium but also dispersion zones associated with the transport of river sediments. Concentrations as high as 4 mg/kg are often found in soils away from any obvious anthropogenic activity and have been suggested to represent the upper baseline level for uranium. However, even higher concentrations of uranium can be found in soils associated with mineralized environments such as those found in the vicinity of deposits of phosphate or in superficial uranium ore deposits. Such deposits are common throughout the world. For example measured levels of uranium in surface soils associated with phosphorite in North Africa and the Middle East, may reach that of the primary phosphate, i.e. approximately 200 mg/kg. In industrialized environments, uranium may also be found associated with uranium processing plants (e.g. British Geological Survey, 1992), mine tailings and process waste streams (e.g. Ledvina et al., 1996; McConnell et al., 1998), and agricultural environments in which uranium-rich phosphate fertilizers have been used (e.g. Zielinski et al., 1997).

3.4 Mobility of uranium in soil and water

The dissolution and mobility of dissolved uranium and hence DU, in soils (e.g. during the infiltration of rainwater and plant uptake) is strongly controlled by the proximity of groundwater to the soil environment, soil and water pH, soil organic carbon content and the presence and abundance of cation exchange sites such as those found in clays (e.g. Ribera et al., 1996; Burns and Finch, 1999 and US EPA, 1999; 2000b). Unlike many heavy metals, such as lead, the mobility of uranium increases under neutral to alkaline conditions, due to the formation of stable negative complexes (oxy-anions) with oxygen and carbon (Figure 3.2). For example, uranium sorption values were found to be very low in soils rich in montmorillinite and low in organic carbon, such as those found in west Anatolia and other semi-arid Mediterranean type environments (e.g. Akcay, 1998; Zielinski et al., 1997).

More accurate insights into the mobility and chemical form of uranium in soils and groundwater require the use of geochemical modelling codes such as PHREEQC (Parkhurst and Appello, 1999) or coupled chemical transport codes in which predictions concerning the physical migration of uranium are coupled to chemical processes that may retard such migration.

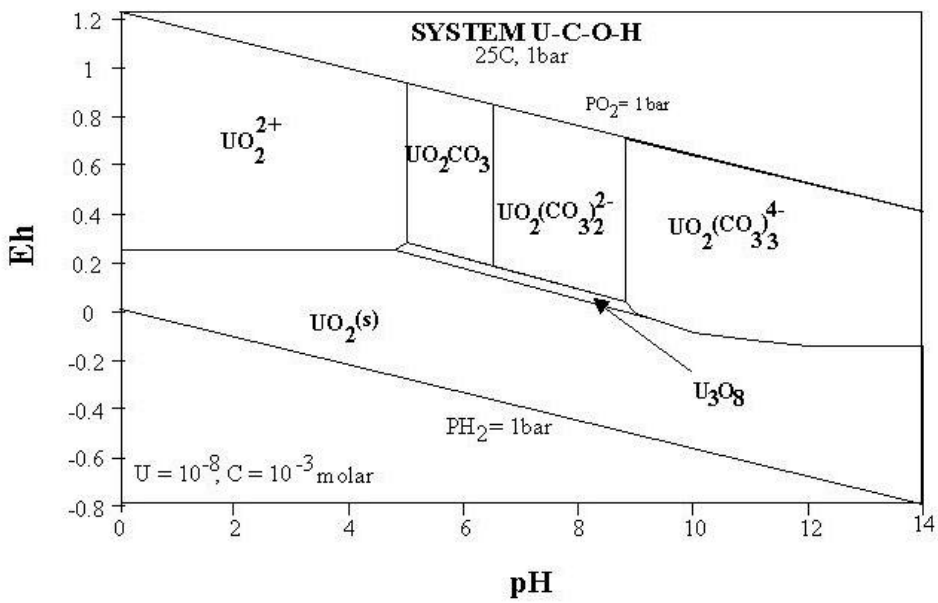

Figure 3.2 Eh-pH diagram showing stability fields for uranium under various Eh and pH conditions. Eh is an indicator of oxidation potential that may be related to the presence of dissolved oxygen. pH is an indicator of acidity. Note the wide stability fields (i.e. the regions bounded by lines) over environmental conditions (moderate Eh and pH) of the dissolved, highly soluble neutral and negatively charged anionic species UO_2CO_3, $UO_2\ (CO_3)_2^{2-}$ and $UO_2\ (CO_3)_3^{4-}$ compared to that of the positively charged, strongly sorbed cation UO_2^{2+} and insoluble UO_2 (s). The diagram has been constructed for a U-C-O-H system adapted from Brookins, (1988) and may be used as a first approximation to predict the chemical form and mobility of uranium species in soils and groundwaters in which Eh and pH have been determined.

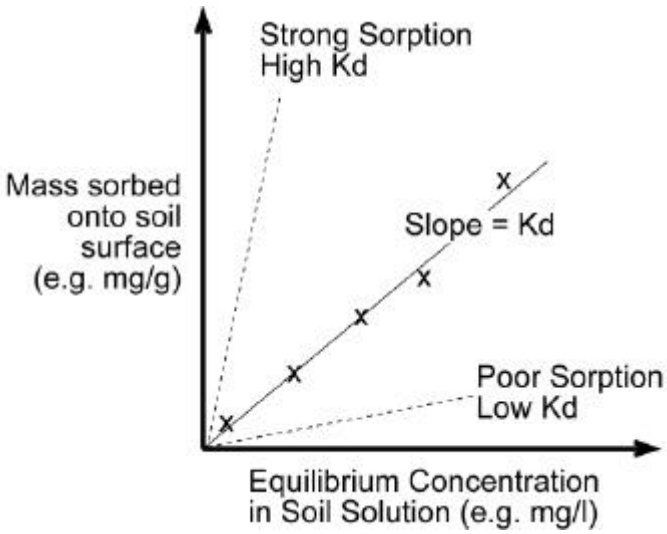

Figure 3.3 Example of a linear Freundlich isotherm illustrating the derivation of the K_d term.

One important indicator of migration potential commonly used in the assessment of pollutant mobility is the soil water distribution coefficient (K_d) (see Figure 3.3). The K_d represents a special case 'linear' Freundlich isotherm (an X,Y plot of the concentration of contaminants such as uranium sorbed onto the soil verses the equilibrium

concentration of the contaminant in associated soil water, where the slope of the resultant line is equal to the K_d), (Domenico and Schwartz, 1990).

Values of the K_d of uranium for various soil pH values are given in Table 3.3. It should be noted that the organic carbon content of a soil also strongly influences pH and the K_d for uranium (soils high in organic carbon having a larger K_d). There should be no differences between the values of K_d for DU and uranium because of their chemical similarity, although the value of the K_d does change with the chemical form of uranium present.

Table 3.3 Values of Kd for various soil pHs (US EPA, 1999). Higher values indicate greater sorption and hence greater retardation. Typical Northern and Central European soils have a pH range of 5 to 7 while those in Mediterranean environments and formed over limestones typically exhibit pH ranges from 7 to 9. Additions of various soil conditioners and fertilizers such as peat, lime or phosphate may significantly effect the behaviour of uranium in soils.

Soil pH	K_d ml/g
3	<1
4	0.4
5	25
6	100
7	63
8	0.4
9	<1
10	<1

3.5 Food

As a component of the natural environment uranium is likely to be present as a trace constituent in all foodstuffs. It may become incorporated into the bulk of the food or may alternatively adhere to the surface of foodstuffs as particulate contamination, with root vegetables often containing higher levels. ATSDR (1999) cites a review of the oral intake of uranium in the US with a typical range of 0.9 to 1.5 µg per day in food and the same range for drinking water, for a total intake of 1.8 to 3.0 µg per day. Harley (1998) cites a review of naturally occurring sources of radioactive contamination undertaken in several European countries and estimates dietary intakes of uranium to range between 0.5 and 2 µg per day. These compare with 0.5 to 3 µg per day in Japan and 0.5 to 0.9 µg per day in the UK which were also cited in Harley (1998). On the basis of this information these authors suggest a worldwide average daily dietary intake of 4 µg although they state that it is often unclear if drinking water exposure has been included in the reviewed dietary assessments. Spencer et al. (1990) measured the total dietary intake of uranium (excluding water and milk) in the strictly controlled diet of four patients partaking in a survey of uranium intake and excretion to be 2 µg/day. This is similar to that quoted by Hamilton (1972) and US Department of Health and Human Services (1990) of 1 to 2 µg/day.

The determination of uranium in a variety of foodstuffs from the USA and UK (Annex-3) indicates that the highest recorded concentrations have been found in shellfish, molluscs and winkles (9.5 to 31 µg/kg), presumably due to the relatively high

concentrations of uranium in seawater. Typical concentrations in staple foods such as bread and fresh vegetables were approximately two orders of magnitude lower (i.e. 2 µg/kg) whereas uranium concentrations in other foods such as rice and meat were in the range of 0.1 to 0.2 µg/kg in meat products.

Concentrations of uranium observed in a variety of tropical staple foods including cassava, matooke, maize and sweet-potato ranged from less than 1 to 11 µg/kg (unpublished data for carefully peeled foods, British Geological Survey). The unusually high phytate (or fibre) content of such diets potentially affects the uptake of uranium from the human gut in a similar manner to the uptake of other minerals (Gibson, 1994). Additionally soil is known to adhere to vegetables, particularly root crops, and the efficiency of washing is a factor that can significantly bias results (particularly as it is not often described in conjunction with the analytical results).

The mean concentration in nine different prepared beverages, including tea and coffee, was found to be 0.98 µg/l (range 0.26 to 1.65 µg/l) and in a series of mineral waters was 9.20 µg/l (Cheng et al., 1993). A survey or 56 randomly sampled bottled mineral waters from Europe by Misund et al. (1999) observed uranium concentrations to range from 0.0104 to 9.45 µg/l.

Like many trace metals, the bioavailability (i.e. gut uptake) of uranium in food may be influenced by the food's phytate (or fibre) content (Gibson, 1994; Golden and Golden, 1981) and the presence of low molecular weight ligands, such as citrate, that may promote uptake.

The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 2000) has estimated that the total annual intake into the human body by adults is 460 µg by ingestion of food and water and 0.59 µg from inhalation.

3.6 Other sources in the human diet

Other sources of uranium in the human diet include dusts and soils both inadvertently and deliberately eaten and uranium derived from cooking and serving wares.

3.6.1 Cooking and serving containers

Landa and Councell (1992) performed leaching studies on the release of uranium from 33 glass items and two ceramic items in which uranium had been used as a colouring agent. The uranium-bearing glasses released a maximum of 30 µg/l uranium. Experiments also showed that when a glazed ceramic plate was kept in contact with a 4% acetic acid solution for 24 hours, the concentration of uranium in the leachate was 3.1 mg/l (Landa and Councell, 1992).

Fortunately, the use of uranium in glazes by artists and potters is now rare, although, as described earlier, uranium powder is still employed in some specialist glazes used principally in the jewellery industry, and this may introduce uranium into the diet through habitual placing of the object into the mouth.

3.6.2 Uranium in dust and soil

Levels of uranium in dust and soil to which individuals and populations may be exposed to through ingestion are varied, and levels depend greatly on the existence of potentially elevated sources. For example baseline levels may be dominated by soil and dust in the

range of less than 1 to 4 mg/kg, as discussed in Chapter 3.4, while levels of uranium in soil and dust intimately associated with areas of man-made contamination or mineralisation may be considerably higher.

Additionally it is important to consider that levels of uranium determined in root crops and drinking water may include contamination from uranium present in soil and dust unless considerable care is taken during sample preparation. Where such material is likely to be present in prepared foods it should be included in the calculation of exposure to uranium.

As discussed later in Chapter 5, three distinct categories of soil/dust ingestion can be defined, and levels of uranium relevant to each category are discussed below.

(1) *Inadvertent ingestion of small quantities of soil and dust.* The high density of uranium and its chemical reactivity may lead to concentration within different particle size/density ranges. This may result in considerably elevated concentrations of uranium with the fine/dense fractions of dusts and soils with a direct impact on levels of uranium ingested as a result of inadvertent exposure.

(2) *Occasional deliberate consumption of soil and dust.* Many young children indulge in this type of exploratory behaviour for a relatively short time. However, unlike inadvertent ingestion, it is generally considered that deliberate consumption is likely to result in ingestion of the bulk soil or dust rather than any particularly mobile size or density fraction (see Figure 3.4).

Figure 3.4 Picture of child eating dirt

(3) *Geophagia*. This term refers to the persistent and purposeful consumption of soil and/or dust, often in relatively large quantities. Historically, it has been recognized as a worldwide phenomena although its prevalence now tends to be associated with traditional communities (both rural and urban) and the poorly nourished. Where geophagia is practised, large quantities of soil (often from a preferred source such as soils associated with termitaria (termite mounds)) may be consumed by children, young adults and pregnant women on a regular basis (Geissler et al., 1997). It is therefore important to determine the concentrations of uranium within preferred sources of soil in such circumstances and any methods or pre-treatment or preparation that might affect levels used in subsequent assessments of exposure.

Toxic metals and uranium in soils are generally considered to be much less bioavailable than similar substances present in food and water. It is therefore important when describing levels of uranium in soils that may be ingested to consider its potential bioavailability in the context of inadvertent and deliberate consumption. The percentage of uranium ingested in soils that might be bioavailable for uptake into the systemic circulation depends upon the physiochemical form of the uranium and the mode by which the soil is ingested. For example, if soil is eaten with foods, particularly those rich in fibre, the bioavailability of uranium may be reduced as a result of sorption by phytate present in such foods. There are also a number of other physiological parameters such as stomach pH, food consumption and stomach/intestinal residence time, which indicate that kinetic constraints of the dissolution of uranium-bearing phases within ingested soil and dust also provide an important control on metal bioavailability. A number of physiologically based extraction tests (e.g. Ruby et al., 1996) have been developed to study the bioavailability of lead and arsenic from mine wastes and other polluted soils; and these methodologies may be extrapolated, with appropriate validation, to investigate the solubility of uranium in contaminated soils.

3.7 Summary

Uranium (U) is a naturally occurring element with an average abundance in the Earth's crust of about 2 mg per kg (range 0.1 to 20 mg per kg). It is more abundant than silver or gold. Typical concentrations in air are low ranging from less than 0.01 to 0.2 ng/m^3. Concentration in air may be influenced by smoking cigarettes and the presence of a range of industrial processes such as mining of uranium ore, gas releases from coal-fired power stations and nuclear fuel manufacturing facilities. Concentrations of uranium in water, food and soil are variable (typically 0.1 to 5 µg/l in water; 0.1 to 2 mg/kg in soil and 0.01 to 2 µg/kg in food) and depend largely on the presence of uranium in associated parent materials (i.e. rocks) or proximity of industries that may introduce uranium into the environment. In all cases, extreme concentrations (up to a factor of about 100 times the typical ranges quoted above) may quite naturally be found in suitable geological environments. Levels in both surface and groundwaters, as well as many bottled mineral waters commonly exceed current WHO drinking water guidelines in many countries.

The 'natural' isotopic ratio of $^{234}U/^{238}U$ may be perturbed by a variety of environmental processes, while the ratio of $^{235}U/^{238}U$ remains largely constant.

4 Industrial, commercial and military applications

Whereas the mining of uranium has taken place since the Middle Ages it is only in the last 100 years, and particularly the last 50 years, that mining has taken place on a large scale. Estimated total production of metallic uranium since recording began in 1920 is estimated at 1.5 million tonnes (British Geological Survey, 2000) although this is only a small fraction of the 10^{14} tonnes estimated to be present in the lithosphere. Recent production figures for a range of acknowledged producers are given in Annex 3.

Uranium by itself has relatively few industrial uses and is commonly removed from raw materials such as fertilizers containing phosphate as a waste product during processing, or because of environmental concerns where it is often removed with other heavy metals such as cadmium.

4.1 Historical uses

Glassware and ceramics Prior to the discovery of radioactivity, uranium was principally used in the colouring of ceramics and glass. Sodium and ammonium diuranates were used as a yellow glaze, although they could also be used at increased concentrations to produce cream, orange, brown, green or black glazes (Chu and Chu, 1975; Conrad, 1973). In glass, uranium is used typically at concentrations in the range of 0.1% to 2% by mass; it produces a fluorescent yellow or light green glow making it possible to easily identify this type of glass. Production of glass containing uranium continued until the middle of the 20th century and was called by various names depending on its colour. For example popular German names include 'Annagelb' for yellow glass and 'Annagruen' for green glass, whereas in the UK and the US such glass is usually referred to as 'Vaseline glass'. Depleted uranium has been and is potentially still used as the basis of a yellow enamel powder used in the manufacture of badges and jewellery (NUREG, 1999).

Dentistry Until the early 1980s uranium and DU were also used in the production of dental porcelains to obtain a natural colour and fluorescence (Sairenji et al., 1982 Noël et al., 1988 and NUREG, 1999). Uranium concentrations of 170 to 13 300 mg/kg were found in 15 types of porcelain powders from one manufacturer (Noël et al., 1988) while Sairenji et al. (1982) determined uranium concentrations of 0.5–24.7 mg/kg in eight types of porcelain powders marketed in Sweden. The US National Regulatory Commission (NRC) standard for uranium in dentures is 500 mg/kg (NUREG, 1999).

Chemical catalysts Uranium has also occasionally been used as a catalyst in certain specialized chemical reactions and in photographic films. For example the oil and gas industry continue to use nickel-U material (10%–65% DU) in relatively large quantities as a catalyst. The nuclear industry has also used small quantities of DU to chemically absorb gaseous tritium for the purposes of transportation, and in the production of plutonium. In the period between the discovery of radioactivity and nuclear fission, uranium ore (typically as pitchblende) was mined principally for the extraction of radium (a decay product of uranium) which was used for medical purposes and the preparation of luminous paints.

Nuclear weapons and power production From 1938, when nuclear fission was first identified, uranium mining expanded due to the need for enriched uranium as a fuel for use in nuclear power stations and for nuclear weapons.

The first step in the process is mining; historically, this has involved subterranean or open-pit ore rock removal and now also involves liquid in-situ leaching of unconsolidated deposits (the latter method has been used extensively in the Czech Republic and the former East Germany). The next steps in the production of uranium fuel, aimed at concentrating the uranium, are usually carried out near the mine to save on transportation costs. Methods used depend on the nature of the ore, and may involve mechanical procedures such as crushing, screening and flotation, followed by acid or alkaline leaching, solvent extraction, or ion exchange and eventual precipitation. The product of these concentration steps contains perhaps 40%–70% uranium by weight and is generally shipped to a central processing plant to be further refined. This purification is either by digestion with nitric acid and extraction of the resulting uranyl nitrate into an organic solvent, or by conversion to UF_6 and fractional distillation of that volatile compound. At this stage, all naturally occurring radioactive progeny have been removed from the uranium which is considered to be chemically pure. However, due to the inherent radioactive nature of uranium, following this purification step the presence of other radioactive elements within the uranium decay chains will start to increase.

Figure 4.1 Nuclear fuel rods being loaded.

Whilst some nuclear reactor types such as the CANDU and Magnox use natural uranium as fuel, others require enriched uranium (see Figures 4.1 and 4.2). If such ^{235}U enrichment is required, the purified uranium in an appropriate chemical form is transferred to an isotope separation plant (see Annex 1). Isotope separation of ^{238}U, ^{235}U and ^{234}U may be achieved by a number of processes including gaseous diffusion, centrifugal or laser enrichment. The enriched uranium is then processed and fabricated into appropriate forms for use in nuclear reactors. The by-product of this enrichment process is DU, often in the form of UF_6, as has been discussed above.

Figure 4.2 Uranium fuel assembly used in a pressurized water type of nuclear reactor.

4.2 Current applications

The four major uses of DU at the present time include radiation shielding, counterbalance weights and military armour and ammunition.

Radiation Shielding The density of DU makes it a suitable material for the shielding of gamma radiation. For this reason, uranium has been used extensively in the medical, research and transport sectors (NUREG, 1999) as radiation beam collimators and containers to transport nuclear sources. Thus, DU has often been used as a shield for radioactive sources in tele-therapy units (figure 4.3) used in the treatment of cancer and in linear accelerators. Typical quantities of DU used in such equipment range from tens to hundreds of kilograms.

Figure 4.3 Cobalt-60 tele-therapy unit

Current developments in waste management have also employed DU as a shielding material. For example, casks used for holding spent fuel in the nuclear power industry

have been constructed by combining DU with concrete (e.g. DUCRETE™ (www.starmet.com, 2001)). This achieves a significant increase in gamma-radiation shielding with thinner shield walls and much lighter weight casks than traditional storage casks. Appreciable quantities (i.e. thousands of kilograms) of DU have been used as shielding material in casks used for the transport of radioactive sources such as those used in the medical and engineering industries.

Counterbalance weights and ballast Vessels and equipment, such as boats and satellites require a large amount of weight to be carried in the form of ballast. The high density and relative availability of DU make it a potentially suitable material for this use by fulfilling the weight requirements while minimising the amount of space taken up by the ballast material.

Industries in which the use of DU has been cited for these purposes include the aircraft industry, military aerospace industry, and the oil and gas exploration and production industry (NUREG, 1999). It has also been suggested that it has been used in the manufacture of keels for yachts (Priest, 2001).

Wide-bodied aircraft such as the McDonnell-Douglas DC–10, Lockheed L–1011 and Boeing B–747 require heavy counterweights on control surfaces (usually, but not exclusively, the aileron) to enable proper flight control. These areas often have low surface clearance with insufficient space available for low density materials. Depleted uranium, lead and tungsten have all been used for counterweights due to their high density. A typical wide-bodied aeroplane such as the Boeing 747 (‘jumbo jet’) requires up to 1500 kg of counterweights (NUREG 1999). Not all of this material is DU however, and DU is now being replaced retrospectively with tungsten.

The plane which crashed into a block of flats in Amsterdam in 1992 carried 282 kg of DU counterweights and the Korean Boeing–747 which crashed near Stansted airport in England in January 2000 was estimated to be carrying approximately 425 kg of DU counterweights (Uijt de Haag et al., 2000). When used in aircraft DU is usually either plated (Ni and/or Cd) and painted or sheathed in an aluminium alloy. No data appears to have been generally issued on DU released from other civilian or military air crashes.

The Nuclear Regulatory Commission’s report (NUREG, 1999) comments that ‘It is unknown how many DU counterweights are currently installed in aircraft. It is estimated that approximately 15,000 weights may be associated with the Boeing–747 fleet (based on 550, Boeing–747 aircraft produced between 1968 and 1981 and spare parts) (Gallacher, 1994). However, the number of aircraft that contain DU counterweights is rapidly decreasing. Rather than refurbishing the DU (during maintenance operations), tungsten counterweights are used as a replacement.’

Similarly DU may be used as weights for geophysical exploration tools, as rotor tips in military helicopters (AEPI, 1995) and experimentally in many different forms of engineering applications such as counterweights in various engines (particularly during the 1980s when its commercial use was seen as a way of reducing stockpiles of such material). There is little evidence that the use of DU has continued into the production of any of these experimental applications, although the pressure to find appropriate commercial outlets for DU is undoubtedly still present.

Military Uses Depleted uranium (and associated uranium-titanium alloys [typically 0.75 wt % Ti]) have been employed by the military as a component of heavy tank armour and armour-piercing munitions (e.g. AEPI, 1995; Rao and Balakrishna Bhat, 1997). The high density and high melting point of DU make it an extremely effective material for neutralising anti-tank weapons. The DU is inserted into a sleeve attached to the regular steel armour, thus isolating the DU from the tank crew and those in contact with the external surfaces of the tank while utilising its protective characteristics.

The high density of DU and its various alloys also makes it suitable material for use in armour piercing munitions and to penetrate hardened targets. Depleted uranium also has advantages over similarly dense alternative materials, such as tungsten, in that it is:

- relatively inexpensive.
- non-brittle unlike tungsten.
- at the high temperatures and pressures involved during the impact of such weapons DU has been found to adiabatically shear (e.g. self sharpen) giving increased penetration.

There are four main types of DU munitions acknowledged as being currently in circulation, the 25 mm, 30 mm, 105 mm and 120 mm anti-tank rounds, although small amounts of DU have been used in the manufacture of other munitions (AEPI, 1995). A 30 mm round fired from ground attack aircraft contains a 0.27 kg DU penetrator. Heavy tanks fire 120 mm rounds containing a 4.85 kg DU penetrator. A 30 mm cannon as used in a ground attack aircraft can fire up to 4200 rounds per minute (although such munitions are typically only fired in relatively short bursts of say 120 to 195 rounds or two to three second bursts (CHPPM, 2000)) and a considerable mass of DU could be distributed in an attack zone, particularly if the attack is performed by a number of aircraft. Some of this DU may be released as particles should the penetrator impact on a sufficiently hard target, but the entire round may stay intact with only surface scaring even when impacting with relatively hard targets such as concrete.

DU counterweights may also be used in missiles (AEPI, 1995), warheads and military aircraft. For example some land-attack cruise missiles (Zajic, 1999; personal web site) and other strategic missile systems, such as the trident ballistic missile system, have been reported as using DU as counterweights, although this has not been substantiated by official sources. One use of DU within missile warheads might be to aid ground penetration. For example the now obsolete Pershing D–38 earth-penetrating missile carried an 80 lb (36.3 kg) DU penetrator. In one case recorded at a missile testing range, the warhead used in this type of missile penetrated the earth to a depth of approximately 200 ft (61 m) (Van Etten and Purtymun, 1994).

Data relating to the use of DU in a wide range of weapons systems produced outside of the USA and NATO are generally lacking, although DU weapons are widely available. For example they are thought to be in the possession of at least nine countries (Harley et al., 1999b). Numerical estimates of the relative proportion of DU munitions amongst arsenals of various countries are equally difficult to obtain, although it has been reported in AEPI, (1995) that US contractors had produced more than 55 million DU penetrators for small-calibre munitions (principally of the 30 mm type) and 1.6 million penetrators for tank ammunition. The potential use of DU in other forms of armaments to enhance hard target penetration is briefly reviewed with respect to the recent conflict in Serbia by Liolios, (2000).

Depleted uranium weapons are regarded as conventional weapons by NATO and are not subject to restriction. However, the extent of their use has, in the past, been unclear. It is now accepted and acknowledged that DU weaponry was employed by US forces in the Gulf War. A letter from Lord Robertson, Secretary General of NATO to the Secretary General of the United Nations, Mr Kofi Annan dated 7th February 2000 also confirmed the use of DU munitions during the Kosovo conflict. The use of DU weapons at various firing ranges associated with the development and proving of munitions and armour (e.g. at the Yuma and Aberdeen proving grounds in the USA (AEPI, 1995)) has also been widely acknowledged.

4.3 Summary

Depleted uranium is used in a variety of products. It major uses include radiation shielding, counterbalance weights and ballast, military munitions and armour. The use of DU has been acknowledged in a number of military conflicts including the Gulf War, Bosnia and Kosovo and in various military firing ranges. Use in such conflicts has clearly demonstrated the military benefits of DU.

5 Factors influencing routes of intake and exposure

5.1 Introduction

Exposure to uranium occurs primarily by inhalation, ingestion or external irradiation, while secondary exposure may result from dermal absorption of particularly mobile forms of uranium. This is the case with many chemicals naturally present in our environment. Because of the identical chemical and biological behaviour of naturally occurring uranium and DU, it is possible to draw on our knowledge of exposure to other naturally occurring heavy metals (e.g. lead, arsenic etc.) in attempting to define the relative importance of exposure pathways.

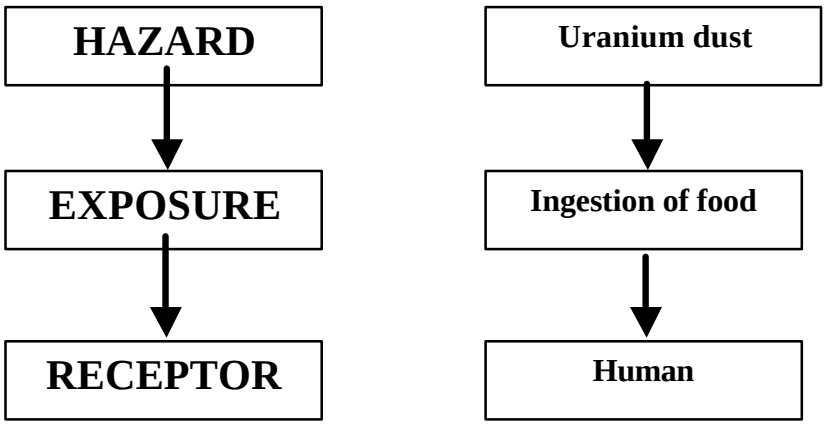

Figure 5.1 Example of a simplified pollutant linkage, for human exposure to uranium. In practice, however, such a linkage only represents one of a much larger number of potential linkages whose relative importance within a specific incident may have to be investigated (Ferguson et al., 1998).

Risk assessment models in which the potential health detriment associated with exposure to a given chemical is calculated are reliant on the definition of a 'pollutant linkage' such as that shown in Figure 5.1. Several different such linkages may occur at any one site associated with the presence of various hazards, environmental pathways and receptors such as humans, animals, groundwater, and buildings.

Exposure by external irradiation is not explicitly dealt with in this chapter, but is discussed in the following chapters where health risks associated with radiological issues are reviewed.

Studies on exposure to uranium have focussed on those chemical forms and modes of exposure of direct relevance to the nuclear industry. In comparison relatively few studies have been conducted on DU. It should be recognized that the extensive database on uranium will be of value in assessing, in many cases, the implications of exposure to DU since they have the same chemical and biological behaviour.

Scenarios used in previous studies on exposure of humans to DU in military situations have focussed on the likely form of compounds and exposure routes by which personnel or the local population may be exposed in the hours or days immediately following the use of DU in munitions and armour. In order to undertake longer term assessments a more in-depth analysis is required of the physiochemical transformations that control

the weathering and transport of uranium through various environmental pathways and compartments by which human exposure may occur. This more detailed analysis should be structured to evaluate the speciation and bioavailability of uranium under the prevailing local environmental conditions. It is well established that the total metal concentration in an environmental medium is an unreliable guide to hazard quantification, as different forms of a metal can have substantially different bioavailabilities (Thornton, 1996; Plant et al., 1996; Elless et al, 1997).

It is noted that regional or local exposure scenarios are often structured to estimate the risk of two different types of health detriment:

(i) population detriment.

(ii) maximum individual detriment.

Population detriment is a traditional public health measure that estimates the number of cases of a particular outcome or disease in an exposed population attributable to a specific source of contamination. The maximum individual detriment relates to the individual who suffers the largest incremental risk due to a particular scenario. The relative importance of different sources and pathways is likely to differ depending on whether population detriment or maximum individual detriment is being calculated. In the case of population detriment it is particularly important that not only is the average exposure estimated, but also that its spatial distribution, and the relative importance of various exposure routes amongst the local population are defined.

Children are not small adults and their exposure may differ from an adult in many ways. Unfortunately, despite their obvious importance little definitive data exists concerning how their uranium exposure differs from that of adults (ATSDR, 1999).

Examples that follow in this chapter illustrate the relative importance of various exposure routes when assessing exposure within the context of population and maximum individual detriment and suggests factors that need to be considered when undertaking more site-specific studies. Such treatment is important in understanding the relative proportion of total exposure that may be allocated to a specific pathway during the assessment of human health risks (WHO, 1994), particularly where substance exposure pathways may exist.

The potential relationships between exposure to uranium and DU and specific forms of health detriment are discussed in Chapters 8 and 9.

5.2 Exposure via inhalation

The pyrophoric nature of uranium is considered to be of special relevance to the assessment of human exposure due to the production of dust containing mixed oxides of uranium. This scenario is especially likely to occur immediately following the use of DU munitions or where DU may be accidentally or deliberately heated (e.g. in the welding of reclaimed battlefield scrap). Its relevance to aviation accidents remains a subject of debate.

The oxides considered to be of principal concern are uranium dioxide (UO_2), uranium trioxide (UO_3) and triuranium octaoxide (U_3O_8) (Harley et al., 1999b; CHPPM, 2000). The size distribution, morphology and exact chemical composition of each particle released during the use of penetrators and armour is highly variable (e.g. Patrick and

Cornette, 1977). Moreover, they may be chemically and mineralogically altered by weathering either following the impact with the target, or their initial release into the environment (e.g. uranium oxides may become hydrated, chemically reacting with other elements and species present in the soil and/or target such as silica, iron, phosphate and vanadium; Patrick and Cornette, 1977 Ebinger et al., 1990). The relative absorption behaviour (lung to blood) for uranium after the inhalation of different chemical forms is given in Table 5.1.

Table 5.1 Absorption types for uranium compounds (ICRP-71, 1995b). Note. Some preparations of U_3O_8 may also be considered as Type S.

Type	Typical compounds
F	UF_6, UO_2F, $UO_2(NO_3)_2$
M	UO_3, UF_4, UCl_4, U_3O_8
S	UO_2

F fast; M moderate; S slow

The biological solubility and bioavailability of the oxides U_3O_8 and UO_2 are relatively low (Type M and S), compared to other forms of uranium to which workers in the nuclear industry may be exposed (e.g. UO_3). This has been reported by Jette (1990) through lung-solubility analysis of particles (< 10 µm in diameter) produced immediately following the impact of DU munitions. Some of these particles may, however, be removed by mucociliary transport into the gastro-intestinal (GI) tract, and reach the intestine where appropriate gut uptake factors need to be considered. For more information on deposition and clearance from the respiratory tract the reader is referred to Annex 4.

There is a lack of detailed mineralogical and chemical analysis of material liberated under battlefield conditions (or other conditions in which uranium dust has been liberated following combustion) and subsequently weathered. This limits the accuracy of exposure assessment. The availability of material specific information would inevitably increase the confidence of such assessments. The lack of such data has also highlighted a significant knowledge gap in performing detailed exposure assessments for scenarios involving military personnel (CHPPM, 2000).

The level of human exposure to dusts and aerosols derived from the impact of possible uncontrolled oxidation of DU is a function of the proximity of the human subject to the source of contamination, the degree to which uranium has become physically and chemically dispersed into the local environment and the particle sizes and density of the dusts produced.

Dust particles with a diameter less than 10 µm activity mean aerodynamic diameter (AMAD) are generally assumed to be respirable, larger particles being trapped in the upper extra-thoracic part of the respiratory tract from where they are either expectorated or swallowed. In the case of radiological exposure the respiratory tract is both a target organ and a route of entry to the systemic circulation. There are two lung models in current use; the ICRP Publication 30 lung model (ICRP-30, 1979) and the ICRP Publication 66 Human Respiratory Tract Model (ICRP-66, 1994a). The latter is used

internationally and is embodied in European Legislation and the Basic Safety Standards (1996). The principals underpinning these models are discussed in more detail in Annex 4. While these models are generally used for radiological exposure, their more detailed treatment of exposure via inhalation offers considerable scope in improving the assessment of chemical exposure via this potentially important route (e.g. CHPPM, 2000).

For the purposes of generalized environmental exposure modelling, approximately 73% of the dust in air is considered to be respirable and the concentration of contaminants in the dust is assumed to be equal to the concentrations in the soil (Muir et al., 1995). Local variations in such factors are high and locally determined factors should, if possible, be used during more extended assessments. The ICRP Human Respiratory Tract Model (ICRP-66, 1994a) offers a more comprehensive treatment in this respect, calculating deposition throughout the entire respiratory tract for a given particle size distribution.

The US EPA estimates that a typical exposure to uranium in air results in a total uranium intake estimated to be from 2 to 20 nanograms ^{238}U/day (US EPA, 2000).

Individuals likely to suffer enhanced exposure to uranium via inhalation are mainly those living or working in close proximity to primary sources of uranium dust such as mine tailings or areas in which DU has been used for various industrial or military activities. Agricultural workers, particularly those involved in cultivation practices such as vines that require the operators nose and mouth to be in close proximity to the soil's surface, or those working with heavy dust-generating machinery, are also likely to be exposed to enhanced levels of uranium containing re-suspended dusts. Limited data are available describing possible re-suspension of DU particles once deposited on soil. Data presented in CHPPM (2000) and cited to have been compiled in a short letter based report by Beyeler, suggests re-suspension factors for DU oxides to range from 3.3×10^{-8} (no mechanical disturbance) to 1.9×10^{-4} (vigorous work activity).

5.3 Exposure via ingestion

The impact on health after ingestion depends on the amount of bioavailable uranium compounds present in ingested material, which in turn is dependent on the concentration of bioavailable uranium in the environment. In most scenarios in which uranium has been released into the environment, it is assumed that it could contaminate the soil, or migrate to surface or groundwater. Using these as sources for drinking water, agricultural land and recreational purposes can lead to human exposure either directly or through the food chain. Therefore, information on the possible concentrations of soluble uranium compounds in these media, in the days and years following the contamination incident is essential. In this context, DU and even anthropogenically redistributed uranium has entered the environment only relatively recently. This, together with a lack of reliable field data where DU has been used for military purposes, significantly limits our knowledge of how, and on what time scale, such materials may become incorporated into the human food chain. This places a high degree of importance on the extrapolation of studies of uranium in natural systems. However, during any such extrapolation it is important to consider likely differences in the bioavailability of the specific chemical forms of DU encountered, compared with those of substances from which extrapolations are to be made.

Because of the diversity of the human food chain it is impossible to suggest a suitably generic dietary balance to cover all populations, scenarios and cultures. WHO has, however, suggested a typical diet to aid in the comparison of exposure; these data are identified in Annex 3.

Like many trace metals the bioavailability of uranium in food, water and soils, affects uptake into the body through the gastrointestinal tract. For example a high-phytate content in the diet may reduce uptake (Gibson, 1994; Golden and Golden, 1981) whereas the presence of lower molecular weight ligands, such as citrate, may promote absorption. However, under the near-neutral conditions of the upper intestine uranium, unlike many heavy metals, is likely to form relatively stable oxy-anion complexes (e.g. Brookins, 1988) that inhibit complexation with organic chelators. Therefore, although the bioavailability of uranium from foodstuffs is an important variable in the exposure assessment process, its extrapolation from the results of other studies on other heavy metals such as Zn, Pb, etc should be avoided. Although unable to determine uptake factors, Spencer et al., (1990) investigated intake and excretion patterns of naturally occurring uranium and calcium in humans. These studies confirmed the similar behaviour of uranium isotopes in the body and confirmed significant elimination of the total dietary intake via faecal excretion (urinary excretion being approximately 2% of total excretion (e.g. Leggett and Harrison, 1995).

ATSDR (1999) considers ingestion to be the major source of environmental exposure to uranium. Typical world-wide dietary intake is estimated at between 0.9 and 4.5 µg/day with an average of 1.5 µg/day (Linsalata, 1994). This is consistent with dietary intakes estimated from the excretion of uranium in urine amongst a group of 12 subjects from Utah (Singh et al, 1990). However, ATSDR (1999) cites a paper describing dietary intake as high as 2.9 to 4.5 mg/day for individuals living near a uranium mine (Yamamoto et al, 1971).

Various gut uptake factors and bioavailabilities have been suggested for ingested uranium and these are discussed in more detail in Chapter 7 with specific reference to the ingestion of water, foodstuff and soil/dust.

5.3.1 Staple foods

There are few data on the concentration of uranium in staple foods from an environment that could be considered to be contaminated with DU. Whereas within the context of the nuclear industry extensive compilations of concentration factors (CF) and concentration ratios (CR) exist in the literature (e.g. ICRP-29, 1978); IAEA, 1982, 1994; see Table-5.2). These have been extensively used in radiological assessments to estimate the likely concentration of a given radionuclide (such as uranium) in a foodstuff from the concentration of that radionuclide in a given substrate (eg. water, food or soil) to which the organism is exposed. In addition to data presented for generalized environments, a significant amount of data has also been collected for more varied local diets such as the Aboriginal homelands of northern Australia (Martin et al., 1998)

Cereals, root vegetables and fruits

Studies with grasses and wheat have shown that in broad terms the majority of uranium appears to be accumulated firstly by the roots, shoot and then seeds (Jain and Aery, 1997). This observation is consistent with observations in other plants that form the basis of some phyto-remediation methods for the removal of uranium from polluted waters and in studies of sites of uranium mineralisation (Basham et al., 1989). In plants used for the purposes of phyto-remediation it has been found that accumulation into

shoots and seeds can be minimized through harvesting during specific periods of growth (Dushenkov et al., 1997).

Consideration of the effects of DU on ecosystem function at military proving grounds highlighted a paucity of data on the impact of DU on the function of non-arable plants. Meyer and McLendon (1997) carried out studies of three species of grasses, *Buchloe dactyloids* (buffalograss), *Schizachyrium scoparium* (little bluestem) and *Aristida puprurea* (purple threeawn). These species were considered to be typical of the vegetation present in the arid and semi-arid conditions of most areas where DU has been tested and deployed. The study concluded that DU was relatively nontoxic to these grasses with no observed toxicity occurring at soil concentrations below 25 000 mg/kg-U. In contrast, Jain and Aery (1997) reported that contamination of agricultural land with uranium may have a detrimental effect on the productivity of wheat. (There is a marked reduction in a number of growth parameters, that included a decrease in seedhead number (spikes), seed number and seed weight (no seed head being produced at an additive level of 625 µg/l U in irrigation water).

Table 5.2 Examples of typical concentration factors for uranium (ICRP-29, 1978; IAEA, 1982, 1994).

Concentration Factor						
Leafy Vegetables*		**Root Vegetables***		**Fruits***		
Minimum	Maximum	Minimum	Maximum	minimum	maximum	
1.2×10^{-4}	1.0×10^{-2}	2.0×10^{-4}	3.0×10^{-2}	4.0×10^{-4}	4	

Grains and Cereals**	
Minimum	Maximum
2.0×10^{-4}	1.3×10^{-3}

* As Bq/g wet plant/Bq/g dry soil
** As Bq/g dry plant and soil

Note: Such factors vary with soil pH, organic carbon content, uranium content etc. (ATSDR, 1999) and site specific data should be applied where possible

To facilitate the assessment of exposure from the ingestion of foodstuffs WHO and various other international and national authorities have compiled a set of typical food consumption patterns. Examples of typical patterns are given in Annex 3. It is emphasized that these data reflect average consumption and that it is important during the assessment of dietary exposure that specific local consumption practices are investigated. Such practices may include the fermentation of food and an unusually strong dietary dependence on one or two specific foods.

Animal products

Like many other heavy metals uranium may become incorporated into the human food chain. The majority of bioaccumulation studies to date have been performed on natural rather than DU. This is an important consideration as the kinetics of bioaccumulation of DU into the food chain will be subject to a lag period, the magnitude of which will be governed by the weatherability and bioavailability of metallic uranium and any mixed oxides. Thomas and Gates (1999) investigated the presence of radionuclides including uranium in the lichen-caribou-human food chain. The observed concentration of

uranium in Caribou muscle were observed to range from 1% to 16% of that observed in lichen from the same environment.

Clulow et al. (1998) investigated and determined the concentration of uranium in water, sediments and fish from lakes near the uranium mining and milling operations at Elliot Lake, Ontario and from control lakes in adjacent non-industrialized watersheds. Bio-concentration of uranium was observed to occur from water, to gut material, to bone in lake trout and white fish. A systematic relationship was not established between sediment levels and those observed in fish tissues. The authors calculated a potential intake of uranium (from fish derived from industrialized areas) of 2.3 mg/annum.

Data on the concentrations of uranium and DU in exposed or unexposed farm animals are scarce. In relation to the assessment of exposure to contamination from anthropogenic sources, such as armed conflict, deposition of mostly insoluble uranium compounds is probably most relevant where it occurs on the soil or on vegetation and is taken up by grazing animals. However, it may also be necessary to investigate the potential for uptake from plants and other sources of animal feed as these uranium oxides become weathered and enter the soil pore water.

Table 5.3 Examples of bioconcentration factors for uranium transfer into animal products (ICRP-29, 1978; IAEA, 1982). Note: Such factors vary with environmental pH, uranium content etc. (ATSDR, 1999) and site-specific data should be applied where possible).

a) uptake from water into fish tissue

Environment	**Biota**	**Concentration Factor**	**Range**
Bq/kg wet weight fish per Bq/l water			
Fresh water	Fish	0.5	0.3 to 50

b) soil to plant transfer factors for all types of pasture, grass, browse and forage vegetation

Concentration Factors (Bq/kg dry plant per Bq/kg dry soil)	
Minimum	Maximum
1.0×10^{-5}	0.2

c) feed to milk transfer factors, fraction of daily intake taken into milk

Transfer Coefficient	
Minimum	Maximum
7.3×10^{-5}	6.1×10^{-4}

Typical soil ingestion values for cattle are estimated to be about 500 g soil per animal per day. Assuming a body weight of 400 kg this corresponds to about 1.25 g/kg of body weight per day. Data on soil intake for sheep and pigs are extremely limited (VHI, 1997) although values of 60 g and 500 g/day for these animals, respectively, have been extrapolated on a basis of body weight. Soil ingestion by goats can be considered to be negligible, as they are very selective grazers typically concentrating on the tops of grass leaves (although ingestion of dust deposited on these leaves could be considered). The uranium content measured in tissues of cattle herds grazing in pastures next to the Rocky Flats Plant in Colorado USA were slightly higher than in other cattle, reflecting

possible contamination from this source (Smith and Black, 1975). Concentrations of uranium in muscle from cattle exposed to elevated levels of forage (440 µg/kg) were similar to controls. Elevated levels of uranium were observed in liver (4 ×), kidney (4 ×) and bone (femur 12 ×) during studies based in New Mexico (Lapham et al, 1989). These results were interpreted as indicating that in cattle the muscle does not concentrate uranium (Lapham et al., 1989).

WHO (1998c) suggests an estimated average daily meat consumption of about 150 g per person per day, although, of course, this may be modified according to local dietary habits and socio-economic factors. For example higher values could be appropriate if a critical group approach were adopted in exposure assessment (see Annex 3).

5.3.2 Drinking water

As discussed earlier in Chapter 3 drinking water can contain naturally occurring uranium over a wide range of concentrations. The potential for additional exposure to uranium through the ingestion of drinking water in environments that are affected by the presence of DU is controlled by a variety of physical, chemical and hydrogeological factors similar to those that control exposure to other sources of chemical contamination. These include the proximity of the DU contamination to potential water resources (that may be currently used or developed in the future), the nature of the water resource (e.g. piped, blended, treated supply versus private water supply; groundwater versus surface water), the local geochemical environment that may promote or inhibit the weathering and transport of DU, physical dispersion during fluid migration and the magnitude of contamination by DU. Due to the complexity of these factors, it is important to undertake a site-specific assessment of the likelihood of DU entering drinking water where such contamination is known to be present.

By necessity, such an assessment is likely to be undertaken in an iterative manner modified in terms of scale and rigour as the actual likelihood and potential impacts of exposure to DU in, or from, the affected drinking-water resource is established. Similar investigations may also be required in areas in which potentially contaminated soils and machinery may be disposed of, or stockpiled. It may also be necessary, under such circumstances, to undertake a review of which water resources are currently used by members of the local population and the potential of water development activities changing this pattern in the future.

The first step in assessing exposure to uranium through drinking water must be the chemical determination of its concentration in water consumed by individuals. This may include monitoring the point of public/household supply, or private supply, or from potential sources such as springs used during camping excursions. It is essential during the collection of samples to be aware of various practices associated with the operation of the supply, such as the amount of water typically run-off prior to consumption, and the use of private water supplies.

The WHO recommends an average water consumption of two litres/day to be used in calculating human exposure to contaminants in drinking water. In practice this consumption rate may vary depending upon age, activity level and the consumption of water in the form of beverages and soft drinks. Where exposure through drinking water is suspected as being an important route of exposure it is recommended that these factors be carefully investigated.

It is unlikely that significant amounts of the more insoluble tetravalent species of uranium will be present in potable water supplies, except where it is carried on particulate material. In such cases the amount of material likely to be ingested should be estimated by sequential filtration procedures and included in subsequent exposure calculations using the appropriate gut uptake factor.

While it is not the objective of this report to specifically review potential remedial scenarios, it should be noted that many treatment methodologies exist for the removal of uranium from water (e.g. Varani et al., 1987) although few have been used to produce potable water.

5.3.3 Soil and dust

How much soil is ingested is a deceptively simple but highly relevant question that has led to a substantial discussion in the literature (e.g. Simon, 1998). Some studies considered to be influential (e.g. Kimborough et al., 1984) have been criticized for using ultraconservative soil ingestion rates with little empirical support (Paustenbach et al., 1986; Gough, 1991). Recent research has been dominated by mass-balance studies of ‘conservative tracer elements’, i.e. chemical elements that are present in soil but which are not significantly absorbed by passage through the gut (van Wijnen et al., 1990; Calabrese, 1989; Davies, 1990).

The ingestion of soil and/or dust occurs both within and outside the household environment and it is important to establish, where possible, the concentration of uranium in both environments. Alternatively, a general relationship between indoor dust and outdoor soil contaminant concentrations may have to be assumed.

For example, Keenan et al. (1989) and Murphy et al. (1989) have reported the proportion of locally derived soil particles in indoor dust to be in the order of 75% to 100%. This estimate was based mainly on a study of land contamination around a series of smelters. However, some studies such as those by Franzen et al. (1988) and Steele et al. (1990) indicated that in mining communities the proportion was much less than this (typically indoor concentrations were 14% to 15% of soil concentrations). This difference was considered to be due to the surface properties and moisture content of smelter particles which allowed them to adhere readily to shoes, clothing and pets, and thus to be tracked indoors more easily than other particles.

For uranium and DU, which may be derived from a variety of sources, it is impossible to suggest one value for the proportion of outdoor-derived dust in the indoor environment. Given the large range of observations and the lack of relevant information, the recommendation of a value of 75% (Keenan et al. 1989) would seem appropriate, even though this is almost certainly cautious in many cases.

Three distinct categories of soil ingestion may be considered, and these are discussed below along with suggested quantities of ingested material. From these examples it can be clearly seen that the amount of soil and/or dust ingested varies greatly, and that it is essential that the likely magnitude of geophagic activity be assessed in potentially exposed populations. During surveys of geophagic behaviour, it is essential that great care is exercised to prevent false negative results being obtained due to cultural taboos associated with this practice (e.g. being considered to be improper or of lower social status).

1. *Inadvertent ingestion of small quantities of soil and dust.*

It is likely that all members of an exposed population will have intakes by this route although exposure is likely to be greatest for children under seven years old. Sources of soil and dust are likely to be derived from both outdoors and indoors and the relative magnitude of exposure will depend greatly on the habits and behaviour of an individual. Despite the wide number of studies considerable uncertainties still exist in data relating to this activity (e.g. Simon, 1998). This is in part due to the difficulty in the methodological use of tracers to estimate such quantities, and also the highly individualistic nature of exposure. Inadvertent soil ingestion rates for studies performed in the USA are given in Tables 5.4 and 5.5 and illustrate the uncertainties and typical values determined in such studies. It should be noted that these values are generally slightly higher than those suggested by WHO (20 mg/day, Annex 3). The use of data based on mass consumed may be inappropriate for such a dense material as DU and its oxides and correction factors accounting for differences in density may need to be applied.

Table 5.4 Examples of soil ingestion estimates for children in the USA (mg/day) derived from tracer studies (note: negative values indicate error in mass balance).

Tracer	(Davies, 1990) N = 101			(Calabrese, 1989) N = 64		
Element	Mean	Median	Range	Mean	Median	Range
Al	39	25	-279 to 904	153	29	-75 to 6837
Si	82	59	-404 to 535	154	40	-53 to 5549
Ti	246	81	-5821 to 6182	218	55	-3069 to 6707

Table 5.5 Soil ingestion rates for adults (mg/day) derived from tracer experiments (N = 6) (Calabrese et al., 1990).

Tracer	Al	Si	Ti
Mean	77	5	377
St Dev	65	55	517
Median	57	0.5	211

2. *Occasional deliberate consumption of soil and dust.*

Most young children indulge in this type of exploratory behaviour for a relatively short time, although there is hardly any quantitative information on the amount of soil deliberately ingested during these activities. This is due in part to the difficulty in separating the occasional consumption of soil from the habitual practice of geophagy. For the group of 64 US children studied by Calabrese et al. (1991) the median soil ingestion rate ranged from 9 to 96 mg/day according to tracer, but one child (a three and a half year old girl) ingested much greater quantities (up to 13.6 g/day). Earlier estimates of the amount of soil deliberately ingested as 5g/day

(US EPA, 1984) and 10 g/day (US EPA, 1989) have generally been based purely on 'judgement'.

3. *Geophagia.*

The term geophagia refers to the persistent and purposeful consumption of soil and/or dust, often in relatively large quantities. It is typically associated with children and pregnant females who are commonly subject to nutrient deficiencies. Geophagia should be considered as being distinct from pica, which also relates to the mouthing or eating of unusual objects, and should not be considered as only occurring in rural environments. Geophagia has been studied in both the United Kingdom and North America within the wider context of pica (e.g. Cooper, 1957; Bicknell, 1975; Barltrop, 1966; Morgan et al., 1988). However as Lacey (1990) comments 'The body of literature on pica is so fragmented that it is difficult to find a precise summary of the knowns and unknowns about the condition. There is little consistency in defining pica, classifying substances ingested, identifying key characteristics of practitioners, recommending treatment or projecting outcomes'. The fragmentary nature of this information therefore makes it extremely difficult to calculate exposure of populations or individuals via this route. The situation elsewhere is even more complicated, particularly in tribal cultures where geophagy is commonly practised. For example studies by Geissler et al. (1998) indicated that a large proportion of male and female children in Kenya practise geophagy up to the age of 16, with an average soil consumption rate of 25 g/day.

5.4 Dermal contact

Exposure to uranium through dermal contact in non-occupationally exposed populations is poorly studied. This is principally due to the relatively low abundance of uranium in the natural environment compared to the concentrations encountered in the workplace.

However, some uranium compounds (e.g. uranyl nitrate, uranyl fluoride, uranium trioxide) have been demonstrated to be chemically toxic to animals through dermal exposure (e.g. Orcutt, 1949, DeRey et al., 1983, Ubios et al., 1997 and Lopez et al., 2000), whereas the radiation dose received through dermal contact is minimal (AEPI, 1995). Potential health effects associated with such exposure are discussed in more detail in Chapter 8.

In the natural environment, dermal exposure to the more soluble forms of uranium is significantly less likely than in an occupational context, as uranium from these compounds is likely to have become translocated and dispersed from soils and sediments into surface waters and groundwater. For less soluble compounds, such as uranium phosphates associated with fertilizers and uranium oxides associated with DU, exposure through dermal contact is likely to result in a greater degree of hand to mouth transfer (see ingestion of soil above).

Human exposure through dermal contact can also result in contaminants and poisons entering the systemic circulation by physical transport following traumatic damage to the skin such as can be encountered in military situations in which DU has entered the body, or perhaps through abrasion of the skin, as has been suggested by the studies of Podoconiosis (Price, 1990) and endemic Kaposi's sarcoma (Ziegler, 1993). In general, dermal contact as a route of uptake of uranium into the body is considered to be unimportant.

5.5 Workplace exposure

Conditions in the workplace where humans are potentially exposed are extremely variable and depend on the prevailing 'health and safety' culture. The potential hazards also depend on the type and chemical form of uranium containing materials handled within the workplace. Six major groups of workers can be identified.

1. Those primarily involved in the mining and milling of uranium ores.

2. Those involved in nuclear fuel fabrication and reprocessing

3. Those concerned with the handling and machining of metallic uranium (see Figure-5.2) and associated compounds during its industrial processing (e.g.-armaments and chemical industries).

4. Those involved in the handling of prefabricated components made of metallic uranium and associated compounds and alloys during assembly and manufacturing of industrial components (e.g. the aircraft industry and medical/research sectors).

5. Those involved in industries in which uranium is present as a contaminant or by-product. For example, in the extraction of other ores containing elevated levels of uranium (e.g. phosporities), in the processing and agricultural application of phosphate fertilizers, or as a worker decommissioning and scrapping military vehicles.

6. Those involved in emergency services in the aftermath of accidents or incidents involving DU (e.g. factory fires, aircraft accidents) or following a plane crash or fire in a uranium storage facility.

Figure 5.2 Workshop for the machining of uranium.

In the second case, there is the potential for contact with pure uranium or uranium compounds such as UF_6 which is extremely toxic due to the release of HF when in contact with water. To date at least one case of attempted acute poisoning (non-fatal) has been recorded in which the subject, a uranium processing worker, deliberately consumed processed uranium (Pavlakis et al., 1996).

Occupational exposure to DU may involve exposure to a number of different physical and chemical forms of uranium. During the production process the solubility of uranium compounds changes dramatically. Uranium can exist in biologically soluble forms such as uranium hexafluoride (UF_6), uranyl nitrate ($UO_2(NO_3)_2$) and uranium trioxide (UO_3), and relatively insoluble forms such as uranium dioxide (UO_2) and triuranium octoxide (U_3O_8). As discussed in Chapters 8 & 9, the severity of health effects may be associated with chemical or radiological toxicity and hence the exposure limits are controlled from the standpoint of both (ACGIH, 2000; ICRP, 1991; BSS, 1996; NIOSH, 1994).

The presence of high concentrations of 'available' uranium compounds in workplaces associated with cases 3 to 6 above is much less likely, although uranium concentrations as high as 400 mg/kg may be reached in ore and dusts associated with phosphate rocks and phosphogypsum. Elevated levels of uranium may also be inadvertently encountered in fly-ash from coal combustion, zircon sands used in the ceramics industry, ores and precipitates used in the production of titanium dioxide pigments and in the metal recycling industry (van der Steen, 1999). In the context of the metal recycling industry, out of a total of 3500 events in which radioactivity was detected in shipments of metals for recycling in the USA, 44 events (1.24%) were ascribed to the presence of uranium and/or DU (Yusko, 1999). These events included the melting of a shipment of recycled zinc containing DU at the Southern Zinc plant in Georgia, USA.

A contaminating incident (involving DU chips and dust) was also reported to have occurred when hammers and chisels were used to remove DU counterweights from an aircraft. Such occurrences highlight the need for promoting awareness of the presence and handling of such materials in the workplace.

5.6 Summary

Exposure to uranium and DU may take place through a wide variety of pathways. Environmental exposure may be due to inhalation and ingestion of food, drinking water and dust/soil. In the workplace exposure is more likely to be the result of dermal contact or inhalation.

In general, it is considered that ingestion of food and drinking water dominate background exposure to uranium. However, this is only likely to occur in the case of DU if it has become well mixed in the food chain and/or contaminated a source of drinking water. This is unlikely to occur in the short term and consequently exposure to DU will be dominated by ingestion and inhalation of any dust derived from the use of DU (exposure by handling metallic DU, for example by picking up penetrators from the battlefield, is dealt with in more detail in Chapter 9).

Data regarding bio-uptake of uranium into plants and animals indicates that bioaccumulation factors, while not being high may be in some cases significant over the longer term, particularly where local consumption patterns indicate a preference for foodstuffs shown to potentially bioaccumulate uranium (i.e. the kidneys of cattle).

In the absence of specific data on the solubility and bioavailability of DU related compounds and mixtures extrapolation from the behaviour of uranium may not always be valid. Similarly natural variations in parameters governing the bioavailability and mobility of uranium and DU are often highly variable indicating the need to collect site specific as well as material specific data.

6 Case studies and exposure scenarios

The aim of this chapter is to outline the factors which determine potential exposure routes for a range of receptors to DU. This is based on an understanding of potential linkages between sources of DU via pathways to environmental receptors, including humans. This chapter describes generic exposure scenarios that may be used to identify the principal exposure pathways for a range of environments and receptors.

6.1 Case studies

6.1.1 Potential exposure from air crashes

Under normal circumstances external radiation exposure of aircrew from intact DU balance weights during flight is insignificant when compared to the additional dose of ionizing radiation received because of exposure to cosmic rays during normal air travel (NUREG, 1999). However, workers may be exposed to DU used in aircraft during routine servicing of components in the immediate vicinity of any DU counterweights or to workers and members of the public as a result of an air crash. Levels of uranium introduced into the environment as a result of such an air crash depend on a wide variety of factors. To date, studies at the two most intensively investigated sites (Amsterdam and Stansted) have indicated that:

- whilst some DU may be released into the environment during air crashes, a large proportion of balance weights may be recovered from such crashes in a near intact condition.

- the varying quantities of DU balance weights used in aircraft, and the potential for production of small fragments and particles make it difficult to estimate the total percentage recovered or accounted for following an air crash.

As discussed in Chapter 4, DU is used as a material for balance weights (typically in rudders, elevators and ailerons) in some commercial aircraft (in weights of between 0.23 and 77 kg). While intact in the aircraft, the DU is either plated (cadmium and/or nickel) and painted or encased in a thin skin of aluminium alloy. Persons most likely to be exposed to DU in these conditions are those working in the manufacture of the components, in which case the conditions are as those discussed in Chapter 5, Section 5.3.5. Aircraft service personnel while unlikely to be directly exposed to DU may be exposed to beta and some weak gamma activity during service of components in the immediate vicinity of control surfaces and during replacement of counterweights (see also Chapter 9). The only environmental exposure route to DU from this source is when it is released as the result of an air crash, or when existing balance weights are inappropriately stored or scrapped. For example the presence of DU in a consignment of 53.5 metric tonnes of aluminium ingots sourced from 'recycled airplane parts' has recently been reported to have occurred (www.nrc.gov/OPA/pn/pn301006.html).

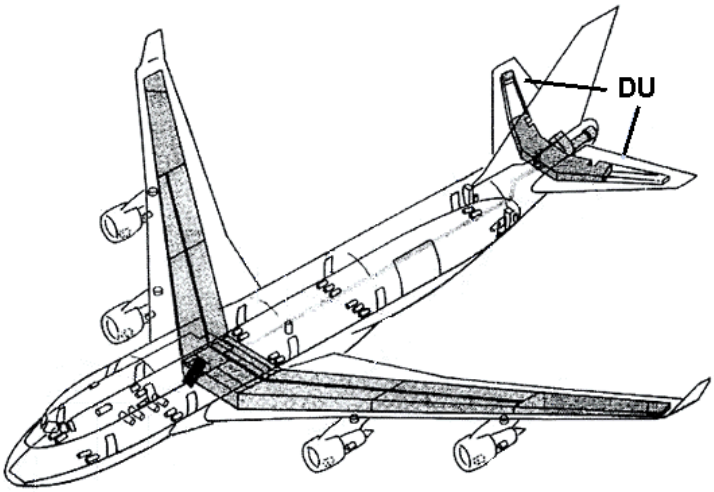

Figure 6.1 Schematic diagram of a Boeing–747 showing locations of DU counterweights (From US Federal Aviation Authority Document (FAA), CAS (2001)). Note this diagram does not acknowledge the use of DU counterweights in the Boeings wings that have been described elsewhere (e.g. NUREG, 1999).

Studies by Elder and Tinkle (1980), on the combustion of DU penetrators, concluded that oxide particles in the respirable size range could be formed when DU metal is exposed to temperatures greater than 500°C for burn times of longer than 30 minutes. Similarly, studies of the corrosion of metallic uranium in air and water vapour have established that corrosion was significantly more rapid in moist air (Haschke, 1998). While it may be that the casing or plating on DU counterweights protects it from fire and exposure to atmospheric corrosion, it is known that such protection degrades with time and that replating can be required. Results of burn tests and the questions raised in relation to the likely fate of DU in air accidents have also been highlighted in the media and in the scientific press (e.g. Parker, 1988, Uijt de Haag et al., 2000).

In the case of the 1992 Amsterdam plane crash (a wide bodied Boeing 747–258F), it has been reported in the press that only 130 kg of the initially estimated 282 kg of DU was recovered by clean-up teams, and that the Dutch commission of enquiry concluded that some of the 'missing' DU may had been released in the form of oxide particles (Kirby, 2000). Data presented in other recent studies such as Haag (2000) confirm that approximately 152 kg of the DU from the crashed plane remained unaccounted for almost eight years after the actual crash. This does not necessarily mean that this quantity entered the local environment, as some of this material could have been removed from the site during general clean-up operations that included the removal of significant quantities of top soil (Haag, 2000).

Using the limited data available Uijt de Haag et al. (2000) calculated potential radiological exposures *to bystanders* (for a 1 hour exposure) from the crash based on a ‘worst case scenario’ that all of the missing 152 kg of DU were oxidized into an insoluble respirable fraction, and a ‘best estimate scenario’ in which only 46 kg of the 152 kg were oxidized during the fire and that only 1% of this was in the respirable fraction. Modelling of airborne dispersion based on these cases resulted in atmospheric levels in the area in which bystanders were present of 3 $\mu g/m^3$ (best-estimate) and

2000 µg/m^3 (worst case estimate). From the results of their calculations they concluded that 'it is therefore highly improbable that exposure of bystanders to uranium would result in the health complaints reported'.

In their calculations, Uijt de Haag et al. (2000) only considered exposure to bystanders present at the crash site for a relatively short period of time, that respirable uranium represented the greatest source of exposure to inhabitants because levels of uranium measured in soils were shown to be consistent with the natural background in the vicinity of the crash site and that 98% of any ingested uranium would be excreted within a few days. Such considerations would be less applicable for the assessment of longer term impacts where contaminated soils have not been removed from the site, although, obviously, levels of DU present in air and dust would have been significantly reduced due to fallout over the short to medium term.

6.1.2 Military uses

The mobility and behaviour of uranium in the environment has been, and continues to be, extensively researched as a result of concerns related to the safe disposal of radioactive nuclear materials derived from power generation, from environmental pollution and regulations associated with the mining of uranium, and from studies assessing the exposure of populations to background levels of radioactivity and heavy metals. Despite this level of research, relatively few studies have been undertaken solely on the presence and environmental mobility of DU released into the environment through actual military conflict.

During the 1990s, the use of DU in test firings, military campaigns (i.e. operation Desert Storm, during the Gulf War, and operation Allied Force, during the Kosovo conflict) was the focus of much attention. This was principally, but not exclusively, due to a suggested or postulated link between exposure of military personnel, who had been in close contact with vehicles and installations that had either been attacked by armour-piercing munitions or had been protected by armour containing DU, and illnesses found in veterans from the Gulf War.

In the Gulf War it has been estimated that approximately 300 tons of DU was used sporadically over a total area in excess of 10 000 km^2 (Fetter and von Hippel, 1999) although another estimate based on data reported in CHPPM (2000) suggests that a total of 338 tons were used. The latter figure comprised 68 tons of large-calibre tank munitions, 260 tons of 30 mm armour-piercing munitions by US Air Force aviators and 11 tons of 25 mm armour-piercing munitions by US Marine aviators. During the air strikes in Kosovo, NATO fired about 10 tons of 30 mm armour-piercing munitions. NATO air operations in Bosnia–Herzegovina fired about 2 tons of 30 mm DU munitions.

The likelihood of exposure to DU and abundance of uranium in the environment following military activities was considered to be related to:

- the type of munitions used (for example single tank rounds with a high probability of impact (80% to 90%) versus strafing runs by ground-attack aircraft with a relatively lower probability of direct impacts).
- the density of munition use.

- the presence of aerosols and dust containing mixed oxides of DU.
- the presence of pieces of residual metallic DU.

Figure 6.2 DU munition used by an A-10 Warthog aircraft and the Gatling gun from which it is fired.

It was considered during these studies that the pyrophoric nature of uranium was of special relevance to the potential health effects and environmental redistribution of uranium resulting from DU use in munitions and armour. Studies of the use of DU munitions have indicated that up to 70% of the DU in a given projectile may be converted to dust and aerosols on impact (AEPI, 1995). Other studies (CHPPM, 2000) indicate a lower estimate of 10% to 37%, depending upon the exact nature of the impact (i.e. with armour or other material such as concrete surrounding the target). This large discrepancy in reported conversion efficiencies may be due to variations in the hardness of the target, the velocity of impact and the angle of impact. The lower conversion figure agrees with information from the Pacific Northwest National Laboratory (formerly Batelle Pacific Northwest Laboratory) and indicates that when DU penetrators were heated under controlled environments (at about 1200°C) about 30% of the uranium was oxidized. In this case, over 99% of the formed uranium-oxide particles were greater than 20 µm AMAD (Mishima et al., 1985) and could therefore be considered as being non-respirable.

It has been observed that, in some cases, the DU projectile went completely through the target without oxidising or producing significant quantities of dust and aerosols, resulting in relatively large pieces of metallic DU entering the environment. Similarly it is likely that projectiles impacting into soft soil, surrounding hardened targets, may penetrate into the ground with minimal production of DU dust.

The percentage of such buried projectiles depends on engagement angles and ranges, soil types and terrain (AEPI, 1995) and is therefore extremely variable. For example during a typical strafing run against a single target, three planes may fire approximately 50 to 100 DU rounds each over an area of about 500 m^2 (10 meters wide by 50 meters long). In US airforce tests prior to the Gulf War a ‘typical’ A10 Thunderbolt strafing attack scenario against a T-62 tank resulted in a 90% miss and 10% hit rate (CHPPM,

2000). This indicates that a substantial mass of DU might become buried in a rural environment and lead to subsequent dispersion in the soil and leaching into groundwater as a result of chemical weathering. Little firm data appears to have been published on the potential penetration depth of projectiles into soils beyond observations that intact 30 mm and 25 mm penetrators have been found at a depth of 30 cm in soft soils typical of the Persian Gulf or Serbia (CHPPM, 2000). This is presumably because of the difficulty of detecting the beta or gamma radiation from buried DU projectiles. Projectiles that miss the target may also ricochet, skipping across the ground with minimal production of dust and aerosols. AEPI (1995) quote that such projectiles usually land within 2 to 4 km of the target.

During military conflict, particulate materials may also be produced during ammunition fires, such as may occur in ammunition depots or factories manufacturing and storing DU components. Elder and Tinkle (1980) have investigated the effects of simulated fires involving penetrators in storage or during transport. Experiments involved the initiation of semi-controlled conditions exposing the penetrators to high temperatures, an oxidising atmosphere and an intermediate wind speed of 2.23 m/s (5 mph). It was observed that penetrators did not tend towards self-sustained burning; this only occurs when finely divided uranium is oxidized. Depleted uranium aerosols were found to disperse in all forced draft oxidation experiments at temperatures in the range 500 to 1000°C. In an outdoor burning experiment with temperatures up to 1100°C, 42 to 47% of the penetrator by weight was oxidized in a three-hour burn. Outdoor burning also produced greater quantities of aerosols in the respirable range (<10 µm AMAD) with 62% of aerosol mass being in this size range compared to a maximum of 14% in the laboratory experiments. In general, DU aerosols in the respirable range are produced when penetrators are exposed to temperatures greater than 500°C for burn times of longer than 30 minutes. Some experiments have also indicated the presence of an ultra-fine particulate fraction (< 0.1 µm) often adhered to larger particles.

As a result of the high temperatures that are created during impact, uranium is converted to a series of oxides which include the relatively insoluble triuranium octaoxide (U_3O_8), uranium dioxide (UO_2), and relatively soluble uranium trioxide (UO_3) (Harley et al., 1999b). It has been stated that the relative insolubility of some of these oxides delays the rapid infiltration of dissolved uranium through the soil zone and into groundwater reserves. However, it does not preclude the physical migration and contamination of surface water resources with particulate uranium, or conversion into more, or less, soluble forms through interaction with other components of the target or soil (Patrick and Cornette, 1977). The exact chemical composition and crystalline structure of particulates and aerosols produced during the impact of DU projectiles also depend upon the composition of target material. For example, studies by Patrick and Cornette (1977) and summary text from CHPPM, (2000) indicate that complex spherical porous particles rich in iron and tungsten can be produced through high velocity collisions with armour. The same authors also state that similarly shaped, complex particles, may be formed by alloying with clay and sand (i.e. containing aluminium, potassium, silicon) as a result of direct impact with soil, or when hot, reactive, secondary particles from the initial impact interact with the soil environment.

Corrosion rates of any remaining metallic DU material in soils have been cited in AEPI (1995) to be in the order of 0.05 cm to 0.10 cm/year. Based on a 1 cm diameter by 15 cm long penetrator (e.g. about the same as a 30 mm round) this equates with the release of approximately 90 g/year. For the larger projectile typical of 120 mm munitions (3 cm×32 cm) this equates with a release of approximately 500 g of DU/year.

Based on these corrosion rates, the remains of such projectiles will only remain as metallic DU for between 5 and 10 years. In areas of low water infiltration it is therefore likely that the rate of migration of DU from the corroding projectile will be controlled by the solubility of secondary corrosion products (i.e. the high concentrations of uranium produced by corrosion in migrating fluids will exceed the solubility of many secondary uranium minerals, thereby promoting precipitation of secondary minerals rather than migration).

Concentrations of uranium in soils associated with the release of DU from military conflict are less well known and are likely to be less predictable than releases measured under more controlled 'proving ground' conditions. However, tests conducted by the US ballistics research laboratory, have shown that, although DU particles thrown into the air can travel downwind, the largest amounts of DU dust created on impact come to rest inside a penetrated vehicle, with significant amounts on the outside surface and within 10 m of the target (SAIC, 1990). Further information citing tests on hard targets at the Nellis Air Force Range in the US, indicates that DU dust from the impact of a 30 mm munition strike was deposited within 100 m of the target. Similar tests with a 120 mm penetrator that perforated a tank resulted in 90% of the airborne DU outside the tank remaining within 50 m of the tank. These dispersal patterns remained typical even after a fire began in a test tank and continued for over 12 hours (AEPI, 1995).

In their calculations based on data from available studies (shown below) CHPPM (2000) used the following data with respect to exposure to DU particulates in and around the immediate vicinity of, a target hit by a DU round:

- airborne Release Fraction 10%–37%.
- respirable Fraction 60%–96%.
- chemical Form UO_2, U_3O_8, UO_3 .
- particle Size 1 to 10 µm.
- solubility characteristics in Lung Fluid 1%–83% Class Y(S); 1%–20% Class W(M); 1%–43% Class D(F).

Note that the values in brackets refer to ICRP-66 (1994a) absorption types (Annex 4). Classes D,W and Y reflect retention half time of day (D), week (W) or year (Y) (ICRP-30, 1979). Also the more soluble a particulate the less radiological hazard and the greater the chemical hazard (see Chapters 7, 8 and 9).

A number of authors have used various theoretical scenarios to assess the likely hazards posed by the use of DU munitions in conflict (e.g. Fetter and von Hippel, 1999; Liolios, 2000; UNEP/UNCHS, 1999; CHPPM, 2000; SSI, 2000). Results of these studies indicate that the people at most risk of exposure to DU munitions are the occupants of vehicles actually attacked and penetrated by DU munitions. Members of the general population including those downwind of battlefields were not considered by these authors to be at risk of significant exposure provided that vehicles struck by DU munitions were made inaccessible to curious civilians (or soldiers).

These studies currently lack validation due to lack of data, and rely on relatively simplistic scenarios, complex modelling or low-resolution broad-scale modelling. However, later studies such as those undertaken by CHPPM (2000) use more recent data and realistic scenarios. As is the case with all such scenarios, they are subject to inaccuracies when considering site-specific issues that may enhance the potential of exposure to DU (i.e. the heavy use of DU munitions in close proximity to important localized water resources or areas of market gardens).

Whilst some authors have suggested that the use of DU munitions are unlikely to add significantly to environmental baseline levels of uranium in soils, it is important to consider that:

- uranium derived from the fragmentation of munitions may be more bioavailable, and possibly mobile, than residual uranium present in weathered soils (as for example demonstrated during investigations of soils contaminated by uranium from the Fernald site by Elless et al. (1997) and at military firing ranges by Becker and Vanta (1995).

- the relative importance of additional anthropogenically derived uranium is dependent upon the degree and rate of mixing, and the depth to which such material is incorporated and redistributed amongst the upper soil horizons.

Figure 6.3 Kosovar Albanian children playing on military equipment following the Kosovo conflict. Such activities may lead to significantly increased probabilities of exposure to DU. (photo used with the permission of National Gulf War Resource Center, see www.ngwrc.org).

For example if DU from the impact of a 4.85 kg penetrator (50% volatilized) were evenly dispersed over a radius of 10 m to a depth of 10 cm it would produce a uranium concentration in soils of approximately 96 mg/kg. This value is above that observed in most natural soils and similar to that observed in dusts in the Amman area of Jordan in which phosphorite has been mined (Smith et al., 1995). However, if a similar release of uranium was restricted to the upper 1 cm of soil, as might be expected from the deposition of atmospheric particulates onto uniform soils of a high clay content, then the resultant concentration, assuming even airborne dispersal, would be in excess of 960 mg/kg. While the presence of elevated concentrations in the near surface region of soil profiles is likely to reduce transfer to plants, it is more likely to facilitate inadvertent exposure to uranium in dusts and other re-suspended forms.

The most extensively researched releases of DU into the environment have occurred in areas used by the military to test munitions (proving grounds). For example, an investigation at the US Department of Energy's Los Alamos National Laboratory, conducted for the US Army, suggested that up to 100 metric tonnes of DU may have been expended. It was estimated that a small canyon with an area of 3.1 square miles had a DU inventory in the region of 35 000 kg (Becker and Vanta, 1995). Similar

quantities of DU were also used at military proving grounds in Yuma, Aberdeen and Jefferson in the USA (Ebinger et al., 1996; Ebinger and Oxenburg, 1997).

The United National Environment Programme in their post-conflict environmental assessment of DU in Kosovo (UNEP, 2001) concluded that:

- There was no detectable, widespread contamination of the ground surface by DU. The corresponding radiological and toxicological risks were considered insignificant or non-existent.
- Detectable ground surface contamination was limited to areas within a few metres of penetrators or penetrator impact.
- There was no significant risk related to these contamination points in terms of possible contamination of air, water or plants. The only risk would be through direct hand contact or the ingestion of contaminated soil. Based on reasonable assumptions on intake of soil the radiological risk would be insignificant while from a toxicological point of view the possible intake might exceed the health limit.

Although studies at such sites are useful for establishing the distribution of uranium immediately following dispersal, they provide little if any information about the longer term mobilisation and distribution of uranium. The most practical way to undertake such studies is to investigate the dispersal at natural sites of uranium mineralisation. A wide range of such 'analogue' studies have been undertaken in support of the nuclear waste disposal industry. They have clearly demonstrated that oxides of uranium, including uraninite and pitchblende (UO_2), may be readily weathered (by oxidation and complexation with inorganic and organic ligands) and converted into more mobile soluble, forms of uranium that become incorporated into local surface waters, groundwaters, micro-organisms and plants (e.g. Basham et al., 1989; Hooker et al., 1989; Burns and Finch, 1999). Currently, there is a lack of comparison between data produced from these studies and that derived from DU alloys and associated particulates and aerosols. However, it should also be noted that weathering rates of particulate dusts produced during the combustion of DU weapons are likely to be enhanced over those of residual metallic uranium (Patrick and Cornette, 1977; Becker and Vanta, 1995) due to their inherently smaller particle size and correspondingly high specific surface area (i.e. area/unit mass of substance)

Military exposure

Modelling of various exposure scenarios has been undertaken as part of environmental monitoring and decommissioning programmes carried out at US Army proving grounds that have become contaminated with DU. The Jefferson Proving Ground decommissioning programme modelled exposure scenarios, which have been documented in several published reports, (Oxenberg et al., 1999; Ebinger, 1998; Ebinger and Oxenburg, 1997; AEPI, 1995; Ebinger and Hansen, 1994). It should be noted that these studies only form examples of the results that may be obtained during case studies and should not be extrapolated to other sites, such as the Balkans and the Gulf, without careful consideration and justification. Three exposure scenarios were generally modelled in these studies in order to consider suitable uses for the site following decommissioning:

i) An occasional user of the site visiting for 4 to 6 weeks of the year to hunt. All food and water would be brought onto the site by the user. Game animals would be consumed by the hunter.

ii) A subsistence farmer consuming vegetables, dairy products and meat from crops and livestock produced on the site. Drinking water would be obtained from uncontaminated off-site sources. A fraction of the drinking water for livestock would be from contaminated groundwater, but the remainder would be from uncontaminated surface water.

iii) As for scenario (ii) except that all drinking water would be obtained from contaminated groundwater.

This modelling exercise concluded that no risk to humans occurred from occasional use of the site, the largest exposure to DU in this scenario being from exposure to contaminated dust.

The farming scenarios showed some risk of exposure due to inhalation of contaminated dust, but by far the largest exposure resulted from the use of contaminated groundwater as drinking water, either by livestock or by humans. The overall conclusions of the modelling exercises were that subsistence farming presented a greater risk of DU exposure than did occasional use. However, in this particular study farming scenarios were not pursued in greater detail, because farming and permanent occupation were considered to be inappropriate end uses due to the presence of unexploded ordnance on both proving grounds.

There are, of course, many cases worldwide where exposure to mines has not prevented the continuation or resumption of farming activities. In such circumstances, it may be desirable to compare the potential risks associated with exposure to uranium with those associated with farming in close proximity to such obvious risks.

The work carried out by Ebinger et al. (1990; 1996) at the Aberdeen and Yuma proving grounds considered exposure to all components of the ecosystem. Depleted uranium was found in almost all samples and was present in most of the ecosystem compartments at Yuma (the semi-arid site) but not so much at Aberdeen. Measurable uranium concentrations were also found in aquatic endpoints (biota) at Yuma and in deer tissues at Aberdeen. Radiological effects were found to be insignificant at both sites, but there was some tentative evidence of toxicological effects. Erosion at Yuma was the primary mechanism of DU transport; wind deposition being of secondary and minor importance. At the wetter Aberdeen site, the main migration pathways were considered to be transport of suspended detritus in surface waters.

Concentrations of uranium in ecosystem components showed kidney content to be below threshold values in all species except for Kangaroo rats at Yuma in which histopathology indicated possible damage to kidney tissue (Ebinger et al., 1996). The consumption of dust, which had become adhered to foliage, was the most important exposure pathway for animals living in these sites.

Model projections of exposure over the next 1000 years at these sites (Ebinger et al., 1996; Ebinger and Oxenburg, 1997) indicate a gradual decline of the importance of particulate exposure together with a gradual increase in exposure to groundwater contamination over the next 100 years, before reaching a reasonably steady state condition between 100 and 1000 years (i.e. uranium particles become weathered releasing dissolved uranium into the water table or are physically removed from the area). Obviously such rates are extremely dependent on mineralogy of the source of uranium, local soil type and hydrological conditions.

Elless et al. (1997) and Elless and Lee (1998) undertook the characterisation of uranium contaminated soils at various US sites. Uranium was found to be associated with the silt and clay size fractions of soil samples analysed in these studies. In addition, mineralogical analysis indicated that the predominant form of uranium contaminant in these soils was an autunite-like phase (e.g. hydrated calcium U(VI) phosphate). In addition to this, major phase uranium minerals such as uraninite (UO_2, U(IV) oxide) and coffinite (U(IV) silicate, $USiO_4$) were also present (IV and VI refers to the ionic charge on the uranium atom). While uraninite and coffinite are generally considered to be insoluble (<0.01 mg/l), autunite the dominant mineral is only slightly soluble (0.1 to 0.2 mg/l) (Langmuir, 1978).

During these studies (Elless et al., 1997 and Elless and Lee, 1998) uranium solubility was determined before and after remedial treatment in support of performing a health-based risk assessment. Solubility of uranium was determined in carbonate-rich soils associated with the contaminated sites, and in background soils, using 75 and 300 day extraction tests performed with rain and groundwater. The results indicate the importance of anionic uranium carbonate complexes in controlling mobility, and that the major factor influencing uranium mobility was solubility control by primary mineralogical phases rather than sorption. The results also indicated that contamination of groundwater resources by DU derived from munitions was possible at the Fernald site, and that this contamination was promoted by the use of carbonate-based erosion control and road building materials.

Risk calculations and biokinetic modelling based on the resultant solubility measurements indicated that the risks were greatest from the soil ingestion pathway and the direct consumption of infiltrating groundwater. Interestingly, the lowest risk in this class was attributed to the inhalation of soil-derived dusts.

From the perspective of kidney toxicity, the greatest source of risk was derived from exposure due to the direct ingestion of infiltrating, contaminated groundwater. In all cases, the calculated level of risk was extremely sensitive to the solubility of uranium and it was recommend by the authors that this parameter must not be overlooked when assessing potential risks associated with exposure to uranium from the environment. It should be noted that whereas a 75 day extraction test may be applicable to the leaching of uranium during infiltration of rainwater, it is inappropriate in assessing solubility within the human gastrointestinal tract, where residence times are in the order of hours (Ruby et al., 1996). Similarly, the use of acid stomach simulants do not adequately account for dissolution of uranium in the neutral environment of the upper intestinal tract.

Short-term leach testing of residues from DU munitions at the Elgin test site, which had been used for test firing of DU munitions for over 20 years, indicated remobilization of uranium from soils and to a more limited extent in drainage sediments over a time scale of 0 to 20 days (Becker and Vanta, 1995). It was hypothesized by these authors that this comparatively rapid leaching of uranium was due to the abundance of small particles released from munitions during the combustion process (the majority of DU particles being associated with the fine clay and silt fractions despite the sandy nature of the soil). Analysis of cores showed transport of DU into the soil profile with baseline composition being reached at a depth of approximately 100 cm.

The longer term durability of relatively insoluble U(IV) oxides has been investigated during studies of the mobilisation of uranium dioxide stored in geological media with particular reference to the direct disposal of spent nuclear fuels (e.g. Cachoir et al.,

1996; Gallien et al., 1996). Under oxidising conditions, a two-step process was defined in the alteration mechanism.

(i) Incorporation of oxygen and hydrogen correlated to a reduction in the volumetric uranium content (kinetic control).

(ii) Formation and dissolution of schoepite (UO_3 $2H_2O$) (thermodynamic control).

Under reducing conditions, preliminary experimental results suggested an alternative mechanism. Gallien et al. (1996) measured the concentration of uranium under reducing conditions to be as low as 10^{-11} molar. Other investigations, again undertaken during studies pertinent to the disposal of nuclear waste, have investigated the occurrence and weatherability of uranium oxides under natural conditions (so called 'natural analogue' studies). Such studies (e.g. Miller et al., 2000; Basham et al., 1989; Hooker et al., 1989) have shown that even reduced uranium oxides may become mobilized into ecosystems and the local environment over a period of tens, hundreds and thousands of years. These are time scales which are impracticable for studies in the laboratory and at proving grounds.

Case studies performed to date emphasize the wide variability in the behaviour of uranium and the comparatively small range of potential end-use scenarios that have been investigated. This is particularly relevant where uranium and/or DU has been released into an environment from which the exclusion of human beings is not a practicable option.

6.2 Environmental exposure scenarios

Factors to be considered for assessing environmental exposures include variables related to soil composition and chemistry, climate, hydrogeology, land use and the mode and magnitude of exposure.

6.2.1 Soil

A range of soil functions may influence, or be influenced by DU contamination (Note that in the context of this work contamination does not imply harm to any given endpoint; see glossary). These are:

- control of substance and energy cycles as compartment of ecosystems.
- basis for the life of plants, animals and humans.
- carrier of genetic reservoir.
- basis for the production of agricultural products.
- buffer inhibiting movement of water, contaminants or other agents into groundwater.

Scenarios associated with some of these functions are discussed in the sections that follow on plants, animals and groundwaters.

Clearly the potential for DU to contaminate soil depends upon the magnitude and nature of exposure. For example, large-bore munitions with a high probability of hitting hardened targets and fires involving DU munitions will potentially introduce more particulate DU and DU-oxides into soils than situations where small-bore DU munitions

have been used in strafing attacks. On the other hand, the amount of metallic DU introduced deeper into the soil profile is likely to be greater for typical strafing attacks, particularly in rural environments.

It is important to consider the influence of scale when considering scenarios. For example, it must be decided whether to consider a battle as a single diffuse source of contamination or as a series of point-source contamination incidents. Such a decision is site-specific, and to a certain extent depends on the proximity, distribution and sensitivity of various receptors.

Whilst the weathering rate of both DU-oxides and metallic DU is low, it is still a relatively rapid process compared with that of many natural soil minerals. However, the mobility of weathered DU in the soil profile is dependent upon sorption and mass transport properties of the soil (i.e. Kd and the water infiltration rate, see Chapter 3). Depleted uranium has been shown to be mobile in environments subject to high surface erosion and low infiltration rates, such as deserts. The variation in Kd of uranium with organic carbon content and soil pH (as described in Chapter 3) indicates that mobility is likely to be greater in semi-arid calcareous environments, or calcareous environments in which neutral to alkaline soil pH combines with a low organic carbon content. While mobility is greater in semi-arid calcareous soils, low net infiltration may significantly reduce the dispersal and mixing of DU.

Both point and diffuse sources of DU will weather and slowly become homogenized with uranium naturally present in the soil environment. Any increased weathering and mobility associated with specific forms of DU can be viewed favourably, due to the reduction of high levels of point-source contamination (i.e. dilute and disperse). Thus the exposure of receptors in the surface environment may be reduced. This implies that the level of contamination in a number of receptors, such as groundwater, and risks associated with any harmful, non-threshold effects, may be increased. Similarly, dispersal may significantly decrease the cost-effectiveness and technical feasibility of clean-up.

To date, difficulties in identifying DU penetrators that have missed their target and become embedded in the soil profile has limited the development of scenarios relating to the exposure of soil to DU from munitions. The collection of data on the depths to which various DU munitions penetrate in generic soil units would greatly assist any such developments and the design of suitable sampling strategies.

6.2.2 Water

When firing of DU munitions occurs over land, DU contamination of water is likely to be dominated by transfer from direct soil deposition, due to the small surface area that freshwater generally covers. The transfer of uranium from the soil, or regolith, will be controlled by physical and chemical processes, which will in themselves be regulated by the climatic and geologic environment in which the contamination occurs.

The nature of DU entry onto the soil surface (e.g. fragmentation from impact with a target) or within the soil profile (e.g. near intact burial) will affect the rate and mode of transfer of uranium to the soil-water, surface-water and ground water environments. Fragmentation will increase the surface area, of any one projectile available to chemical and physical weathering. Small particles may be entrained in the near-ground atmosphere during dry (dusty) conditions. Overland water flow, from rainfall or snow thaw, will cause the physical movement of particulates to surface watercourses, and

ultimately into estuaries and near-shore environments. Physical translocation of particulate material into groundwater may occur through the regolith and within aquifers that have secondary fracture flow mechanisms. The entire burial of DU weapons, from a 'soft' impact with soil, will lead to little fragmentation, but potentially contaminate groundwater resources through dissolution and migration into aquifers.

The mobility of DU in the near-surface environment will be controlled by factors such as the pH of soil minerals and water, and the sorption potential of soil minerals (Chapter-3). Thus where soil strongly binds the uranium in secondary phases or on surfaces (e.g. iron oxides, clay minerals or organic carbon), its release into soil water, and translocation to groundwater, should be minimal. In deeper environments mobility and attenuation are controlled by the composition of fracture coatings and water chemistry. Where uranium is highly mobile, water resources may be more vulnerable to contamination.

The vulnerability of water to uranium contamination will be controlled by the geological, soil conditions and mobility encountered. The primary factors affecting vulnerability, assuming that uranium is mobile, are the depth of the unsaturated zone (i.e. proximity of the contamination to the water table) and the infiltration rate of recharge. For example, vulnerability of water resources hosted in river gravels may be high due their proximity to the surface. Whilst, the vulnerability of those obtained from deeper, possibly confined, aquifers will be lower.

6.2.3 Plants

Plants, while generally considered to be poor accumulators of uranium, may be affected by the presence of elevated uranium concentrations in the environment. Exposure to uranium can occur through water (soil pore water, irrigation water or rainfall), from the atmosphere (i.e. wet and dry deposition to foliage), from leaching of soil by root exudates or from the direct incorporation of particulate matter. During studies at the Yuma proving ground, the association of uranium and DU with vegetation and plant litter was clearly established, although the majority of determined uranium was considered to be associated with particulate contamination of samples that was easily removed by washing.

Any scenario exposure to plants occurs primarily through:

1) Atmospheric fallout of particulate material. This mode of contamination would therefore be more likely where conversion efficiencies of metallic DU into particulates and aerosols are high (i.e. tank attack onto armoured targets) or following an intense fire in which DU is present. There is clear evidence that such contamination may be minimized through thorough washing of vegetables, greens and fruit.

2) Uptake of contaminated water. The mobility of uranium in the soil zone (see 6.2.1 above) and the proximity of uranium to the root zones of plants principally control this mode of contamination. The relative importance of infiltrating uranium being carried in from the dissolution of surface particulates compared to that derived from the weathering of buried DU penetrators is therefore dependent upon the depth of penetration. Although uptake into the plant may be minimal, it is possible that uranium may become concentrated into the skin of vegetables and tubers during the exclusion process (this has been demonstrated in some non-food plants; e.g. Basham et al., 1989). Exposure may therefore again be reduced by careful peeling and washing.

6.2.4 Animals

Animals (domestic or undomesticated) may become exposed to DU through similar routes to humans. Domestic animals are more likely to be given fodder grown outside the affected area than wild stock, lessening their exposure. Animal exposure is likely to be greater than that of humans, due to their often monotonous and less spatially diverse diet. Herbivores typically ingest considerable quantities of soil while browsing and may be particularly vulnerable to particulate DU present in soils and adhered to vegetation surfaces. Carnivores may be affected by bio-accumulation from species lower down the food chain.

6.3 Human exposure scenarios

When considering human exposure to uranium in the environment, it is important to consider the relative importance of the various exposure routes outlined above and in the previous chapter. An awareness of the most significant exposure pathway in any given circumstance allows the prioritisation of hazard assessment and control. For example, if it is recognized that the most important exposure route in a given scenario is posed by the inhalation of dust, immediate measures can be taken to prevent inhalation by suitable respiratory protection, exclusion from the area or by minimising wind-blown dust through irrigation. If, however, the greatest probability of exposure is posed by ingestion of contaminated food, measures can be taken to obtain alternative food sources.

The first six scenarios consider exposure routes associated with the use of DU munitions. Scenarios 7 and 8 represent exposure as a result of an air accident involving an aeroplane carrying DU counterweights. Scenario 9 considers exposure in the controlled environments of the uranium mining and processing industries.

Scenario 1: Person in attack zone

A person is in an area where the active use of DU munitions is occurring. The potential for dermal contact is high and this may even take the form of wounding with fragments of DU. The likelihood of exposure to DU aerosols and fine particulates as the result of impacts is high. Amounts of ingestion are considered low and would be principally associated with particles adhered to food.

Scenario 2: Relief worker in a war zone

A relief worker is entering a war zone to attend injured persons and assist with the clearing of associated debris. The exposure potential is similar to that in Scenario 1; the difference is that dermal contact is slightly reduced, and there is no risk posed by flying fragments of DU munitions or armour. There is likely to be greater contact with dust and soil, as people and debris are moved as part of the relief work. It has been suggested that levels of DU in re-suspended dusts are much lower than those encountered immediately after a high-energy impact as in Scenario 1 (CHPPM, 2000).

Scenario 3: Metal reclamation

Damaged armoured vehicles are decommissioned after military operations. Vehicles have often been transported and stood in the open air for periods of time. Unused munitions are removed, and in some cases vehicle parts may be salvaged for re-use. It is also possible that local inhabitants may take metal parts for their own use. This may

include armour containing DU. In this scenario welding and cutting equipment may be used (note this exposure scenario has been quoted in AEPI (1995) as potentially producing unacceptable levels of particulate DU). Dermal contact directly with DU metal and dust represents a potentially significant mode of exposure. Secondary mobilisation of dust may lead to inhalation, and welding, and cutting activities may result in the formation of fumes or aerosols, which may also be inhaled.

Scenario 4: Local inhabitant

Following the cessation of military activities in an area, subsistence and commercial farming may recommence. A farmer/plot holder and family will cultivate the soil and consequently be exposed to fine particles contaminated with uranium that may be inhaled or ingested. Contaminated soils may also be ingested as a result of hand to mouth activities. There is some potential of dermal exposure to metal fragments and dust. A subsistence farmer and his family will also rely on food and water that may be contaminated with uranium. The time scale of this will vary and be dependent on the bioavailability and mobility of the uranium in the environment. This will need to be assessed on a site-specific or regionally specific basis. Current estimates indicate that DU munitions and armour degrade and chemically weather over a period of 100 to 1000-years (Erickson, 1990), although this is highly dependent on the particle size or composition of any metallic uranium or uranium alloy; conceivably in some environments this could be considerably less. Certain traditional farming methods involve close contact with the soil and a stooping position that may encourage the inhalation of dust. Other more mechanized methods may result in the enhanced mobilisation and thus exposure by inhalation. Potential exposure to young children accompanying adults may be high.

An inhabitant in an urban area where a plane crash has occurred or has been subjected to bombardment may obtain some food from a vegetable patch or allotment garden tended by themselves. The key differences from a rural situation are that drinking water and dairy products are likely to be obtained from uncontaminated sources.

Scenario 5: Children playing

The return to normal activities in an area where DU munitions have been deployed will include children playing. This may be in areas where derelict military equipment remains. The hand to mouth and inquisitive activities of children may lead to significant dermal contact with metal fragments and dust. Ingestion of contaminated dust and soil will be likely and ingestion of contaminated food and water may also occur. Secondary mobilisation of fine contaminated particles may also increase potential exposure from inhalation. This scenario will be of much greater importance in regions in which geophagia is practised.

Scenario 6: Villager

Again this considers exposure following the cessation of military activities. People returning to villages in areas which have been affected by military activities may be exposed to DU in this environment (e.g. through the resuspension of dust or presence of metallic penetrators). The most important exposure routes are those related to the ingestion of drinking water and soil and dust, which are likely to be of greater importance than foodstuffs. It is likely that in most cases foodstuffs will be derived from a variety of local sources (or possibly from even wider afield). Drinking water and soil

are probably more consistent factors controlling exposure although contamination of drinking water supplies will vary according to source and method of supply etc.

Scenario 7: Relief worker at an aircraft accident

A relief or salvage worker, or possibly an investigation team, will work in the area in and around an aircraft crash site. Exposure routes are generally similar to those associated with relief workers in war zones. The magnitude of exposure is likely to be reduced because of the nature of the DU involved. Counterweights are large pieces of encased metal, whereas munitions consist of uncased metal which is designed to impact, and penetrate tank armour. Therefore, volatilisation and fragmentation are less likely and the relative importance of associated exposure pathways should be reduced. In the case of food and water ingestion, relief workers will be obtaining sustenance from external sources, and eating and drinking are likely to take place in areas removed from the crash site.

Scenario 8: Local inhabitant near a plane crash

An inhabitant of an area where a plane crash has occurred is assumed to obtain some food from a vegetable patch or allotment garden tended by themselves. This is a similar scenario to that of a subsistence farmer in a war zone. The key differences are that in the air crash zone drinking water and dairy products are likely to be obtained from uncontaminated sources.

Scenario 9: Industrial exposure

The potential exposure of workers would be expected to be controlled through national legislation and international recommendations which are designed to limit exposure. The greatest potential source of intake is likely to be by the inhalation of dust and fumes, although uptake through contaminated wounds, ingestion and dermal absorption cannot be excluded. Any contamination of food and drinking water will result from the inadvertent presence of dust while at work; or alternatively from off-site transport of dusts or waste products to the household environment. Because of the specific nature of these industrial processes, appropriate monitoring of the working environment and of the individual may be necessary. A guide on the assessment of exposure to uranium from mining and associated activities is available from the IAEA (1989a) which, despite ignoring the chemical toxicity of uranium, represents a useful approach in calculating and minimising exposure. Advice on individual monitoring procedures is given in ICRP-78 (1997).

Scenario 10: Background exposure

Background exposure illustrates the relative potential importance of various exposure pathways in an area unaffected by man-made sources of uranium or DU.

Scenario summary The above scenarios give an indication of the most likely exposure routes in a given situation. However, the specific conditions in an actual situation may have a major influence on these assessments and must be considered. For example, the potential magnitude of exposure posed by inhalation and ingestion of dust and aerosols in scenarios 1, 2 and 7 above would be greatly reduced if the events took place at a time of heavy rain. All exposure assessment and subsequent risk assessment exercises require the consideration of material and site-specific factors.

There are several key factors that can be noted from the scenarios:

- Exposure is likely to be greater under uncontrolled conditions.

- The potential for exposure during military conflict in which DU munitions and armour is used is more significant than that posed by the release of DU through air accidents.

- The relative importance of a variety of exposure routes in a war zone situation is similar for a range of scenarios. The actual health risk may however be of a very different magnitude, depending on concentrations in air, water etc. and pattern of exposure (e.g. acute or chronic).

- Outside of direct exposure to DU in active warfare, those with a large potential for exposure to DU are children returning to normal activities within a war zone. The hand to mouth action and inquisitive play of children mean that they are the most likely to be exposed in a wide variety of exposure pathways.

6.4 Summary

Humans and animals may become exposed to uranium by inhalation, ingestion, dermal sorption or injury (e.g. embedded fragments). The relative importance of each of these exposure routes depends on the physical and chemical nature of the uranium to which individuals may be exposed. For example exposure to uranium in a typical baseline environment away from anthropogenic or geological sources is likely to be dominated by ingestion of drinking water. In some cases, exposure may be dominated by the deliberate consumption of soil where geophagy is practised. Alternatively, for coastal populations, exposure may be dominated by the ingestion of shellfish. Estimates of baseline exposure to uranium range from 0.0005 to 0.001 mg/day.

The pyrophoric nature of uranium means that when DU munitions are used in warfare or training, a quantity of dust composed of mixed uranium oxides may be liberated in the immediate vicinity of any impact. Such dust may be inhaled, or enter the food chain either directly through the ingestion of dust on food or by plant uptake from surface water, via the deliberate or inadvertent ingestion of soil by young children, via the use of surface water for drinking or following migration of uranium into local groundwaters which may be used for drinking water or crop irrigation. While the ingestion of foods or soil and dust are likely to lead to exposure immediately following a period of conflict incorporation and migration to groundwater may occur over a time span of tens, hundreds of thousands of years.

During or after a military conflict exposure is also likely to be controlled by the type of engagement and the type of DU munitions used. For example strafing attacks may produce markedly different exposures than those likely to occur as a result of tank battles.

Due to the wide variation in potential exposure scenarios to uranium from natural and anthropogenic sources, it is recommended that exposure assessments are performed in a tiered manner (Tier 1: desk assessment and review; Tier 2: field study and analysis; Tier 3: detailed site-specific exposure assessment/validation) prior to any decisions being

made on the use of remedial measures or likelihood of potential health outcomes.

Current gaps in knowledge include:

- Weathering of DU combustion products and bioavailability (long-term and short-term, appropriate climatic scenarios).
- Relative importance of soil ingestion (regional dependence).
- Validation of actual exposure (natural and following conflict).
- Ecological cycling (food stuffs and plant uptake).

7 Behaviour of uranium in the body

7.1 Introduction

Although ubiquitous in the environment, uranium has no known metabolic function in humans and animals and has been generally regarded as a nonessential element. However, as in the case of many heavy metals, the search for health effects related to short-term and long-term exposure to uranium has an extended history. The first recorded reference to studies on uranium exposure dates back to 1824, only 40 years after its discovery. Later in the same century, studies on human subjects showed that uranium could be administered as a therapeutic agent for diabetes as it had been shown to increase glucose excretion (Hodge et al., 1973). During the final decades of the 20th-century, research into the occurrence and toxicity of natural uranium continued to be undertaken, with increasing emphasis being placed on understanding natural and baseline exposure to uranium and uranium-series radionuclides, on the toxicity of short-lived decay products such as radon and its progeny, and on the effects of chronic long-term exposure to natural uranium. These developments occurred in parallel with the development of increasingly sensitive and robust analytical techniques for the determination of uranium in soil, water and stream sediments primarily developed for application in the exploration and exploitation of uranium resources. Such techniques have also proved of great value in improving our understanding of how uranium behaves in the body.

As has been indicated in numerous scientific and general interest publications over the past decade, very little health-related research has been undertaken specifically on DU. The major reason for this is the extensive research undertaken on natural and enriched uranium, both of which pose a greater radiological hazard and an identical toxicological chemical hazard to depleted uranium. However, it is also important to realize that any resultant health effects from exposure to depleted uranium, due to radioactive decay or chemical interactions, do not necessarily occur in isolation. For example, although no direct information appears to be available on this subject, ATSDR (1999) points out that co-exposure to other nephrotoxins (e.g. lead and cadmium) could have an additive effect. Similarly, little reliable data exists on synergistic effects that may lead to enhanced uptake or excretion of uranium compounds.

7.2 Biodistribution and toxico-kinetics

Although uranium has no known metabolic function, its strong affinity for many physiological compounds suggest that it is unlikely to exist, except transiently, as free ions. For example, given the near-neutral pH of blood and many body fluids, it is generally accepted that the most important species controlling uranium mobility in the systemic circulation are the carbonate, bicarbonate species and citrate complexes of U(VI) (Cooper et al., 1982). For example, about half of the U(VI) circulating in blood is present as carbonate complexes (e.g. UO_2CO_3 and $UO_2(CO_3)_2^{2-}$) (Durbin, 1984) or associated with bicarbonate and citrate complexes (Cooper et al., 1982). However, at slightly lower pH, in the range 5 to 6, uranium is also known to complex strongly with a wide range of organic ligands and these might be expected to impact on the assimilation of uranium into body tissues from the systemic circulation. In urine, uranium is present predominantly as the bicarbonate complex (Cooper et al., 1982)

Uranium has been shown to form strong complexes *in-vitro* with biological molecules containing phosphate (e.g. glucose phosphate, phospholipids and nucleic acids and for glucosamine, acetylglucosamine and related polymers) (Guibal et al., 1996; Wedeen, 1992), although the presence of some of these compounds has yet to be established *in-vivo*.

The ICRP biokinetic models for predicting the behaviour of uranium in the body are described in more detail in Annex 4. Other toxico-kinetic models covering the systemic behaviour of uranium include those proposed by Sontag (1986), Fisher et al. (1991) and Wrenn et al. (1988).

7.3 Ingestion

Absorption of uranium from the gastrointestinal tract (GIT) depends on the bio-solubility of the uranium compound, previous food consumption and concomitant exposure to oxidising agents. Wrenn et al. (1985) quoted a value of 1% to 2% average human gastrointestinal absorption. This has gained general acceptance and the currently recommended generic average human gastrointestinal absorption value for uranium is considered to be about 2% (WHO, 1998b; Leggett and Harrison, 1995; ICRP-72, 1996). Studies of GIT uptake factors in animals showed variance with quantities of uranium administered, age, dietary stress such as iron deficiency, and fasting. For example, various studies cited in ATSDR (1990) indicate factors of 3 to 4 times enhancement under these conditions compared to values obtained during studies on well-nourished adult animals. Uptake factors for specific modes of ingestion are discussed below.

Drinking Water: Uranium dissolved in water is almost exclusively present as the hexavalent species, and hence uptake factors for this form should generally be applied during the calculation of uptake from the human gut. For example the IAEA (1989a) suggested a gut uptake factor for uranium in water of 5% for most hexavalent compounds and a factor of 0.2 % for UO_2, U_3O_8 and most tetravalent compounds. More recent data from Harduin et al. (1994) suggest that 5% may be a conservative estimate and that a lower uptake factor of 2% for soluble components in water should be adopted.

Food: Wrenn et al. (1985) reviewed uranium uptake factors across a range of average diets and suggested an uptake factor of 2% to 3%. However, these authors report that values as high as 20 % have been quoted in the literature. It should be noted that these authors also report a significantly greater uptake factor in rats than other species, and suggest that rats should not be used to determine uptake factors for man.

Soil: The bioavailability of uranium in soils depends on the physical–chemical form in which the uranium is present. It is therefore impossible to suggest a single value or range of values associated with soils, particularly as the diversity of potential chemical forms of uranium and soil types is large. Characterisation and solubility measurements of uranium contaminated soils undertaken by Elless et al., (1997) confirm these general points and emphasize the wide variability in solubility (and inferred bioavailability) of uranium from various sources. However, given our current knowledge of conditions in the human gut and the aqueous chemistry and mineralogy of U, it is reasonable to hypothesize that uranium mobility (and thence bioavailability) is likely to be higher in the neutral conditions of the upper intestine, than in the acid conditions of the gut. This may be influenced by the formation of stable oxy-anion complexes. Consequently, the use of acidic leachates to assess solubility as described in Elless (1997) may yield

different results from those obtained using other forms of physiologically based extraction tests (e.g. Ruby et al., 1996)

7.4 Inhalation

Clinical postmortem studies of an occupationally exposed worker indicate that significant amounts of uranium are present in lung tissues (Kathren et al., 1989). This indicates inhalation has been an important source of accumulation.

The amount of particulate material deposited in the respiratory tract will depend on several factors, which include particle size and shape, breathing rates etc. These factors have been described in detail in the ICRP Human Respiratory Tract Model (HRTM) (ICRP-66, 1994a), see also Annex 4. Absorption of inhaled uranium into the systemic circulation will depend on the rate at which particles dissolve in the lungs and on their interactions with the ligands present in lung fluid.

Generic absorption parameters for soluble (Type F), moderately soluble (Type M) and poorly soluble (Type S) compounds have been published by ICRP. These are also summarized in Annex 4, along with factors influencing lung retention.

Table 7.1 Absorption types for uranium compounds (ICRP-71, 1995b).

Type	Typical Compounds
F	UF_6, UO_2F, $UO_2(NO_3)_2$
M	UO_3, UF_4, UCl_4, U_3O_8
S	UO_2,

F Fast; M Moderate; S Slow

Note some preparations of U_3O_8 can be assigned to Type M while for others their behaviour lies between Type M and Type S.

The behaviour of all the compounds formed in the nuclear fuel cycle has been studied extensively. Recently, much of this work has been reviewed and material-specific values have been reported in terms of HRTM parameters (Hodgson et al., 2000; Ansoborlo et al., 2001). When sound experimental data are available ICRP has recommend for many years that these should be used instead of the default given in the HRTM model. Material-specific biokinetics and toxico-kinetics of uranium oxides are discussed further in Chapters 10 and 12.

In the absence of material-specific *in-vivo* data, *in-vitro* dissolution studies (Edison, 1994) can provide much useful information which can be of use in assessing the implications to exposure.

It is recognized that there is a paucity of data on the dissolution and absorption characteristics of aerosols formed as a consequence of the combustion and thermal oxidation of DU. In this context, *in-vitro* studies and an improvement in our understanding of the mineralogical nature of these particles will provide rapid and cost-effective information given the physical–chemical variability of such materials resulting from different scenarios and outcomes.

In-vitro solubility analysis of particles (< 10 µm AED in diameter) produced immediately following the impact of DU munitions found that between 24% and 43% of the total particulate load dissolved rapidly. The remainder of the particles were relatively insoluble with a predicted lung retention half time of longer than 100 days (Jette, 1990). While these data may be used to estimate the radiological dose received from inhalation of such particles immediately following impact, further study is required to assess the impact of weathering on particle solubility class.

7.5 Injury, insult and dermal sorption

Increased concentrations of urinary uranium (up to 150 times compared to those observed in control groups without fragments) have been observed in soldiers with retained DU fragments (Hooper et al., 1999). These studies also revealed a slow continuing release of uranium from individuals with fragments over one year later, suggesting a slow controlled release of DU. Parallel studies undertaken on rats have reported increased concentrations of DU in the kidneys and bone, although detectable amounts were also observed in the brain, testicles and lymph nodes of exposed animals (Pellmar et al., 1999a) (see Chapter 8 for further details). Further validation and research into the implications of this data are required.

It has been demonstrated in animal studies that soluble uranium compounds such as nitrate can be absorbed through the skin (Orcutt, 1949; DeRey et al., 1983, 1984). In studies with rabbits, death due to renal failure was observed to occur via this mode of exposure with a lowest LD_{50} value of 28 mg uranium per kg as uranyl nitrate in an ethereal solution (Orcutt, 1949). Rats and guinea pigs were observed to be significantly less sensitive. More recent studies of sub-acute dermal exposure to uranyl nitrate (typical applied concentrations 0.6 g/ml uranyl hexahydrate to skin areas of between 0.5 and 16 cm^2) by Lopez et al. (2000) confirm the observations of earlier studies of acute exposure citing histological alterations of the kidney that increased in severity with the magnitude of exposure. Parameters describing dermal absorption coefficients for various compounds have not been reported although studies indicating changes in skin permeability with exposure to uranium (thereby favouring the entry of uranium into the body) were cited by Lopez et al. (2000).

7.6 Excretion and elimination

Given the relatively low uptake of uranium from the gut it follows that most ingested *non-absorbed* uranium is excreted in faeces. Experimental evidence has shown, that once uranium enters the systemic circulation (i.e. passes through the gut wall and enters the blood) about 90% of it will be excreted through the kidneys as urine over a period of a few days. The exact proportion of uranium excreted depends upon its chemical speciation in blood. Retention of uranium in the kidney has been attributed to the creation of complexes with proteins and phospholipids in the proximal tubule (Wedeen, 1992). Faecal excretion typically accounts for less than 1% of the uranium absorbed from the gut ICRP-69, (1995a).

Clearance from the skeleton is considerably slower; half-lives of 300 and 5000 days have been estimated, based on a two-compartment model (WHO, 1998b; Kathren et al., 1989). A more in-depth discussion of the kinetics of retention following uptake into the systemic circulation is given in ICRP-69, (1995a) and/or ATSDR, (1999) to which the reader is referred.

Because of its rapid and substantial urinary excretion the concentration of uranium in urine can form the basis of assessing intake (e.g. Hooper et al., 1999; ICRP-78, 1997). Once measurements in urine have been made, biokinetic models may then be used to calculate intake. However, for inhalation, lack of information on the temporal pattern of exposure and chemical form and variation in natural excretion (e.g. Dang et al., 1992) reported urine concentrations of 0.0128 µg/l as an average for 'unexposed individuals' whereas Medley et al. (1994) observed values of 0.004 to 0.057 µg/l) can result in appreciable errors in such estimates (e.g. Stradling et al., 1998). For the specific case of DU, it may be possible to utilize differences in isotopic ratio to evaluate the upper limit of intake that corresponds to doses at the mSv level (Roth et al., 2001). Similar considerations should also apply for assessing the upper limits of DU in other tissues. These issues are discussed further in Chapters 10, 11 and 12.

For military veterans containing embedded DU, urine excretion levels of between 10 and 20 µg/l have been reported (Hooper et al. 1999). Negative finding regarding renal injury have been reported amongst such individuals (McDiarmid et al, 2000). This issue is discussed later in the following chapter. Gulf war veterans exposed to DU from inhalation, ingestion and wounds, showed average urinary excretion, 7 years post exposure, of 0.08 µg U/g creatinine, with the highest rates around 30 µg U/g (McDiarmid et al., 2000). Normal excretion of creatinine is considered to be 1.7 g/day (Jackson, 1966).

The occupational exposure decision level used for uranium workers at one facility in the United States is 0.8 µg/l of uranium in urine (FEMP, 1997). This value assumes an acute inhalation intake of moderately soluble uranium and a 60-day urine sampling frequency. For investigational purposes a value of 4 µg/l of urine is used for UK workers (information from Dr M Bailey, UK NRPB).

7.7 Accumulation

In autopsies of chronically exposed individuals, uranium has been observed in the skeleton, liver and kidneys in the average ratio of 63:2.8:1 (Kathren et al., 1989). Variations in this ratio are common and are dependent on the pattern and nature of exposure (Fisenne, 1993; ATSDR, 1999). The ratios are consistent with studies performed on mine workers and members of the public (Wrenn et al., 1985) and reflect the affinity of uranium for phosphate, which is abundant in bone. A similar distribution would be expected for DU and uranium provided the patterns of intake are comparable and the delay between cessation of exposure and autopsy are similar.

In the studies performed by Pellmar et al. (1999a) rats were surgically implanted with sterilized DU and/or tantalum pellets within the gastrocnemius muscle. As early as one month after pellet implantation and at subsequent sample times (six months), brain concentrations of uranium were statistically elevated in DU-implanted rats compared to controls implanted with tantalum (e.g. less than 2 to approximately 120 ng U/g tissue after six months implantation with 20 DU 1×2 mm pellets). The authors also observed that levels of uranium were statistically elevated in the testes of exposed animals when compared to those in the control group (e.g. less than 50 to approximately 600 ng U/g tissue after 18 months implantation with 20 DU 1×2 mm pellets). Levels of uranium in both testes and brain tissues were observed to be positively correlated with exposure (number of implanted pellets). Significant amounts of uranium were excreted in urine throughout the study (e.g. 1010 ± 87 ng uranium per ml urine in high-dose rats at 12-months exposure). The study suggests that in a rat model, uranium can accumulate

within the central nervous system and testicles. The accumulation of uranium in brain tissues has also been observed by Ozmen and Yurekli (1998).

No treatment-related effects (brain lesions) were identified during histopathological analysis of brains from animal studies performed by Gilman et al., (1998a, 1998b, 1998c).

7.8 Summary

Uranium may enter the body through the skin, lungs or gut. Once it has entered the systemic circulation it is distributed throughout the body, where it may become absorbed onto the surface of bone, accumulate or most likely be excreted through the kidneys.

Absorption via the inhalation route depends upon the size and chemical composition of the inhaled particulates and their biological solubility. While absorption through the gut and skin largely depend upon the bioavailability of the various DU compounds to which an individual has been exposed. Typical gut absorption factors for uranium in food and water are in the order of 2% for U(VI) compounds and less for the generally more insoluble compounds of U(IV). Soluble uranium and DU compounds may be absorbed through the skin.

A number of biokinetic models exist that describe and model the biokinetics of uranium and hence DU in the body. The most recent are the ICRP models for the lung, systemic circulation and gut (all summarized in Annex 4). Whilst these models describe the distribution of uranium amongst major organs, they tend to be orientated to radiological protection issues and have not addressed more recent data relating to the distribution of uranium into testes and brain.

Current gaps in knowledge include:

- Distribution (modelled and experimentally determined) of uranium at minor concentrations and in minor organs.
- Validation of animal data to man on biodistribution into brain, liver and gonads.
- Uranium distributions at a cellular level; bio-uptake of uranium derived from DU munitions throughout all exposure pathways in comparison with typical non-munition derived uranium and DU.

8 The chemical toxicity of uranium

8.1 Introduction

The chemical toxicity of a given elemental compound is related to the interaction of the compound with the biochemical processes of the human body. Some of these interactions may be beneficial or even essential, whereas others may be detrimental. For example, metal ions may interact in a positive or negative way with important functional sites of enzymes. Others may compete with essential metals for uptake or incorporation into proteins. Often the mechanisms of these effects are poorly understood particularly where different species of the same metal are likely to exist in different environments within the body across a wide variety of spatial and temporal scales. The chemical action of all isotopes and isotopic mixtures of uranium are identical, and independent of the specific activity, because chemical action depends only on chemical properties. Thus the chemical toxicity of natural, depleted, and enriched uranium are identical (ATSDR, 1999). The health effects from exposure to uranium have been recently reviewed (WHO, 1998a and b; ATSDR, 1999; Fulco et al., 2000, Durakovic`, 1999).

In the 1990s, the subjective symptoms and signs of disease among Gulf War veterans have been extensively studied, and the role of exposure to uranium as a possible underlying causative agent has been explored (Fulco et al., 2000; Harley et al., 1999b; CHPPM, 2000; Durakovic`, 1999).

The health risks caused by chemical effects of uranium exposure, that is effects not related to ionizing radiation, can be assessed using the IPCS guidelines for derivation of guidance values for health-based exposure limits (WHO, 1994), which are the basis of the risk estimates in the IPCS Environmental Health Criteria Document and Concise International Chemical Assessment Document series.

Tolerable intake (TI: usually expressed as mg/kg of body weight per day) in these guidelines is defined as ‘an estimate of the intake of a substance which can occur over a lifetime without appreciable health risk’. For chemicals like uranium, for which it is likely that a threshold below which no adverse health effect will occur, the approach used is based on a perceived No Observed Adverse Effect Level (NOAEL) or Lowest Observed no Adverse Effect Level (LOAEL) and uncertainty factors (UF).

The NOAEL is defined (WHO, 1994) ‘as the greatest concentration or amount of a substance, which causes no detectable adverse alteration of morphology, functional capacity, growth, development or life span of the target organism under defined conditions of exposure. Alterations of morphology, functional capacity, growth, development or life span of the target may be detected which are judged not to be adverse’.

In order to derive a tolerable intake from a NOAEL, the NOAEL is divided by an uncertainty factor, which is (WHO, 1994) ‘a product of several single factors by which the NOAEL or LOAEL of the critical effect is divided to derive a TI. These factors account for adequacy of the pivotal study, interspecies extrapolation, inter-individual variability in humans, adequacy of the overall database, and nature of toxicity’.

Components of the applied total uncertainty factor are based on ‘best judgement’ from available data; when no adequate data exist for a specific factor, a default value is used. For example, for the extrapolation between species, the default uncertainty factor is 10,

which is composed of factors of 4.0 for toxico-kinetic and 2.5 for toxico-dynamic uncertainties. Similarly in the assessment of uncertainties associated with extrapolation between various human sub-populations (comprising all age groups, and healthy as well as sick people) a default inter-individual human uncertainty factor of 10 has been recommended (WHO, 1994). Combination of these factors leads to a total default uncertainty factor of 100. Other uncertainty factors may be applied to account for inadequacies in the database and/or critical study (WHO, 1994).

The consideration of uncertainty factors described above relates primarily to exposure of a general population. While similar principals may be used to estimate tolerable daily intakes for occupational exposure this has not gained general acceptance (WHO, 1994). Two reasons for this are:

(i) that the more vulnerable members of the human population (children, the sick and the elderly) do not form part of the generally exposed occupational population.

(ii) that workplace exposures can be controlled and monitored.

Methodologies for deriving occupational exposure limits for uranium and DU are presented separately in Chapter 10 and in Annex 5.

8.2 Toxicity in experimental animals and humans

8.2.1 Experimental animals

Inhalation

The dose response behaviour of a specific inhaled substance is highly dependent on the particle size distribution and chemical nature of a given inhalation experiment. While some useful data in this respect is given in the early literature, in almost all cases a significant amount of such contextual data relating to the conditions of the exposure are lacking. Because of these factors it is difficult in the case of inhalation to interpret LOAEL and NOAEL data solely from the quantities and chemical form of inhaled material. Because of this, supplementary data giving concentrations for a specific target organ is especially useful when deriving a LOAEL or NOAEL for that particular organ. Such data is tabulated later in this Chapter for the kidney in animal experiments.

Pulmonary toxicity of uranium varies among species and is dependent on the chemical form of uranium (Tannenbaum et al., 1951). Mortality can be induced in rats and guinea pigs at high concentrations of uranium hexafluoride (about 26 to 35 mg U/m^3). The cause of acute death is apparently irritative damage to the respiratory tract and this is probably due not to uranium but to hydrofluoric acid, a hydrolysis product of uranium hexafluoride (Spiegel, 1949; Leach et al., 1984) although mortality may be due to kidney effects. Pulmonary edema, haemorrhages, inflammation and emphysema were also observed in rats, mice and guinea pigs after 30 days exposure to 13 mg U/m^3 as uranium hexafluoride (Spiegel, 1949).

Slight degenerative changes in lung histology were observed in rats and dogs exposed to uranium trioxide and dogs exposed to uranyl nitrate hexahydrate at exposure levels of approx. 10 mg U/m^3 for 4 to 5 weeks, but not after similar exposure to uranium dioxide or triuranium octaoxide (Roberts 1949; Rothstein 1949; Dygert 1949).

Rabbits are sensitive to uranium-induced pulmonary damage: pulmonary edema and haemorrhages were reported after exposure to ammonium diuranate, uranium peroxide, uranium trioxide, and carnotite, but not after exposure to uranium dioxide (Dygert, 1949; Rothstein, 1949; Pozzani, 1949).

In long-term studies, with exposure up to one year with several animal species (rats, rabbits, guinea pigs, hamsters and dogs) and various uranium compounds (soluble and insoluble), no signs of pulmonary changes were observed in a concentration range of 0.05 to 10 mg U/m^3 (Cross et al., 1981a, 1981b). Chronic exposure of rats, dogs and monkeys to 5 mg U/m^3 for 1 to 5 years, as uranium dioxide did not reveal histological changes in the lung nor damage to the kidneys. A post-exposure follow-up study showed slight interstitial and vascular fibrosis in dogs and some pulmonary fibrosis in monkeys (Leach et al., 1970, 1973). However, investigators stated that radiation rather than chemical toxicity was believed to have caused the injuries noted.

Renal effects can be produced in animals after acute-duration and intermediate-duration inhalation exposures to uranium. A 10-minute exposure to 675 mg U/m^3 as uranium hexafluoride produced severe degeneration of the cortical tubules 5 to 8 days later in rats (Spiegel, 1949). These same effects were observed in dogs 1 to 3 days after a 1-hour exposure to 250 mg U/m^3 as uranyl fluoride (Morrow et al., 1982). Proteinuria and glucosuria were also observed in rats after 2 to 10-minute exposures to uranium hexafluoride (Leach et al., 1984).

In intermediate-duration studies with guinea pigs, mice, rats, cats, rabbits, and dogs, inhalation exposures to a variety of uranium compounds were damaging to the kidneys. The effects were compound-dependent and concentration-dependent and ranged from minimal microscopic lesions in tubular epithelium (for low concentrations) to severe necrosis of the tubular epithelium (for high concentrations) in several species (Dygert, 1949; Pozzani, 1949; Roberts, 1949; Rothermel, 1949; Spiegel, 1949; Stokinger et al., 1953). In one of these studies, mice were exposed to uranium tetrachloride dust for 30-days. The exposure resulted in severe degeneration and necrosis of the renal-cortical tubular epithelium, and mortality, in the 11 mg U/m^3 group by the third day. At the end of the study, moderate tubular degeneration was observed in the 2.1 mg U/m^3 group and minimal degeneration in the 0.1 mg U/m^3 group.

The nephrotoxic effects of uranium in animals may include damage to the glomerulus as evidenced by histopathological signs in the kidneys of rats and rabbits exposed to 15.4 mg U/m^3 as uranium dioxide for 23 days (Dygert, 1949) and dogs exposed to 15 mg U/m^3 as uranyl fluoride for five weeks and to 16 mg U/m^3 as uranium trioxide for four weeks (Rothstein, 1949).
In long-term inhalation studies with rats and dogs, soluble and insoluble uranium exposures as low as 0.05 mg U/m^3 and as high as 10 mg U/m^3 for 1 to 5 years were damaging to the kidneys. Nephrotoxic effects found in these animals ranged from minimal microscopic lesion in tubular epithelium (for low concentrations) to acute tubular necrosis (for high concentrations) (Leach et al., 1970; Stokinger et al., 1953). For further comments on issues regarding inhalation toxicity, its incorporation into ICRP methodologies and occupational exposure standards see Annex 5.

Oral

The derivation of dose response data from ingestion is less dependent on experimental conditions and toxio-kinetics than that derived from inhalation. Making the commonly

used derivation of TDI values directly from concentrations of ingested material less subject to uncertainty.

The oral toxicity of uranium compounds has been evaluated in several animal species. Oral LD_{50} (dose producing 50% mortality rate) values of 114 and 136 mg U/kg have been estimated for rats and mice, respectively, following single gavage administrations of uranyl acetate dihydrate (Domingo et al., 1987).

Rats exposed to a single average dose of 5.6 mg U/kg suffered slight renal dysfunction and minimal microscopic lesions in the tubular epithelium (Domingo et al., 1987, 1989a). In intermediate-duration animal studies, exposure to uranium (uranyl fluoride, triuranium octaoxide, uranyl nitrate hexahydrate, uranium tetrachloride, uranium peroxide, ammonium diuranate) at oral doses as low as 0.05 mg U/kg/day and as high as 7858 mg U/kg/day for 30 days were damaging to the kidneys. Nephrotoxic effects found in these animals ranged from minimal microscopic lesions in the tubular epithelium (for low doses) to extensive necrosis in the tubular epithelium (for high doses of soluble compounds) (Maynard and Hodge, 1949).

Rats exposed to uranium as uranyl nitrate in drinking water for 91 days were found to have renal lesions of the tubules, glomeruli, and interstitium observed in the lowest exposure groups (males 0.06 mg U/kg/day; females 0.09 mg U/kg/day) (Gilman et al., 1998a). The studies by McDonald-Taylor et al. (1992, 1997) produced similar renal lesions (thickened glomelular basement membrane) in rabbits.

For various endpoints and animal species ATSDR reported minimal effect levels in the range of 1 to 10 mg/kg of body weight per day (ATSDR, 1999). For example Ortega, (1989) observed adverse effects with rats at exposure levels, via ingestion, of 1.1 mg/kg per day. For cattle and sheep Puls (1990) reported that minimal effects are associated with a daily uranium intake of 400 or 50 mg U, respectively (corresponding to 1mg U/kg of body weight for both species).

For rabbits exposed to uranium as uranyl nitrate in drinking water for 91 days, dose-dependent histopathological changes were primarily limited to the kidney. Dose-dependent differences consisted of histopathological changes limited primarily to kidney; changes were more pronounced in male rabbits (Gilman et al., 1998b). In another study, male New Zealand rabbits were exposed to uranium as uranyl nitrate in drinking water for 91 days, and were then allowed to recover for several weeks (Gilman et al., 1998c). The lowest-observed-adverse-effect-levels (LOAELs) in these studies were 0.05 mg U/kg/day for non-Pasteurella free rabbits and 0.49 mg U/kg/day for Pasteurella free rabbits. While not being essential for deriving a tolerable intake, data relating to the concentration of uranium in kidney and bone is useful for linking LOAELs to organ-specific data produced from biokinetic models, commonly used for assessing radiological and chemical effects in radiological protection (e.g. Spoor and Hursh, 1973; also see Chapters 10, 12 and Annex 5). This data is summarized from the recent studies by Gilman et al. (1998a, 1998b, 1998c) in Table 8.1.

Table 8.1 Kidney and bone concentrations observed in experiments performed by Gilman et al., during the 1990s.

Study	SEX/Type	LOAEL mg U/kg body wt / day	Kidney µg/g	Bone µg/g
1	M Rat	0.060	<0.2	<1.78
	F Rat	0.090	<0.2	<1.78
2	M Rabbit	0.050	0.04 ± 0.03	0.09 ± 0.05
	F Rabbit	0.490	0.019 ± 0.01	0.053 ± 0.004
3	M Rabbit	<1.360	0.18 ± 0.13	0.20 ± 0.05
	F Rabbit	<1.360	0.18 ± 0.13	0.20 ± 0.05

1 Gilman et al. (1998a) 91 day experiment Sprague-Dawley Rat
2 Gilman et al. (1998b) 91 day experiment New Zealand White Rabbits (Specific Pathogen Free (SPF) derived)
3 Gilman et al. (1998c) 91 day experiment New Zealand White Rabbits (SPF)

The pathogenesis of the kidney damage in animals indicates that regeneration of tubular epithelium occurs in survivors upon discontinuation of exposure to uranium (Bentley et al., 1985; Dygert, 1949; Maynard and Hodge, 1949; Pozzani, 1949; Rothemel, 1949; Rothstein, 1949; Spiegel, 1949; Stokinger et al., 1953).

Leggett (1989) cites that tolerance may develop following repeated exposure to uranium, but this tolerance does not prevent chronic damage to the kidney, as the regenerated cells are quite different. Persistent changes in the proximal tubules of rabbits have been reported to be associated with the kidney's ability to store uranium (McDonald-Taylor et al., 1997). In another study Gilman et al. (1998c) describes a recovery study performed on New Zealand White Rabbits exposed to uranium nitrate (24 or 600 mg/l corresponding to 1.4 mg U/kg body wt/day and 41 mg U/kg body wt/day respectively) for 91 days. Renal tubular injury with degenerative nuclear changes, cytoplasmic vacuolation, and tubular dilation were seen in the high dose group without consistent resolution even after 91 days. Kidney concentrations observed in the high exposure group decreased from 3.48 ± 1.54 to 0.02 ± 0.01 µg/g over the 91 day recovery period in an exponential manner.

Reproductive and developmental toxicity In several studies with mice given soluble uranium compounds (uranyl nitrate hexahydrate, uranyl acetate dihydrate), the teratogenic, embryotoxic and reproductive effects of uranium have been studied (Domingo, 1989a,1989b). Exposure-related fetotoxicity, reduced fetal body weights, external and internal malformations, increased incidence of developmental variations, and decreased fertility were observed. In rats, unspecified degenerative changes in the testes have been reported following chronic administration of uranyl nitrate hexahydrate and uranyl fluoride in the diet (Maynard and Hodge, 1949; Maynard et al., 1953; Malenchenko et al., 1978).

Carcinogenicity Although bone cancer has been induced in experimental animals by injection or inhalation of soluble compounds of high-specific-activity uranium isotopes or mixtures of uranium isotopes, no carcinogenic effects have been reported in animals ingesting soluble or insoluble uranium compounds (Wrenn et al., 1985). However, given the nature of ionizing radiation damage to DNA, retention of any radioactive material in the body will have associated an increase in the probability of cancer; albeit small and depending on the radiation dose.

8.2.2 Implanted depleted uranium fragments

The chronic long-term health consequences of exposure to depleted uranium (DU) fragments have been addressed by Benson and Schnieder (1998) and Pellmar et al. (1999a).

Pellmar et al. (1999a) undertook studies in rats surgically implanted with sterilized DU and/or Tantalum pellets (see Chapter 7). The results of these studies concluded that in a rat animal model, uranium could accumulate within the central nervous system and testicles. A follow-up study by the same group (Pellmar et al., 1999b) assessed the potential for electrophysiological changes in the hippocampus of rats implanted with DU fragments. At 12 months, the amplitudes of synaptic potentials were significantly greater in tissues derived from high-dose DU-implanted rats compared with controls. But, in the same animal model, uranium did not affect locomotive activity, discrimination learning, or the results of a battery of general functional measures (Pellmar et al., 1997), which makes it difficult to interpret the significance of uranium accumulation in the brain. No nephrotoxicity was observed in these animals or in studies of kidney function performed in female rats implanted with depleted uranium pellets for a period of 84 days (Benson and Schneider, 1998). These observations are markedly inconsistent with observations made on rats and other mammals in which exposure to uranium occurred through oral ingestion (e.g. those discussed in 8.2.1 above).

8.2.3 Dermal absorption

Soluble uranium compounds such as nitrate can be absorbed through the skin (Orcutt, 1949; DeRey et al., 1983, 1984). In studies with rabbits, death due to renal failure was observed to occur via this mode of exposure with a lowest LD_{50} value of 28 mg U/kg as uranyl nitrate in an ethereal solution (Orcutt, 1949). Rats and guinea pigs were observed to be significantly less sensitive. In the specific case of acute exposure of animals to uranyl nitrate, penetration into the intracellular space between the granular and horny layers of the skin was observed to occur within a period of 15 minutes; after 48 hours no residual uranium was observed in the skin (DeRey et al., 1983). These authors considered this to be due to absorption of uranium into the systemic circulation resulting in weight loss and, in severe cases, death. More recent studies of sub-acute dermal exposure to uranyl nitrate (typical applied concentrations 0.6 g/ml uranyl hexahydrate to skin areas of between 0.5 and 16 cm^2) by Lopez et al. (2000) confirm the observations of earlier studies of acute exposure. In these studies histological alterations of the kidney that increased in severity with the magnitude of exposure were noted along with a dose-dependent reduction in bone volume and bone alteration. Parameters describing dermal absorption coefficients for various compounds have not been reported although studies indicating changes in skin permeability with exposure to uranium (thereby favouring the entry of uranium into the body) have been reported. For example Ubios et al. (1997) have determined that application of acute levels of soluble uranium compounds (i.e. 0.012 g U/day) to the skin can significantly reduce the thickness of the epidermis (41 ± 14 to 21 ± 10 µm). Such thinning of the epidermis was also observed to be present 60 days after the cessation of a 31 day, daily application regime. Results of these tests were considered by the authors to be due to the chemical rather than radiological effects of U.

8.2.4 Humans

Inhalation

Inhalation of dusts of various uranium compounds will have different chemical toxicity depending mainly on the biological solubility and chemical reactiveness with body tissues.

Despite evidence indicating lethal effects of uranium to various animals (including rabbits, which appear to be particularly sensitive to the toxic effects of uranium), epidemiological studies indicate that routine exposure to airborne uranium is not associated with increased mortality (ASTDR, 1999). Brief accidental exposure to high concentrations of uranium hexafluoride has caused acute respiratory illness, which may be fatal. However, this is most likely to be due to the hydrogen fluoride liberated from uranium hexafluoride upon hydrolysis.

In studies on uranium miners and those mining other ores (e.g. tin and iron), an increased risk of lung cancer has been attributed mainly to exposure to radon decay products (see Chapter 9.4). Studies of these underground miners indicate that risks of a few other types of cancer and of chronic respiratory disease might be increased, although not due to radon (Darby et al., 1995; NRC, 1999; Fulco et al., 2000). However, it is unclear which out of the other toxicants in mines (including engine exhausts, silica, nickel oxide, cobalt oxide, vanadium pentaoxide, inhalable dust particles and uranium) might be relevant to the aetiology of these diseases

Studies of uranium workers other than miners while reducing the importance of these confounders may also be sensitive to other data inadequacies and/or confounders. For example the largest exposures are likely to have occurred in the 1940s and 1950s at a time when safety requirements were less stringent, record keeping by employers was poorer and testing was less commonplace. Similar deficiencies exist in the accuracy by which various health outcomes (i.e. nephritis) are codified and/or recorded, although generally this is less of a problem for cancer than for other diseases. In over 10 studies on such workers no excess of respiratory cancer or non-malignant respiratory disease has been established in relation to uranium exposure. (ATSDR, 1999 studies also cited and reviewed in Fulco et al., 2000). However, the statistical power of these studies was generally low. In particular, Fulco et al. (2000) concluded that there was limited/suggestive evidence of no association between exposure to uranium and lung cancer for cumulative internal doses below 200 mSv, but that there was inadequate/insufficient evidence to determine whether or not there is an association at higher doses. Fulco et al. (2000) also concluded that there was inadequate/insufficient evidence to determine whether or not there is an association between uranium and either lymphatic cancer or bone cancer.

Several epidemiological studies have found no increased mortality in uranium workers due to renal disease (Archer et al., 1973a, 1973b; Brown and Bloom, 1987; Checkoway et al., 1988; Polednak and Frome, 1981). Also, case studies showed that workers accidentally exposed to high levels of uranium did not suffer renal damage, even up to 38 years post-exposure (Eisenbud and Quigley, 1956; Kathren and Moore, 1986), although the tests for renal damage used in these studies were not very sensitive. A recent comparison of kidney tissue obtained at autopsy from seven uranium workers and six control subjects with no known exposure to uranium showed that the groups were indistinguishable by pathologists experienced in uranium-induced renal pathology

(Russell et al., 1996). One study on the kidney function of uranium mill workers chronically exposed to insoluble uranium (uranium dioxide) revealed renal tubular dysfunction as manifested by mild proteinuria, aminoaciduria, and a concentration-related clearance of $\$_2$-microglobulin relative to that of creatinine when compared to a reference group of cement workers. The incidence and severity of these nephrotoxic signs correlated with the length of time that the uranium workers had spent in the area where insoluble uranium oxide yellowcake was dried and packaged (Saccomanno, 1982; Thun et al., 1985). The data from this study are indicative of reduced re-absorption in the proximal renal tubules.

Delayed renal effects were observed after a male worker at a uranium enrichment plant was accidentally exposed to a high concentration of uranium tetrafluoride powder for about five minutes in a closed room (Lu and Zhao, 1990). Renal effects were not observed in another accidental exposure (Fisher et al., 1990) in which 24 of 31 workers were followed for two years. However, an increased standardized mortality rate has been observed for chronic nephritis amongst a 2514-strong cohort of uranium processing workers, although this was based on just six deaths and was not statistically significant (Dupree-Ellis et al., 2000).

Oral

Few data are available that adequately describe the dose-response toxicity of uranium after an oral exposure in humans. Although the negative findings regarding renal injury among workers exposed over medium to long time periods to insoluble compounds (McDiarmid et al., 2000 and Eisenbud and Quigley, 1956) and shorter periods of exposure to relatively soluble uranium compounds (Kathren and Moore, 1986) are particularly significant in view of the high levels of exposure reported in these studies.

A recent review of human toxicity undertaken by Fulco et al. (2000) covering both epidemiological and experimental studies concludes that ‘although uranium is a heavy metal that causes transient renal dysfunction, the preponderance of evidence indicates little or no clinically important renal effects of exposure to uranium’. However, at least two studies have shown changes in renal function (e.g. Lu and Zhao, 1990 and Zamora et al., 1998). Whilst, Dupree-Ellis et al. (2000) have recently reported an increased rate of chronic nephritis and a dose-response relationship between external radiation and kidney cancer amongst a cohort of 2514 uranium processing workers, these findings are based on small numbers of deaths and neither increase is statistically significant. As pointed out by Dupree-Ellis et al. (2000) potential inaccuracies in data, even amongst a relatively large cohort, can lead to a significant degree of uncertainty in the interpretation of epidemiological data.

In the study by Lu and Zhao (1990) delayed renal effects were observed after a male worker at a uranium enrichment plant was accidentally exposed to a high concentration of uranium tetrafluoride powder for about five minutes in a closed room. A trend towards increasing excretion of urinary $\$_2$-microglobulin, as indicator for an early tubular defect, and increasing concentration of uranium in well-water was observed during clinical studies performed in Canada by Moss et al. (1983). Although it was suggested that the suspected tubular defect might well be rapidly reversible. Elevated levels of protein and $\$_2$-microglobulin have also been observed in the urine of uranium mill workers (study of 39 exposed individuals to 36 unexposed controls) and presented data was considered to be consistent with uranium nephrotoxicity (Thun et al., 1985). In

a further study, a statistically significant association (p = 0.03) was observed between increasing but normal levels of urine albumin and the uranium cumulative index (Mao, 1995).

In the study of Zamora et al. (1998) two groups of biomarkers were used as indicators of kidney function and cellular toxicity between two communities. One community (the control) was supplied with well-mixed mains water containing typically less than 1 µg U/l, whereas the other community represented exposed individuals whose water supply contained between 2 and 780 µg U/l. The total number of individuals partaking from each community was 20 and 30, respectively. Estimated total intakes of uranium in each group (including uranium from drinking water) were 0.3 to 20 and 3 to 570 µg/day. Urinary glucose was found to be significantly different and correlates positively with uranium intake for males, females, and pooled data. Increases in alkaline phosphatase and $$_2$-microglobulin were also observed to be correlated with uranium intake for pooled data. In contrast, the indicators for glomerular injury, creatinine and protein were not significantly different between the two groups nor was their urinary excretion correlated to uranium intake. Because of the uncertainty of the clinical significance and possible reversibility of the observed changes, their clinical significance has been questioned (e.g. Harley et al., 1999b; Fulco et al., 2000). In addition, a possible role of confounding factors such as other compounds in drinking water has not been clarified.

Embedded DU fragments

Dipino et al. (1998) compared five measurements of premorbid intellectual functioning amongst a group of patients injured by DU munitions during the Gulf War. Unfortunately only inter-group comparisons were made and it was considered impossible to compare scores with those suffering from injuries not associated with depleted uranium.

McDiarmid et al. (2000) studied a cohort of Gulf War veterans who had fragments of depleted uranium in their soft tissues. Results from a battery of computer-based neurocognitive tests suggest a statistical relationship between elevated urinary uranium levels and 'problematic performance on automated tests assessing performance efficiency and accuracy' (McDiarmid et al., 2000). Traditional tests of neurocognitive function (pen-and-pencil tests) did not show any statistical difference in performance between the veteran cohort and a control group. However, as discussed by the Committee on Health Effects Associated with Exposure During the Gulf War (Fulco et al., 2000), because of methodological problems, it is difficult to draw firm conclusions from this study. Kidney function was normal in Gulf War veterans with embedded DU fragments, years after exposure, despite urinary uranium concentration up to 30.7 µg U/g creatinine (McDiarmid et al., 2000).

8.3 In-vitro studies

In-vitro studies on human osteoblast cells have indicated that they may be transformed to the tumorigenic phenotype (i.e. exhibiting morphological changes, anchorage independent growth in soft agar, induction of tumours when implanted into nude mice, and differences in *ras* oncogene expression and pRb phosphorylation) by DU administered as uranyl chloride (Miller et al., 1998a). The authors considered this transformation to be primarily due to chemical rather than radiological effects; such as the interaction of uranium with phosphorus-containing groups in DNA, and consider the magnitude of activity to be similar to that observed for nickel sulphate and lead acetate (both known transforming metals).

Uranyl nitrate was cytotoxic and genotoxic in Chinese hamster ovary cells. There was a dose-related decrease in the viability of the cells, a decrease in cell cycle kinetics, and increased frequencies of micronuclei, sister chromatid exchanges and chromosomal aberrations (Lin et al., 1993). The genotoxic effects in this study were thought to have occurred through the binding of the uranyl nitrate to the phosphate groups of DNA. It was suggested that these results provide a possible mechanism for the observed teratogenic effects (WHO, 1998b). Miller et al. (1998b) also observed mutagenic activity (Ames *Salmonella* reversion assay) in the urine of rats implanted with DU pellets (in muscle tissue), although no significant mutagenicity was observed in serum.

In-vivo and *in-vitro* testing on the clam *Corbicula fluminea*, the worm *Eisenia fetida andrei* and the teleost fish *Brachydanio rerio* by (Labrot et al., 1996) was used to determine if changes in the activities of various biomarkers (lipid peroxidation, acetylcholinesterase, catalase and glutathione peroxidase) and other postmitochondrial fractions could be identified. Results of these studies indicated that exposure to uranium resulted in an increase in malondialdehyde (an indicator of oxidative stress) whereas no increase in lipid peroxidation was observed *in-vivo*. With some exceptions exposure to uranium was also observed to result in decreased activities of acetylcholinesterase, catalase and glutathione peroxidase in all three species. However acute toxicity was only observed in the case of *Eisenia fetida andrei* and *Brachydanio rerio.*

8.4 Derivation of a tolerable intake for uranium

The renal toxicity of various uranium species given in repeated oral doses is given in Table 8.2 and in repeated inhalation is given in Table 8.3 (from ATSDR, 1999).

8.4.1 Soluble uranium compounds (group F: $UO_2(NO_3)_2$, uranium carbonates)

Oral A LOAEL has been established as 50 µg/kg in male rabbits in 91-day studies (Table 8.2) with the end point being slight microscopical changes in the kidney. From this, a TI can be derived using a 100 fold total uncertainty factor (3 for LOAEL to NOAEL, 3 for toxico-dynamic and toxico-kinetic differences between species (comparative data from animals and humans indicate that the absorption in humans is no greater than in animals; therefore a full 10 interspecies uncertainty factor is not applied), and 10 for intraspecies variation. As uranium seems to have a biological half life (time for half the material to be eliminated from the body) of approximately 15-days, steady state already having been reached in the 91 day study, and no adjustment for subchronic to chronic studies is required (WHO, 1998a). Thus the TI is 0.5 µg/kg bw/d.

Inhalation A NOAEL can be approximated as 0.1 mg/m^3 from several medium- and long-term studies (Table 8.3). Adjusting this to the exposure difference in the experimental studies and general population exposure (24 hour exposure rather than 5 to 6; 7 day/week rather than 5.5 to 6 day/week), this gives an effective air concentration of 20 µg/m^3. As the inhalation volume of rat is 0.1 l/min, i.e., 150 l/d, this means an inhalation dose of 15 µg/d, which, using 250 g as an average weight of a rat, translates to 60 µg/kg/d. Using the default uncertainty factor of 100 (10 for interspecies and 10 for intraspecies variation) as no information is available for inhalation exposure that would allow use of a specific uncertainty factor, this means a TI of 0.6 µg/kg/d, which is in good agreement with the figure derived from the oral studies above.

Table 8.2. Renal toxicity (chemical) of uranium species in repeated doses –oral (ATSDR, 1999)

	Reference Chemical Form	Species	Exposure /duration /frequency	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Reference
F	UO2(NO3)2*6H2O	Rat	28d W	35.3 M	40.0 F	Gilman et al. 1998a
F	UO2(NO3)2*6H2O	Rat	30d F	3.3	16.6	Maynard & Hodge 1949
F	UO2(NO3)2*6H2O	Rat	91d W		0.06M, 0.09F	Gilman et al. 1998a
F	UO2(NO3)2*6H2O	Rat	2y F	16.6	33	Maynard & Hodge 1949, Maynard et al. 1953
F	UO2(NO3)2*6H2O	Rabbit	30d F		2.8	Maynard & Hodge 1949
F	UO2(NO3)2*6H2O	Rabbit	91d W		0.05M, 0.49F	Gilman et al. 1998b
F	UO2(NO3)2*6H2O	Rabbit	91d W		1.36 M	Gilman et al. 1998c
F	UO2(NO3)2*6H2O	Rabbit	91d W		0.93 M	McDonald-Taylor et al. 1992, 1997
F	UO2(NO3)2*6H2O	Dog	138d F	47	95	Maynard & Hodge 1949
F	UO2(NO3)2*6H2O	Dog	1y F	47	95	Maynard & Hodge 1949
F	UO2F2	Rat	30d F	5.4	27	Maynard & Hodge 1949
F	UO2F2	Mouse (C3F	48w ad lib F		452 M	Tannenbaum & Silverstone 1951
F	UO2F2	Mouse (DB/	48w ad lib F		452 M	Tannenbaum & Silverstone 1951
F	UO2F2	Dog	30d 6d/w F	7.7	15.4	Maynard & Hodge 1949
F	UO2F2	Dog	1y F	8		Maynard & Hodge 1949, Maynard et al. 1953
M	UCl4	Rat	30d F	88	438	Maynard & Hodge 1949
M	UCl4	Dog	30d 6d/w F	63	313	Maynard & Hodge 1949
M	UCl4	Dog	1y F	6.3	31	Maynard & Hodge 1949, Maynard et al. 1953
M	UF4	Dog	30d 6d/w F		3790	Maynard & Hodge 1949
M	UF4	Rat	2y F	1061	10611	Maynard & Hodge 1949, Maynard et al. 1953
M	UO3	Rat	30d F	11650M		Maynard & Hodge 1949
M	UO3	Dog	30d 6d/w F		83	Maynard & Hodge 1949
S	U3O8	Dog	30d 6d/w F		5653	Maynard & Hodge 1949
S	UO2	Rat	30d F	12342		Maynard & Hodge 1949
S	UO2	Dog	30d 6d/w F	17.6	441	Maynard & Hodge 1949
S	UO2	Rat	2y F	12341		Maynard & Hodge 1949, Maynard et al. 1953
	(NH4)2U2O7	Dog	30d 6d/w F		38	Maynard & Hodge 1949
	Na2U2O7	Dog	30d 6d/w F		37	Maynard & Hodge 1949
	UO2(C2H3O2)2*2H2O	Rat	30d F	786 M	7858 M	Maynard & Hodge 1949
	UO2(C2H3O2)2*2H2O	Rat	4w W		1.1 M	Ortega et al. 1989a
	UO4	Rat	30d F	55	138	Maynard & Hodge 1949
	UO4	Dog	30d 6d/w F		15.4	Maynard & Hodge 1949

Type: This reflects the absorption rate of uranium compounds in the lung. F (fast)=rapid, almost total, absorption into the blood usually within 10 minutes, M (moderate)=about 70% of deposited material reaches blood eventually, S (slow)=about 10% reaches blood eventually (ICRP 66, 1994)

Exposure/duration/frequency: y=year(s), w=week(s), d=day(s), h=hour(s), ad lib=ad libitum, F=food, W=water
NOAEL=no-observed-adverse-effect level, LOAEL=lowest-observed-adverse-effect level, M=male, F=fe,male

Table 8.3. Renal toxicity (chemical) of uranium species in repeated doses – inhalation (ATSDR, 1999)

Type	Reference Chemical Form	Species	Exposure/duration/ /frequency	NOAEL (mg U/m3)	LOAEL (mg U/m3)	Reference
F	UF6	Rat	30d 6h/d	0.2		Spiegel 1949
F	UF6	Rat	1y 5.5d/w 6h/d	0.05	0.2	Stokinger et al. 1953
F	UF6	Mouse	30d 6h/d	2	13	Spiegel 1949
F	UF6	Gn Pig	30d 6h/d	2	13	Spiegel 1949
F	UF6	Gn Pig	36w 5.5d/w 6h/d	0.2		Stokinger et al. 1953
F	UF6	Rabbit	36w 5.5d/w 6h/d		0.25	Stokinger et al. 1953
F	UF6	Dog	1y 5.5d/w 6h/d		0.05	Stokinger et al. 1953
F	UO2(NO3)2*6H2O	Rat	30d Cont.		0.13	Roberts 1949
F	UO2(NO3)2*6H2O	Rat	1y 5.5d/w 6h/d	0.15	0.25	Stokinger et al. 1953
F	UO2(NO3)2*6H2O	Rat	2y 5.5d/w 6h/d		2	Stokinger et al. 1953
F	UO2(NO3)2*6H2O	Gn Pig	26w 5.5d/w 6h/d	2 M		Stokinger et al. 1953
F	UO2(NO3)2*6H2O	Rabbit	30d Cont.		0.13	Roberts 1949
F	UO2(NO3)2*6H2O	Rabbit	26w 5.5d/w 6h/d		0.25	Stokinger et al. 1953
F	UO2(NO3)2*6H2O	Dog	30d Cont.		0.13	Roberts 1949
F	UO2(NO3)2*6H2O	Dog	1y 5.5d/w 6h/d	0.15	0.25	Stokinger et al. 1953
F	UO2(NO3)2*6H2O	Dog	1y 5.5d/w 6h/d	0.15	0.25	Stokinger et al. 1953
F	UO2(NO3)2*6H2O	Dog	2y 5.5d/w 6h/d		2	Stokinger et al. 1953
F	UO2F2	Rat	5w 6d/w 6h/d	0.5	2.2	Rothstein 1949a
F	UO2F2	Gn Pig	5w 6d/w 6h/d	2.2	9.2	Rothstein 1949a
F	UO2F2	Cat	5w 6d/w 6h/d	2.2	9.2	Rothstein 1949a
F	UO2F2	Dog	5w 6d/w 6h/d		0.15	Rothstein 1949a
M	UCl4	Rat	1y 5.5d/w 6h/d		0.2	Stokinger et al. 1953
M	UCl4	Gn Pig	30w 5.5d/w 6h/d		0.2	Stokinger et al. 1953
M	UCl4	Dog	1y 5.5d/w 6h/d	0.05	0.2	Stokinger et al. 1953
M	UF4	Rat	30d 6h/d	4	18	Dygert 1949a
M	UF4	Rat	1y 5.5d/w 6h/d		0.5	Stokinger et al. 1953
M	UF4	Gn Pig	30d 6h/d	4	18	Dygert 1949a
M	UF4	Gn Pig	34w 5.5d/w 6h/d		3	Stokinger et al. 1953
M	UF4	Dog	30d 6h/d	0.5	3	Dygert 1949a
M	UF4	Rabbit	30d 6h/d		0.4	Dygert 1949a
M	UF4	Rabbit	34w 5.5d/w 6h/d		2	Stokinger et al. 1953
M	UF4	Cat	30d 6h/d		18	Dygert 1949a
M	UO3	Rat	4w 6d/w 6h/d	16		Rothstein 1949c
M	UO3	Rabbit	4w 6d/w 6h/d		16	Rothstein 1949c
M	UO3	Cat	4w 6d/w 6h/d		16	Rothstein 1949c
M	UO3	Dog	4w 6d/w 6h/d		16	Rothstein 1949c
S	U3O8	Rat	26d 4-6h/d		4.8	Dygert 1949c
S	UO2	Rat	1y 5.5d/w 6h/d	1	10	Stokinger et al. 1953
S	UO2	Mouse	5w 6d/w	19.4		Rothstein 1949b
S	UO2	Gn Pig	28w 5.5d/w 6h/d	10		Stokinger et al. 1953
S	UO2	Rabbit	5w 6d/w	9.2	19	Rothstein 1949b
S	UO2	Rabbit	30w 5.5d/w 6h/d		1	Stokinger et al. 1953
S	UO2	Dog	5w 6d/w	1.1	8.2	Rothstein 1949b
S	UO2	Dog	1-5y 5d/w 5.4h/d	5.1		Leach et al. 1970
S	UO2	Dog	1-5y 5d/w 5.4h/d	5.1		Leach et al. 1973
S	UO2	Monkey	5y 5d/w 5.4h/d	5.1		Leach et al. 1970
S	UO2	Monkey	1-5y 5d/w 5.4h/d	5.1		Leach et al. 1973
	(NH4)2U2O7	Rat	30d 6h/d		6.8	Dygert 1949b
	(NH4)2U2O7	Rabbit	30d 6h/d		6.8	Dygert 1949b
	Carnotite U ore	Mouse	30d 4.4-6h/d		2.9	Pozzani 1949
	Carnotite U ore	Gn Pig	30d 4.4-6h/d	0.8	2.9	Pozzani 1949
	Carnotite U ore	Dog	30d 4.46h/d		0.8	Pozzani 1949
	Carnotite U ore	Rabbit	30d 4.4-6h/d	0.8	2.9	Pozzani 1949
	Na2U2O7	Rat	5w 5.5d/w 6h/d		15	Rothstein 1949d
	Na2U2O7	Rabbit	5w 5.5d/w 6h/d		15	Rothstein 1949d
	UO4	Rabbit	23d 5d/w 5h/d		15.4	Dygert 1949d
	UO4	Cat	23d 5d/w 5h/d		15.4	Dygert 1949d

Type: This reflects the absorption rate of uranium compounds in the lung. F (fast)=rapid, almost total, absorption into the blood usually within 10 minutes, M (moderate)=about 70% of deposited material reaches blood eventually, S (slow)=about 10% reaches blood eventually (ICRP 66, 1994)

Exposure/duration/frequency: y=year(s), w=week(s), d=day(s), h=hour(s), ad lib=ad libitum
F=food,W=water. M=male, F=female
NOAEL=no-observed-adverse-effect level, LOAEL=lowest-observed-adverse-effect level.

8.4.2 Uranium compounds with limited solubility (Type M: UO_3, UF_4, UCl_4)

The database is rather limited, and it is not apparent that there is a justification for the generation of this type: differences within the group are large, and not much different from those between this group and the other two types, notably when the exposure is by inhalation. This is, however, in part, due to the fact that there is a major difference in the toxic potency observed in the early studies, and more recent studies: the recent studies observe effects at levels that are generally lower than those where effects are reported in the old studies. For the Type M uranium species, no recent studies are available. It does not seem justified to derive a separate TI for the Type M; rather, the TI derived for Type F should be used.

8.4.3 Uranium types practically insoluble in water (Type S: UO_2, U_3O_8)

Again, the database is limited, and only old studies are available. However, it would seem that both in inhalation and oral studies, the doses that induce effects are higher than in the case of the Types F or M. This difference is especially marked for oral exposure. For oral exposure, however, only one study, using two species and two uranium compounds is available. For inhalation exposure, the database is somewhat more extensive, and it may be estimated that the toxicity of the group S uranium species is approximately one tenth that of the group F. This would lead to a TI of 5 µg/kg/d. Note U_3O_8 may also have Type M absorption behaviour (see Chapter 7).

8.4.4 Other uranium compounds

Limited information on the different chemicals in this group (e.g., UO_4, $UO_2(C_2H_3O_2)_2$, $(NH_4)_2U_2O_7$, $Na_2U_2O_7$, Carnotite ore) and apparent variation between the different chemicals of this group make it impossible to give a separate TI value for either this group, or any uranium species in this group. It would be prudent to use the TI of the group F, 0.5 µg/kg for all uranium species other than those in group S.

8.5 Uncertainties of chemical risk assessment

The studies in humans cannot be used in quantitative risk estimation, as the information of exposure, both qualitatively and quantitatively, is inadequate.

The database on the toxicity of uranium is limited; most of the studies are old, meaning that not all present methods available to assess renal toxicity were available at the time of these studies. Information, especially on long-term effects of different uranium species, is based on studies from a limited number of researchers. Information is very limited for many uranium species, especially those with limited water solubility. The different studies tend to give rather different results *vis a vis* the quantitative risk estimates. In many studies, dose-response and dose-effect relationships cannot be assessed because of limited dose levels studied. In inhalation studies, the physical–chemical characteristics of the aerosols are often not well characterized, and are likely to be different for different uranium species. There appear to be differences in the sensitivity of different species to uranium toxicity, but no general picture seems to emerge.

The tolerable intake derived is applicable to long-term exposure, intakes lower than the tolerable intake should not lead to adverse health effects. It is apparent that single or short-term exposures lower than the tolerable intake similarly do not adversely affect health. It is likely that in single exposures and short-term exposures, even higher

exposure levels will be well tolerated. However, quantitative information to assess, how much the long-term tolerable intake values may be temporarily exceeded without risk, is not available.

In the extrapolation from experimental animals to humans, comparative information on the toxico-dynamics is not available. Similarly, for inhalation exposure, reliable comparative information is not available on the toxico-kinetics. Thus for these parameters, default values for the extrapolation (10) have to be used. On the other hand, available information would tend to indicate that the oral absorption in humans is not greater than that in experimental animals and the default value for toxico-kinetics in this setting can be replaced by unity. Very limited information is available on the inter-individual variation in uranium toxicity within the human species, and thus the default uncertainty factor for the general population, 10, has to be applied.

8.6 Summary

The primary routes of exposure to uranium for humans are through ingestion or inhalation. The effects of embedded shell fragments containing depleted uranium (among other things) have also been studied (e.g. Fulco et al., 2000).

The target organ to be considered for uranium toxicity is the kidney (also considered to be the primary target organ for ingested uranium in WHO (1998b).

Uranium hexafluoride induces irritative effects at high doses; some uranium compounds may cause pulmonary effects at relatively high inhalation exposures. However, long-term exposure to lower concentrations (generally less than 10 mg/m^3) has usually not resulted in pulmonary toxicity. Carnotite mineral dust causes haemorrhages in dog lungs. Other factors such as diverse inorganic inhalable dust particles, radium, or radon progeny may contribute to these effects. No increase in malignant or nonmalignant respiratory disease mortality has been established in cohorts exposed to uranium in uranium processing. However, the available epidemiological data are generally limited by low statistical power, uncertainties in the assessment of uranium exposure, and/or the paucity of data on exposures to other agents.

In the kidney, proximal tubules are considered to be the main target (ATSDR, 1990, 1999). Currently, uranium is regarded as a less potent nephrotoxin than the classical nephrotoxic metals (cadmium, lead, mercury) (Goodman, 1985). No kidney toxicity related to urinary uranium concentrations was observed in people with embedded DU fragments.

Tolerable intakes for soluble (F and M type) and insoluble (S type) compounds can be derived for inhalation and ingestion. The TI for soluble uranium compounds is 0.5-µg/kg of bw/day and is 5.0 µg/kg of bw/day for insoluble compounds.

9 Health effects due to the presence of radioactivity

9.1 Mechanisms and background

Study of the toxicological and medical effects of radiation on human beings, and the control of exposure to radiation is generally undertaken under the broad disciplines known as radiobiology and health physics. In such studies, it is generally assumed that any resulting health detriment (e.g. cancer, cellular damage etc.) is brought about by exposure to a radioactive substance, and is primarily a function of the amount of energy (as ionizing radiation) absorbed per unit mass of tissue through which it passes. The detailed mechanisms by which radiation interacts with biological materials are the subject of continuing research. However, it is thought that one of the ways in which the energy deposited by radiation may damage cells is by causing changes to occur in deoxyribonucleic acid (DNA), a biologically important molecule that controls cell structure and function, and which is found mainly in cell nuclei. The type of change and the likelihood of error-proof repair depend upon the amount of energy (or dose) deposited. Thus biologically significant effects that lead to the development of cancers or inherited genetic defects may result.

Based on these fundamental principles the assessment of the effects of exposure to radioactive substances has developed in a somewhat different, although often parallel, manner to those commonly used in the assessment of chemical toxicity. The primary concept in radiation protection is that of radiation dose (energy absorbed from ionizing radiation per unit mass by a target organ or tissue). For further information on the usage of terms related to radioactivity and radiological protection the reader is directed to BSS, (1996), ICRP-67 (1993) and ICRP-60 (1991b) onwards or to the RASANET home page at www.iaea.org.

Ionizing radiation emitted by different radionuclides differ in their ability to penetrate matter depending both on the type of radiation emitted and its energy. Alpha particles are hardly able to penetrate the outer layer of skin and do not constitute a hazard when emitted outside the body. Beta particles are able to penetrate the outer layers of skin and can give rise to a localized dose to the skin when in contact. Gamma radiation is potentially more penetrating and can deposit energy to internal organs when outside the body, the magnitude of which depends on the energy of the gamma radiation emitted. Thus, exposures from radionuclides may be both external and internal to the body and the relative importance of these exposure pathways depends upon the type of radiation and the radionuclides involved.

The amount of energy deposited per unit mass of material, such as human tissue, is called the *absorbed dose* and is given the unit gray, symbol Gy (1 Gy is equivalent to 1 J/kg). However, since different types of ionizing radiation differ in the ways in which they interact with biological materials, equal amounts of energy may not result in the same level of biological effects. For example, when alpha-particles are emitted within the body, they deposit energy more densely than either beta particles or gamma radiation, with the result that 1 Gy of alpha radiation is more harmful than 1 Gy of gamma radiation. This potential for causing harm is taken into account in the quantity *equivalent dose*, called sievert and given the symbol Sv (by using a weighting factor of 20 for alpha particles, and 1 for beta and gamma radiation). Another quantity that is commonly referred to in radiological protection is *effective dose*, which is also given in sieverts (or often mSv which is one thousandth of a sievert). In calculating this quantity

the equivalent doses to organs are multiplied by tissue weighting factors that relate to the relative risk of cancer associated with each organ or tissue. This quantity has the advantage that it gives a general indication of the level of risk implied by a given dose, and allows internal and external exposures and uniform and non-uniform irradiation to be quantified on the same basis.

The effective dose, while useful in providing a measure of the health detriment implied by a radioactive substance, is not a directly measurable quantity. A detailed explanation of the dose quantities for measurement purposes is beyond the scope of this report. In this context it is sufficient to note that effective doses are often calculated on the basis of measured dose rates (e.g. in Gy/h) and activity concentrations (e.g. in Bq/kg). Activity is given the name becquerel (symbol Bq) and is equivalent to the number of disintegrations per second. To calculate external dose, it is possible to use dose rate information or calculated coefficients that relate activity to effective or equivalent dose to the skin (e.g. in $Sv/Bq/m^2$). Internal doses are generally calculated from standardized tables of coefficients that give the effective (or equivalent organ doses) arising from a unit intake of activity (Sv/Bq). These coefficients are included in the BSS (1996). Coefficients are given separately for inhalation and ingestion and for different age groups. They take account of the biokinetics and the forms of emission, of both the principal radionuclide and its decay products.

Explanation of the basis behind the calculation of radiation dose is out of the scope of this document and the reader is guided to various publications of the International Commission for Radiological Protection (ICRP) and the International Atomic Energy Agency (IAEA) (for example see ICRP-66 (1994a), ICRP-60 (1991b) and IAEA (1989a) and the principals laid out and described in the Basic Safety Standards for protection against Ionizing Radiation and for the Safety of Radiation Sources (commonly referred to as the BSS) which was jointly agreed by the FAO, IAEA, ILO, NEA, PAHO, and WHO (BSS, 1996). The BSS have been designed to be fully applicable to the occurrence of any isotopic combinations of radionuclides, including DU.

Two categories of health effect have been shown to result from exposure to ionizing radiation; deterministic and stochastic effects. Deterministic effects are those that occur at high doses and dose rates. These effects occur at dose levels far higher than those encountered from the use of, or exposure to, radioactive materials under normal environmental conditions and exposures to the general public. Erythema, or reddening of the skin, is a form of deterministic effect that may result from skin exposure (at instantaneous absorbed doses of 5 Gy or more). Above the dose threshold, the likely severity of such effects is affected by the dose received.

The primary stochastic effect associated with radiation exposure is cancer induction. Most of the information relating radiation doses to an increased risk of cancer is derived from situations in which people have been exposed at higher doses and dose rates than normally encountered (e.g. Nagasaki and Hiroshima bomb survivors). At lower levels of dose and dose rate, it is difficult to demonstrate an increased cancer incidence from radiation exposure because of the high natural incidence of cancer, which is a major confounding factor in epidemiological studies, particularly at low doses and dose rates. Information about the way in which radiation interacts with cells, however, supports what has become known as the linear no-threshold hypothesis. Thus, for radiation protection purposes, it is assumed that there is no level of dose below which there is no risk of a radiation-induced cancer and that the probability (and not the severity) of

cancer increases in proportion with an increase in radiation dose. This assumption has the implication that different sources of radiation can be considered separately, and that limits, or other action levels set for protection purposes, are not based on a borderline between what is safe and unsafe, but on a balance of risk and benefit. As a result, different dose limits or action levels are used for different protection situations.

Dose limits have been recommended by the ICRP-60 (1991b) for controlling the additional radiation doses that arise from normal operations. These limits are based on studies of the 'acceptability of risk' and represent the upper bound on the additional level of risk which may tolerated on a continuing basis from practices involving deliberate application of ionizing radiation. These limits have been incorporated into international and national standards, including the International Basic Safety Standards (BSS, 1996).

9.2 Dose Limits

The International Basic Safety Standards for Protection against Ionizing Radiation and for Safety of Radiation Sources (BSS,1996) require that:

- the occupational exposure of any adult worker shall be so controlled that the following limits are not exceeded
  a. an effective dose of 20 mSv per year averaged over five consecutive years
  b. an effective dose of 50 mSv in any single year.

- the estimated average doses to the relevant critical groups of members of the public that are attributable to practices shall not exceed the following limits
  a. an effective dose of 1 mSv in a year
  b. in special circumstances, an effective dose of up to 5 mSv in a single year provided that the average dose over five consecutive years does not exceed 1 mSv per year; the special circumstances are not defined.

These limits are based on the 1990 recommendations of the International Commission on Radiological Protection (ICRP-60, 1991b) and are also embodied in European Legislation (OJEC, 1996). For a detailed explanation of the models, the reader is referred to the original publications but brief synopses are given in Annex 4.

These limits apply to additional doses from normal planned operations, where the additional level of dose received can be controlled at source; they are not applicable to situations, for example when there is pre-existing contamination, when the decision about whether it is necessary to apply measures to reduce doses are based on the balance between the risk and benefit implied by the intervention measure (and not the source). The recently published ICRP-82 (1999b), includes dose levels above which intervention would be justified to reduce prolonged exposures to members of the public from, for example, radionuclides present in the environment. These recommendations imply that intervention measures are unlikely to be justified at annual doses below than 10 mSv and almost always likely to be justified at annual doses exceeding 100 mSv. An additional annual dose level of 1 mSv is recommended for derivation of intervention exemption levels for commodities (e.g. building materials).

9.3 External radiation exposure

In addition to exposure to anthropogenically introduced substances such as DU, all life on earth is exposed to the presence of external irradiation through a wide range of radiation sources outside the body. Recently processed DU or chemically pure unenriched uranium, consisting solely of naturally occurring uranium isotopes, principally decays through the emission of alpha particles. However, as discussed in Chapter 2 relatively rapid ingrowth of beta- and gamma- emitting progeny occurs in the months following chemical and isotopic separation. Any potential for external exposure is generally considered to be limited to a localized dose to the skin from direct physical contact with DU mainly due to beta emissions of ^{234m}Pa, a progeny of ^{238}U and gamma radiation (AEPI, 1995; Danesi, 1990). This may either occur to the hands when physically handling DU metal or through physical contact with dust derived from the oxidation of DU. AEPI, (1995) states that all DU weapons systems used by the USA army are shielded to control beta radiation emitted to DU when handled by military personnel prior to firing.

The most obvious target for external radiation exposure from DU deposited on the body is the skin. The outermost layer of skin is composed of a layer of dead cells described collectively as the epidermis, while the lower and basal layers are composed of living cells and are therefore susceptible to the effects of ionizing radiation. The average thickness of the epidermis, measured in males of between 26 and 30 years is considered to be around 50 µm with a thickness range between 20 and 100 µm ICRP-23 (1974) and ICRP-59 (1991a). This compares to a typical range in tissue for an alpha particle from ^{238}U of 28 µ, and, therefore, provided that redistribution or sorption of uranium into the epidermis does not occur, alpha particles cannot penetrate to the sensitive lower or basal layers. However, DU greater than 24 days old has quantities of ^{234m}Pa which emits a 2.29 MeV average energy beta that is capable of irradiating the basal layers of the skin. Effects of acute dermal exposure to DU are unlikely but could result in erythema (redness of the skin) or epilation (loss of hair) (Upton, 1992).

Case studies confirm this observation, generally indicating that exposure to basal cells even during prolonged, often aggressive, physical contact does not occur to a significant degree. For example, skin cancer was only rarely observed during the study of 11-cohorts of underground uranium mine workers (Sevcova, 1978). Where skin cancer was observed in these workers, it was considered more likely that this was caused by the presence of arsenic in ores mined from Czechoslovakia and China. In addition there have been no recorded incidents of skin cancers being observed in nuclear workers whose skin has been exposed to occasional hot spots of alpha-emitting radioactive materials (Harley et al., 1999a). During studies undertaken by the US Army it has been established that US occupational exposure standards (NRC–10 CFR 20.1201) covering radioactive exposure of skin (in this case due to exposure to a combination of alpha, beta and gamma radiation) would only be exceeded by holding an unshielded DU projectile in the hand for a period in excess of 250 hours (AEPI, 1995).

It has been estimated that the maximum radiation dose rate from DU armour and DU munitions received by a tank commander, gunner and/or loader working in a fully loaded Mark 1 Abrams battle tank is 0.1 to 0.2 µSv/h (AEPI, 1995). Because of the configuration of the tank and its armour, the driver of the tank may receive a slightly higher dose (1.3 to 0.3 µSv/h). The skin dose rate received when handling a bare penetrator is estimated to be 2 mSv/h (200 mrem/hr; AEPI, 1995).

Penetrator, bullet and armour are contained in a protective coating and adsorption through the skin can therefore be considered to be negligible in these cases. The potential external dose received in the vicinity of a target following attack by DU munitions has been theoretically estimated to be in the order of 4 µSv/year (UNEP/UNCHS, 1999) based on gamma ray exposure. Such doses are small when compared to recommended guidelines for human exposure to ionizing radiation (20 mSv/annum for a worker for penetrating whole body radiation or 500 mSv/year for skin (BSS, 1996).

The radiation doses received from handling other uranium products, which are not made of depleted or chemically pure uranium, may be higher than those described above, particularly where the material also contains progeny of uranium, such as radium. It is impossible to quantify the dose received unless the composition of the material is known and hence both chemical and radiochemical determinations are required in such circumstances, prior to calculation of any potential radiation dose.

9.4 Internal exposure

Internal exposure to ionizing radiation is a function of the route of a given nuclide through the body and its residence time amongst various organs. Doses are therefore calculated, based on the use of biokinetic models that describe the passage and kinetics of various given radionuclides throughout the body. A brief overview of biokinetic models currently employed to calculate dose coefficients in radiation protection is given in Annex 4, and output from these models that may be applied to exposure to DU and uranium in an occupational and public context are described in the following Chapters-10, 11 and 12.

Current epidemiological evidence concerning the carcinogenicity of uranium, both natural and enriched, comes from studies of uranium miners and studies of nuclear workers in fuel enrichment and production facilities.

Among uranium miners, epidemiological studies provide consistent and convincing evidence of excess lung cancer, but not of leukaemia. The information comes from numerous mortality studies of miner cohorts in Australia, Canada, China, Europe and the USA (IARC, 1988; NRC 1999). The lung cancer risk is associated with alpha particle exposure from ^{222}Rn and its decay products, which arise in uranium mines from the decay of ^{238}U. In these studies, the quality of the information on the level of exposure to radon varies across cohorts and over time, from a few air measurements of radon gas and radon decay products in the early years, or re-creation of early mining conditions, to real time individual exposure estimates (taking into account ventilation patterns, ore characteristics, mining methodology, weekly surveys of gamma radiation, radon and dust levels and location and duration of work of individual miners), and even to individual estimates from personal alpha dosimeters in more recent years in France (Tirmarche et al., 1993). The risk of lung cancer appears to be proportional to the radiation dose received. Critical reviews of these studies can be found in IARC (1988) and NRC (1999). There are also a number of epidemiological studies of other alpha emitting radionuclides (e.g. radium, thorium) that also show very clear increases of cancer risk in specific organs (IARC, 2001; NRC, 1999). On the basis of the 1988 review, IARC has classified radon and its decay products as carcinogenic to humans. IARC, in its most recent review of ionizing radiation, has also classified “Internally deposited radionuclides that emit α particles” as carcinogenic to humans (Group 1) (IARC, 2001).

Studies of cancer risk among nuclear workers in nuclear fuel enrichment and production facilities are fewer, although the body of literature is increasing rapidly (Fulco et al, 2000; Cardis and Richardson, 2000). At uranium fuel production facilities, inhalation of airborne uranium dust may represent an important potential source of radiation exposure. Workers in these facilities therefore have two main possible sources of radiological exposure to tissues of the whole body; external gamma-ray exposure which results in a fairly uniform distribution of dose and internal depositions that deliver radiation doses (mainly from alpha-particles) primarily to the lung and lymphatic system. If the uranium dust is solubilized, exposure may also result in other tissues such as the liver, the kidney and the bone. Tumours occurring in these organs are therefore, *a priori*, of particular interest in epidemiological studies of workers at uranium production facilities.

Comparison of findings between uranium processing facilities is, however, complicated by the fact that processes and historical periods of operation have differed between facilities, leading to differences in exposure conditions and follow-up between cohorts. Further, assessment of past internal uranium exposure of nuclear workers is complicated by methodological difficulties of internal dosimetry, as well as by inadequate historical information with which to accurately quantify internal radiation doses. These exposure measurement problems pose significant problems for epidemiology: the inability to accurately classify workers by level of internal radiation exposure may lead to confounding of the analyses of radiation-cancer associations, since workers with significant dose from internal contamination are often persons with substantial external exposure.

Lung cancer has been the primary outcome of interest in studies of workers in fuel enrichment and production facilities. Lung cancer mortality was found to be significantly elevated, compared to national rates, among workers in nuclear fuel processing facilities (Loomis and Wolf, 1997; Checkoway et al., 1988; Frome et al., 1990), but not in others (Dupree et al., 1995; Polednak and Frome, 1981; Hadjimichael et al., 1983; Waxweiler et al., 1983; Stayner et al., 1985; Brown and Bloom, 1987; Ritz, 1999; Dupree et al., 1987). An association between external radiation dose and lung cancer mortality was observed in two cohorts in the US (Ritz, 1999; Checkoway et al., 1988) and an association with lung cancer incidence and radiation dose (using a 20 year lag) in one study in the UK (McGeoghegan and Binks, 2000a). No association was found in other studies on US (Ritz et al., 2000; Hadjimichael et al., 1983;) and UK (McGeoghegan and Binks, 2000b) cohorts where this was studied. An association with estimated dose from internal contamination was observed by Checkoway et al.(1988) but not in another US cohort (Ritz et al., 2000). In contrast, a US multi-facility case control study of lung cancer among workers exposed to uranium dust found no such association, however, there was a suggestion of positive associations among workers hired at ages over 45 years (Dupree et al., 1995). Future research with these cohorts may help to understand the role of uranium dust exposure in cancer risk.

10 Biokinetics for uranium after internal exposure

10.1 Introduction

The aims of this Chapter are to:

- calculate intakes of natural uranium and DU that correspond to dose limits that apply to occupational and public exposure (20 mSv and 1 mSv, respectively) both in terms of radioactivity and mass.
- ascertain whether intakes should be restricted by radiation dose or mass.
- describe the biokinetics of uranium (DU will be identical) in man after inhalation using the generic models recommended by ICRP for soluble (Type F), moderately soluble (Type M) and relatively insoluble (Type S) compounds after deposition in the respiratory tract; emphasis is placed on chest retention, and urinary and faecal excretion rates which are the parameters normally used to assess intake and dose.
- describe the biokinetics of inhaled uranium octoxide (U_3O_8), uranium dioxide (UO_2), uranium trioxide (UO_3) and mixed uranium oxides in man using material-specific parameters derived experimentally; these oxides are the ones of immediate concern during the testing and use of DU munitions.
- comment on the limits on annual intake by ingestion for members of the public.

For ease and consistency of presentation, it is emphasized that the biokinetic data are presented in terms of either unit intake (acute exposure), or unit intake per day (chronic exposure). Hence, in principle, the data can be scaled to the intake of choice, or conversely chest retention or excretion rates can be scaled for the purpose of predicting the intake. However, both approaches should be treated with caution if the temporal pattern of intake is unknown, or the contribution to the excretion rates is influenced by extraneous and variable sources such as uranium present in the diet. Dose limits are expressed in terms of the annual intake limit by mass. However it should be emphasized that these should be reduced as far as is practicable (see Chapter 9).

Compliance, or otherwise, with the limits for internal contamination can be achieved by assessing intakes and doses using the following procedures:

- combining measurements of DU in the environment (airborne concentrations, aerosol size, bioavailability, concentrations in contaminated food and drink etc.) with the suite of biokinetic models recommended by ICRP, namely, the Human Respiratory Tract Model (ICRP-66, 1994a), the systemic model for uranium (ICRP-69 1995a), and the model for uptake via the gastrointestinal tract (ICRP-30, 1979).

- using these biokinetic models to interpret external measurements of uranium in the chest or in excreta.

10.2 Inhalation dose coefficients and annual intake limits

For the purpose of recommending dose coefficients, or dose per unit intake for radionuclides, ICRP considered three types of compounds (ICRP-66, 1994a) in which the absorption rates from the lungs to blood are deemed to be fast (Type F), moderate (Type M) or slow (Type S). The default absorption parameter values are given in Table 10.1.

Table 10.1 Default absorption parameters for Type F, M and S materials

ICRP -66 absorption type	F (fast)	M (mod.)	S (slow)
Model parameters:			
Fraction dissolved rapidly, f_r	1	0.1	0.001
Dissolution rate:			
Rapid (per d), s_r	100	100	100
Slow (per d), s_s	-	0.005	0.0001

Notes

F (fast) materials that are readily absorbed into blood (corresponding to 'Class D'). There is significant absorption from ET_2 and BB_1 (see Annex 4), but some material in these regions will remain in solution in mucus and be swallowed, rather than be absorbed through the epithelium. Hence the default for such materials is s_r=100 per d ($t_{½}$ ~ 10 min).

M (moderate) materials with intermediate rates of absorption (corresponding to 'Class W'). For such materials the percentage absorbed rapidly is on the order of 10%, and the slow-phase retention time of the order of 100 d. This is represented by f_r = 0.1%; s_r = 100 per d; and s_s = 0.005 per d.

S (slow) relatively insoluble materials (corresponding to 'Class Y'). It is assumed that for most of the material the rate of absorption to blood is 0.0001 per d. This equals the particle transport rate from the most slowly cleared AI compartment. However, it is characteristic of even very insoluble materials that some rapid uptake to blood occurs immediately after inhalation. As a default it is assumed that 0.1% of the deposited material is rapidly absorbed. While the effect of this on doses is likely to be negligible, it may significantly affect the interpretation of measurement of activity in urine. This is represented by f_r = 0.001; s_r = 100 per d; and s_s = 0.0001 per d.

Based on a considerable amount of published biokinetic data (ICRP-71 1995b, ASTDR, 1999, Scripsick et al. 1985a, 1985b) obtained primarily from animal studies, uranium compounds were assigned to one of these types. The biokinetics of UO_3 are mostly consistent with assignment to Type M compounds and UO_2 to Type S compounds. For U_3O_8, some studies indicate Type M behaviour and others Type S (ICRP-71, 1995b).

Based on the human respiratory tract, systemic and gastrointestinal tract models referred to above, ICRP–68 (1994b), ICRP-72 (1996) and BSS (1996) have recommended dose coefficients, or dose per unit intake, expressed as Sv/Bq for all the important isotopes of uranium. These dose coefficients take account of several age groups within the population, but only those for adults and isotopes appropriate for natural and DU are listed here (Table 10.2).

The values for individual isotopes can be used to derive the dose coefficients for any isotopic composition of uranium. The Annual Intake Limits (AIL) for uranium and DU for adults, which correspond to dose limits of 20 mSv and 1 mSv, expressed as Bq, and calculated by dividing the dose limit (Sv) by the dose coefficient (Sv/Bq) are given in Table 10.3. Also included in the table are the equivalent masses of uranium and DU based on the isotopic composition and specific activities given with Table 10.3. These comparisons are important since they can be used for assessing whether exposure criteria for radiotoxicity or chemical toxicity will be more important for different chemical forms of uranium and DU.

Table 10.2 Dose coefficients, Sv/Bq, for uranium isotopes: public and occupational exposure (OJEC, 1996; BSS, 1996; ICRP-68, 1994b)

Type/Isotope[a]	**AMAD** 1µm (Public)	**AMAD** 5µm (Occupational.)
Type F	10^{-7}	10^{-7}
Uranium-234	5.6	6.4
Uranium-235[b]	5.2	6.0
Uranium-238[b]	5.0	5.8
Type M	10^{-6}	10^{-6}
Uranium-234	3.5	2.1
Uranium-235[b]	3.1	1.8
Uranium-238[b]	2.9	1.6
Type S	10^{-6}	10^{-6}
Uranium-234	9.4	6.8
Uranium-235[b]	8.5	6.1
Uranium-238[b]	8.0	5.7

Notes
a Uranium-236 is not considered, as it contributes less than 0.0003% of total activity. Other transuranics excluded as contributing less than 1% of dose (CHPPM, 2000; Annex 2)
b Includes contribution from short-lived progeny

Table 10.3 Dose coefficients[a] and AIL for natural uranium and DU: occupational and public exposure (rounded values).

Isotopic composition	**Worker** (5 µm, dose limit 20 mSv)		**Public** (1 µm, dose limit 1 mSv)	
	U-nat	DU	U-nat	DU
ICRP Type F compound				
Dose coefficient (Sv/Bq) 10^{-7}	6.15	5.92	5.26	5.06
Intake limit (Bq) 10^{3}	32.5	33.8	1.90	1.98
Intake limit (mg U)	1290	2270	75	133
ICRP Type M compound				
Dose coefficient (Sv/Bq) 10^{-7}	18.5	16.8	31.6	29.6
Intake limit (Bq) 10^{3}	10.8	11.9	0.32	0.34
Intake limit (mg U)	430	800	13	23
ICRP Type S compound				
Dose coefficient (Sv/Bq) 10^{-7}	62.9	59.2	87.1	82.6
Intake limit (Bq) 10^{3}	3.18	3.38	0.12	0.12
Intake limit (mg U)	130	230	4.5	8.1

Note
a Includes contribution from short lived progeny

Composition	by mass (%)			By activity (%)			Specific activity of mixture
	^{234}U	^{235}U	^{238}U	^{234}U	^{235}U	^{238}U	Bq /mg
a: U-nat	0.5×10^{-3}	0.72	99.3	48.9	2.2	48.9	25.2
b: DU	1.0×10^{-3}	2.0×10^{-1}	99.8	15.5	1.1	83.4	14.8

Chemical toxicity is discussed further in Chapter 8 and Annex 5. However it is important to recognize at this juncture that based on an occupational exposure limit of 0.2 mg/m^3 for soluble and insoluble forms of uranium (ACGIH, 2000; NIOSH, 2000; FRA, 1988; HSE, 2000), equivalent to 2 mg/d. It can be deduced from Table 10.3 (see also Stradling et al., 1997, Stradling et al., 1998, ICRP-78, 1997) that:

- daily intakes of Type F and Type M compounds will always be limited by chemical toxicity; daily intakes of Type M compounds will always be limited by chemical toxicity; annual intakes of Type M compounds will be limited by consideration of radiation dose
- daily intakes of Type S compounds will be limited by chemical toxicity while annual intakes will be limited by radiation dose

On the basis of the OSHA (1989) limit for soluble uranium of 0.05 mg/m^3, annual intakes of Type M compounds would also be restricted by chemical toxicity. Similar considerations should apply to exposure of the public using the appropriate recommendations for airborne dust and dose limit.

10.3 Biokinetics of Type F, M and S compounds of uranium after inhalation.

Because uranium and DU are identical chemically, their biokinetic behaviour will be the same. For workers, the predicted time-dependent retention in the chest and systemic tissues, and excretion, can be modeled using the default absorption parameters (Table-10.1) with other default parameter values for particle size, density, aerosol deposition in the respiratory tract and exercise levels (ICRP-66 1994a). These are given in Table 10.4.

The default absorption parameters, and density, are also used for members of the public. The other default parameter values, also given in Table 10.4 are different.

Table 10.4 Deposition of inhaled aerosols in the human respiratory tract: occupational and public exposure.

Region[c]	**Worker**[a] (%)	**Adult male**[b] (%)
ET_1	33.9	14.2
ET_2	39.9	17.9
BB	1.8 (33% in BB_2)	1.1 (47% in BB_2)
Bb	1.1 (33% in bb_2)	2.1 (49% in bb_2)
AI	5.3	11.9
Total deposit	82.0	47.3

a **Occupational exposure** 5 μm AMAD (σ_g = 2.5), 3.5 μm AMTD, density 3.0 g/cm^3, shape factor 1.5 (see Chapter 5); fraction breathed through nose is 1.31% sitting and 69% light exercise; mean ventilation rate is 1.2 m^3/h. (ICRP-66, 1994a).

b **Environmental exposure** 1 μm AMAD (σ_g = 2.47), 0.69 μm AMTD, density 3.0 g/cm^3, shape factor 1.5; fraction breathed through nose is 1.55% ventilation rate is 0.78 m^3/h. 33.3% sleeping, 25% sitting, 40.6% light exercise and 1.0% heavy exercise.

c The extrathoracic airways consist of the anterior nasal passages (ET_1) and posterior nasal and oral passages, pharynx and larynx (ET_2).

The thoracic regions are bronchial (BB and bb) and alveolar-interstitial (AI). For the purposes of external monitoring, the retention in the chest would be the activity retained in the thoracic regions.

10.3.1 Acute exposure

As illustrative examples, the chest retention for workers and members of the public after unit intake (radioactivity or mass) are shown in Figure 10.1. The figure emphasizes the difference in retention in the chest due to different particle size, breathing pattern and exercise level. The chest retention of Type F compounds within a few hours of exposure is negligible, and hence is not included in the figure.

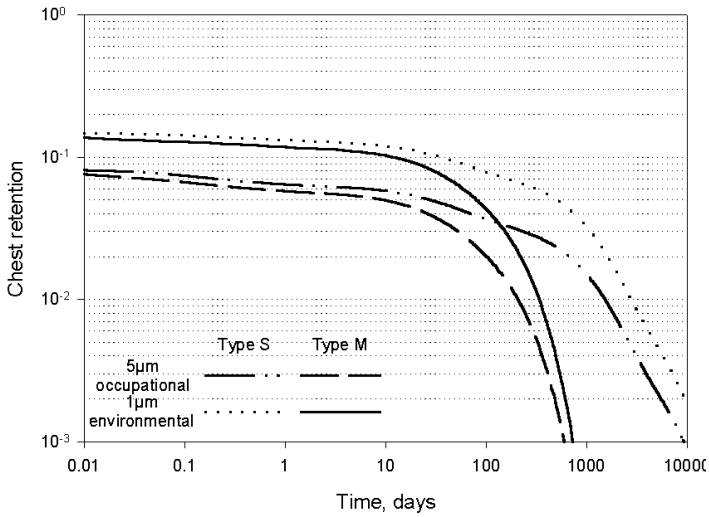

Figure 10.1 Fractional retention of inhaled uranium in the chest after acute inhalation of Type M and S compounds: occupational and public exposure.

The predicted urinary excretion rates for occupational exposure is given in Figure 10.2 and for public exposure in Figure 10.3.

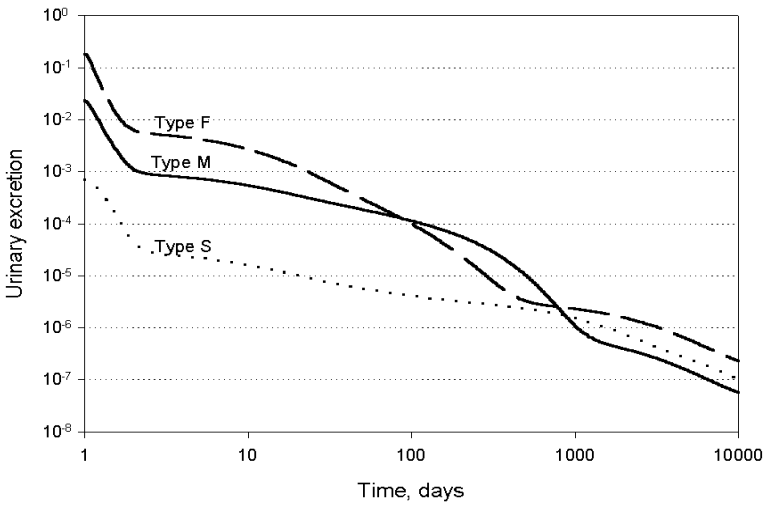

Figure 10.2 Fractional urinary excretion rate of inhaled uranium after acute inhalation of Type F, M and S compounds: occupational exposure.

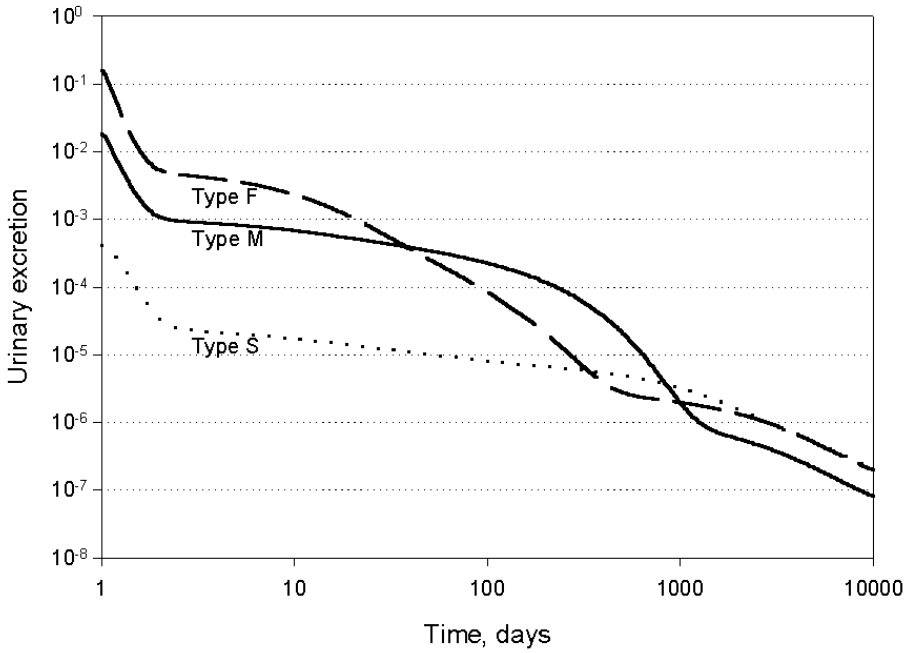

Figure 10.3 Fractional urinary excretion rate of inhaled uranium after acute inhalation of Type F, M and S compounds: public exposure.

The predicted faecal excretion rates for occupational exposure is given in Figure 10.4 and for public exposure in Figure 10.5.

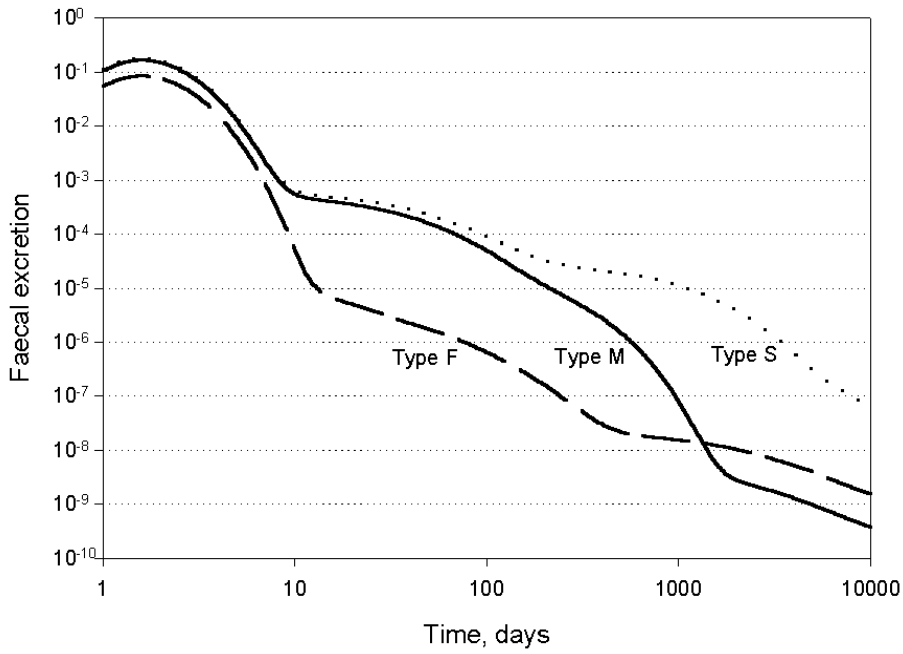

Figure 10.4 Fractional faecal excretion rate of inhaled uranium after acute inhalation of Type F, M and S compounds: occupational exposure.

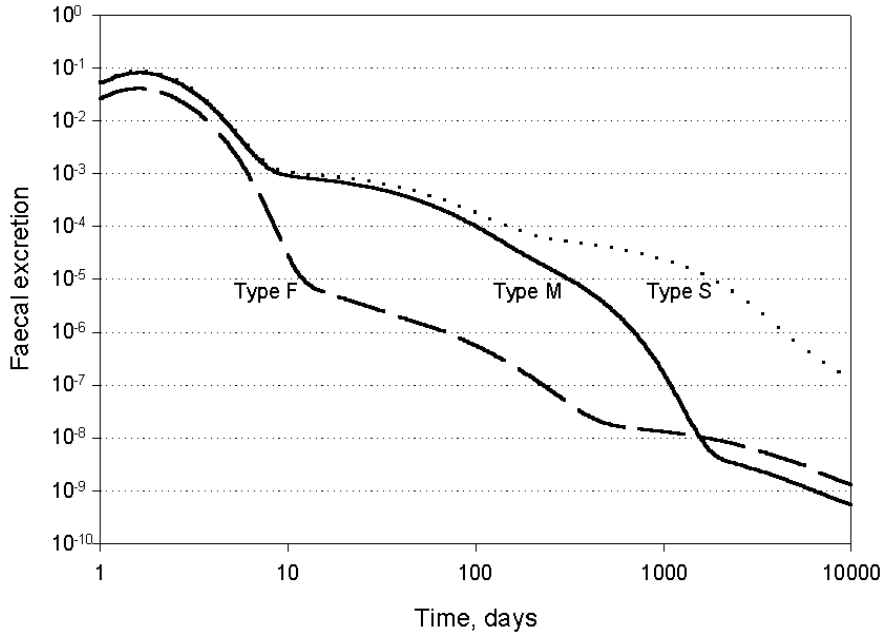

Figure 10.5 Fractional faecal excretion rate of inhaled uranium after acute inhalation of Type F, M and S compounds: public exposure.

Figure 10.2 shows that while there are large differences in the fractional excretion rates at early times after exposure, the rates beyond about 700 d are closely similar for the three absorption types. A comparison of Figures 10.2 and 10.3 shows that the fractional rate for occupational exposure is always higher than for public exposure. However, the curves in Figure 10.3, like those in Figure 10.2, converge at about 700 d. It is noteworthy that the faecal excretion curves for Type M and Type S compounds in Figure 10.4 are almost coincident up to about 100 d after exposure. Figures 10.4 and 10.5 demonstrate that the faecal rates after occupational and public exposure exhibit the same trends. However, in the latter case, the rates for Type M and S compounds are slightly higher beyond about one week after exposure. The rates for Type F compounds after public exposure are slightly lower than after occupational exposure during the first week; thereafter the curves are coincident.

In principle, assessments of intake and dose can be extrapolated from measurable amounts of uranium and DU in the chest or in urine or faeces a long time after intake. This approach should be treated with caution since the actual pattern of intake and airborne concentrations are unlikely to be known with certainty, and normal dietary intakes of natural uranium, could substantially distort the assessment. For example, a urinary excretion rate of say 1 µg/d of uranium observed several years after an assumed occupational intake of DU, a value which could be accounted for by a small and recent intake of a soluble form of uranium from the normal diet, would suggest that the original intake may have been about 1 g.

On the other hand, the urinary excretion curve could be used with advantage if very low levels of DU can be detected. For example, if the excretion of DU in urine several years after an assumed occupational exposure was 10 ng/day, then the maximum predicted intake would be only about 10 mg. This amount equates to a committed effective dose of only about 1 mSv for a Type S compound. (see Table 10.3).

10.3.2 Chronic intake

In this chapter, consideration is given only to members of the public. The biokinetics of uranium after chronic intake have been based on unit intake per day, say 1Bq or 1 μg. The data may be interpolated for any other intake rate if warranted. For example, in some cases it may be appropriate to consider chronic intake rate corresponding to the annual intake limit (see Table 10.3) i.e. at a rate of (annual intake limit) / 365 per day. While it is impossible to consider all the alternative exposure scenarios for continued exposure, this model is perfectly acceptable for illustrative purposes, and is consistent with one of the approaches used by ICRP-78 (1997)

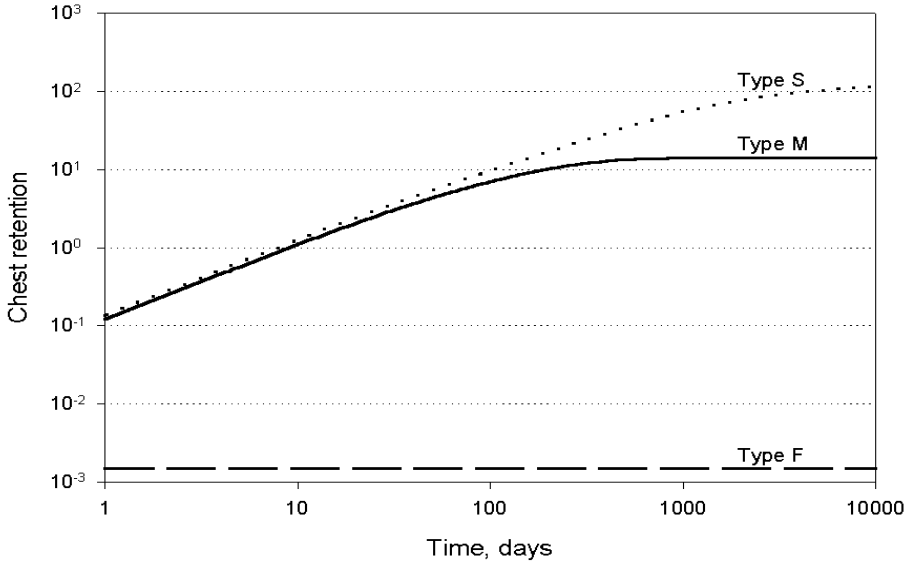

Figure 10.6 Chest retention of inhaled uranium after chronic inhalation of Type F, M and S compounds: public exposure.

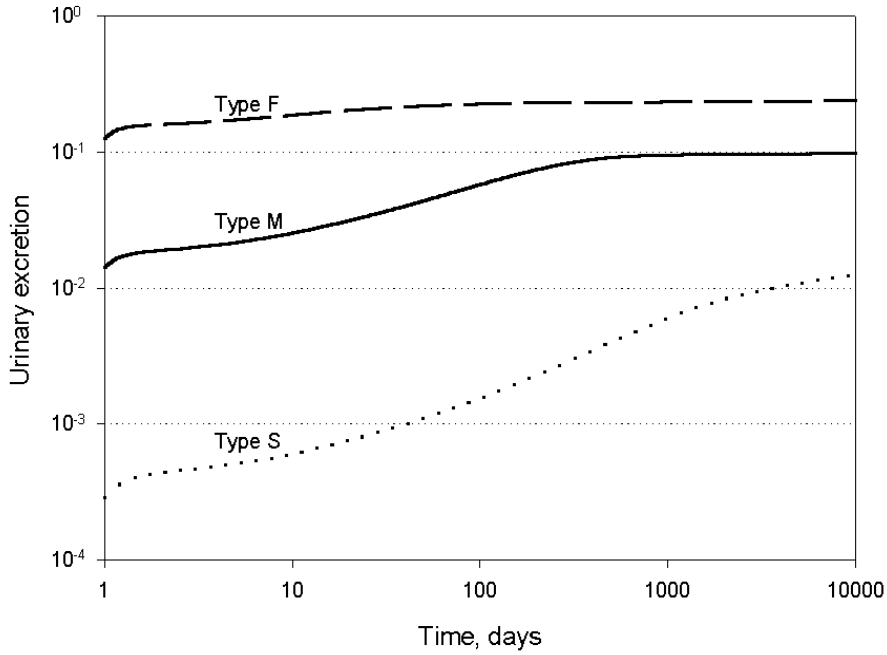

Figure 10.7 Fractional excretion rate of inhaled uranium in urine after chronic inhalation of Type F, M and S compounds: public exposure.

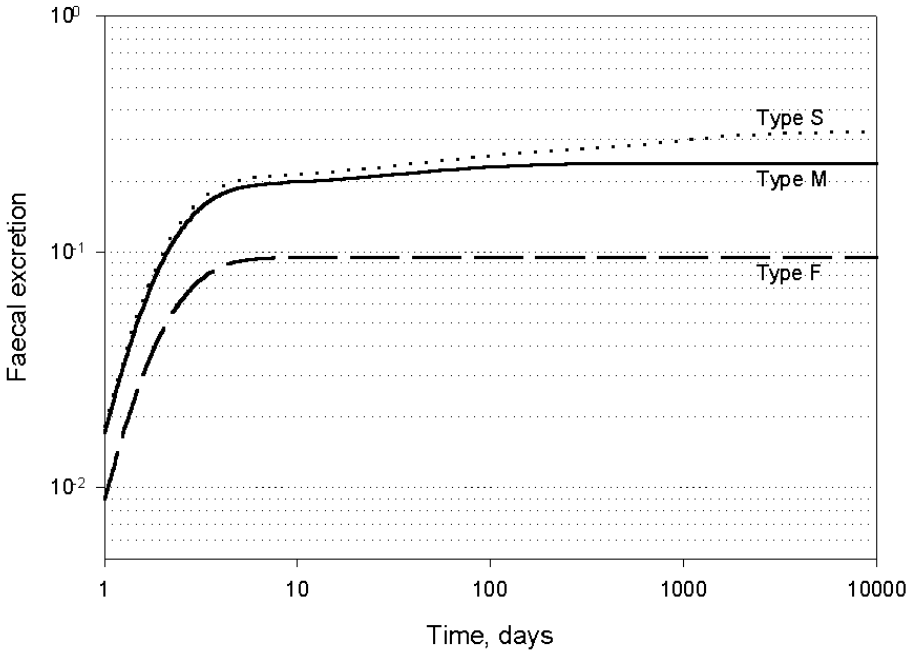

Figure 10.8 Fractional excretion rate of uranium in faeces after chronic inhalation of Type F, M and S compounds: public exposure.

Figure 10.6 shows that the amounts present in the chest after the inhalation of Type M and S compounds are closely similar up to about 100 d after the commencement of exposure. The data in Figure 10.7 show that the urinary excretion rates differ by about an order of magnitude over the first 100 or so after the commencement of exposure before slowly converging. Figure 10.8 shows that the faecal excretion rates are much closer than those for urinary excretion; indeed they only differ overall by about three-fold after 10 000 d. However it should be borne in mind that the curves represent idealized intake and excretion rates.

10.4 Material specific biokinetic behaviour of inhaled uranium oxides

Biokinetic studies have been conducted on all natural uranium compounds present in the nuclear fuel cycle (e.g. Hodgson et al., 2000, Ansoborlo et al., 2001; ICRP-71, 1995b). In comparison, very few studies have been conducted with DU. However since uranium and DU are identical chemically, the database on the former is relevant for the biokinetics of DU. However the discussion here is limited to the oxides which are reported to be present in the air in the aftermath of the use of DU munitions, or after a uranium fire. For DU used in warfare, U_3O_8 and to a lesser extent UO_2 and UO_3 are the compounds of most immediate interest. In addition, consideration is also given to the biokinetics of mixed uranium-iron (U/Fe) oxides, which may also be present.

Whilst many studies on the biokinetics of uranium oxides have been reported, very few of them have been designed to assess the absorption parameters of uranium as defined in the human respiratory tract model (ICRP-66, 1994a). For the purpose of this monograph, the values used have been obtained for materials formed during the fabrication of nuclear fuels in the UK (Hodgson et al., 2000), although similar data have been derived for similar compounds in the French nuclear industry (Ansoborlo et al., 2001). This latter publication also provides extensive dissolution data. The absorption parameters for reprocessed UO_3 (Moody et al., 1997) are similar to those obtained for

the compound formed in nuclear fuel fabrication (Hodgson et al., 2000). Information on mixed uranium-iron oxides has been obtained from an industrial source (Ansoborlo et al., 1998).

The values obtained from these studies are used here for illustrative purposes. For more detailed information on the application of the ICRP respiratory tract model, the reader is referred to a forthcoming technical document (ICRP, 2001)

It should be noted that the material specific absorption parameters are quite different from the default values (Table 10.1), which is not unexpected. Indeed, ICRP have continually acknowledged the importance of such data, and these results support this recommendation. In calculating these data it has been assumed that the absorption parameters in animals are the same as those in man. The evidence available at present suggests that this assumption is justifiable, since in other studies the observed biokinetic behaviour of U_3O_8 and UO_2 in workers is closely similar to that predicted by extrapolation from animal studies (Stradling et al., 1989, Bertelli et al., 1998).

The material specific absorption parameter values (Table 10.5) have been used with the other default parameter values for members of the public (Table 10.4) to calculate exposure limits (Table 10.6).

Table 10.5 Absorption parameter values for uranium oxides and DU default material.

Material	f_r	s_r, per d	s_s, per d	f_1	
				Inhaled	Ingested
U_3O_8	0.044	0.49	3.5×10^{-4}	0.002	0.002
UO_2	0.011	0.95	6.1×10^{-4}	0.002	0.002
UO_3	0.92	1.4	3.6×10^{-3}	0.02	0.02
U/Fe oxide[a]	0.12	1.45	2.6×10^{-3}	0.02	0.02
DU default[b]	0.2	1	1.0×10^{-3}	0.02	0.002

Notes

a mixture of UO_2, U_3O_8, UO_3, FeO, Fe_2U

b best judgement values. The value for f_r is based on DU with a large soluble fraction as determined from *in-vitro* studies (Scripsick, 1985a, 1985b); the value for s_r is based on the results of several experimental studies with uranium oxides and other compounds (Hodgson et al., 2000, Ansoborlo et al., 2001); s_s is representative of values obtained from several experimental studies with U_3O_8 (Hodgson et al., 2000, Ansoborlo et al., 2001). A material density of 9 is assumed since this is considered more appropriate than the ICRP default value of 3. The high value for f_r may be due to the presence of ultrafine metal or oxide particles; this has been demonstrated for other aerosols (Ansoborlo et al 1998)

Table 10.6 Dose coefficients[a] and annual intake limits for industrial uranium compounds and DU with default parameter values[b]: occupational and public exposure (rounded values).

Isotopic composition	**Worker** (5 µm, dose limit 20 mSv)		**Public** (1 µm, dose limit 1 mSv)	
	U-nat	DU	U-nat	DU
UO_3				
Dose coefficient (Sv/Bq) 10^{-7}	3.88	3.63	6.60	6.25
Intake limit (Bq) 10^3	51.6	55.0	1.51	1.60
Intake limit (mg U)	2040	3700	60	110
UO_2				
Dose coefficient (Sv/Bq) 10^{-7}	43.9	41.0	59.0	55.6
Intake limit (Bq) 10^3	4.56	4.88	0.17	0.18
Intake limit (mg U)	180	330	6.7	12.1
U_3O_8				
Dose coefficient (Sv/Bq) 10^{-7}	48.5	45.5	65.3	61.6
Intake limit (Bq) 10^3	4.12	4.40	0.15	0.16
Intake limit (mg U)	160	300	6.1	10.9
U/Fe Oxide				
Dose coefficient (Sv/Bq) 10^{-7}	25.7	23.8	36.6	34.3
Intake limit (Bq) 10^3	7.77	8.40	0.27	0.29
Intake limit (mg U)	310	560	10.8	19.6
DU default [b]				
Dose coefficient (Sv/Bq) 10^{-7}	35.7	33.2	55.6	52.3
Intake limit (Bq) 10^3	5.60	6.03	0.18	0.19
Intake limit (mg U)	220	410	7.1	12.8

Notes
a includes contribution from short lived progeny
b see Table 10.5 for absorption parameters

It is noteworthy that the exposure limits for uranium and DU in Table 10.6 for U_3O_8 and UO_2 lie between those of Type M and S compounds, and those for UO_3 between Type F and M compounds. The retention kinetics of uranium in the chest and excretion rates in urine have also been predicted using the material-specific absorption parameters in Table 10.5.

10.4.1 Acute exposure

The retention kinetics of uranium in the chest after unit intake for workers is shown in Figure 10.9 and for members of the public in Figure 10.10.

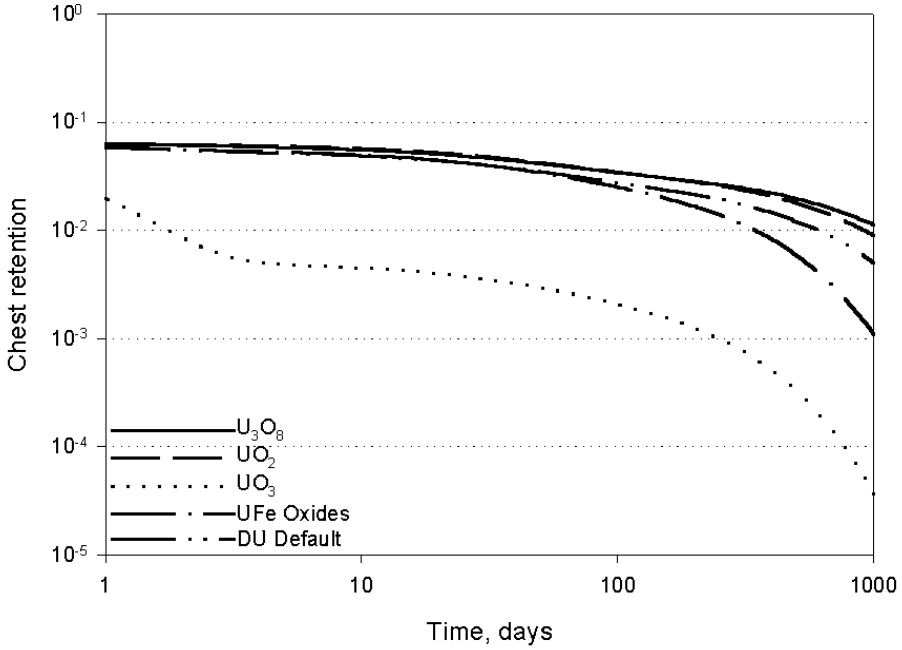

Figure 10.9 Fractional retention of inhaled uranium in the chest after acute inhalation of uranium oxides and the DU default: occupational exposure.

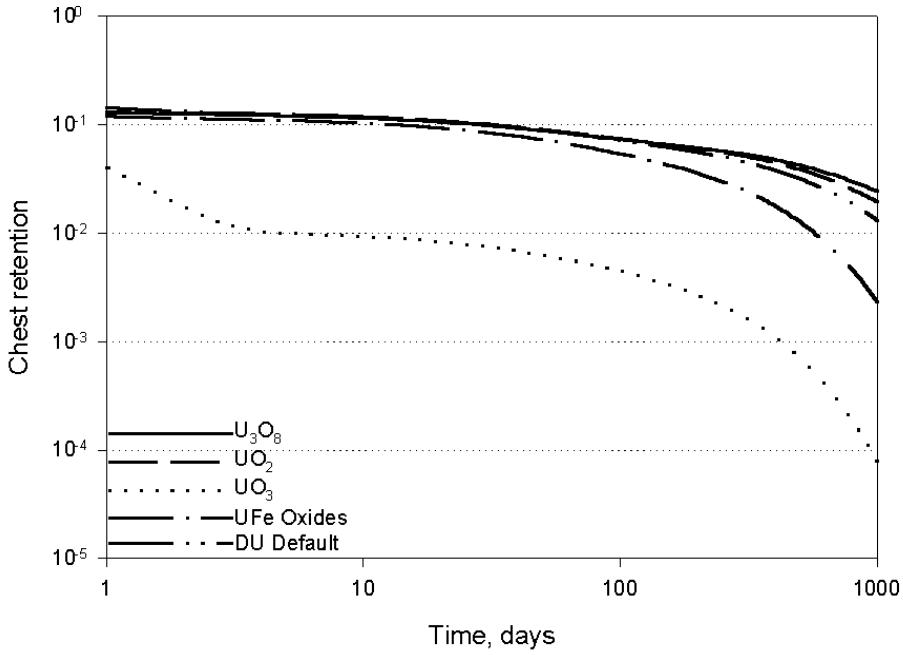

Figure 10.10 Fractional retention of inhaled uranium in the chest after acute inhalation of uranium oxides and the DU default: public exposure.

Figure 10.9 shows that apart from UO_3, the retention in the chest is similar for all the materials up to about 100 d after exposure. For the oxides, the chest retention kinetics lie between those for Type M and Type S compounds (Figure 10.1). The chest retention kinetics for each material after public exposure, shown in Figure 10.10, are closely similar to those after occupational exposure, reflecting the fact that most of the clearance from the respiratory tract occurs by particle transport to the gastrointestinal tract.

The fractional urinary excretion rates for uranium oxides after acute occupational and public exposure to uranium oxides and default DU are shown in Figures 10.11 and 10.12 respectively.

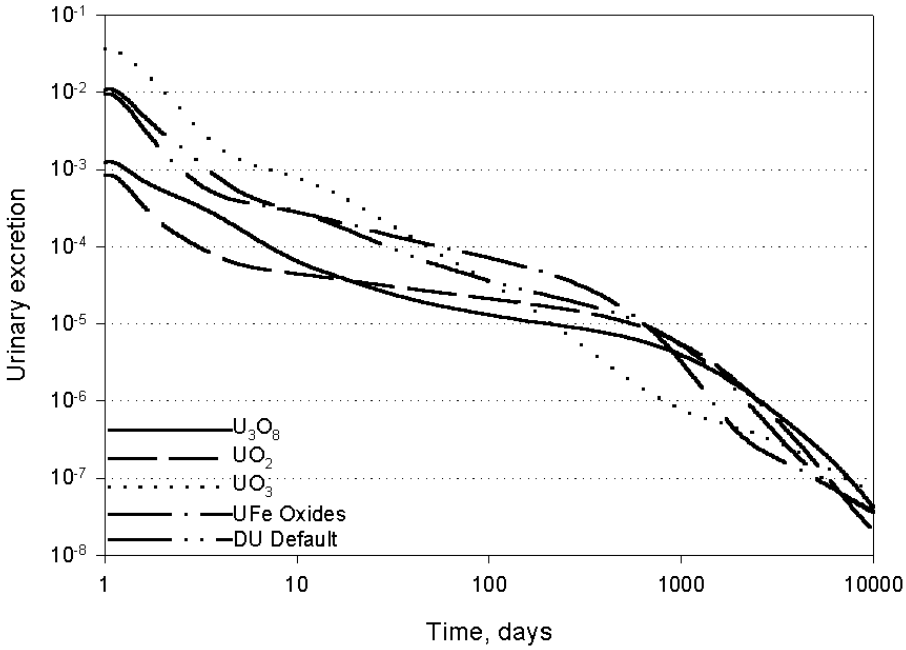

Figure 10.11 Fractional urinary excretion rate of inhaled uranium after acute inhalation of uranium oxides and the DU default: occupational exposure.

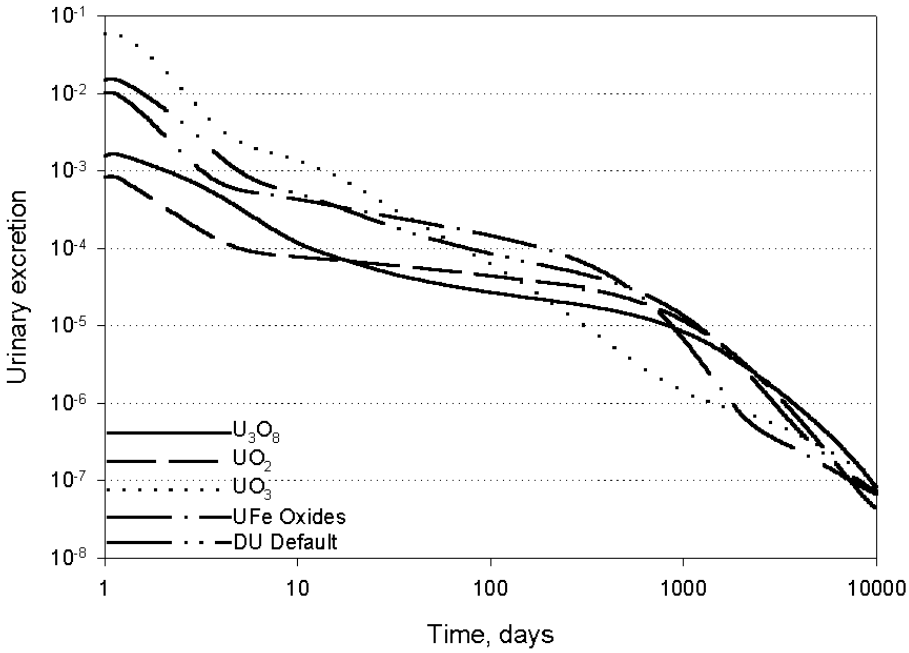

Figure 10.12 Fractional urinary excretion rate of inhaled uranium after acute inhalation of uranium oxides and the DU default: public exposure.

Figure 10.11 shows that up to 100 d after exposure, the urinary excretion rates for the various oxides can differ by an order of magnitude or more. It is noteworthy that, as for Type F, M and S compounds (Figure 10.2), the rates at about 1000 d are closely similar. However, it should be noted that for the oxides, the excretion rates are higher than for their assigned absorption type, due probably to a higher value of f_r (see 10.4). This

should facilitate interpolations from excretion data in so far that lower values on the assessment of intake should be possible.

Figure 10.12 shows that the urinary excretion rates are similar to those predicted after occupational exposure. From two days onwards, the rate for public exposure is always between 1.5 to 2.5 faster than for occupational exposure when the same material is inhaled.

The fractional faecal excretion rates for uranium oxides after acute occupational and public exposure to uranium oxides and default DU are shown in Figures 10.13 and 10.14 respectively.

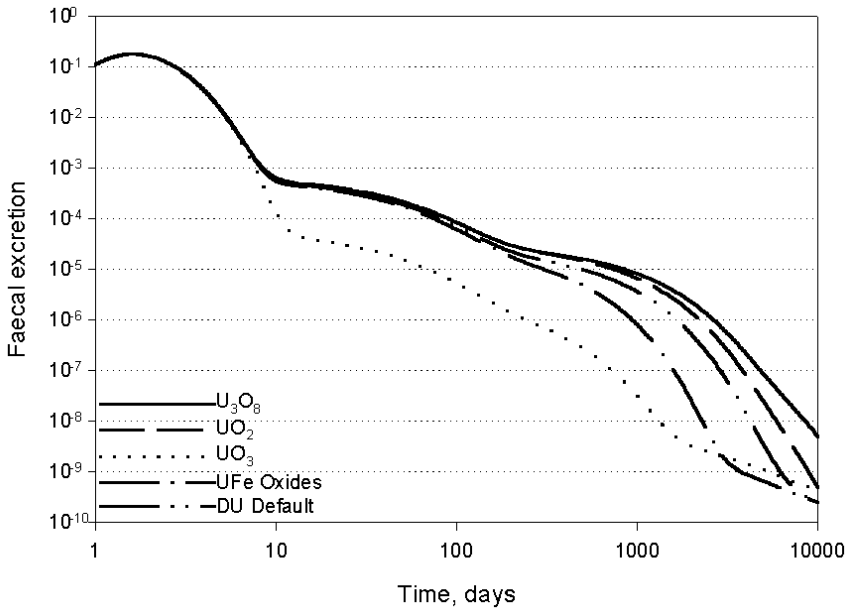

Figure 10.13 Fractional faecal excretion rate of inhaled uranium after acute inhalation of uranium oxides and the DU default: occupational exposure.

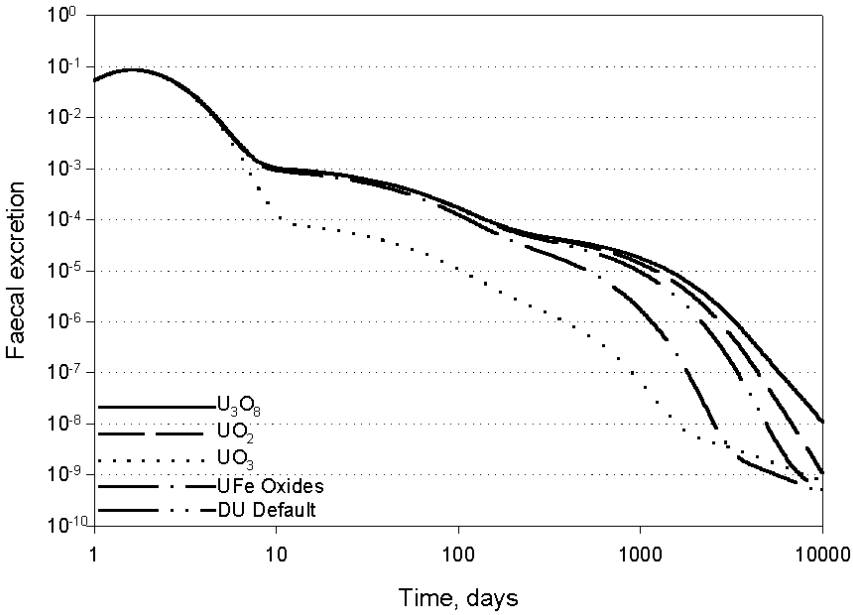

Figure 10.14 Fractional faecal excretion rate of inhaled uranium after acute inhalation of uranium oxides and the DU default: public exposure.

Figure 10.13 shows that, other than for UO_3, the faecal excretion rates are the same for the first week after exposure and then closely similar up to several hundred days. The faecal excretion rates after public exposure, shown in Figure 10.14 are closely similar to those obtained after occupational exposure.

This Chapter has shown that the differences in biokinetic behaviour when using default or material specific values may be important for optimising the assessment of intake from chest monitoring or the assay of excreta. This subject is discussed in later in Chapter 11.

10.4.2 Chronic exposure

The fractional retention of the uranium oxides and default DU after continuous chronic intake by adult members of the public is shown in Figure 10.15.

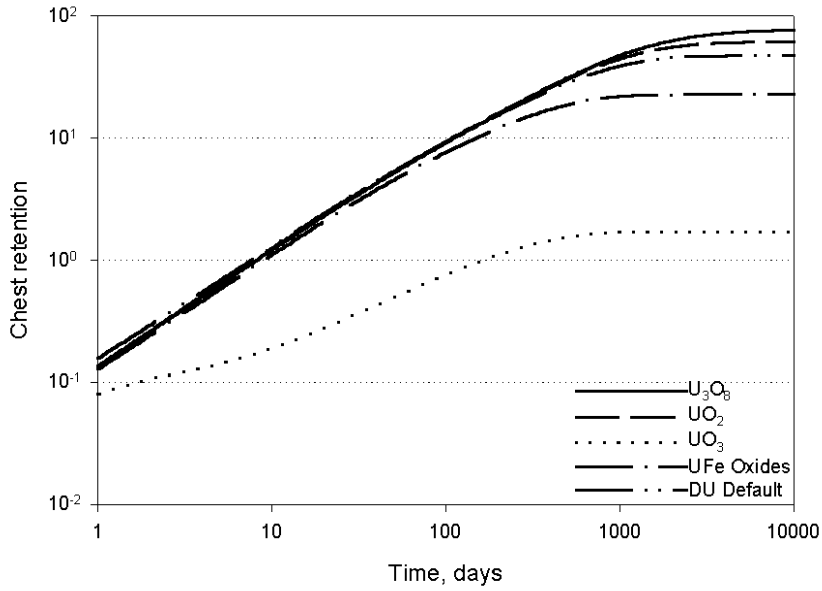

Figure 10.15 Fractional retention of inhaled uranium in the chest after chronic inhalation of uranium oxides and the DU default: public exposure.

Figure 10.15 shows that, apart from UO_3, the retention of uranium in the chest is closely similar for all materials up to about 600 d after the commencement of exposure. The fractional urinary and faecal excretion rates after chronic exposure are shown in Figures 10.16 and 10.17.

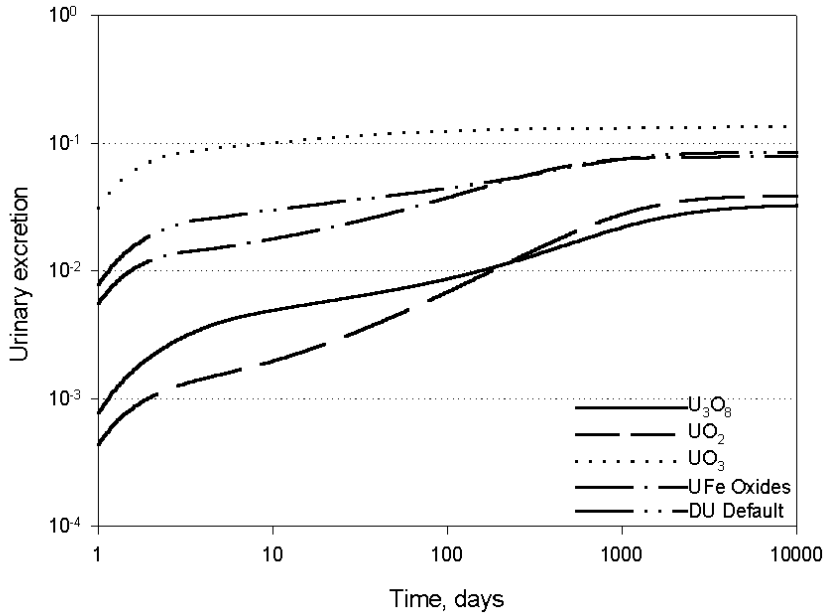

Figure 10.16 Fractional urinary excretion rate of inhaled uranium after chronic inhalation of uranium oxides and the DU default: public exposure.

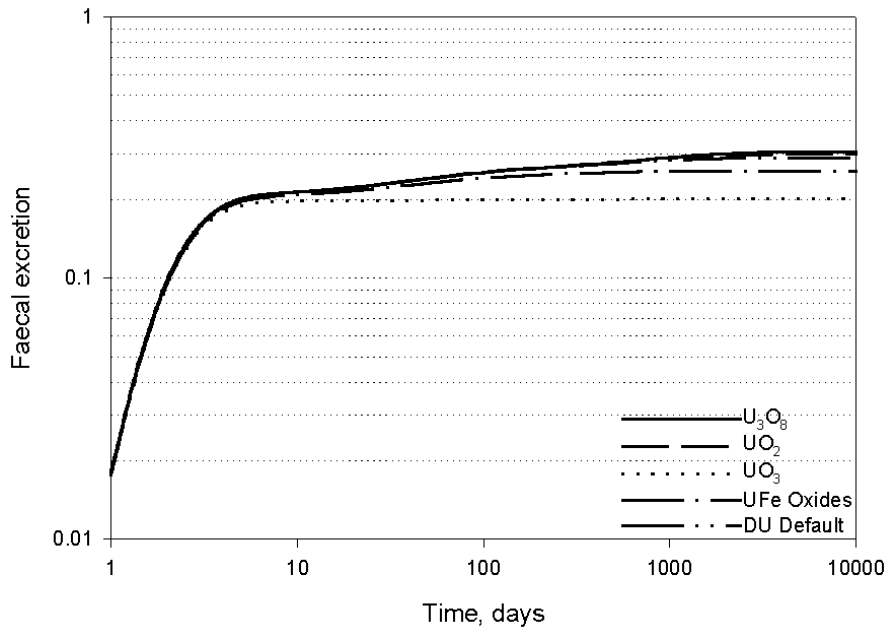

Figure 10.17 Fractional faecal excretion rate of inhaled uranium after chronic inhalation of uranium oxides and the DU default: public exposure.

Figure 10.16 shows that differences in the urinary excretion rate of an order of magnitude or more occur up to 100 d after the commencement of exposure, the curves slowly converging thereafter. The data for the faecal excretion rate in Figure 10.17 show that they are closely similar up to 10 000 d after exposure.

It should be emphasized that usually, there is less certainty about the chemical form of the uranium and DU inhaled by the public than during occupational exposure. In such circumstances, and until further information becomes available, it may be prudent to assume that for the purpose of radiological dose assessment the dust is assigned to inhalation Type S. It is also emphasized that from a toxicological standpoint, the dust should be assigned to inhalation Type F if a conservative assessment is required.

10.5 Ingestion coefficients and annual intake limits for adult members of the public

This Chapter concerns uptake from the gastrointestinal tract by adult members of the public from food and drink. As for inhalation, ICRP-72 (1996) and IAEA 1996) have recommended dose coefficients, or dose per unit intake, expressed as Sv/Bq for all the isotopes of uranium. For this purpose, the model of the gastrointestinal tract, summarized in Annex 4, is coupled to the systemic model for uranium (ICRP–69, 1995a). The generic values for natural uranium and DU are given in Table 10.7. In general, there is less certainty about the chemical form of the uranium and DU after ingestion rather than after inhalation. In such circumstances it may be prudent to assume that the dust is of inhalation Type F.

Table 10.7 Dose coefficients and annual intake limits for Type F compounds of natural uranium and DU after ingestion (f_1=0.02)

Isotopic composition	U-nat	DU
Type F		
Dose coefficient (Sv/Bq) 10^{-8}	4.82	4.77
Intake limit (Bq) 10^{3}	20.7	21.0
Intake limit (mg U)	820	1400

10.6 Wound contamination.

At present, there is no appropriate biokinetic model, which describes the behaviour of radionuclides after entry into the body from superficial or deep-seated wounds, or from embedded DU fragments. However in such cases estimates of the systemic tissue content can be made by extrapolating from the urinary excretion rate using the ICRP systemic model for uranium (ICRP-69, 1995a).

10.7 Summary

In order to make the amount of data manageable for the reader, this chapter has been limited to intakes and doses for only two generic potentially exposed groups of people, namely workers and adult members of the public as defined by ICRP. It is concluded that:

- chemical toxicity and radiotoxicity should be considered carefully in assessing the risk to the individual since either could dominate under different exposure scenarios.
- the likely exposure pattern should be identified as far as possible since this will substantially affect body retention and excretion parameters, and hence assessment of intake and dose.
- the chemical forms of uranium or DU, and their proportions, in the aerosol should be identified as far as possible since this will considerably improve assessments of intake and dose.
- material-specific data should be used whenever possible for predicting the biokinetics of uranium and DU; while much of this information has yet to emerge, the known behaviour of the likely constituents of the aerosol will make a substantial contribution towards realistic assessments of intake and dose.
- until more information on the chemical form of uranium and DU in the environment is obtained, it would be prudent to assume that it is in a soluble form (ICRP Type F).

- the lack of an appropriate wound model should not prejudice estimates of the uranium content of systemic tissues, notably the kidneys (See Chapter 12).

Currently there are gaps in knowledge in the following areas:

- Biokinetic data on DU aerosols with emphasis on the effect of variable physical-chemical composition resulting from the use of munitions.
- Bioavailability of uranium after dispersion and re-suspension of DU dusts and aerosols.

11 Monitoring for internal exposure to depleted uranium

In principle, the deposition of DU in the respiratory tract after inhalation and its subsequent uptake to organs in the body can be assessed from:

- external radiation measurements of DU in the chest
- the assay of DU excreted in urine
- the assay of DU excreted in faeces

The introduction of DU into the body via ingestion can be assessed using similar techniques for urine and faecal assessment. The specific case of DU ingestion is described in more detail in Chapter 12.

For wounds, DU can be measured directly at wound sites, and uptake can be assessed by excretion measurements.

The choice and efficacy of each procedure is dictated by the route of intake, the pattern of exposure, the physical and chemical form of the uranium, the time between intake and measurement, and the limit of detection of the analytical procedure used. The use of bio-indicators for assessing nephrotoxicogical effects in the kidneys is discussed in Chapter 8.

11.1 External monitoring of the chest

Measurements can be made of DU in the respiratory tract by external radiation counting. Such monitoring is sometimes referred to as 'Whole Body Monitoring'. To make a positive identification of DU (rather than natural uranium), it is necessary to measure both the ^{235}U and ^{238}U lung contents. The main gamma-emissions for these two radionuclides are at 186 keV (^{235}U), 63 keV and 93 keV (^{238}U). The last two are actually from the ^{234}Th daughter of ^{238}U, which will, in practice, always be present. All are low intensity emissions: the gamma-emission for ^{235}U is relatively high yield, but the activity fraction of ^{235}U in DU is low (~ 1.1%), while the gamma-emissions for ^{238}U are both low yield. The low intensities, taken together with the relatively low gamma energies, mean that specialized counting systems (for example using germanium (Ge) semiconductor detectors) are required.

Under optimum conditions, the minimum detectable amount of natural uranium in the chest may be as low as 2 to 3 mg (Lane et al., 1985, Palmer and Rieksts, 1985, Pomroy and Malm, 1985; Toohey et al., 1991). For DU, the value is in practice likely to be rather higher, at about 8 mg for a 45 minute measurement (see Table 11.3). Chest monitoring using Ge detectors is feasible if measurements can be made soon after the exposure took place. However, for exposures taking place years previously, only a small fraction of the amount initially deposited remains in the lung. Significant intakes may then result in lung activities that are below the limit of detection for chest monitoring, and so in these circumstances chest monitoring may not be useful.

Amounts remaining in the respiratory tract at various times following inhalation have been calculated under two separate conditions, broadly characterized as occupational exposure (Table 11.1) and public exposure (Table 11.2). The occupational exposure case is appropriate for military personnel who may have been exposed to DU as a result of a single incident on the battlefield (i.e. 'acute' exposure) or remediation. The public

exposure case is appropriate for the general population who may have been exposed over a protracted period, up to the time of measurement, to DU in the form of general environmental contamination (i.e. 'chronic' exposure). Calculations were performed using the ICRP-recommended default model parameters for occupational or public exposure, and default absorption parameters for moderately soluble (Type M) and insoluble (Type S) materials; and also using estimates of the most appropriate model parameters for DU (Tables 10.1, 10.4, 10.5). The time-dependent retention for Type M and Type S materials in the lungs after acute or chronic exposure are shown in Figures 10.1 and 10.6.

A measure of the sensitivity of the measurement technique is the minimum detectable intake of DU. This depends not only on the minimum detectable amount (MDA) in the respiratory tract, but also on the time between intake and measurement. Tables 11.3 and 11.4 give the minimum detectable intakes for lung measurements made at the times given in Table 11.1, for an acute occupational exposure and for chronic public exposure.

Table 11.1 Uranium lung retention after an acute intake by inhalation: occupational exposure.

Time after intake (d)	**Lung retention [c] (% of intake)**		
	Type M [a]	**Type S [a]**	**DU [b]**
1	5.76	6.43	5.98
7	5.18	5.95	5.03
30	3.84	4.94	4.02
365	0.40	2.65	1.56
3650	~ 0	0.33	0.01

a For Type M and Type S, ICRP-recommended default model parameter values for occupational exposure were used (see Tables 10.1 and 10.4 for values)
b For DU, estimates for the most appropriate model parameter values for occupational exposure were used (see Table 10.4 and 10.5 for values)
c Retention in the tracheo-bronchial airways and thoracic lymph nodes is included

Table 11.2 Uranium lung retention after a chronic intake by inhalation: public exposure.

Time after start of intake (d)	**Lung retention [c] (% of daily intake)**		
	Type M [a]	**Type S [a]**	**DU [b]**
1	12.2	13.6	15.8
7	79.2	89.4	92.3
30	292	348	346
365	1260	2710	2340
3650	1410	9430	4670

a For Type M and Type S, ICRP-recommended default model parameter values for public exposure were used (see Tables 10.1 and 10.4 for values)
b For DU, estimates for the most appropriate model parameter values for public exposure were used (see Table 10.4 and 10.5 for values)
c Retention in the tracheo-bronchial airways and thoracic lymph nodes is included

Table 11.3 Minimum detectable acute intakes for DU by lung counting: occupational exposure.

Time after intake (d)	Minimum detectable intake (mg)					
	Based on detection of ^{238}U [a]			Based on detection of ^{235}U [b]		
	Type M	Type S	DU	Type M	Type S	DU
1	140	130	130	380	340	370
7	160	140	160	420	370	440
30	210	160	200	570	440	550
365	2000	300	520	5500	830	1400
3650	2×10^{11}	2400	79 000	4×10^{11}	6600	210 000

a MDA for ^{238}U in lung is estimated to be 100 Bq (~ 8.0 mg DU), based on counting statistics
b MDA for ^{235}U in lung is estimated to be 3.5 Bq (~ 22 mg DU), based on counting statistics

Table 11.4 Minimum detectable chronic intakes for DU by lung counting: public exposure.

Time after start of intake (d)	Minimum detectable intake (mg) [a]					
	Based on detection of ^{238}U [b]			Based on detection of ^{235}U [c]		
	Type M	Type S	DU	Type M	Type S	DU
1	66	59	51	180	160	140
7	71	63	61	190	170	170
30	82	69	70	230	190	190
365	230	110	130	640	300	340
3650	2100	310	630	5700	850	1700

a Sum of daily intakes
b MDA for ^{238}U in lung estimated to be 100Bq (~ 8.0 mg DU), based on counting statistics
c MDA for ^{235}U in lung estimated to be 3.5 Bq (~ 22 mg DU) , based on counting statistics

As indicated in Table 10.6, for the acute occupational exposure case, an intake of 410 mg DU would result in a committed effective dose equal to the annual dose limit of 20 mSv. Thus, if a positive identification of ^{235}U is required, it can be seen that the sensitivity of chest monitoring is barely adequate even if monitoring takes place within the first few days. However, if the assessment of intake can be based on the measurement of ^{238}U only, chest monitoring could be usefully employed for a few months after the exposure. This would be the case where the exposure to DU could have been the only inadvertent exposure to uranium; note that exposure to natural sources of uranium would be extremely unlikely to result in amounts in the chest approaching the MDA.

It should be noted, however, that even under optimum conditions, the minimum detectable intake corresponds to a dose that is a significant fraction of the annual dose limit for occupational exposure. Furthermore, it should be noted that chest monitoring would have inadequate sensitivity whatever the time of measurement if the material were to be more soluble than has been assumed here. This would be the case if a large fraction of the DU was in the form of UO_3, rather than UO_2 and U_3O_8.

For the chronic public exposure case, chest monitoring does not have sufficient sensitivity to detect intakes that would result in a committed effective dose of 1 mSv.

11.2 Urine and faecal monitoring

In principle, the assay of uranium in urine can be used for dose assessment after the uptake of any chemical form of uranium after inhalation and ingestion, or from wounds. However, the use of faecal assay is confined to intakes by inhalation of relatively insoluble forms such as UO_2 and U_3O_8.

Measurement of uranium excreted in urine at known times after the exposure is potentially a more sensitive method than chest monitoring for determining the amount of DU inhaled. However, uncertainties in the assessed intake can be quite large, because many assumptions concerning the aerosol size, solubility, and rates of movement around the body must be made. Another problem is that natural uranium is present in urine because of the ingestion of natural uranium in food and drink. Typically, an individual may excrete between 10 and 400 ng of natural uranium in urine each day, but levels can be much higher (ICRP–23, 1975, Roth et al., 2001). Differentiating between uranium that is excreted as a result of dietary intake of natural uranium, and uranium, that is excreted as a result of exposure to DU is a significant problem for any measurement technique.

The amounts of DU present in urine following a DU exposure may well be similar to, or even less than, the naturally occurring amount. For instance, an intake of 100 mg of DU by inhalation would give rise to about 25 ng DU in a 24-hour urine sample taken ten years after the exposure (based on default model parameters for occupational exposure to DU, Table 10.5). It is therefore necessary to measure the $^{235}U : ^{238}U$ isotopic ratio to determine the fraction of the measured uranium in urine that arises from a DU intake. Tables 11.5 and 11.6 give the excretion rates of uranium at various times following inhalation, as a percentage of the intake, for an acute occupational exposure and for chronic public exposure. The time-dependent excretion of DU after acute occupational exposure is shown in Figures 10.2 and 10.11, and after chronic exposure of the public in Figures 10.7 and 10.16.

Table 11.5 Urinary and faecal excretion rates of uranium after an acute intake by inhalation: occupational exposure.

Time after intake (d)	Urine (% / day) Type M [a]	Urine (% / day) Type S [a]	Urine (% / day) DU [b]	Faeces (% / day) Type M [a]	Faeces (% / day) Type S [a]	Faeces (% / day) DU [b]
1	2.32×10^{-0}	7.04×10^{-2}	1.09×10^{-0}	10.7	11.4	11.2
7	6.50×10^{-2}	1.93×10^{-3}	3.67×10^{-2}	2.26×10^{-1}	2.47×10^{-1}	2.35×10^{-1}
30	2.65×10^{-2}	7.70×10^{-4}	9.87×10^{-3}	2.72×10^{-2}	3.50×10^{-2}	3.04×10^{-2}
365	2.81×10^{-3}	2.64×10^{-4}	1.61×10^{-3}	3.45×10^{-4}	2.21×10^{-3}	1.29×10^{-3}
3650	2.21×10^{-5}	3.56×10^{-5}	2.46×10^{-5}	1.46×10^{-7}	8.16×10^{-5}	2.62×10^{-6}

a For Type M and Type S, ICRP-recommended default model parameter values for occupational exposure were used (see Tables 10.1 and 10.4 for values)

b For DU, estimates for the most appropriate model parameter values for occupational exposure were used (see Table 10.4 and 10.5 for values)

Table 11.6 Urinary and faecal excretion rates of uranium after a chronic intake by inhalation: public exposure.

Time after start of intake (d)	Urine (% of daily intake / day)			Faeces (% of daily intake / day)		
	Type M [a]	Type S [a]	DU [b]	Type M [a]	Type S [a]	DU [b]
1	1.42	2.84×10^{-2}	7.80×10^{-1}	1.71	1.82	1.81
7	2.32	5.45×10^{-2}	2.82	19.5	21.0	20.9
30	3.61	8.78×10^{-2}	3.60	21.2	23.1	23.0
365	8.70	3.29×10^{-1}	5.93	23.7	27.6	27.0
3650	9.67	1.00	8.40	23.8	32.0	28.7

a For Type M and Type S, ICRP-recommended default model parameter values for public exposure were used (see Tables 10.1 and 10.4 for values)
b For DU, estimates for the most appropriate model parameter values for public exposure were used (see Table 10.4 and 10.5 for values)

The main techniques available for measurement of uranium in urine are fluorimetry, alpha spectrometry and mass spectrometry. The capabilities of these techniques for DU monitoring are discussed below.

11.2.1 Fluorimetry

The minimum detectable amount for fluorimetry, based on empirical formulae, is in the range 1.5 to 7 ng of total uranium in a 24-hour urine sample, using the Kinetic Phosphorescence Analysis method. However fluorimetry cannot provide isotopic analysis, and so cannot differentiate between DU and natural uranium in urine. One possible use would be to screen samples for unusually high levels of uranium; the isotopic composition of samples above a screening level could then be measured using another technique. However, caution would need to be exercised, because the potentially large range of natural uranium urine levels could mean that screening levels would have to be set so high that significant levels of DU in urine could be missed.

11.2.2 Alpha spectrometry

The minimum detectable amount for alpha spectrometry, based on counting statistics, is approximately 0.1 mBq of ^{234}U, ^{235}U or ^{238}U in a 24-hour urine sample. Count times of approximately one week are required to achieve this sensitivity. For natural uranium, a measurement of 0.1 mBq of either ^{234}U or ^{238}U would correspond to about 8 ng of total uranium. Thus, naturally occurring levels of uranium in urine could be detected, although not with high precision. A sample containing natural uranium would need to contain about 200 ng of uranium before ^{235}U could be detected, because of this isotope's low activity fraction.

For DU, measurement of the ^{238}U activity would again allow the detection of about 8 ng total uranium. However, to make a positive identification of DU, either the ^{234}U or ^{235}U activities would have to be measured. The minimum masses of DU necessary to detect ^{234}U or ^{235}U are about 40 ng and 600 ng, respectively. If the uranium present was predominantly DU rather than natural, it should be possible to make a positive identification of DU at these levels. However, if a significant fraction of the uranium were natural, higher levels would be needed in order to determine the amount of DU present. The amount of DU that could be detected depends on the content of natural uranium and the accuracy of the determination of the ^{235}U (or ^{234}U) and ^{238}U levels.

Because of the relatively poor sensitivity of alpha spectrometry to ^{235}U in either natural uranium or DU, the determination of the fraction of the uranium in urine that is DU would normally be done on the basis of the measured ^{234}U : ^{238}U ratio. However, a problem arises because this ratio measured in urine can vary from 1:1 up to 3:1 (Hurtgen, 2001). The increase in this ratio above its expected value of 1:1 arises because of the influence of geochemical processes on uranium in groundwater. In order to use the technique, it would be necessary to establish the isotopic ratio of natural uranium in urine for each potentially contaminated individual measured, perhaps by the use of control measurements on uncontaminated individuals living at the same location. It is necessary to take a sufficiently large number of baseline measurements so that the background distribution can be reasonably established. For an occupational monitoring program, the measured distribution (typically log-normal) can be used to establish a confidence interval above which exposures are considered to be occupationally derived. The frequency of sample collection can then be adjusted accordingly.

11.2.3 Mass spectrometry

Specialized mass spectrometric techniques (such as Multi Collector Inductively Coupled Plasma MS, MC-ICP-MS; High Resolution Inductively Coupled Plasma MS, HR-ICP-MS; or Thermal Ionization MS, TIMS) can provide isotopic analysis at levels lower than can be achieved by alpha spectrometry. Depending on the technique, amounts of total uranium in a 24-hour urine sample in the range 1 fg to 5 pg can be detected. However, larger amounts are required to detect the change in isotopic ratio that would indicate the presence of DU. Again, the amount of DU that could be detected depends on the content of natural uranium and the accuracy of the determination of the ^{235}U and ^{238}U levels. On current information, it appears that the minimum detectable reduction in $^{235}U/^{238}U$ ratio from its natural value is approximately 10% (Harwell, 2001). For typical levels of natural uranium, amounts of DU in a 24-hour urine sample as low as about 10 ng (perhaps lower than this in some cases) could therefore be detected. Caution is needed since further development of their methods is needed to allow a satisfactory determination of the $^{235}U/^{238}U$ ratio in urine samples that contain natural uranium levels typical of healthy subjects (Roth et al., 2001).

On the assumption that 10 ng of DU in a 24-hour sample can be detected, Tables 11.7 and 11.8 give the corresponding minimum detectable intakes for measurements of 24-hour urine and faecal excretion made at the times given in Table 11.5, for an acute occupational exposure and for chronic public exposure.

Table 11.7 Minimum detectable acute intake for DU by bioassay/mass spectrometry: occupational exposure.

Time after intake (d)	Minimum detectable intake (mg)					
	Urine excretion			Faecal excretion		
	Type M	Type S	DU	Type M	Type S	DU
1	0.00043	0.014	0.00092	0.000093	0.000088	0.000089
7	0.015	0.52	0.027	0.0044	0.0040	0.0043
30	0.038	1.3	0.10	0.037	0.029	0.033
365	0.36	3.8	0.62	2.9	0.45	0.78
3650	45	28	41	6800	12	380

Minimum detectable amount of DU assumed to be 10 ng in a 24-hour sample

Table 11.8 Minimum detectable chronic intake for DU by bioassay/mass spectrometry: public exposure.

Time after start of intake (d)	Minimum detectable intake (mg) [a]					
	Urine excretion			Faecal excretion		
	Type M	Type S	DU	Type M	Type S	DU
1	0.00070	0.035	0.0013	0.00058	0.00055	0.00055
7	0.0030	0.13	0.0025	0.00036	0.00033	0.00033
30	0.0083	0.34	0.0083	0.0014	0.0013	0.0013
365	0.042	1.1	0.062	0.015	0.013	0.014
3650	0.38	3.7	0.43	0.15	0.11	0.13

a Sum of daily intakes
Minimum detectable amount of DU assumed to be 10 ng in a 24-hour sample

For the acute occupational exposure case, it can be seen that mass spectrometric measurements of DU in urine can detect intakes as low as about 40 mg DU, ten years after the exposure. Such an intake would give rise to a committed effective dose of about one tenth of the annual dose limit for occupational exposure.

For the chronic public exposure case in which the intake is spread uniformly over a period of exposure of up to ten years and continues up to the time of measurement, mass spectrometric measurements of DU in urine can detect intakes as low as 0.43 mg. In practice, the exposure pattern is unlikely to be so well characterized, but sensitivity should be adequate to determine an intake resulting in a committed effective dose of 1 mSv.

The data given in Table 11.7 and 11.8 for DU indicate that faecal monitoring has comparable sensitivity to urine monitoring, and furthermore that if the material were more insoluble than expected, would actually have significantly greater sensitivity. However, faecal monitoring has some significant disadvantages. Firstly, the natural variability in the amount of uranium ingested results in a large day-to-day variability in faecal excretion, which would present additional problems for the determination of the amount of DU. Secondly, the difficulty in obtaining samples in most cases would probably mean that its potential advantages could not be exploited, except where a special investigation was being carried out.

11.3 Wound monitoring

DU fragments present in the body as a consequence of wounds have been identified by clinical examination using X-rays (Hooper et al., 1999; McDiarmid et al., 1999). The results suggest that small fragments can penetrate deep into soft tissue without clear evidence of a superficial wound. Some fragments about 20 mm in diameter could be identified by this procedure, but most were less than 1mm. In some cases, elevated urinary excretion of uranium was observed, although DU fragments could not be identified by this procedure. Hence, X-ray techniques should not be regarded as definitive, and at best can provide only a semi-quantitative indication of internal contamination.

Following the Gulf War, probes have been developed for the specific purpose of assessing wound contamination with DU (Chandler, 2000). Tissue equivalent wound

phantoms containing various amounts of DU metal embedded at various depths (0.97 cm, 3.91 cm, 7.73 cm) were used to compare the efficiencies of bismuth germinate (BGO) and sodium iodide (NaI(Tl)) detectors. The results showed that the lowest minimum detectable activities for the shallow, medium and largest depth phantoms were 5.8 kBq, 3.5 kBq and 10 kBq respectively.

11.4 Monitoring for individuals potentially exposed to DU aerosols

The data presented in Tables 11.1 to 11.8 demonstrate the importance of carrying out monitoring as soon as possible after a potential acute exposure. In the case of chest monitoring, useful results are obtainable for a few months after an intake, although sensitivity greater than that which is presently achievable would be desirable. For urine monitoring with isotopic analysis by mass spectrometry, adequate sensitivity is probably achievable for samples taken ten years or more after a potential acute exposure, but uncertainties in assessed intakes are likely to be quite large. With the present state of knowledge of long-term uranium excretion, it is difficult to quantify uncertainties, but errors of up to a factor of 10 are possible. Uncertainties would be significantly reduced if monitoring were to take place soon after a potential exposure.

For retrospective assessment of potential exposures taking place more than one year previously, measurements of DU in urine using high sensitivity mass spectrometry (e.g. High Resolution Inductively Coupled Plasma Mass Spectrometry, HR-ICP-MS; Multi-Collector Inductively Coupled Plasma Mass Spectrometry, MC-ICP-MS; or Thermal Ionization Mass Spectrometry, TIMS are recommended. However, there remains considerable uncertainty about the capabilities of these techniques for this particular application, and it will be important for methods to be validated and performance-tested before they are used as part of a screening programme.

Initial screening using lower cost, lower sensitivity methods (e.g. Quadrupole ICP-MS) could be considered, but it would be necessary to confirm that such methods have the capability to detect the presence of (if not to quantify) significant levels of DU in urine. If DU is detected in urine in significant amounts, chest monitoring could provide additional useful information for intakes that could have taken place up to a few months previously. However, for potential exposures occurring approximately ten years before a measurement, chest measurements are unlikely to provide useful information.

11.5 Monitoring for those with health effects attributed to exposure to depleted uranium

All of the monitoring techniques available for retrospective assessment of intake have large inherent uncertainties. For the small number of people for whom it has been suggested that observed health effects are a result of exposure to DU, all possible monitoring methods should be considered.

For *in vivo* (body) monitoring, chest monitoring should be performed; consideration should also be given to the measurement of skeletal activity, although there appears to be no publications on this matter at present. Assessment of activity in tracheo-bronchial and broncho-pulmonary lymph nodes should also be considered (although it is recognized these would be difficult measurements to perform and interpret).

For bioassay monitoring, assessments of DU urine and faecal excretion rates using high sensitivity mass spectrometry techniques should be performed.

11.6 Summary

Monitoring for internal exposure to DU, particularly where single intakes may have occurred more than about one year previously, presents significant difficulties. High sensitivity mass spectrometric measurement of uranium isotopes excreted in urine is recommended as the main monitoring method. However, the capabilities of these techniques for the measurement of DU in urine should be confirmed before they are used as part of a screening programme. Body monitoring, to determine the amount of any uranium contamination in the lungs, may also be of some use for the assessment of occupational exposure during the first few months following an intake.

12 Biokinetics of uranium species from the standpoint of nephrotoxicty

The behaviour of uranium in the body predicted using ICRP biokinetic models, in association with default absorption parameter values for Type F, M and S compounds, and material-specific absorption parameter values for the oxides have been described in Chapter 10. In this Chapter, the focus is on the potential nephrotoxicity of uranium after inhalation of these materials, or after ingestion of soluble forms of the element.

12.1 Inhalation of uranium

Using the same default parameter values for uranium Type F, M and S compounds as given in Table 10.1, the time-dependent retention of uranium in the kidneys after an acute unit intake (Bq or µg) by occupationally exposed workers and chronic intake (Bq/d or µg/d) by members of the public are given in Figures 12.1 and 12.2. For comparison, the predicted kidney retention kinetics after the inhalation of U_3O_8, UO_2, U_3O_8, mixed U/Fe oxide and DU default using the same absorption parameters as given in Table 10.5 are shown in Figures 12.3 and 12.4.

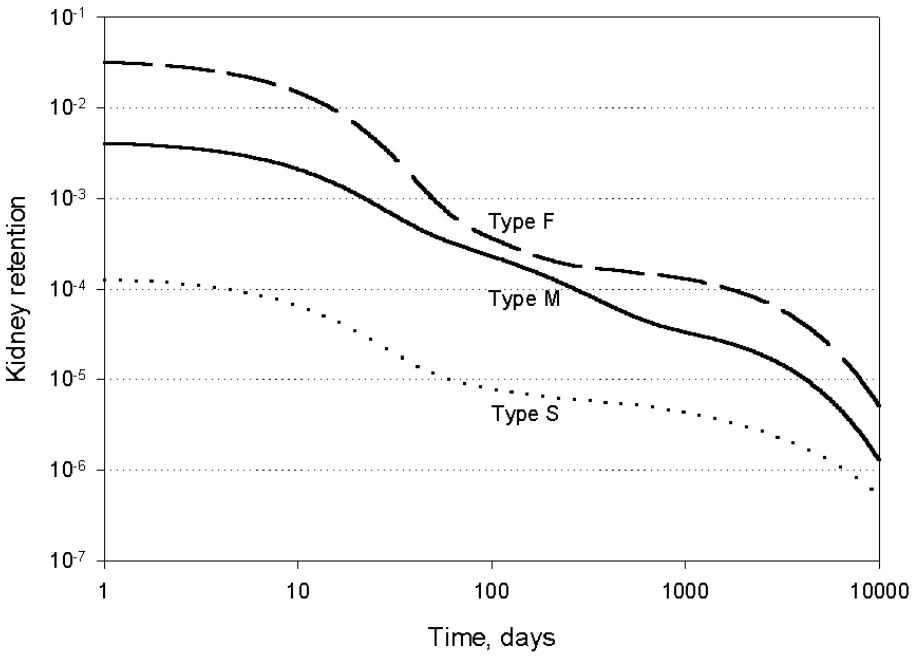

Figure 12.1 Fractional retention of inhaled uranium in the kidneys after acute inhalation of Type F, M and S compounds: occupational exposure.

After acute intake by workers, the maximum concentration of uranium in the kidneys occurs about one day after exposure. The intake that will result in a specified amount, or concentration in the kidneys (assuming that the mass of the organ is 310 g), can be deduced from Figures 12.1 and 12.3. The intake values given in Table 12.1 refer to the usually accepted kidney limit of 3 µg/g; the intake for any other specified concentration e.g. 0.3 µg/g of kidney will of course be proportional. Table 12.1 shows that for workers, the permissible concentration of uranium in the kidneys for U_3O_8 and UO_2 in particular are most unlikely to be exceeded in any exposure scenario. Similar conclusions may apply for members of the public, even if the acceptable concentrations in the kidneys were reduced by an order of magnitude to 0.3 µg/g.

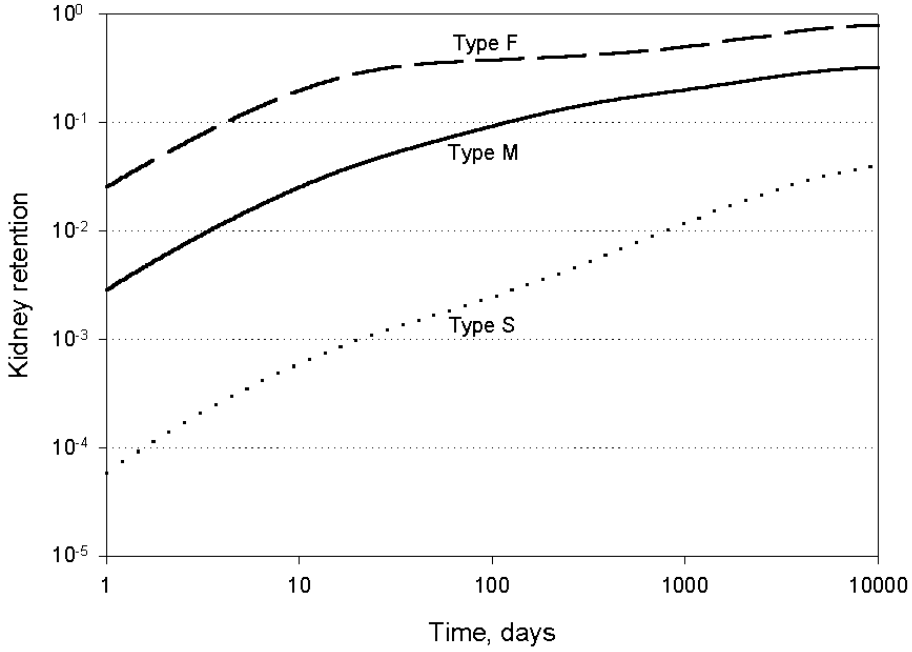

Figure 12.2 Fractional retention of inhaled uranium in the kidneys after chronic inhalation of Type F, M and S compounds: public exposure.

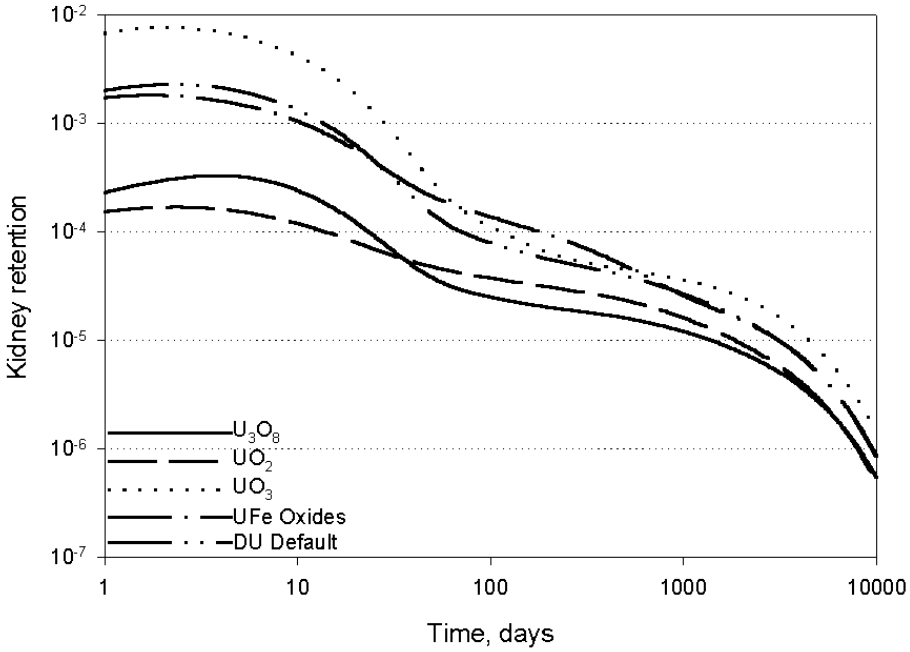

Figure 12.3 Fractional retention of inhaled uranium in the kidneys after acute inhalation of uranium oxides and the DU default: occupational exposure.

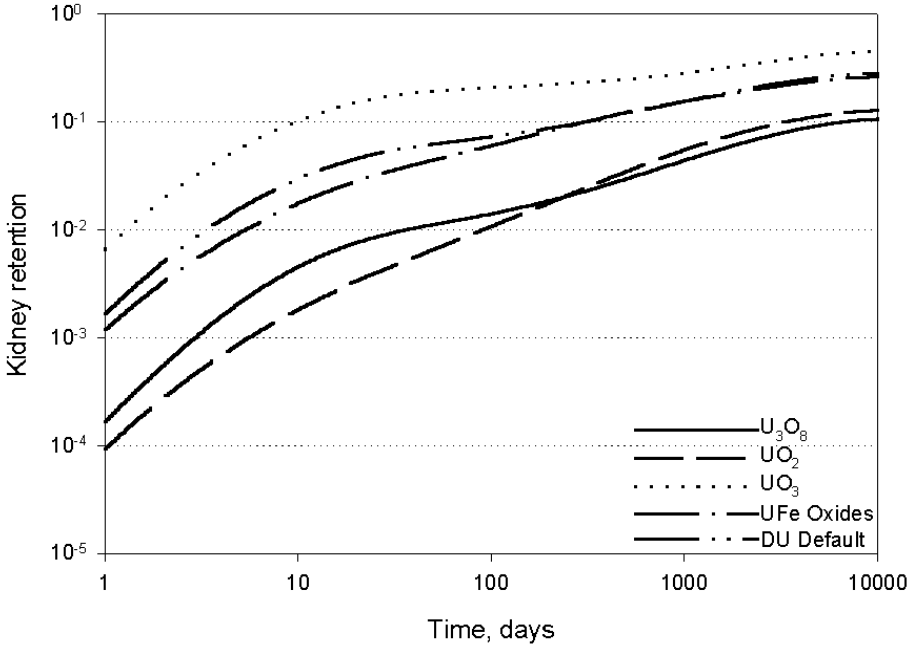

Figure 12.4 Fractional retention of inhaled uranium in the kidneys after chronic inhalation of uranium oxides and the DU default: public exposure.

Table 12.1 Inhalation intakes of uranium which result in a maximum concentration of 3 μg/g in the kidneys: occupational and public exposure.

Compound	**Intake (mg)**	
	Occupational (5μm)	Public (1μm)
Type S	7400	12510
Type M	230	290
Type F	30	35
U_3O_8	4010	3050
UO_2	6060	5920
UO_3	140	85
UFe oxide	540	490
DU default	460	325

Interestingly, workers whose intakes have exceeded the daily intake limit for moderately soluble (Type M) compounds of uranium (2 mg) by about five-fold have shown no evidence of nephrotoxicity or kidney malfunction (Boback, 1975). This would appear to support the choice of 3 μg/g as a 'safe' limit for the uranium concentration in the kidneys. However, the information given in Table 12.1 indicates that, using the current ICRP models, the amounts deposited in the kidneys as a consequence of intakes in excess of 10 mg or so would have been considerably less than those estimated using the historical model on which the occupational exposure standard was based (see Annex 5).

After chronic intake by members of the public, the concentration in the kidneys will increase progressively with time (Figures 12.2, 12.4). For an assumed intake of 1 mg/d of uranium it can be deduced from Figure 12.2, that the concentration in the kidneys will exceed 0.3 μg/g (93 μg total) after about four days for a Type F compound and about 100 days for a Type M compound. In both cases a concentration of 3 μg/g

(930-µg total) would not be exceeded until after 10 000 days. Values for the oxides can be calculated from Figure 12.4. Clearly material-specific information is vital for assessing the potential nephrotoxicity of uranium.

12.2 Ingestion of uranium in drinking water and foods

The information given in Figure 12.5 shows the amount of uranium present in the kidneys after a continuous chronic intake of a unit amount, say 1 µg/day. For comparison, the amounts present in the skeleton and excreted daily in urine are included. The calculations are based on the ICRP systemic model for uranium, assuming that the fractional uptake from the gastrointestinal tract is 0.02 (ICRP-69, 1995a). Figure 12.5 shows that at equilibrium the fractional amount present in the kidneys is 0.067. In other words, a continuous daily intake of 4 µg/d (the current WHO recommended guideline, Chapter 14) would result in a kidney content of 0.27 µg, and a kidney concentration of 0.0009 µg/g. This value is substantially below what is generally regarded as a safe value.

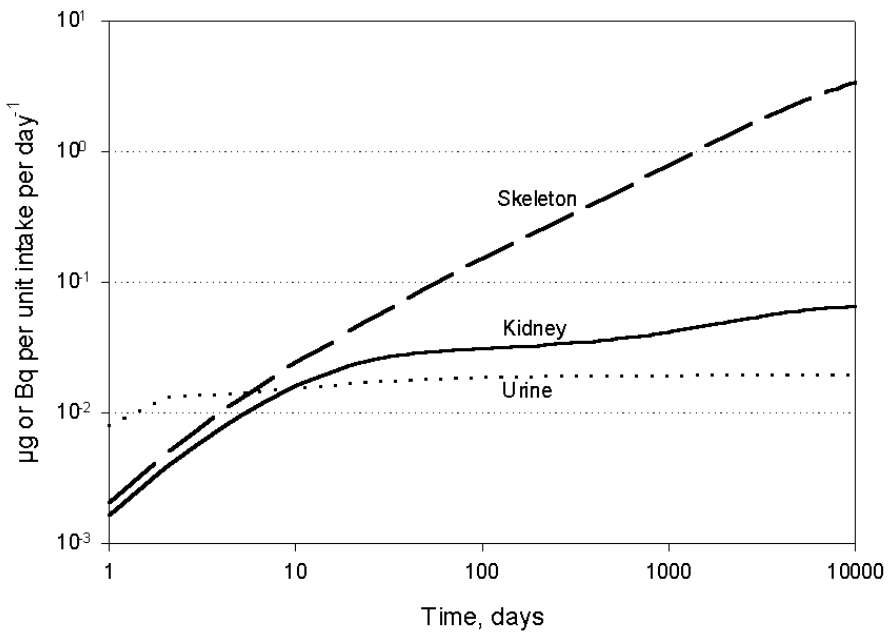

Figure 12.5 Fractional retention of ingested uranium in the kidneys and skeleton, and urine excretion rate after chronic ingestion of a Type F compound.

Whilst the discussion here has been concerned with drinking water, similar considerations would apply to the total dietary intake of uranium.

12.3 Relationship between kidney content and urinary excretion

The relationship between contemporary kidney content and urinary excretion after the acute and chronic inhalation of Type F and M compounds, using the HRTM (ICRP-66, 1994a) and systemic model for uranium (ICRP-69, 1995a) is shown in Figures 12.6 and 12.7.

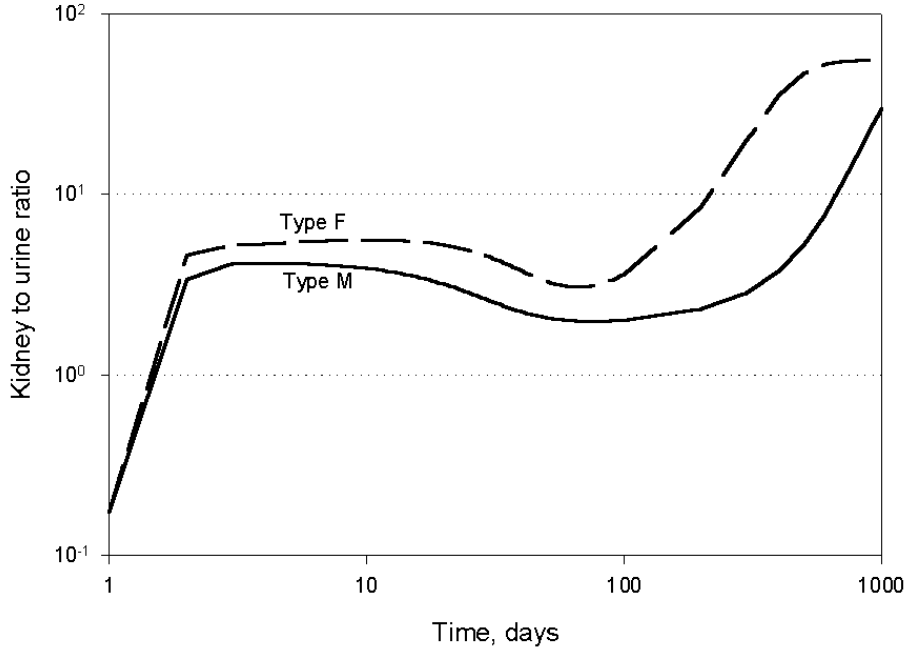

Figure 12.6 Kidney to urine ratio of uranium in the kidneys after acute inhalation of Type F and M compounds: occupational exposure.

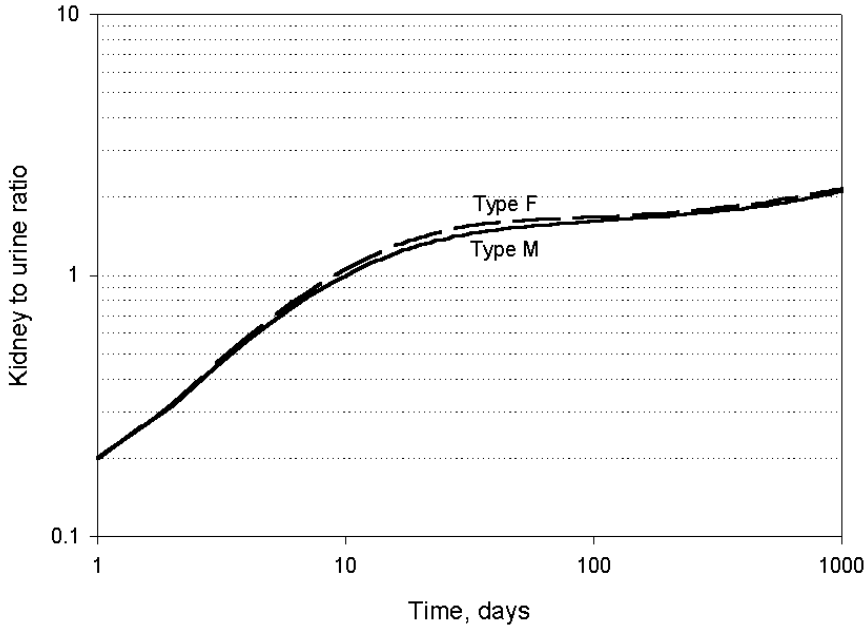

Figure 12.7 Kidney to urine ratio of uranium in the kidneys after chronic inhalation of Type F and M compounds: public exposure.

It is noteworthy that the ratios for Type F and M compounds up to 100 days after acute intake and up to 10 000 days after chronic exposure are closely similar.

It can be deduced from Figures 12.6 and 12.7 that between 7 days and 100 days the kidney to urine ratio is reasonably independent of the chemical form of intake, particle size of the aerosol or pattern of exposure. The variation in ratio during this interval is from about 1:1 to about 5:1. An assumed average value of 2.5:1 would appear to be a useful compromise. Thus, judgements on the health status of the individual could be

readily made by comparing the calculated kidney content with that obtained using the threshold value of 3 μg/g or an acceptable fraction of it, say 0.3 μg/g (see Annex 5) .

Similar calculations have been made after direct intravenous injection of uranium into the blood, simulating absorption from a contaminated wound. The values for the kidney:urine ratios are similar to those obtained after inhalation (information from A Phipps, UK-NRPB 2001).

It is concluded therefore that the kidney:urine ratio could be used with advantage in some circumstances for assessing the likelihood of potential nephrotoxic effects. However, it should be recognized that excessive concentrations in the kidneys may suppress the kidney:urine ratios. Studies with rats have indicated that this is less than two fold even when the kidney concentration is 3 μg/g (Hodgson et al., 2000).

12.4 Modelled kidney concentrations resulting from WHO standards

Uranium concentrations in the human kidney ranges from 0.000 33 to 0.0055 μg/kg with a median of 0.0022 μg U/kg kidney (converted from Bq/kg as ^{238}U from UNSCEAR, 2000). The results of the modelling of derived WHO tolerable intakes are (see Chapter 8 and WHO, 1998b) described in the following sections.

12.4.1 Oral consumption at the TI (soluble compounds*)*

In Chapter 8 WHO derives a tolerable daily intake of 0.5 to 0.6 μg/kg/d. For a 60 kg adult this corresponds to a daily intake of 36 μg/day (13.14 mg/annum). Based on a fractional intake at biokinetic equilibrium for the kidney of 0.067 (Figure 12.5) this would result in a kidney content of 2.41 μg and a resulting kidney concentration (assuming a kidney weight of 310 g) of 0.008 μg/g.

Estimated radiological dose (to kidney or whole body) resulting from the ingestion of 13.14 mg/y of DU to an adult member of the public and based on ICRP biokinetic models is 9.4×10^{-3} mSv/year.

12.4.2 Ingestion at the WHO drinking water guideline value

The current WHO recommended drinking water guideline (for natural uranium, Chapter 14) is 2 μg/l which is considered to be protective, and is based on the assumption that allows 10% of the TI intake of uranium to come from uranium derived from drinking water (at a drinking water consumption of 2 l/day, 60 kg adult). Consumption at the guideline level gives a daily uranium intake of 4 μg (1.46 mg/y). Based on a fractional intake at equilibrium for the kidney of 0.067 (Figure 12.5) this would result in a kidney content of 0.268 μg and a resulting kidney concentration (assuming a kidney weight of 310 g) of 0.0009 μg/g. This value is substantially below what is generally regarded as a safe value within the occupational context. However, it is recognized that this standard is not applied universally and is significantly lower than that suggested in other countries (see Chapter 14).

Estimated radiological dose (to kidney or total body) resulting from the ingestion of 1.46 mg/y of DU by an adult member of the public is 1×10^{-3} mSv/year.

Consumption of drinking water containing natural uranium at levels significantly above this is possible in a number of countries (See Annex 3 and Chapter 3). This could result in significantly greater kidney concentrations (at which changes in kidney function, that

might be considered to be detrimental, may be observed e.g. Zamora et al., 1998). For example exposure to uranium in drinking water at non atypical vales (20 µg/l) and maximum concentrations (1600 µg/l) of U observed in studies cited in Annex 3 would result in kidney concentrations of 0.009 µg/g and 0.72 µg/g respectively.

Estimated radiological dose to an adult member of the public resulting from the ingestion of drinking water containing 20 µg/l and 1600 µg/l of DU is 1×10^{-2} and 0.8 mSv/year respectively.

12.4.3 Inhalation at the TI for Type F (soluble)

The recommended TI for inhalation for Type F compounds in Chapter 8.4 (0.6 µg/kg/day) is equivalent to an intake of 36 µg/day (13.14 mg/y, DU Type F, Table-10.3). Using the information from Figure 12.2, for chronic exposure, it can be deduced that the kidney content at equilibrium will be 36 µg, and the concentration 0.12 µg/g of U. This is reasonably consistent with the suggested value of about 0.3 µg/g (Annex 5).

Estimated radiological dose resulting from the inhalation of 13.14 mg/y of DU by a member of the public is 9.9×10^{-2} mSv/year.

12.4.4 Inhalation at the TI for Type M (moderately soluble) compounds

The recommended TI for inhalation of Type M compounds in Chapter 8.4 (0.6 µg/kg/day) is equivalent to an intake of 36 µg/day (13.14 mg/y), DU Type M, Table 10.3). Using the information from Figure 12.2, for chronic exposure, it can be deduced that the kidney content at equilibrium will be 30 µg, and the concentration 0.10 µg/g.

Estimated radiological dose resulting from the inhalation of 13.14 mg/y DU (Type M) by an adult member of the public is 0.58 mSv/year.

12.4.5 Inhalation at the TI for Type S (insoluble) compounds

The TI for inhalation of Type S compounds in Chapter 8.4 (5 µg/kg/day) is equivalent to an intake of 300 µg/day (110 mg/y, DU Type S, Table 10.3). Using the information from Figure 12.2, for chronic exposure, it can be deduced that the kidney content at equilibrium will be 21 µg, and the concentration 0.12 µg/g. This is reasonably consistent with the suggested value of 0.07 µg/g (Annex 5).

Estimated radiological dose resulting from the inhalation of 110 mg/y of DU by an adult member of the public = 13.4 mSv/year. (Note whilst unacceptable for a member of the public this dose may be acceptable for a worker (see Chapter 9 and Annex 5).

12.5 Summary

The time-related retention of uranium in the kidneys has been discussed after acute and chronic inhalation of compounds with widely different solubility characteristics by workers and adult members of the public. The chronic ingestion of soluble uranium in drinking water has also been addressed. It is concluded that:

- the likely exposure pattern should be identified since this will substantially affect the time-related amounts retained in the kidneys, and hence judgements on nephrotoxicity.
- nephrotoxicity after intakes of Type S compounds, and uranium octoxide and dioxide is considered most unlikely in view of the considerable amounts that would need to be inhaled; even the inhalation of other materials such as mixed U/Fe, and a DU mixed oxide with a highly soluble component (20%) would need to be appreciable.
- in certain circumstances effective judgements on the likely nephrotoxicity of uranium can be deduced from kidney content extrapolated from the urinary excretion rate; the kidney to urine ratio is reasonably independent of the chemical form and exposure pattern from a few days to several months
- upper values for the kidney content of uranium can be deduced from consideration of its concentration in drinking water and daily intake.

Our current knowledge gaps include:

- Chemical composition and solubility of the aerosol inhaled.
- Chemical forms and solubility of uranium in drinking water.

13 Protective measures, health monitoring, and medical management

This chapter is intended for medical officers and health physicists who are not familiar with the detailed health and radiological protection issues associated with DU, but who need guidance on these matters. Hence, it is anticipated that it may be consulted in isolation. Inevitably there will be some repetition of material contained in other chapters, and the reader is referred to them when more information is required.

This chapter addresses practical questions concerning:

- background information useful for considering the nature and extent of contamination and response actions
- protective measures needed to prevent possible health effects from DU exposure
- health monitoring or medical surveillance of people living or working in areas where DU has been or is being used
- medical management of persons contaminated internally with DU

Protective measures, health assessment, and medical monitoring and management of public and occupational exposures are considered.

13.1 Background information

13.1.1 Public exposure

Area of contamination The area and levels of DU contamination in the environment should be determined. DU munitions produce fragments and dust of uranium metal and oxides, the extent of which depends on the magnitude of the impact, heat generated and explosive force. Heavier fragments and dust settle close to the site of impact while finer dust is carried further afield. Consequently, contamination levels decrease rapidly with distance. Finer dust produced on impact may be carried tens to hundreds of metres from the impact site. However, with increasing distance from the impact site, the DU dust concentrations will decrease rapidly. DU can also be released into the air from production and processing facilities. DU released to water through factory effluent, direct fallout, or erosion may settle onto silt or dissolve and be transported into groundwater. (For further information see Chapter 6).

Routes of exposure and magnitude of intake Assessment of the levels of intake of uranium and DU provide a basis for determining the potential for adverse health effects since every person exposed to DU is also exposed to uranium in air, food, and water. When civilians inhabit areas that contain DU, exposure could occur by inhalation of airborne contamination or resuspended dust, or ingestion of drinking water, contaminated food or soil, or by dermal contact. The human body contains about 90 μg of uranium from the normal intake of food, air and water. About 66% is present in the skeleton, 16% in the liver, 8% in the kidneys and 10% in other tissues. For further information on routes of exposure see Chapters 3, 5 and 6, and for the assessment of levels of intake see Chapters 10, 11 and 12.

13.1.2 Occupational exposure

In occupational situations the primary principle is to identify potential hazards and then control them with engineering and administrative measures supported by environmental monitoring and, if necessary, biological monitoring.

Hazard identification A hazard identification and control program should be established for workplaces when DU exposures are likely to occur. Occupational exposure to DU can occur during the production and handling of DU containing materials (shells, armour, ballast, shielding), and in the remediation and reclamation of contaminated areas. Additionally, people working in previously contaminated sites in occupations such as construction, farming, and other dust-producing activities may also be at increased risk of exposure to DU. Military personnel in combat situations are clearly at potential risk of DU exposure

Any operation or activity that handles or processes un-encapsulated DU should consider measures to protect workers from the potential chemical and radiological hazards. The level of protection to be used and the extent of the required monitoring program will depend on:

- the quantities of material being handled or processed.
- the physical and chemical properties of the material (e.g. chemical form and particle size).
- the nature of the operations being conducted.

Under normal circumstances, appropriate occupational health standards (see Chapter 14 and Annex 5) will apply to workers in both production facilities and environmental remediation activities.

Inhalation The most likely route of exposure at the workplace is the inhalation of uranium or DU. As indicated in Table 14.4, exposure limits exist for the control of uranium inhalation (HSE, 2000; ACGIH, 1993; NIOSH, 1994; ICRP, 1994a; NRC 1991). To verify that contamination control measures are effective, and to confirm that worker exposure is maintained within the appropriate limits, routine measurements of the levels of airborne DU should be conducted. Depending on the nature of the exposure, these measurements may need to be supplemented with urine sampling, faecal sampling and/or *in vivo* monitoring of the lungs for uranium.

The following sections highlight some of the major elements of an adequate monitoring program. Since a number of guidance documents exist for implementing such monitoring programs (ANSI, 1995; ICRP-54, 1988; ICRP-78, 1997; Rich, 1988; Stather, 1994), the reader is referred to these documents for further discussion, and other relevant chapters in this monograph.

13.2 Protective measures

Identifying appropriate protective measures for exposed individuals first involves establishing a framework for decision making by defining the range of protection measures and then identifying relevant protective actions.

Measures to protect the public from DU exposure are divided into the broad categories of locality-based, environment-based and medical-based actions, as described below, in response to a perceived, potential, or actual health threat. The initial response may be generic in nature, with actions becoming more focused and appropriate as data are collected and assessed as to level, chemical and physical form, and solubility relative to prescribed standards.

13.2.1 Locality-based protective measures

Locality-based protective measures are taken when data on which to base decisions are insufficient to determine if individuals are being exposed to DU, and that the situation indicates a reasonable probability of exposure. This could occur following the military or civilian use of DU, when a DU processing facility is suspected of releasing quantities of DU above allowable limits, when environmental monitoring results indicate new or elevated levels in any medium e.g. drinking water, or when medical symptoms may suggest an over exposure.

Precautionary actions include general advice published or reported by national or international organisations to reach as wide a cross-section of the population as possible. These should be coordinated with environmental and medical monitoring activities and be subject to constant assessment and revision following any specific environmental or medical investigations of the situation.

13.2.2 Environment-based protective measures

Environment-based protective measures are taken when sampling and survey results demonstrate the presence of excessive levels in a specified environmental medium to which the public is or may be exposed, or when a direct exposure pathway has been established. This occurs when levels measured in air, food, or water exceed regulatory limits or guideline levels (see Chapter 14 for more detail) or are likely to do so based on the anticipated spread of known contamination. Environmental response actions include limiting human and animal exposure to the contamination, or providing access to alternative clean or less contaminated supplies. These actions will need to be revised as further information becomes available.

13.2.3 Medical-based protective measures

Medical-based protective measures are taken when medical results confirm that an individual's health is being, or likely to be, compromised by exposure to DU. This could include measurements of uranium in the chest, biological samples (generally urine) along with assessment of renal proximal tubule damage (see Chapters 11, 12). Since no biomarker is specific to DU, and it is often impossible to quantify the measured value and potentially toxic effect, a combination of laboratory tests may be useful (Chapter 8). Care should be taken not to overlook other potential causes of adverse health effects, such as other toxicants or stressful conditions.

Screening for DU-associated disease in asymptomatic individuals or populations is generally not recommended. In individuals with high levels of uranium in urine, medical assessment should be undertaken.

13.2.4 Occupational measures

Control of DU exposure in an occupational environment also requires management directives on organizational and individual worker practices, such as restricting eating, drinking, or smoking in areas where DU exposures could occur, restriction of admittance to various work areas, training, record keeping, and hazard communication. The choice of appropriate monitoring programmes will be essential for adequate protection of the worker. These include:

Air monitoring Workplace air monitoring is the primary means available to demonstrate that the airborne levels of DU in a facility or project are within the desired exposure limits. When it is believed that air concentrations have the potential to exceed a pre-established fraction of the applicable exposure limits, a general area air-sampling program should be in place. The fraction of the regulatory limit at which monitoring is required will vary depending on local regulatory requirements. In the absence of local regulatory guidance, a value of 10% is commonly used.

For work practices that are difficult to cover using a fixed sampling location, such as remediation/clean-up activities, consideration should be given to the use of a personal air sampling device. These portable units have sampling heads that can be attached directly to a worker's collar, thereby providing a more accurate estimate of exposure than is possible with a fixed head unit. However sampling rates are low, typically 2l per minute, and results can be biased unless a centripeter device is used to remove non-respirable particles.

Internal exposure monitoring (biological monitoring) The worker protection program should include measurements that are capable of evaluating the extent to which workers are internally exposed to uranium. These measurements, which include urine sampling, faecal sampling and other *in vivo* examinations such as external monitoring of the chest, are referred to as bioassay measurements. When used in conjunction with appropriate metabolic models (ICRP-54 1988, ICRP-78 1997), and ideally, material-specific data on particle size and absorption parameter values for absorption to blood, these bioassay samples can be used to evaluate the extent of a worker’s internal exposure to DU (see Chapters 10,11). It is important that measurements are undertaken, and assessments of exposure are undertaken by organisations validated to do so. As part of the bioassay program, action levels should be clearly defined. The intent of these action levels is to ensure that acute effects due to chemical toxicity do not occur and that a worker’s radiological exposure is minimized. A number of guidance documents on establishing a bioassay program (with associated recommendations for chemical and radiological action levels) have been published (Rich, 1988; Stather, 1994 (see comments above); ANSI, 1995; ICRP-54, 1988; ICRP-78, 1997).

The choice of bioassay sample type and monitoring frequency is dependent on the solubility of the material being evaluated (see Chapter 11). For highly soluble uranium compounds, such as UF_6, urine samples will need to be collected as soon as possible after known or potential acute exposures. For routine monitoring purposes, and when exposures are likely to be significant, sampling intervals of about one week may be necessary. On the other hand, assessment of intake of poorly soluble forms such as UO_2, might be better evaluated using faecal sampling. In either situation, it may be desirable to supplement urine and/or faecal monitoring with external measurements of the chest. Being a direct measurement of the lungs, it ensures that any large intakes of uranium are not missed. Chest monitoring can be used to assess the contemporary lung content (limit

of detection about 8 mg) but if measurements are made soon after exposure, doses of a few tens of mSv can be identified (see Chapter 11).

Because uranium is naturally present in both urine and faecal samples, care should be exercised in the interpretation of positive results. All positive bioassay results should be interpreted in the light of the natural distribution of uranium present in the worker population. Prior to the initiation of work in a contaminated area, all workers should be required to submit a baseline bioassay sample. All bioassay results should be provided to workers with an explanation and counselling about their meaning and implications.

In some cases, it may be determined that bioassay monitoring is insufficient to evaluate a worker's exposure. This can be the case for facilities that handle highly insoluble forms of DU. In this situation, the use of supplemental breathing zone air samplers (BZAs or personal air samplers) should be considered. Many commercially available BZAs are capable of sustained flow-rates of up to five litres per minute. If one assumes a worker's breathing rate of 20 litres per minute, the BZA can be considered as a surrogate bioassay sample, which measures 25% of a worker's intake.

External exposure monitoring While the primary hazard from working with un-encapsulated DU is from internal exposure, as discussed in Chapter 9, large quantities of DU may present an external radiation hazard. In particular, exposures to the whole body (by gamma radiation from uranium and the progeny) and to the skin (from ^{234m}Pa, a beta-emitting member of the uranium decay series) can be significant. When large quantities of uranium are stored, there is also a potential for penetrating whole body exposure from DU which can reach a surface dose rate of 0.1 mSv/hr

If an environmental characterization indicates that there is a potential for workers to receive external doses or dose rates that exceed those identified in relevant regulations, then workers should be provided with personal dosimeters. These limits may vary with the regulatory agency. Dosimeters should be selected so that their energy response characteristics are appropriate for the radiation field being evaluated. Guidance documents for the implementation of an external dosimetry monitoring program are available (e.g. NCRP, 1999; Rich, 1988).

13.3 Preventative actions

This section covers a range of preventative actions available to communities and individuals for protection against DU over exposure, which may be achieved through changes to habits and activities.

13.3.1 Air

When the public have been advised that DU levels in air exceed regulatory or guidance values (such as may occur during military activities in which DU is used or released from a DU facility) some level of protection may be offered by closing windows and doors, securing ventilation, wearing a filtered respirator or mask or breathing through a damp cloth. Resuspension of DU dusts through wind erosion and both foot and vehicle traffic may be a source of continued low-level exposure. Exposure in these situations may be reduced through minimizing activities likely to resuspend dusts (e.g. removal of surface materials, fixation of the contamination, or watering and tilling to dilute the surface material available for resuspension or limiting access)

13.3.2 Children

Children should not play in areas where DU has been used or transported until the soil is acceptably clean. Two reasons for concern are that children exhibit hand-to-mouth activities leading to soil ingestion (especially geophagic children who can eat large amounts) and military activities may contaminate surface soil and dust accessible to children.

13.3.3 Concerned individuals

Individuals concerned about potential over exposure to uranium should consult a physician.

13.3.4 Contaminated items

Outer clothing and shoes that have possibly become contaminated should be removed outdoors. Before washing, segregate gently to minimize airborne levels, then wash groups of these clothes in succession, double-washing the most soiled, and including a clothes-free cycle to prepare the washer for uncontaminated clothing. Contaminated tools and vehicles, including the underside of vehicles and tires, should be washed in designated areas, where particulate contamination is removed in a suitable trap and disposed of appropriately.

13.3.5 Drinking water

Consumption of contaminated drinking water should be limited or avoided when total uranium (natural and DU) exceeds established limits or guidelines (see Chapter 14). This may be resolved by using appropriate filters, changing to bottled water that is known to contain acceptably low levels of uranium, or collectively on a community basis through mixing and blending with other uncontaminated water sources.

13.3.6 Exposed skin

If work or play occurs on contaminated soil, carefully wash hands, face and other exposed skin areas (e.g. the soles of the feet) thoroughly and again before eating or drinking.

13.3.7 Food

Food grown in DU contaminated areas may contain DU primarily attached to the surface of roots or leaves. Much of the DU should be removable by thoroughly washing or peeling all edible surfaces and removing any rough outer membrane. The drying of foods on potentially contaminated soils should be avoided. Governmental advice should be sought on levels of contamination that may restrict food consumption (Chapter 14).

13.3.8 Impacted areas

Where practicable, areas where significant DU contamination actually or potentially exists should be cordoned off until a survey has determined that it is safe for habitation. If levels warrant a clean-up of the area, the cordons should be retained and appropriately adjusted for actual conditions until results of a final status survey show the area is safe for unrestricted access.

13.3.9 Metal fragments, depleted uranium munitions, scrap metal and souvenirs

In all locations where DU has been used militarily, it is recommended that the public do not handle remnants of armaments (not least because of other more immediate hazards associated with unexploded ordinance). While the radioactivity of DU is low, large pieces of DU are capable of delivering significant doses to the skin (see Chapter 9).

Thus collecting of intact or fragmented DU penetrators, unexploded munitions/armour or other equipment containing DU for souvenirs or fabrication into other products should be actively discouraged (see Chapter 4).

13.4 Environmental monitoring

Environmental monitoring includes conducting radiation-level surveys and chemical-contamination surveys with portable equipment, and virtually always will include the collection of samples of environmental media (see Chapter 6 and Annex 6).

13.4.1 Radiation surveys

Surveys for DU may typically involve the use of hand-held monitors (such as an NaI(T1) type scintillation detector) with determinations being performed on a regular grid or being undertaken based on a local knowledge of areas of particular concern or vulnerability. Samples may also be taken from the field for more accurate chemical and radiochemical analysis in the laboratory (see below). Alternatively more complex surveys may be undertaken from the air or in specially equipped vehicles. In general, radiation surveys must only be undertaken by qualified professional bodies or persons who can demonstrate both professional competence in the detection of DU and have appropriate quality assurance and quality control in place.

13.4.2 Chemical contamination surveys

Contamination surveys for DU typically involve the removal of representative samples of material (water, soil, food, vegetation, dust, airborne dust etc.) from various locations of interest and transferring the samples in sealed containers back to a laboratory for detailed characterisation. The 3-dimensional location (ideally a recognized spatial grid coordinate and sample depth) being accurately recorded and selected based on the likelihood of including contamination or the vulnerability of a particular media to contamination (e.g. water sources used by many people). Sampling for DU could include air samples up- and down-wind of the impact or discharge point, surface soil samples for recent fallout or thicker samples for aged fallout, occasional core soil samples (for uranium and DU depth profiles and uranium reference levels for that area), river sediment and water up- and down-stream of any air plume or water discharge point (for accessibility to potable water for humans, food animals, and food crops), surface drinking water supplies, and locally produced food. Groundwater can also be collected, but its contamination from any source of DU is less likely; however, it is more likely than surface water to contain high levels of natural uranium and its decay products (see Chapter 3). Surveys of chemical contamination may be complex and if inappropriately conducted may lead to misleading positive and/or negative results. For this reason such studies are best undertaken by qualified professional bodies or persons which can demonstrate both professional competence in environmental sampling techniques and the chemical analysis of DU, and have appropriate quality assurance and quality controls in place. Some methods appropriate for the determination of DU and uranium in environmental media are described in Annex 6.

13.5 Health assessment

To determine if a health assessment program might be beneficial, an investigation should assess whether realistic circumstances exist for DU exposure. For example could a person, during the course of their activities, have ingested, inhaled or come into contact with significant amounts of DU? In such an appraisal it may be appropriate to reference international standards and guidelines (Chapter 14), provided that information relating to levels of actual exposure are available.

The conclusions presented in this report indicate that consideration should be given to both the radiological toxicity and chemical toxicity of DU. Dose limits for limiting effects due to ionizing radiation are given in Chapter 9 and their implications to various levels of DU exposure are described in Chapters 10 and 11. While the question of chemical toxicity is discussed in Chapters 8, 12 and Annex 5.

Health assessments may be conducted on overexposed individuals immediately following releases of DU through military operations or from processing plants or storage areas etc. It is also implemented in a phased manner on other individuals based on their potential for overexposure to DU from any source. This potential can be assessed, based on environmental monitoring results and medical monitoring of patients who present with symptoms that relate to DU over exposure, which can be further assessed through biological sampling. In essence, environmental and biological samples are analysed, the results used to assess exposure, which provides feedback to the sampling programs, ultimately ending in a risk assessment for individuals or population groups as a function of estimated exposure and potential health endpoint.

The sections below address the diagnosis, monitoring and treatment of medical conditions associated with exposure to DU or U.

13.5.1 Medical diagnosis

Pathophysiology

The pathophysiology of uranium and DU are discussed briefly below and in more detail in Chapters 7, 10 and 12.

Inhalation

Under various circumstances DU and uranium may become liberated in the form of dust and aerosols. Particles typically less than 10 µm AMAD will enter the deep lung, while the larger ones will be trapped by the upper respiratory tract and then either expectorated or swallowed. The amount of uranium or DU entering the blood and the rate of absoprtion will depend upon the particle size and chemical form (the smaller, more biologically soluble, particles tending to enter more efficiently and quickly). Elimination from the blood occurs primarily through the kidneys. During the first 24 hours after incorporation, up to 60% will be excreted and about 90% in the following few days. Most of the remainder will be deposited in the skeleton (see Chapters7, 9, 10, 11 and 12)

Ingestion

Absorption from the intestinal tract may vary from a few per cent to less than 0.2% depending on its chemical form. The remainder is excreted in faecal material. About 90% of the absorbed part will be eliminated by the kidneys, while the remaining part will mainly accumulate in the skeleton, with a biological half-life of about 300 days (see Chapter 7, 10, 11 and 12)

In the kidneys, uranium accumulates mainly in the proximal tubules. At elevated levels uranium is considered as nephrotoxic and can cause glucosuria and an albuminuria (see Chapters 8 and 12).

Dermal contact and uranium in wounds
Prolonged and direct contact with uranium may cause skin erythema, but at DU exposure levels it is not considered likely to reach a significant level of toxicity (see Chapter 8).

Embedded fragments
Soldiers with retained DU fragments typically demonstrate an enhanced level of urinary excretion of uranium than control groups. This finding may still be present seven years after exposure, suggesting a slow controlled release from the disintegrating fragments (see Chapter 12).

Long term pathophysiology
Below a certain concentration, kidney damage may be transitory (see Chapter 8). The only known human pathology due to the chemotoxicity of uranium is the destruction of renal tubules, without clinical manifestation, but causing a diminution of the kidney reserve function.

Theoretically, exposure to DU or uranium may lead to effects associated with ionizing radiation. Considering the amount of radioactivity involved, deterministic acute effects are excluded. Long term effects are represented by the increased probability of developing a cancer, although this risk remains extremely low (see Chapter 8). Modelling of exposure in extreme conditions (inhalation of dust shortly after the impact of projectiles) suggests that a calculated radiation dose of 10 mSv would occur, representing half of the maximum yearly dose limit for workers.

Diagnosis
Persons exposed to uranium normally show no specific clinical sign, apart from those non-specific symptoms associated with exposure to many other heavy metals.

If it is strongly suspected or known that a person has either inhaled or ingested large amounts of uranium dust, a presumption of uranium exposure can be gained by a thorough anamnesis. Details such as the time of the exposure, the exact location of the person, the distance from the impact etc, need to be determined to properly assess the extent of exposure. If the exposure extends over a long period of time and occurred mainly through ingestion, alimentary habits must be checked.

If the physician is convinced that there has been an exposure to uranium, the following routine laboratory tests should be performed:

- routine urine analysis (especially checking for albumin and glucose).
- blood urea and creatinine for kidney function.
- a complete blood count for anaemia.
- chest X-ray in case of a possible inhalation exposure to exclude lung damage.

In cases of severe exposure, the kidney may be the critical organ. Signs indicating a tubulopathy should be looked for. If the tubules are damaged, there will be many low molecular weight proteins that appear in the urine, among which the β_2-microglobulin is the most commonly assessed. Therefore the diagnostic procedure should be:

- determine the dosage of β_2-microglobulin in a 24-hours urine collection. Most major hospitals and medical laboratories are able to perform this analysis. The patients should be informed how to correctly collect the 24 hours urine sample. It should be

remembered that this protein is unstable in acid urine, so that it is important that the collection of the urine is performed according to the instructions of the laboratory

- if the result of this determination is pathologic, the amount of uranium excreted in urine in 24 hours should be determined. However, as only few laboratories are equipped for the determination of uranium in the urine, it is absolutely necessary to contact such a laboratory before collecting the urine and to follow their instructions. A spot urine analysis is of considerably less value, even when coupled with a creatinine determination. These factors are discussed in detail in Chapter 11, Annex 6 and the following sections.

Prognosis

So far only very few cases of acute uranium overdose have been reported in the literature. They are characterized by an acute renal failure, which may lead to a complete anuria and need temporary dialysis. An incomplete Fanconi syndrome, in the form of a renal tubular acidosis, may persist more than six months after exposure, requiring a daily supplement of sodium bicarbonate (Pavlakis et al., 1996).

For chronic exposure at low uranium levels no permanent damage has been reported. Occupational exposure of uranium mine workers is not known to have been detrimental to their health; only a few have developed transitory anaemia. Soldiers who have incorporated uranium fragments have not shown any renal problems. The radiological hazard is likely to be very small. No increase in leukaemia or other cancers has been established following exposure to uranium or DU (see Chapter 8).

13.5.2 Medical monitoring

Medical monitoring may include examination by a physician and the collection and analysis of biological samples for DU and indicators of potential health problems. At low levels of exposure to DU, no links to disease have been identified from pathology but there are data that subclinical indicators of renal function may be altered. There is a low risk of cancer from any radioactive material but no clear link has been established between exposure to DU and any specific type of cancer (See Chapter 8).

As discussed in Chapter 11 medical monitoring for exposure to DU via inhalation or via ingestion can be achieved by determining the presence of DU in the chest, urine or faecal material. Sampling and analysis methods should be appropriate to the task, and these may be different for assessing total uranium content relative to threshold levels and actual DU present in any medium. Kinetic phosphorescence analysis (KPA) is the standard, low-cost method to determine total uranium, and is preferred if total uranium content relative to a limiting concentration is sufficient. DU analysis, however, requires more sophisticated equipment and techniques that are capable of determining isotope composition and proportions. Accurate measurement of these proportions enables the calculation of the DU portion of total uranium present in each environmental and biological sample.

Results should be high quality, demonstrating adequate quality assurance and quality control and provide suitably low detection limits along with total analytical uncertainty. Appropriate reference background samples (free of DU but with typical levels of natural uranium) should be analysed so that the excess due to DU is determinable for each sample analysed. The selection of appropriate techniques is discussed in more detail in Chapter 11 and Annex 6.

13.5.3 Treatment of human contamination

Once uranium or DU has entered the body there is little that can be done to remove it or increase the excretion rate. The patient can only be treated symptomatically.

Acute exposure When acute intake of DU or uranium has occurred various methodologies have been tried to remove the contaminant from the human body. Sodium bicarbonate has been recommended, and other substances such as Ca-ETDA and Zn-DTPA have been suggested, for enhancing the elimination of uranium from the body (Bhattacharyya et al., 1992). While temporary increases in urinary excretion are likely to occur after their administration, there appears to be no evidence that they cause a marked reduction in the content of the kidney or skeleton.

Animal experiments have shown that some phosphonates are able to reduce the uranium content in the kidneys to about 10% of those in controls when treatment commences within a few minutes of uranium administration; however under realistic conditions, when treatment is delayed for several hours, or longer, these substances are ineffective (Henge-Napoli et al., 2000)

Inhalation Lung lavage is a recognized procedure for the treatment of obstructive lung diseases. However, for reducing the radiation risk from inhaled insoluble compounds of uranium, it is unlikely to have much impact. In these circumstances, lung lavage is not normally advised unless the lung dose is likely to exceed 5 Sv over a few weeks (Henge-Napoli et al., 2000) or the intake is in excess of 100 times the annual limit (Battaacharyya et al., 1992). The information given in Chapter 10 (Table 10.3) for Type S compounds of DU shows that the mass inhaled would need to exceed 23 g, a most improbable scenario. On the other hand, the use of lung lavage may be justifiable if lower intakes of DU are likely to seriously impair lung function.

Long-term follow up If significant incorporation of uranium has been diagnosed, clinical follow up over subsequent years for early detection of any cancer may be recommended. Patients however should be told that the probability that they would develop a cancer due to irradiation through uranium is extremely low, based on current knowledge.

Of greatest value is the psychological therapeutic approach. Patients should be told that the only established consequence of uranium exposure is damage to the kidneys, and that at anticipated levels of exposure this would most likely be transitory and not even clinically detectable. To relate the patient’s problems to uranium contamination, especially the presence of any malignancy, is highly improbable and would be contradictory to current medical knowledge.

14 Health standards, guidelines and recommendations

This chapter summarizes available information on public health standards, guidelines and recommendations relating to exposure to uranium and DU. Analytical methods that may be used to determine the concentration of uranium in environmental materials, prior to applying these standards, are summarized in Annex 6.

From available biological and health effects data, some countries and organisations, such as WHO, have adopted a tolerable intake (TI) approach to derive a guideline value for the chemical toxicity of uranium. Others have proposed minimal risk levels or suggested acceptable intakes based on chemical toxicological and radiological studies. It should be noted that, in all of the cases described below, the health-related exposure standards, guidelines and recommendations have incorporated uncertainty factors into the TI to allow for unknowns.

14.1 Generic

The generic standards, guidelines and recommendations given in Table 14.1 apply to the total intake from all sources and are commonly used together with a knowledge of the relative importance of various exposure routes as a basis for defining specific guidelines for the individual exposure routes.

In constructing many of the generic standards, guidelines and recommendations given below, it was assumed that chemical toxicity effects outweigh potential radiological effects. It should also be noted that exposure to soluble uranium via lung uptake should be included as a component of ingested uranium when applying these standards, guidelines and recommendations.

Table 14.1 Generic standards, guidelines and recommendations for total intake from all sources.

WHO—Chemical*	For oral exposure, a Tolerable Intake (TI) for uranium of 0.0006 mg per kg of body weight per d was established by the WHO (1998a, b) based on the adverse effects observed by Gilman, (1998a). In this report this has been slightly modified to 0.0005 mg per kg of body weight per day. The uncertainty factors used in this calculation were: three for LOAEL to NOAEL, three for extrapolation from animals to humans and ten for human variability. Giving a total uncertainty factor of ninety.
US NRC **	Occupational annual intake limit of 14.8 g natural uranium (CFR 20, 1991)
US—Agency for Toxic Substances and Disease Registry (ATSDR, 1999)	A minimal risk level for intermediate-duration ingestion has been proposed by ATSDR of 0.002 mg per kg of body weight per day based on the study of rats by Gilman et al. (1998a). This minimum risk level is also considered to be protective for chronic exposures. The uncertainty factors used in this calculation were: three for use of a minimum LOAEL, one for extrapolation from animals to humans and ten for human variability. Giving a total uncertainty factor of thirty.
United States Environmental Protection Agency	US EPA have defined a reference dose (RfD) For uranium (soluble salts) of 0.003 mg per kg of body weight per day based on a 'critical effect' of losses in initial body weight and moderate nephrotoxicity. The EPA considers that intake of this amount or less over a lifetime is unlikely to result in the occurrence of chronic non-cancer effects. EPA considers that there is insufficient knowledge to suggest guidelines based on reproductive or developmental effects, while also acknowledging that animal studies have reported foetal toxicity and degenerative changes in the testes from oral exposure to uranium.
Germany	Jacob et al. (1997) proposed a TDI of 0.0007 mg per kg of body weight per day based on effects observed by McDonald-Taylor et al. (1997)

* Based on derivation of water guideline value by WHO (1998a; 1998b) and this work.

** Occupational limit

14.2 Drinking water

For drinking water guidelines WHO (1998a, b) using an earlier study on rats as the basis, in which the NOAEL was 60 µg/kg bw/d, derived a provisional TI value of 0.6 µg/kg.

Standards, guidelines and recommendations for water quality (Table 14.2) are generally derived from generic standards based on total ingestion by apportioning a specific fraction of the total exposure that may be allowed to originate from the ingestion of drinking water. Radiologically based guidelines for the level of uranium in drinking water may be derived in a similar manner or alternatively through the application of separate regulations based on the gross alpha and beta activity in water.

Table 14.2 Standards, guidelines and recommendations for water.

WHO Radiological (Member of the Public, excess dose 0.1 mSv/year)*	Pure natural uranium = 0.16 mg per l DU = 0.28 mg per l
WHO—Chemical	The WHO has derived a provisional** guideline for drinking-water quality of 0.002 mg/l. This value is considered to be protective for subclinical renal effects reported in epidemiological studies (WHO, 1998a).
US EPA—Chemical	Based on the Clean Water Act, US EPA has proposed a drinking water standard for naturally occurring uranium of 0.020 mg/l (US EPA, 1990). This standard is currently under review (56 FR 33050) and may be set at a practicable level of 0.030 mg/l. See also US EPA (1995a).
Canada—Chemical	Canadian limit 0.020 mg/l.
US EPA Groundwater standards for remedial actions at inactive uranium processing sites	Maximum combined limit for ^{234}U and ^{238}U of 1.11 Bq/l (US EPA, 1995b). This is equivalent to 0.044 mg/l assuming secular equilibrium between ^{238}U and ^{234}U.
Australia	National Health and Medical Research Council (NHRMC) guideline value = 0.020 mg/l (1996).
Russia	Maximum allowable concentration in bottled mineral water = 1.7 mg/l (Misund et al., 1999).

* Calculated for an adult using ICRP-72 (1996) and WHO guidelines, which states that excess radiological dose due to radioactivity in drinking water should be limited to 0.1 mSv (WHO, 1993).

** This term is used for constituents for which there is some evidence of a potential hazard, but where the available information on health effects is limited; or where an uncertainty factor of greater than 1000 has been used in the derivation of the tolerable daily intake (TDI). Other guideline values for inorganic water quality parameters listed as being provisional by (WHO, 1998a) include, boron, copper, nickel and chronic exposure to nitrite.

14.3 Food

Health standards for the quality of food may be derived in a similar way to that suggested for drinking water (i.e. derived from generic standards based on total ingestion by apportioning a specific fraction of the total exposure that may be allowed to originate from the ingestion of the given food). Radiological quality may be derived in a similar manner or alternatively through the application of separate regulations based on the gross alpha and beta activity of foods.

Because of the diversity of foods and their importance to human nutrition it is assumed that levels of uranium derived from all food should not exceed the total generic intakes described in 14.1 above.

The WHO has recently derived intervention levels for radionuclides in food to be used following nuclear accidents (WHO, 1998d). The calculated values (Table 14.3) are, of necessity, based on an effective dose of 5 mSv for each nuclide in isolation in a single food category, since it is not possible to generalize regarding which nuclides will be most important in each food category after an accident. Similar generic guidance following emergency situations is given in BSS (1996)

Table 14.3 **G**uideline values for radioactivity in food following a nuclear emergency in Bq/kg (WHO, 1998d). Note uranium isotopes present in DU fall between high and low dose unit intake factors, in the classifications (see Table 5.2). For comparison 1 ppm depleted uranium (1 mg/kg) is approximately equivalent to 14.8 Bq/kg.

Class of radionuclide	**Cereals**	**Roots and tubers**	**Vegetables**	**Fruit**
High dose per unit intake factor (10^{-6} Sv/Bq)	35	50	80	70
Low dose per unit intake factor (10^{-8} Sv/Bq)	3500	5000	8000	7000
Class of Radionuclide	**Meat**	**Milk**	**Fish**	**Drinking Water**
High dose per unit intake factor (10^{-6} Sv/Bq)	100	45	350	7
Low dose per unit intake factor (10^{-8} Sv/Bq)	10 000	4500	35 000	700

14.4 Soil

Soil and dust derived from soil may either be readily ingested or inhaled and as such may be required to meet both a chemically and radiologically defined quality.

In the case of ingestion the presence of soluble compounds of uranium maximize both the radiological and chemical hazards. The opposite is true when dust containing

uranium is inhaled, as soluble uranium is more readily excreted and not stored in the lungs.

There are currently no generally accepted guideline concentration values relating to the quality of soils or dusts in respect of contamination from DU.

14.5 Air

Table 14.4 Standards, guidelines and recommendations for air.

United Kingdom Occupational exposure standards, EHE/40, HSE, (2000)	United Kingdom Occupational exposure standards for soluble natural uranium compounds (HSE, 2000); Long term—0.2 mg/m^3 Short term—0.6 mg/m^3
Agency for Toxic Substances and Disease Registry	ATSDR derived a Minimal Risk Level (MRL*) for chronic inhalation exposure of 0.008 mg/m^3 and 0.0004 mg/m^3 for intermediate duration inhalation of insoluble and soluble uranium respectively (ATSDR, 1999). For chronic inhalation of soluble uranium the MRL is 0.0003 mg/m^3. The uncertainty factors used in this calculation were: three for extrapolation from animals to humans and ten for human variability. Giving a total uncertainty factor of thirty.
Germany	Jacob et al. (1997) proposed a tolerable air concentration of 0.000 07 mg/m^3 of uranium, based on studies of exposure of rats to uranium. Currently in Germany 0.25 mg/m^3 for insoluble uranium compounds (Roth et al., 2000)
American Conference of Governmental Industrial Hygienists **	ACGIH adopted the maximum permissible concentration of 0.2 mg/m^3 for medium term exposure to soluble and insoluble natural uranium in air, and a short-term exposure limit of 0.6 mg/m^3 (ACGIH, 1993)
US Occupational Safety and Health Administration (OSHA)	OSHA recommends a limit of 0.25 mg/m^3 for insoluble uranium and 0.05 mg/m^3 for soluble uranium based on an 8-hour working day.
National Institute for Occupational Safety and Health **	NIOSH recommends a limit of 0.2 mg/m^3 for insoluble uranium (time-weighted average) for chronic occupational exposure, and a short-term exposure limit of 0.6 mg/m^3 in air. For soluble uranium the corresponding levels are 0.5 mg/m^3 and 10 mg/m^3 respectively, (NIOSH, 1994).

* MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health

assessors and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites.

** Note, based on occupational exposure corresponding levels for population exposure after correction for a 24 hour, 7 day per week are 0.069 and 0.21 mg/m^3 for short-term exposure and long-term exposure (based on insoluble compounds as might be encountered immediately following combustion of uranium). This correction from occupational exposure is based on that suggested by the US EPA where:

$$Level_{pub} = Level_{occ}\ (mg/m^3) \times VEho/VEh \times L_{occ}/\ L_{pub}$$

Where: $Level_{pub}$ = public exposure level; $Level_{occ}$ = occupational exposure level; VEho = human occupational default respiration rate (9.6 m^3 per day); VEh = human ambient default respiration rate (20 m^3 per day); L_{occ} = length of a standard working week (5 days) and L_{pub} = length of standard week (7-days)

Chemical health risks are considered to be negligible when exposure levels in air are below these exposure limits (Table 14.4). It is difficult to assess the potential health risk for humans at higher levels of exposure because information describing dose response relationships for potential deleterious effects in humans is lacking. However, animal studies point to concentrations showing no or minimal effects (particularly kidney damage) following short to chronic inhalation exposure in the range of 0.15 mg/m^3. Assuming that humans are equally sensitive, this indicates that levels above 0.15 mg/m^3 would be required to induce clinically significant toxic effects in humans.

15 Summary, Conclusions and Research Needs

15.1 Summary

Objectives

The principal objective of this monograph is to assess any health impacts of exposure to DU using existing knowledge about uranium and DU, and to provide a framework for identifying the likely consequences of public and occupational exposure to DU.

To achieve this objective, information is given on situations where exposures might arise, the likely routes of intake, the potential risks from radiological and chemical toxicity, and future research needs. Background data and scenarios are also provided from which numerical estimates may be constructed from knowledge of local exposure.

Uranium and depleted uranium

- Uranium is a naturally occurring heavy metal found in various chemical forms in all soils and rocks, seas and oceans, and in drinking water and food.
- Natural uranium consists of a mixture of three radioactive isotopes ^{238}U (99.27% by mass), ^{235}U (0.72%) and ^{234}U (0.0054%). All these isotopes were present when the Earth's crust was formed.
- Uranium is used primarily in nuclear power plants. However, most reactors require uranium in which the ^{235}U content is enriched from 0.72% to about 3%.
- The uranium remaining after removal of the enriched fraction contains about 0.25% of ^{235}U, 99.8% ^{238}U and 0.001% ^{234}U by mass; this is referred to as depleted uranium or DU.
- The behaviour of uranium and DU in the body is identical. However for the same mass, the radiation dose from DU is about 60% of that from uranium (without daughters).
- Spent uranium fuel and other forms of uranium from nuclear reactors are reprocessed by placing them in the same enrichment plants as is used for natural uranium enrichment. This can cause some reactor-created radioisotopes to contaminate the reprocessing equipment. These reactor-created radioisotopes can then contaminate the DU from natural uranium enrichment. Under these conditions another uranium isotope, ^{236}U, may be present together with very small amounts of the transuranic elements plutonium, americium and neptunium, and the fission product technetium-99. However, the increase in radiation dose following uptake into the human body of these additional radioisotopes in DU will be less than 1%.
- As a radioactive element, freshly produced DU emits mainly alpha (α) radiation. DU produces progeny or daughter radio-isotopes that emit beta (β) and only a small amount of gamma (γ) radiations. Because α and β radiations are not very penetrating, external radiation exposure resulting from DU is mainly limited to the skin. However, α radiation is most important for internal exposures.
- As a consequence of using DU munitions, the predominant chemical forms of uranium present in the atmosphere are likely to be uranium trioxide (UO_3), uranium octoxide (U_3O_8) and uranium dioxide (UO_2), although the presence of aerosols containing mixtures of metals with uranium has been established. In the general environment, uranium trioxide (UO_3), and soluble forms such as carbonates, are most likely to predominate in due course.

Applications of depleted uranium

- The main civilian uses of DU include counter weights in aircraft, radiation shields in medical radiation therapy machines and containers for the transport of radioactive materials.
- Due to its high density, which is about twice that of lead, and other properties, DU is used in munitions designed to penetrate armour plate and in other forms of armour plate for protection of military vehicles such as tanks.

DU exposure and exposure pathways

Exposure to DU

On average, approximately 90 µg of uranium exists in the human body from normal intakes of food, water; and air; approximately 66% is found in the skeleton, 16% in the liver, 8% in the kidneys and 10% in other tissues. The average annual intake of uranium by adults has been estimated to be 460 µg from ingestion and 0.59 µg from inhalation.

Smoking two packets of cigarettes per day may allow inhalation of up to 50 ng of uranium per day. Coal fired power stations have been reported to produce 3ng/m^3 of uranium downwind from their discharges.

Until recently, the public was not exposed to DU. With the use of DU counterweights in aircraft, there is a possibility that people near an aircraft crash may be exposed to DU dusts if the counterweights were to combust on impact. Significant exposure to people in this situation is unlikely. Exposure of clean-up and emergency workers following aircraft accidents is possible but normal occupational protection measures should prevent any significant exposure occurring.

Since 1991, when DU weapons were first used in conflict, exposure may occur to people working or living in areas where DU munitions were used and where they hit targets and formed various uranium compounds, predominantly oxides A recent UNEP report giving field measurements taken around selected impact sites in Kosovo found that contamination by DU in the environment was localised to a few tens of metres around impact sites. Contamination by DU dusts to local vegetation and water supplies was found to be extremely low. Thus the possibility of significant exposure to the local populations was found, at least where measurements were made, to be very low.

DU exposure pathways

Individuals can be exposed to DU in the same way they are exposed to natural uranium i.e. by inhalation, ingestion, dermal contact or injury (e.g. embedded fragments). The relative contribution from each of these pathways to the total DU uptake into the body depends on the physical and chemical nature of the DU as well as the level and duration of exposure. Each of these exposure situations needs to be assessed to determine any potential health consequence.

Most (>95%) uranium entering the body entering the body via inhalation or ingestion is not absorbed, but is eliminated via the faeces. Of the uranium that is absorbed into the blood, approximately 67% will be filtered by the kidney and excreted in the urine within 24 hours and about 90% in a few days. Typical gastrointestinal (GI) tract absorption rates for uranium in food and water are about 2% for soluble uranium compounds and down to 0.2% for insoluble uranium compounds.

- **Intake by ingestion** is important for populations having their drinking water or food contaminated by DU. In addition, the ingestion of soil by children via geophagia or hand-to-mouth activities is also potentially important.
- **Intake by inhalation** can be important following the use of DU munitions during or immediately following conflict or when DU deposits in the environment are re-suspended in the atmosphere by wind or other forms of disturbance. Accidental inhalation may also occur as a consequence of a fire in a DU store, an aircraft crash, or the decontamination of vehicles from within or close to conflict areas.
- **Intake** through intact skin is very low and considered to be relatively unimportant.
- **Intake from wound contamination** or embedded fragments in skin tissues allows DU to enter the systemic circulation.

Behaviour of DU in the body

In this monograph, consideration is given to occupational and public exposure, short- and long-term intakes and uptake by inhalation and ingestion of compounds with widely different solubility characteristics. The behaviour of uranium in the body has been described using both reference and material specific absorption parameters for the uranium compounds likely to be present in DU aerosols; calculations have also been performed for a reference DU aerosol. Emphasis has been placed on lung retention, and urinary and faecal excretion rates, since assessments of intake are usually based on such measurements.

The amounts and concentrations of uranium in the kidneys after exposure have also been derived from information on the known nephrotoxicity of uranium. In certain circumstances effective judgements on the likely nephrotoxicity of uranium can be deduced from kidney content extrapolated from the urinary excretion rate; the kidney to urine ratio is reasonably independent of the chemical form and exposure pattern from a few days to several months.

ICRP Models

The models recommended by the International Commission on Radiological Protection (ICRP) are embodied in the internationally recommended Basic Safety Standards for radiation protection and in the legislation of many countries, including those in the European Union. In the context of this report, the appropriate generic models are those for the human respiratory tract (not specific for uranium), the systemic behaviour of uranium and uptake from the GI tract.

- These ICRP models can be combined to calculate the radiation dose from intakes of uranium and DU, and for a given intake to predict the retention of DU in the important organs of the body e.g. lungs, bone, kidneys, and urinary and faecal excretion rates.
- The models consider materials assigned to one of three types that describe absorption from the lungs to the blood, or uptake from the GI tract to blood. These materials are referred to as Type F (fast absorption), Type M (moderate absorption) and Type S (slow or poor absorption). In broad terms UO_2 is considered a Type S compound, UO_3 a Type M compound and uranyl carbonate a Type F compound. The absorption characteristics of U_3O_8 can vary between those for Type M and S compounds.
- The human respiratory tract model has reference values assigned for deposition and particle transport to the GI tract. Reference values for uptake of uranium from the GI tract are given for Type F, M, or S compounds.

- The ICRP has recommended that material specific values for aerosol parameters such as size and density, absorption parameter values from the lungs to blood, together with appropriate exercise levels and breathing patterns should be used whenever possible. Most of these factors are considered in the monograph.

Health effects

DU has both chemical and radiological toxicity with the two important target organs being the kidneys and the lungs.

Kidney

Retention of uranium in the kidney has been attributed to the creation of complexes with proteins and phospholipids in the proximal tubules; considered to be the main site of kidney damage. Animal studies have shown that long-term exposure to uranium causes nephrotoxic effects that ranged from minimal microscopic lesion in the tubular epithelium (low concentrations) to tubular necrosis (high concentrations).

Long-term studies of workers chronically exposed to uranium have reported impairment of the kidneys (proximal tubular epithelium) that depended on the level of exposure. Studies of members of the public chronically exposed to uranium in drinking water have also shown similar signs of impairment of kidney function. There is some evidence that kidney function returns to normal once the source of excessive uranium exposure has been removed.

Lung

Pulmonary toxicity of uranium varies depending on the animal species studied and the chemical form of the uranium. Some early studies on animals reported pulmonary oedema and haemorrhage following exposure to some uranium compounds (e.g. uranium peroxide, uranium trioxide) but not others (uranium dioxide). However, more recent long-term studies using a range of animals inhaling various uranium compounds, both soluble and insoluble, did not reveal any histological damage to the lungs.

In a number of studies on uranium miners, an increased risk of lung cancer has been demonstrated but this has been attributed to exposure from radon decay products. There is a possibility of lung tissue damage leading to a risk of lung cancer if a high enough radiation dose results from insoluble DU compounds remaining in the lungs over a prolonged period (many years).

Skin

Erythema or other effects on the skin should not occur even if DU is held against the skin for prolonged periods (weeks). There are no established data to suggest that skin cancer occurs from skin contact with uranium dusts.

Liver and skeleton

Autopsies of individuals chronically exposed to uranium have found that the average ratios of the amount of uranium in the skeleton, liver and kidney was 63:2.8:1. The uranium content in the skeleton may reflect its affinity for phosphate which is abundant in the bone. No consistent or confirmed adverse effects have been reported for the skeleton or liver. However, few studies have been conducted.

Reproductive and developmental effects

Reproductive and developmental effects have been reported in rodent studies ingesting or being exposed via dermal contact to extremely high levels of soluble uranium compounds. No such effects have been reported in humans; however very few studies are available. Further studies are needed to clarify if these effects occur in other animals and whether they are likely to occur in humans.

Central nervous system

Although uranium released from embedded fragments may accumulate in CNS tissue, and some animal and human studies are suggestive of effects on CNS function, it is difficult to draw firm conclusions from the results available. Better designed and focussed studies are needed to clarify if any effects on CNS occur from exposure to uranium.

International limits for radiological exposure

This monograph uses the radiation dose[1] limits published in the International Basic Safety Standards (BSS) for Protection against Ionizing Radiation and for the Safety of Radiation Sources. The following doses are in addition to those from normal background exposures. They should not be exceeded:

Public exposure

- an effective dose of 1 mSv in a year
- in special circumstances, an effective dose of up to 5 mSv in a single year provided that the average dose over five consecutive years does not exceed 1 mSv per year
- an equivalent dose to the skin of 50 mSv in a year

Occupational exposure

- an effective dose of 20 mSv per year averaged over five consecutive years
- an effective dose of 50 mSv in any single year
- an equivalent dose to the extremities (hands and feet) or the skin of 500 mSv in a year

Guidance on exposure based on chemical and radiological toxicity

Chemical toxicity of a given material is related to its detrimental interaction with biochemical processes in the human body. WHO has guidelines for determining the values of health-based exposure limits or tolerable intakes for chemical substances. Tolerable intake (TI) is an estimate of the intake of a substance that can occur over a lifetime without appreciable health risk. The TI is usually expressed as mg per kg of body weight per day.

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[1] The amount of energy deposited per unit mass is called the *absorbed dose* and is measured in gray where 1 Gy is equivalent to one joule per kilogram. Since alpha particles can deposit more energy in tissue than gamma or beta radiation, they have a greater ability to do more damage. Thus, a weighting factor is given to all radiations (20 for alpha and 1 for beta and gamma radiations) and is multiplied by the *absorbed dose* to give the *equivalent dose*. The unit of *equivalent dose* is the sievert (Sv). Another quantity used in radiation protection is the *effective dose*. It is calculated by multiplying the *equivalent dose* by tissue weighting factors that relate to the relative risk of cancer associated with each organ or tissue. Thus the *effective dose*, also in Sv, gives a general indication of the level of risk implied by the dose; a measure of health detriment. The *collective dose* is an expression for the total radiation dose incurred by a population and is expressed in man-sieverts (man.Sv).

The effect observed at the lowest exposure level is used to determine the TI value. This is usually due to chemical toxicity when intake is by ingestion or inhalation of Type F or M uranium compounds and due to radiological toxicity for Type S uranium compounds after exposure by inhalation. These are discussed below for both public and occupational exposure. The TIs derived are applicable to long-term exposure. In single and short-term exposures, higher exposure levels can be tolerated without adverse effects. However, quantitative information is not available to assess how much the TI values may be temporarily exceeded without risk. Limits derived for uranium compounds are equally applicable to DU.

Public exposure by ingestion

- Based on medium-term and long term toxicity studies in experimental animals, a TI of 0.5 µg per kg of body weight per day (i.e. about 11 mg/y for an average adult) was derived for Type F uranium compounds.
- The same TI should be used for Type M uranium compounds as the data available do not consistently demonstrate lower nephrotoxicity for these compounds.
- The value of the TI would, on the basis of the ICRP biokinetic models, result in an effective dose of about 10 µSv.
- Uranium compounds with low absorption (Type S) are markedly less nephrotoxic, and a tolerable intake of 5 µg per kg of body weight per day is applicable.
- When the solubility characteristics of the uranium species are not known, which is often the case in exposure to DU, it would be prudent to apply the more stringent tolerable intakes, i.e., 0.5 µg per kg of body weight per day for oral exposure.

Public exposure by inhalation

- The data on the nephrotoxicity of Type F and M uranium compounds are consistent with a TI for oral exposure of 0.5µg per kg of body weight per day; this translates to an airborne concentration of uranium of about 1.5 µg/m^3, or approximately 1-µg/m^3 (in the respirable fraction).
- For Type S compounds, exposure corresponding to a daily intake of 5 µg per kg of body weight (as derived for the exposure by ingestion above) would lead to a total radiation dose of about 13 mSv per year. As the accepted upper limit for radiation exposure to the general public is 1 mSv/year, it would be appropriate to reduce the TI to 0.5 µg per kg of body weight per day, equivalent to a tolerable concentration in ambient air of 1 µg/m^3 (respirable fraction).

Occupational exposure by inhalation

- The ICRP model indicates that an effective radiation dose limit for workers of 20-mSv per year, averaged over five consecutive years, leads to an 8-hour time-weighted average (TWA) inhalation limit of 0.05 mg/m^3 for Type S uranium compounds.
- As consideration of chemical toxicity led to an inhalation exposure limit for the more soluble uranium compounds that is identical to that derived for Type S compounds based on radiological toxicity for the general population (see above), it seems appropriate to apply an 8-hour TWA inhalation exposure limit of 0.05 mg/m^3 for exposures from Type F and M uranium compounds.

Monitoring and treatment of exposed individuals

For the general population, neither civilian or military use of DU is likely to produce exposures to DU much above normal background levels produced by uranium. Thus an exposure assessment for DU will not normally be required.

When an individual is suspected of being exposed to DU at a level significantly above the normal background, an assessment may be required. In principle this assessment may be undertaken by extrapolation from the amounts of DU excreted in urine and faeces and by external radiation monitoring of the chest.

- **Urine measurement:** Provided that the dietary concentrations of uranium are low, assessment of DU exposure is best achieved by analysis of daily urine excretion. The amount of DU in the urine is determined from the ^{235}U:^{238}U ratio, obtained using sensitive mass spectrometric techniques. Assessment of DU intake is then determined by back extrapolation using appropriate graphs and information in this monograph. In such circumstances it should be possible to assess doses from DU intake at the mg per day or mSv level.
- **Faecal measurement** can give useful information on intake if samples are collected soon after exposure (a few days).
- **External radiation measurements** over the chest, using a whole-body radiation monitor for determining the amount of DU in the lungs, has limited application since it requires specialist facilities and can only assess relatively large amounts of DU in the lungs.

Treatment of overexposure

There are no specific means to decrease the absorption of uranium from the gastrointestinal tract or lungs, or increase its excretion. Thus general methods appropriate to heavy metal poisoning could be applied. Similarly there is no specific treatment for uranium poisoning and the patient should be treated based on the symptoms observed. Dialysis may be helpful in extreme cases of kidney damage.

15.2 Conclusions

This review concludes that:

- Limitation on public intake of soluble DU compounds (Type F and M) should be based on a TI value of 0.5 µg per kg of body weight per day and for insoluble (Type-S) DU compounds on 5 µg per kg of body weight per day.
- The TI value of 0.5 µg per kg of body weight per day leads to a limitation on public inhalation of soluble DU compounds to 1 µg/m^3 DU in air; the same guideline air concentration for insoluble DU compounds comes from the radiation limit dose of 1-mSv/year.
- The 8-hour time-weighted average (TWA) limitation on worker inhalation of soluble and insoluble DU compounds is 50 µg/m^3 DU in air.
- Under most circumstances, use of DU will make a negligible contribution to the overall natural background levels of uranium in the environment. However, levels of DU may be significantly raised over background levels in close proximity to DU contaminating events. Over the days and years following such an event the contamination will become dispersed into the wider natural environment.
- The greatest potential for DU exposure will follow conflict where DU munitions are used and people living or working in these areas inhale dusts and consume contaminated food and drinking water. Measurements of DU in conflict areas indicate only localised (within a few tens of metres from impact sites) contamination at the ground surface. However, levels of contamination in food and drinking water could rise after some years and should be monitored where it is considered that there

is a reasonable possibility of significant quantities of DU entering the ground water or food chain.

- Where possible, clean up operations in impact zones should be undertaken where there are substantial numbers of radioactive projectiles remaining and DU contamination levels are deemed unacceptable by qualified experts. If very high concentrations of DU dust or metal fragments are present, then areas may need to be cordoned off until removal can be accomplished.
- Guidance on the necessity for clean up of radioactive materials has been provided by the ICRP (1999b). Similar methodologies employed during the cleanup of land contaminated with heavy metals resulting from industrial activity are also appropriate, particularly as radiation dose levels of DU in conflict areas would not normally exceed those recommended for clean up by the ICRP.
- Young children could receive greater exposure to DU when playing in or near DU impact sites. Typical hand-to-mouth activity could lead to high DU ingestion from contaminated soil. Necessary preventative measures should be undertaken.
- General screening or monitoring for possible DU related health effects in populations living in conflict areas where DU was used is not recommended. Rather individuals who believe they have had excessive intakes of DU should consult their medical practitioner for an examination and treatment of any symptoms.
- Since DU is a radioactive metal, restrictions are needed on the disposal of DU. There is the possibility that DU scrap metal could be added to other scrap metals for use in refabricated products. DU is a pyrophoric metal that can produce oxides that can be inhaled when heated (welded). Disposal of DU should normally come under appropriate national or international (IAEA) recommendations for use of radioactive materials.

15.3 Research needs

Priorities for research that would significantly enhance knowledge and lead to better assessments of health risks from exposure to DU are given below.

- Studies are needed to clarify our understanding of the extent, reversibility and possible existence of thresholds for kidney damage in people exposed to DU. Important information could come from studies of populations exposed to naturally elevated concentrations of uranium in drinking water.
- WHO, through its International Agency for Research on Cancer (IARC), continues to study the effects of low-level exposure to ionizing radiation in order to improve the scientific base for health risk assessment and radiation protection. The utility and feasibility of studies to assess whether there has been an increased rate of cancer amongst military personnel who served in the Gulf or Balkans conflicts, and to evaluate the possible role of DU if an increase is found, should be investigated.
- Studies are needed that will allow better exposure assessments of children. This is particularly important given their unique exposure scenarios such as geophagia and hand-to-mouth activities.
- Studies are required to validate transfer coefficients for DU compounds entering the human food chain. For example, this is important given the amount of soil ingested by many livestock during browsing.
- There is a lack of information about the possible biological action of uranium or DU in the following areas:
  - Neurotoxicity: Other heavy metals, e.g. lead and mercury are known neurotoxins, but only a few inconsistent studies have been conducted on uranium. Focused studies are needed to determine if DU is neurotoxic.

- Reproductive and developmental effects have been reported in single animal studies but no studies have been conducted to determine if they can be confirmed or that they occur in humans.
- Haematological effects: Studies are needed to determine if uptake of DU into the bone has consequences for the bone marrow or blood forming cells.
- Genotoxicity: Some *in vitro* studies suggest genotoxic effects occur via the binding of uranium compounds to DNA. This and other mechanisms causing possible genotoxicity should be further investigated.

- Investigations are needed on the chemical and physical form, physiological behaviour, leaching and subsequent environmental cycling of specific forms of uranium from various industrial and military sources (e.g. depleted uranium alloys, phosphate by-products). Particular attention should be paid to where the bulk of DU finally goes.

Annex 1 Process of uranium enrichment

After mineral extraction, processed uranium ore (or more exceptionally uranium generated from the recycling of nuclear fuel or reactor materials) undergoes a variety of chemical processes leading to enhancement with respect to both the total concentration and chemical purity of the produced uranium compounds. Following this chemical treatment, the purified uranium may be isotopically enriched, if required, by a number of processes including gaseous diffusion, centrifugation or laser excitation (e.g. Weigel, 1983). Such enrichment is now almost exclusively for the nuclear power industry. Depleted uranium is produced in various forms as a by-product of the isotopic enrichment of uranium (Figure A1.1). In the most common isotopic enrichment process (gaseous diffusion) uranium is required in the form of uranium hexafluoride (UF_6) which may be produced via the following chemical reactions:

$$U_3 0_8 + 2H_2 = 3UO_2 + 2H_2O;\ UO_2 + 4HF = UF_4 + 2H_2O;\ UF_4 + F_2 = UF_6$$

At atmospheric pressure, UF_6 exists as a gas above 57°C, and as a solid below this temperature. It sublimes, as does carbon dioxide without passing through a liquid state.

Chemically, UF_6 is highly reactive with water forming water soluble hydrofluoric acid (HF) and uranyl fluoride (UO_2F_2), the former of which is considered to be highly toxic. For this reason DU, produced as a by-product of the enrichment processes in the form of UF_6, is often re-converted to less hazardous forms. These include uranium oxides (UO_2 or U_3O_8) which are more chemically stable and suitable for long-term storage or disposal, or to U metal (AEPI, 1995).

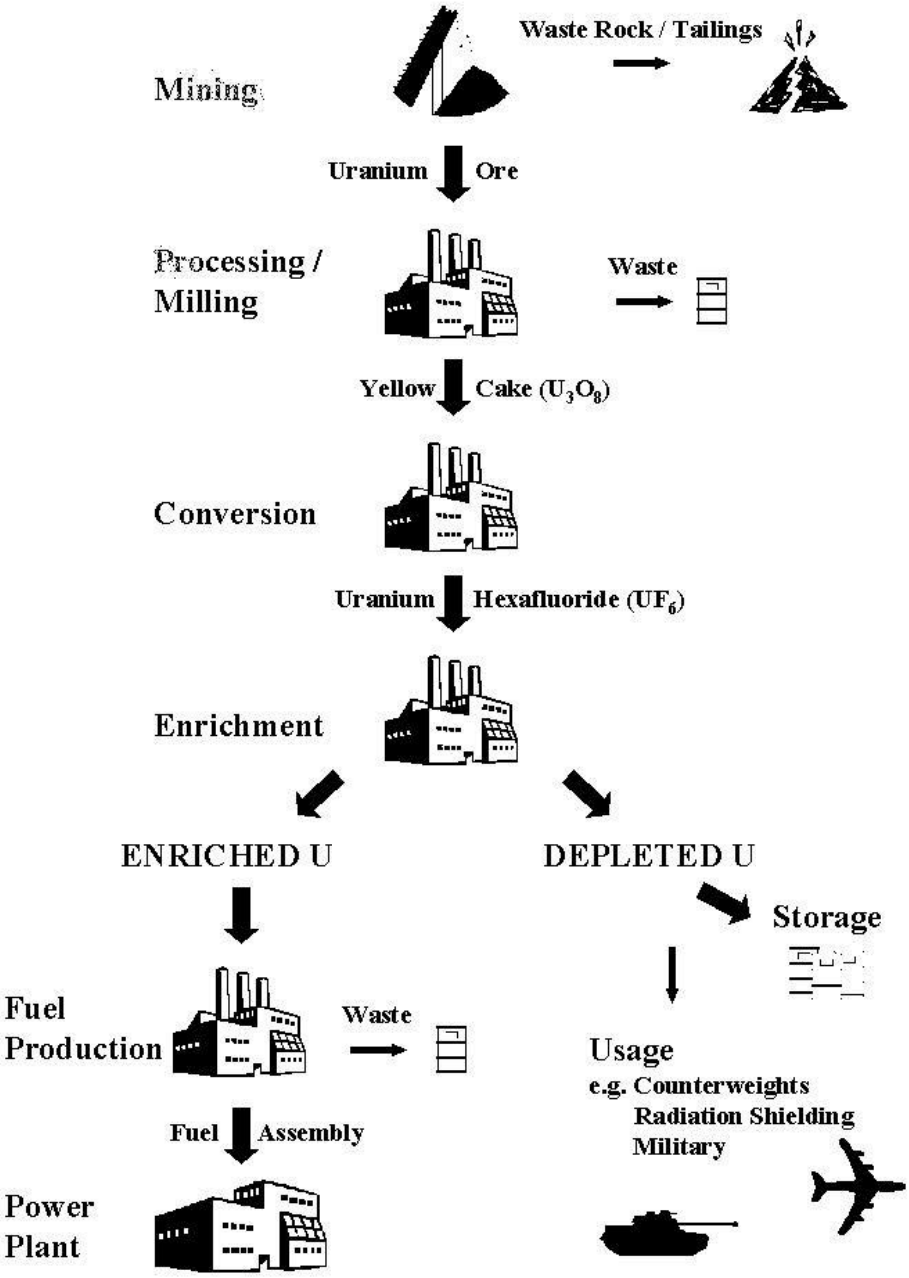

Figure A1.1 Schematic diagram showing various manufacturing routes leading to the production and use of enriched and DU. Note (a) DU is often stored as UF_6 and typically converted to metallic DU prior to the production of fuel, weapons, counterweights etc. (b) yellowcake is a generic term and is often used to describe other uranium compounds such as diuranate or mixtures of diuranate and oxides, (c) reprocessed uranium may enter the process at the enrichment stage.

Annex 2 Radiological dose due to other nuclides

The OSAGWI report has stated that the trace levels of transuranics and fission products in DU contribute less than 1% of the total dose. A simple assessment has been carried out based on measurements of the specific activity of ^{99}Tc, ^{237}Np, ^{238}Pu, $^{239/240}$Pu and ^{241}Am given by TACOM (Bhat, 2000). TACOM analysed 60 samples of DU from three different sources, in this assessment the highest measured value is used for each radionuclide, even though these values did not usually occur in the same sample. Committed effective doses were calculated on the basis of the default dose coefficients given in ICRP-68 (1994b), assuming Type M solubility and an aerosol of 5m AMAD. It is found that the dose from transuranics and fission products is about 0.1% of the dose from DU itself, thus confirming the OSAGWI assertion. Although ^{99}Tc has by far the highest specific activity (as high as 500 pCi/g DU compared to less than 3 pCi/g DU for ^{239}Pu), its lower dose coefficient means that it is not as important as the transuranics. Each of the transuranic radionuclides given above contributes roughly equal parts to the total dose from transuranics.

Table A2.1 Radioactive dose from transuranics and fission product contaminants in DU (for inhalation by worker of 5 µm; Type M particles)

Nuclide	pCi/g*	Bq/mg	e(50)**	E/mg DU
^{241}Am	19	7.03×10^{-4}	2.70×10^{-5}	1.90×10^{-8}
^{237}Np	3.7	1.37×10^{-4}	1.50×10^{-5}	2.05×10^{-9}
^{238}Pu	2	7.40×10^{-5}	3.00×10^{-5}	2.22×10^{-9}
$^{239/240}$Pu	2.7	9.99×10^{-5}	3.20×10^{-5}	3.20×10^{-9}
^{99}Tc	540	2.00×10^{-2}	3.20×10^{-9}	6.39×10^{-11}
Total				2.62×10^{-8}
% of DU dose				0.11%
DU		14.8	1.68×10^{-6}	2.49×10^{-5}

* highest of highest values of the 3 billets (R K Bhat, Encl. 1; Bhat, 2000)

** ICRP-68, Inhalation, Type M, 5µm AMAD

Note: % of total DU dose for (a) worker 1µm particle Type M = 0.13% and (b)member of the public1µm particle, Type M = 0.09%

Annex 3 Uranium in the environment, food and reference data.

A3.1 Uranium production

Table A3.1 Uranium production in 1998 (British Geological Survey, 2000)

Country	Tonnes
Argentina	35
Australia	4 901
Canada	11 041
China	500
Czech Republic	611
France	508
Gabon	731
India	200
Kazakhstan	1 250
Namibia	2 778
Niger	3 713
Pakistan	23
Portugal	19
Romania	100
Russia	2 000
South Africa	965
Spain	255
Ukraine	500
USA	1 872
Uzbekistan	1 930
World Total	33 900

A3.2 Uranium levels in the environment

Table A3.2 A selection of typical data on uranium concentrations determined in groundwaters, surface waters and drinking waters post-1980.

Country	Comments
Argentina	Bomben et al. (1996) determined the uranium concentrations in bottled mineral waters from Argentina to lie in the rage 0.04 to 11µg/l with a mean of 1.3µg/l.
Brazil	DeCamargo and Mazzilli (1996) determined the concentrations of uranium in mineral waters from a high background region in Brazil to be 0.08 to 2.0 µg/l.
USA	Fisenne et al., (1987) determined the mean concentration of uranium in drinking water from New York City, USA, to range from 0.03 to 0.08 µg/l. A mean uranium concentration of 2.55 µg/l was reported in drinking water from 978 sites in the USA surveyed during the 1980s (US EPA, 1990; 1991). In Salt Lake City, USA the mean intake of uranium per day from drinking water has been estimated to be 1.5 µg (Singh, 1990).
Canada	In a survey of 130 sites in Ontario Canada conducted between 1990 and 1995, the mean of the average uranium concentrations in treated drinking water was 0.40 µg/l with a range of 0.05 to 4.2 µg/l (OMEE, 1996). In other areas of Canada private water supplies have been found with concentrations of up to 700 µg/l (Moss et al., 1983 and Moss, 1985). On the basis of the results from the OMEE survey of Ontario in 1996 the average daily intake of uranium from drinking water in Canada was estimated to be 0.8 µg (i.e. that based on the consumption of two litres of drinking water per day).
Himalayas	Virk, (1997) determined uranium concentrations in waters (springs, groundwaters and surface streams) from the western Himalayas to range from 0.89 to 63.4 µg/l.

Country	Comments
India	Singh et al (1995) Measured uranium in drinking waters from Punjab (Bathinda and Amritsar). Observed concentrations ranged from 11 to 113 µg/l. Singh et al, (1996) Measured uranium concentrations in 24 water samples collected from various important sources of drinking water in Upper Pradesh. Concentrations were found to lie between 0.87 and 11 µg/l.
Finland	The daily intake of uranium from waters in Finland, that are often sourced from fractured aquifers in granitic rocks or overlying sediments, has been estimated to be 2.1 µg (Kahlos and Asikainen, 1980). Salonen (1988) measured natural radionuclides in groundwaters in Finland and recorded an arithmetic mean concentration of uranium of 166 µg/l, a geometric mean of 26 µg/l and a maximum of 2900 µg/l amongst 58 water samples derived from wells drilled into bedrock.
Norway	A survey of groundwater derived from 28 private water supplies in Norway yielded uranium concentrations in the range <0.02 to 170 µg/l (Banks et al., 1995) An investigation of 145 hard-rock groundwater samples collected from private drinking water wells in the area of Oslo and Bergen yielded a median uranium concentration of 3.514 µg/l. The median values for the Oslo and Bergen areas were 3.378 µg/l and 3.720 µg/l respectively (Reimann et al., 1996).
Japan	In five Japanese cities, a mean uranium concentration of 0.009 µg/l was determined in potable drinking water supplies (Nozaki, 1970).
Jordan	Studies by Gedeon et al. (1994) and Smith et al. (2000) of 168 drinking water supply boreholes, principally in karstic limestone aquifers, gave a mean uranium concentration in drinking water supply wells of 2.4 µg/l with a wide range of <0.04 to 1400 µg/l. Average intakes were considered to be inappropriate because of the supply system in which supply to one area was often fed directly from a limited number of wells.
Cyprus	Studies undertaken in Cyprus in a variety of underground aquifers hosted in limestone, sandstone and alluvial deposits indicated an average uranium concentration in 215 drinking water and agricultural supply wells of 0.86 µg/l with a range of <0.005 to 38 µg/l Smith et al. (2000).
Kuwait	Studies by Bou-Rabee (1995) measured uranium concentrations in six drinking waters following the Gulf War (1991). Concentrations ranged from 0.02 to 2.48 µg/l and all samples had a $^{235}U/^{238}U$ ratio of 0.007 (typical for natural uranium = 0.007 25).
UK	Surveys of 35 groundwaters and spring waters in the United Kingdom (Edmunds et al., 1989) showed uranium concentrations in the range <0.1 to 10 µg/l across a variety of aquifers including those developed in sandstones, millstone grits, chalk, limestone and greensand. A high degree of variation was observed in most aquifers. Analysis of spring-waters from Derbyshire and closely associated millstone grits have shown ranges of <2 to 13 µg/l (Banks et al., 1997). The mean uranium concentration in over 100 000 surface waters throughout the UK has been determined to be 0.65 µg/l with a maximum observed concentration of 233 µg/l.
Pakistan	In the analysis of 16 groundwaters from Pakistan, an average uranium concentration of 1.6 µg/l was measured with a range of <0.05 to 5 µg/l (British Geological Survey, unpublished data)
Sea Water	Uranium is present in sea water at concentrations of about 3.3 µg/l (Kaye and Laby, 1993). In estuaries concentrations are generally positively correlated with salinity and in open Oceans range from 3.0 to 3.6 µg/l (Ivanovich and Harmon, 1982). Experimentation has shown it may be removed by a number of techniques such as ion exchange, ultra-filtration and reverse osmosis (Pramauro et al., 1996; Raff and Wilken, 1999).
Spain	Herranz et al. (1997, 1999) determined the mean uranium content of drinking water at four treatment plants serving municipalities in northern Spain to be 0.11 µg/l. The same authors observed a 60% drop in the uranium content of sewage effluent during the treatment process, indicating an increased uranium content in the sewage sludge.
Syria	Othman and Yassine (1996) measured uranium in drinking waters from 48 sites in Southern and Middle Syria. Highest observed values (approximately 14 µg/l) were observed in the vicinity of phosphate mines.
Uganda	Analysis of water samples collected from 139 drinking water supplies, mainly sited in highly weathered terrains overlying undifferentiated granitic basement rocks during studies of water quality in Uganda (Smith et al., 1996; Smith et al., 1998) indicate a range of uranium concentrations <0.05 to 17 µg/l with a mean of 0.59 µg/l (British Geological Survey, unpublished data).
Europe (Bottled Waters)	In a survey of 56 randomly selected bottled mineral waters Misund et al. (1999) observed uranium concentrations to range from 0.0104 to 9.45 µg/l.

Table A3.3 Concentrations of uranium observed in soils during recent studies.

Country	Comments
Czech Republic	Ledvina et al. (1996) undertook an analysis of uranium within soils in the former state farm of Trebon in Southern Bohemia which was felt to be potentially contaminated with uranium from a uranium processing plant and from naturally mineralized soils. Concentrations in the range of 14 to 50 mg/kg were observed in the most contaminated soils with an apparent baseline concentration of 2 to 4 mg/kg.
Brazil	Perez et al. (1998) determined the total uranium concentration in the A and B horizons of 15 Brazilian soils and found positive correlation between uranium concentration and the proportion of clay and iron oxides. A range of 0.001 to 2.115 mg/kg and 0.003 to 2.298 mg/kg were observed for A and B horizon soils respectively.
India	Singh et al. (1998) determined the uranium concentration in a range of contaminated soils (due to releases from fertilizer and thermal power plants) and soils from background areas of Uttar Pradesh, Rajastan and Kerala (India). Their results indicated levels of between 0.24 and 9.20 mg/kg with the most elevated levels being recorded in the proximity of a coal-fired power plant.
Central Barents Region	Reimann et al. (1998) collected a suite of geochemical samples from 617 catchment areas 132 in Norway, 191 in Finland and 294 in Russia. Topsoil concentrations (0-5cm) were in the range of <0.5 to 30 mg/kg with a median of less than 0.5 mg/kg.
Lithuania	A geochemical survey of Lithuania by Kadunas et al. (1999), analysed 2700 topsoil samples which indicated levels across the country of between 0.7 and 5.0 mg/kg with a median of 1.7 mg/kg.
Kuwait	Bou-Rabee (1995) measured uranium in 12 soil samples collected following the gulf war. Uranium concentrations ranged from 0.30 to 1.85 mg/kg and had $^{235}U/^{238}U$ isotopic ratios in the range 0.006 to 0.007. Samples of dry deposition and oil fly-ash were also measured. In these samples concentrations of uranium ranged between 0.19 and 0.25 mg/kg in the fly-ash samples and 0.95 to 1.79 mg/kg in the dry deposition samples $^{235}U/^{238}U$ ratios ranged between 0.006 and 0.007 (typical natural uranium = 0.007 25).
Slovenia	Brajnik et al., (1988) measured natural radioactivity at a number of locations in Slovenia. Sites studied included soils associated with the underground uranium mine at Zirovski vrh, natural radioactivity associated with the coal-fired power stations at Ljubljana, Trbovlje and Sostanj and natural radioactivity (U, Ra and Rn) associated with phospho-gypsum wastes. The latter source was found to dominate with respect to the contamination of sediment and water of the river Sava even in comparison with the impact of the uranium mine.
United Kingdom	Uranium concentrations in rural topsoils and profile soils have been determined as part of the British Geological Surveys G-BASE programme. Concentrations of uranium ranged from 0.05 to 76 mg/kg in profile soils over central and eastern England. Urban soils sampled in 5 major UK cities ranged from 0.25 to 5.50 mg/kg in topsoils and 0.25 to 9.2 mg/kg in profile soils (e.g. British Geological Survey, 1997)

A3.3 Uranium levels in food

Table A3.4 Average concentrations of uranium in various foodstuffs from New York City, adapted from ([1]Fisenne et al., 1987; [2]NCRP, 1984; [3]US EPA, 1985).

Food Stuff	**Concentration U (ng/kg)**
Fresh vegetables	1900[1]
	520–920[2]
Canned vegetables	340[1]
	90–180[2]
Root vegetables	620[1]
	940–1200[2]
Potatoes	72[1]
	2660–2920[2]
	15 000–18 000[3]
Dry Beans	2200[1]
	1500–3670[2]
Fresh fruit	160[2]
	710–1290[2]
Canned fruit	81[1]
	180–290[2]
Fruit juice	49[1]
	40–120[2]
Bakery products	1900[1]
	1320–1500[2]
	12000[3]
Flour	390[1]
	250–680[2]
Whole grain products	1400[1]
	1450[2]
Macaroni	300[1]
	400–630[2]
Rice	240[1]
	1430–6000[2]
	15000[3]
Meat	190[1]
	580–1320[2]
	20000[3]
Poultry	64[1]
	140–420[2]
Eggs	150[1]
	230[2]
	9600[3]
Fresh fish	110[1]
	430–850[2]
	11 000[3]
Shellfish	160 000[1]
	9500–31 000[2]
Dairy products	59[1]
	80–310[2]
Tea	5000[3]
Coffee	6000[3]
Drinking Water	49

Table A3.5 Typical concentrations of uranium in various foodstuffs and animal feed (wet weight) sampled in the United Kingdom, adapted from MAFF (1999). Where possible data, with the exception of grass and silage, refers to sampling sites remote from nuclear facilities.

Food Stuff	Concentration U (ng/kg)
Milk[1]	< 291 (max = 433)
Lettuce[2]	5500
Potatoes[2]	<1300
Strawberries[2]	<670
Cabbage[2]	<790 - 5500
Grass[3] (Drigg soil mean [U] = 1380000)	3000 (max = 3250)
Grass[3] (Capenhurst soil mean [U] = 1575000)	7100 (max = 18 500)
Grass[3] (Springfields soil mean [U] = 3900000)	78 700 (max = 300 000)
Silage[3] (Capenhurst)	5100 (max = 8700)
Silage[3] (Springfields)	95 000 (max = 283 000)
Sea Fish[4]	150
Crustaceans[4]	1400
Crabs[4]	1800
Lobsters[4]	1400
Molluscs[4]	35 000
Winkles[4]	35 000

[1]From MAFF (1999, table 11.7)

[2]From MAFF (1999, table 11.8)

[3]From MAFF (1999, tables 4.10, 4.15 and 4.16)

[4]From MAFF (1999, table A6.1)

A3.4 Dietary information for exposure assessment

Table A3.6 Typical per capita food consumption levels (Muir et al., 1995), based on North American families as used in US EPA risk assessment calculations. For information on regional diets and associated variations the reader should consult WHO (1997) – Food consumption and exposure assessment of chemicals) and WHO (1998c) – GEMS/FOOD Regional Diets.

Foodstuff	Consumer Group	Consumption (kg/y)
Vegetables (spring mix)	Average American Family	96
Vegetables (summer mix)	Average American Family	160
Vegetables (autumn mix)	Average American Family	430
Vegetables (winter mix)	Average American Family	160
Fruit (spring mix)	Average American Family	66
Fruit (summer mix)	Average American Family	170
Fruit (autumn mix)	Average American Family	95
Fruit (winter mix)	Average American Family	73
Total milk	Average Infant (age under 8)	180
Total milk	Average Adult (age 19–64)	110
Total meat	Average Infant (age under 8)	40
Total meat	Average Adult (age 19–64)	86
Total poultry	Average Infant (age under 8)	5.1
Total poultry	Average Adult (age 19–64)	10

Table A3.7 Consumption rates for terrestrial foods in the United Kingdom based on national statistics (MAFF, 1999) For more detailed information on regional diets and associated variations see WHO (1997) – Food consumption and exposure assessment of chemicals) and WHO (1998c) – GEMS/FOOD Regional Diets.

Foodstuff	**Average / Above Average Consumption rates (kg/y) (Above Average = 97.5th percentile across all consumers)**	
	Adult	Infant
Beef	15 / 45	3 / 10
Cereals	50 / 100	15 / 30
Eggs	8.5 / 25	5 / 15
Fruit	20 / 75	9 / 35
Game	6 / 15	0.8 / 2.1
Green vegetables	15 / 45	3.5 / 10
Honey	2.5 / 9.5	2 / 7.5
Lamb	8 / 25	0.8 / 3
Legumes	20 / 50	3 / 10
Milk	95 / 240	130 / 320
Mushrooms	3 / 10	0.6 / 1.5
Nuts	3 / 10	1 / 2
Offal	5.5 / 20	1 / 5.5
Pork	15 / 40	1.5 / 5.5
Potatoes	50 / 120	10 / 35
Poultry	10 / 30	2 / 5.5
Root crops	10 / 40	5 / 15
Wild fruit	7 / 25	1 / 2

Table A3.8 Body weights and volumes for intake for 'reference man' to be used when site specific data is unavailable as described in WHO (1994). Note: these data are based on ICRP-23, (1974) unless otherwise indicated.

Body weight, kg: Adult male = 70; Adult Female = 58; Average = 64a

Daily fluid intake (milk, tap water, other beverages), ml/day
Normal conditions:
Adults = 1000–2400, representative figure = 1900[b] (excluding milk: 1400[c])
Adult male =1950
Adult female = 1400
Child (10 years) = 1400
High average temperature (32° C):
Adults = 2840–3410
Moderate activity:
Adults = 3700

Respiratory volumes
8–h respiratory volume, m^3

Resting	Light/non-occupational activity:
Adult man = 3.6	Adult man = 9.6
Adult woman = 2.9	Adult woman = 9.1
Child (10 years) = 2.3	Child (10 years) = 6.24

Daily inhalation volume, m^3
(8–h resting, 16–h light/non-occupational activity)
Adult male = 23
Adult female = 21
Child (10 years) = 15
Average adult = 22
Proportion of time spent indoors[c] = 20 h/day

Amount of soil ingested[c]: 20 mg/day

Dietary intake[d]
Cereals = 323 g/day (flour and milled rice)
Starchy roots = 225 g/day (sweet potatoes, cassava and other)
Sugar = 72 g/day (includes raw sugar, excludes syrups and honey)
Pulses and nuts = 33 g/day (includes cocoa beans)
Vegetables and fruits = 325 g/day (fresh equivalent)
Meat = 125 g/day (includes offal, poultry and game in terms of carcass weight)
Eggs = 19 g/day (fresh equivalent)
Fish = 23 g/day (landed weight)
Milk = 360 g/day (excludes butter; includes milk products as milk equivalent)
Fats and oils = 31 g/day (pure fat content)

Footnotes:
[a] WHO uses 60 kg for calculation of acceptable daily intakes and water quality guidelines (WHO, 1987, 1993).
[b] WHO uses a daily per capita drinking-water consumption of two litres in calculating water quality guidelines (WHO, 1993)
[c] from Health and Welfare Canada (1992)
[d] based on average of estimates for seven geographical regions (ICRP, 1974)

A3.5 Uranium chemistry

Table A3.9 Details of some selected uranium and uranium compounds.

Compound	CAS	NIOSH/ RTECS	UN	Molecular formula	Molecular weight	Solubility * in cold water	Solubility class**
Uranium	7440-61-1	YR3490000	2979	U	238	i	Insoluble
Uranium dioxide	1344-57-6			$U0_2$	270	i	Moderately soluble
Uranium hexafluoride	7783-81-5	YR4720000	2977	UF_6	352	s	Highly soluble
Uranium peroxide	19525-15-9			UO_4	302	-	Moderately soluble
Triuranium octaoxide	1317-99-3	YR3400000		U_3O_8	842	i	-
Uranium tetrachloride	10026-10-5	YR4025000		UCl_4	380	-	-
Uranium tetrafluoride	10049-14-6	YR4710000		UF_4	314	-	Highly to moderately soluble
Uranium trioxide	1344-58-7			UO_3	286	i	Moderately soluble
Uranyl acetate	541-09-3	YR3675000	9180	$UO_2(C_2H_3O_2)_2$	388	8	Moderately soluble
Uranyl chloride	7791-26-6	YR420000	2981	UO_2Cl_2	341	320	Moderately soluble
Uranyl nitrate	10102-06-4	YR3805000		$UO_2(NO_3)_2$	394	119	Highly soluble
Uranyl sulphate	1314-64-3			UO_2SO_4	366	19	Slightly soluble
Uranium carbonate				UO_2CO_3	330	-	Highly soluble

i = insoluble

s = soluble

* The solubility (S) are expressed as 100 times the mass of anhydrous compound soluble in a unit mass of water at a temperature between 13 and 20 ^{o}C (data from Kaye and Laby, 1986). Note the solubility in biological fluids may differ significantly from those observed in pure water.

** These solubility classes are taken from Mirto et al., (1999).

Annex 4 ICRP biokinetic models

A4.1 The Human Respiratory Tract Model (HRTM)

The ICRP Publication 66 Human Respiratory Tract Model for Radiological Protection (HRTM) (ICRP-66, 1994a) has been applied with the new generation of systemic models (ICRP-67, 1993; ICRP-69, 1995a) to calculate general-purpose dose coefficients. The effective dose coefficients for workers and the public given in ICRP-68, (1994) and ICRP-72 (1996) were adopted in the International Basic Safety Standards (BSS, 1996) and in the Euratom Directive (EC, 1996).

The HRTM is described in detail in ICRP-66 (1994a). Summaries are given in the ICRP Publications in which it is applied (ICRP-68b, 1994; ICRP-71, 1995b; ICRP-72, 1996; ICRP-78, 1997), and elsewhere (Bailey, 1993, 1994). Only an outline is given here. The main functions of the HRTM are to provide:

- a qualitative and quantitative description of the respiratory tract as a route for radionuclides to enter the body
- a method to calculate radiation doses to the respiratory tract for any exposure
- a method to calculate the transfer of radionuclides to other tissues

The HRTM is comprehensive. It applies to:

- assessing doses from exposures, and assessing intakes from bioassay measurements
- radionuclides associated with particles (aerosols) of all sizes of practical interest (0.0006–100 μm) and to gases and vapours
- all members of the population, giving reference values for children aged 3 months, 1, 5, 10 and 15 years, and adults. Guidance is provided for taking into account the effects of factors such as smoking, diseases and pollutants

A4.1.1 Morphometry

In the HRTM the respiratory tract is represented by five regions, based on differences in radio-sensitivity, deposition and clearance. The extrathoracic (head and neck) airways (ET) are divided into ET_1, the anterior nasal passage, and ET_2, which consists of the posterior nasal and oral passages, the pharynx and larynx. The thoracic regions (the lungs) are Bronchial (BB, airway generations 1–8), Bronchiolar (bb), and Alveolar-Interstitial (AI, the gas exchange region). Lymph nodes are associated with the extrathoracic and thoracic airways (LN_{ET} and LN_{TH} respectively). Target cells are identified in each region: for example the basal cells of the epithelium in both ET regions; basal and secretory cells in the bronchial epithelium. Reference values of dimensions are given which define the mass of tissue containing target cells in each region for dose calculations. They are assumed to be independent of age and sex.

A4.1.2 Physiology

The breathing rate (frequency and volume) is the main factor in the model that depends on age and physical activity. This is also one aspect for which there are comprehensive data relating to women and children. Reference values of important parameters are

recommended for the population groups noted above, for four levels of exercise: sleep, sitting, light and heavy exercise, and taking account of both nose- and mouth-breathing. These have been combined with habit survey data to give the reference volumes inhaled per working shift or per day. Thus light work is a combination of light exercise and sitting. These parameters determine intakes per unit exposure (time-integrated air concentration) but are also used with the deposition model to determine regional deposition.

A4.1.3 Deposition of particles

The deposition model evaluates the fraction of activity in the inhaled air that is deposited in each region. Deposition in the ET regions was determined mainly from experimental data. For the lungs, a theoretical model was used to calculate particle deposition in each region, and to quantify the effects of the subject's lung size and breathing rate. For particles larger than 1 μm, the 'aerodynamic' mechanisms of gravitational settling (sedimentation) and inertial impaction, which increase with particle size and density, dominate. For particles smaller than 0.1 μm, the 'thermodynamic' mechanism of diffusion, which increases with decreasing particle size, dominates. In the range 0.1–1 μm all are important. The aerodynamic equivalent diameter of a particle (d_{ae}) is the diameter of a unit density sphere with the same sedimentation velocity as the particle. The thermodynamic equivalent diameter of a particle (d_{th}) is the diameter of a sphere with the same diffusion coefficient as the particle.

Regional deposition for each age group and exercise level was calculated for aerosols with lognormal particle size distributions, and tabulated as a function of the median size. This may be the activity median aerodynamic or thermodynamic diameter, AMAD or AMTD. (50% of the activity in an aerosol is associated with particles with d_{ae} greater than the AMAD, or with particles with d_{th} greater than the AMTD). AMAD is used when deposition depends on sedimentation and inertial impaction, typically when AMAD less than 0.5 μm. AMTD is used when deposition depends on diffusion, typically when AMAD greater than 0.5 μm. In general, values of regional deposition are lower than corresponding values using the ICRP-30 model (ICRP-30, 1979), and do not vary markedly with age.

The ICRP default values for deposition in the respiratory tract after occupational and public exposure are shown in Table A4.1.

A4.1.4 Gases and vapours

Unlike deposition of particles, respiratory tract retention of gases and vapours is material specific. Virtually all inhaled gas molecules contact airway surfaces, but are usually re-esuspended in the air unless they dissolve in, or react with, the surface lining. The fraction of an inhaled gas or vapour that is retained in, or absorbed from, each respiratory tract region thus depends on its solubility and reactivity and, except in simple cases, has to be treated on an individual basis. The model assigns gases and vapours to three classes:

i) SR-0. Insoluble and non-reactive. No deposition, or uptake to blood. In most cases external radiation from the surrounding cloud dominates exposure.

ii) SR-1. Soluble or reactive, some exposure to all airways, and absorption into blood. They require individual evaluation, but the most important parameter is often the fraction absorbed into blood.

iii) SR-2. Highly soluble and reactive. Complete and instantaneous uptake assumed.

Table A4.1 Deposition of inhaled aerosols after occupational and public exposure

Region[c]	Occupational[a] (%)	Public[b] (%)
ET_1	33.9	14.2
ET_2	39.9	17.9
BB (bronchial)	1.8 (33% in BB_2)	1.1 (47% in BB_2)
bb (bronchiolar)	1.1 (33% in bb_2)	2.1 (49% in bb_2)
AI	5.3	11.9
Total deposit	82.0	47.3

Notes

a Occupational exposure. Worker, 5-µm AMAD (σ_g = 2.5), 3.5-µm AMTD, density 3.0 g/cm^3, shape factor 1.5 (see Chapter 5); fraction breathed through nose is 1. 31% siting and 69% light exercise; mean ventilation rate is 1.2m^3/h. (See Table 6.)

b Environmental Exposure (indoors at home). Adult male, 1-µm AMAD (σ_g = 2.47), 0.69-µm AMTD. density 3.0 g/cm^3, shape factor 1.5; fraction breathed through nose is 1. 55% ventilation rate is 0.78 m^3/h. 33.3% sleeping, 25% sitting, 40.6% light exercise and 1.0% heavy exercise.

c The extrathoracic airways consist of the anterior nasal passages (ET_1) and posterior nasal and oral passages, pharynx and larynx (ET_2). The thoracic regions are bronchial and bronchiolar (BB and bb) and alveolar-interstitial (AI). For the purposes of external monitoring, the retention in the chest would be the activity retained in the thoracic region.

A4.1.5 Clearance

The model describes three clearance pathways (Figure A4.1). Material deposited in ET_1 is removed by nose blowing. In other regions clearance results from a combination of movement of particles to the gastrointestinal (GI) tract and lymph nodes (*particle transport*), and movement of radionuclides into the blood (*absorption*). It is assumed that:

- all clearance rates are independent of age and sex.
- particle transport rates are the same for all materials.
- absorption into blood, which is material specific, occurs at the same rate in all regions except ET_1, where none occurs.
- fractional clearance rates vary with time, but to simplify calculations are represented by combinations of compartments that clear at constant rates. Since particle transport rates are the same for all materials, a single compartment model applies to all, and it was based, so far as possible on human experimental data.

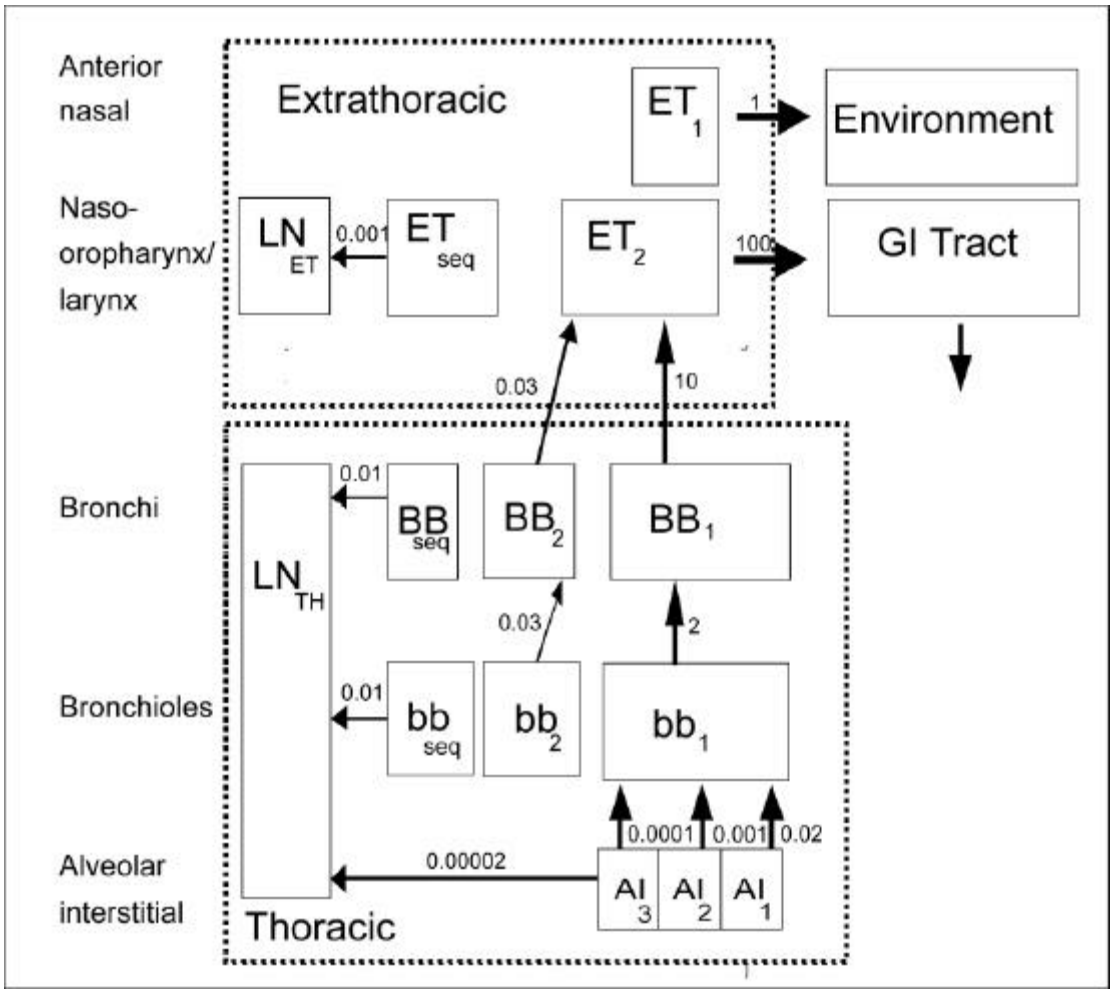

Figure A4.1 Compartment model to represent time dependent particle transport from each region of the respiratory tract. The rate constants down beside the arrows are reference values expressed as d^{-1}.

Absorption to blood is a two-stage process (Figure A4.2): dissociation of the particles into material that can be absorbed into blood (*dissolution*); and absorption into blood of soluble material and of material dissociated from particles (*uptake*). Both stages can be time-dependent. The simplest representation of time-dependent *dissolution* is to assume that a fraction (f_r) dissolves relatively rapidly, at a rate s_r, and the remaining fraction ($1–f_r$) dissolves more slowly, at a rate s_s. Provision is made in the HRTM for two fractions, to avoid undue complexity. *Uptake* to body fluids of dissolved material can usually be treated as instantaneous. In some situations, however, a significant fraction of the dissolved material is absorbed slowly. To enable this to be taken into account, the HRTM includes compartments in which activity is retained in each region in a 'bound' state. However, it is assumed by default that uptake is instantaneous, and the 'bound' state is not used and hence is not included in Figure A4.2.

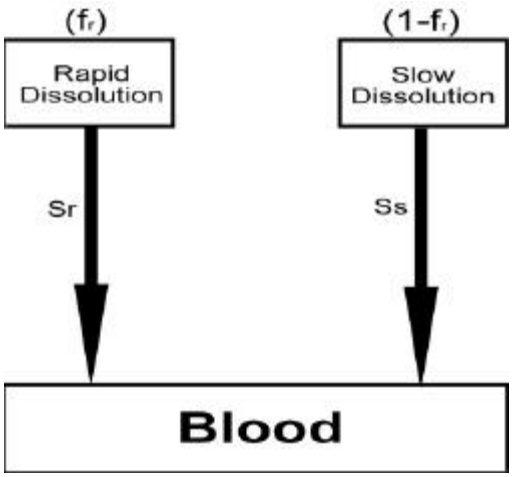

Figure A4.2 Model for time dependent absorption into blood.

It is recommended that material-specific rates of absorption should be used for compounds for which reliable experimental data exist. For other compounds, default values of parameters are recommended (Table A4.2), according to whether the absorption is considered to be fast (Type F), moderate (M) or slow (S) (corresponding broadly to inhalation Classes D, W and Y in the ICRP-30 model).

Table A4.2 Default absorption parameter values for Type F, M and S materials

ICRP Publication 66 absorption type	F (fast)[a]	M (moderate)[b]	S (slow)[c]
Model parameters:			
Fraction dissolved rapidly, f_r	1	0.1	0.001
Dissolution rate:			
Rapid (per day), s_r	100	100	100
Slow (per day), s_s	-	0.005	0.0001

Notes

a) F (fast) – materials that are readily absorbed into blood (corresponding to 'Class D'). There is significant absorption from ET_2 and BB_1, but some material in these regions will remain in solution in mucus and be swallowed, rather than be absorbed through the epithelium. Hence the default for such materials is s_r=100 per day (t ½ approximately 10 minutes).

b) M (moderate) – materials with intermediate rates of absorption (corresponding to 'Class W'). For such materials the percentage absorbed rapidly is on the order of 10%, and the slow-phase retention time of the order of 100 per d. This is represented by f_r = 0.1%; s_r = 100 per day; and s_s = 0.005 per day

c) S (slow) – relatively insoluble materials (corresponding to 'Class Y'). It is assumed that for most of the material the rate of absorption to blood is 0.0001 per day. This equals the particle transport rate from the most slowly cleared AI compartment. However, it is characteristic of even very insoluble materials that some rapid uptake to blood occurs immediately after inhalation. As a default it is assumed that 0.1% of the deposited material is rapidly absorbed. While the effect of this on doses is likely to be negligible, it may significantly affect the interpretation of measurement of activity in urine. This is represented by f_r = 0.001; s_r = 100 per day; and s_s = 0.0001 per day.

A4.1.6 Dose calculation

In accordance with the general approach taken by ICRP, the dose to each region is given by the average dose to the target tissue in that region. To take account of differences in sensitivity between tissues, the dose to each region *i* is multiplied by a factor A_i representing the region's sensitivity relative to that of the whole organ. The weighted sum gives the equivalent dose to the extrathoracic or thoracic airways.

A4.2 The systemic model for uranium

The fate of uranium that enters the bloodstream and systemic tissues cannot be observed or easily measured. Therefore, models are used to represent the movement of material around the body. These models can be used to calculate radiation doses to tissues and to predict the retention and excretion of the element.

The model used for uranium (Figure A4.3) is that recommended by ICRP-69 (1995a). This model describes the deposition of material from the blood into various organs or regions, the transfer from region to region, the return of material to blood, and the eventual excretion of the material. In keeping with ICRP's move towards physiological realism in its models, the uranium model includes recycling, i.e. the possibility for material to pass from region to region via the blood stream (Leggett, 1994). Previous models were simple catenary, or 'straight chain' models; the current uranium model is thus a marked improvement on earlier models.

The model is based on a number of sources which include data from both animal experiments (using baboons, dogs and rats) and studies on humans. Clearly human data is to be preferred, and for uranium, ICRP can draw on a large database, which is not the case for many other elements. In particular, there are data from the so-called Boston Subjects, a group of terminally ill patients who were injected with uranium in the 1950s. A brief overview of the human data that support the ICRP model is given in ICRP-69 (1995a). Other reviews are provided by Leggett and Harrison (1995) and Leggett (1989, 1992).

The principal sites of uranium deposition in the body are the kidneys, the liver and bones. In addition, some material is deposited in various other tissues generally at lower concentrations than the main sites of deposition; these are usually referred to as 'soft tissues'. Of the amount absorbed into the blood stream, the model assigns 30% to soft tissues (rapid turnover, ST0), this represents a pool of activity distributed throughout the body which exchanges rapidly with the blood stream. The remaining activity is apportioned as follows, kidneys 12%, liver 2%, bone 15%, red blood cells 1%, soft tissue (intermediate turnover, ST1) 6.7%, soft tissue (slow turnover, ST2) 0.3%, with 63% being promptly excreted in urine via the bladder.

Some of the material initially deposited in these regions can be returned to the blood stream while some is transferred to other regions of tissues (Figure A4.3). For example, material in the soft tissue compartments is returned only to blood while material in liver can be exchanged with blood or transferred to other regions of the liver (Liver 2 in Figure A4.3). The bone warrants additional comment. Material is initially deposited on the bone surface (either trabecular or cortical), from where it can be transferred to bone volume (exchangeable) or returned to the blood stream. Material which does reach the exchangeable bone volume can be buried deeper in the bone volume (non-exchangeable) or returned to the surface. Material in non-exchangeable volume is transferred slowly to blood. All the pathways used in the model are illustrated in Figure

A4.3. In time, most of the systemic uranium is excreted in urine via the bladder, a small fraction is also excreted in faeces.

The length of time that material remains in these regions is partly governed by a removal half-time, i.e. the time that it takes to remove half of the material present. This time varies from organ to organ, for example the removal half-time for ST0 is as little as two hours, while for ST2 it is one hundred years. The net or apparent time that is takes to halve the amount of material in an organ, however, can be very different from the removal half-time, since material is continually being re-deposited by the recycling nature of the model. The net half-time thus results from a combination of removal of existing material and deposition of new material from the blood stream. It is difficult to simply state values for net half-times. Table A4.3 complements Figure A4.3. It gives retention in liver, kidneys, bone (comprising the six skeleton compartments of Figure A4.1) and the whole body at a number of times after an acute intake directly into the blood stream. It also gives the amount of activity excreted in urine and faeces per day.

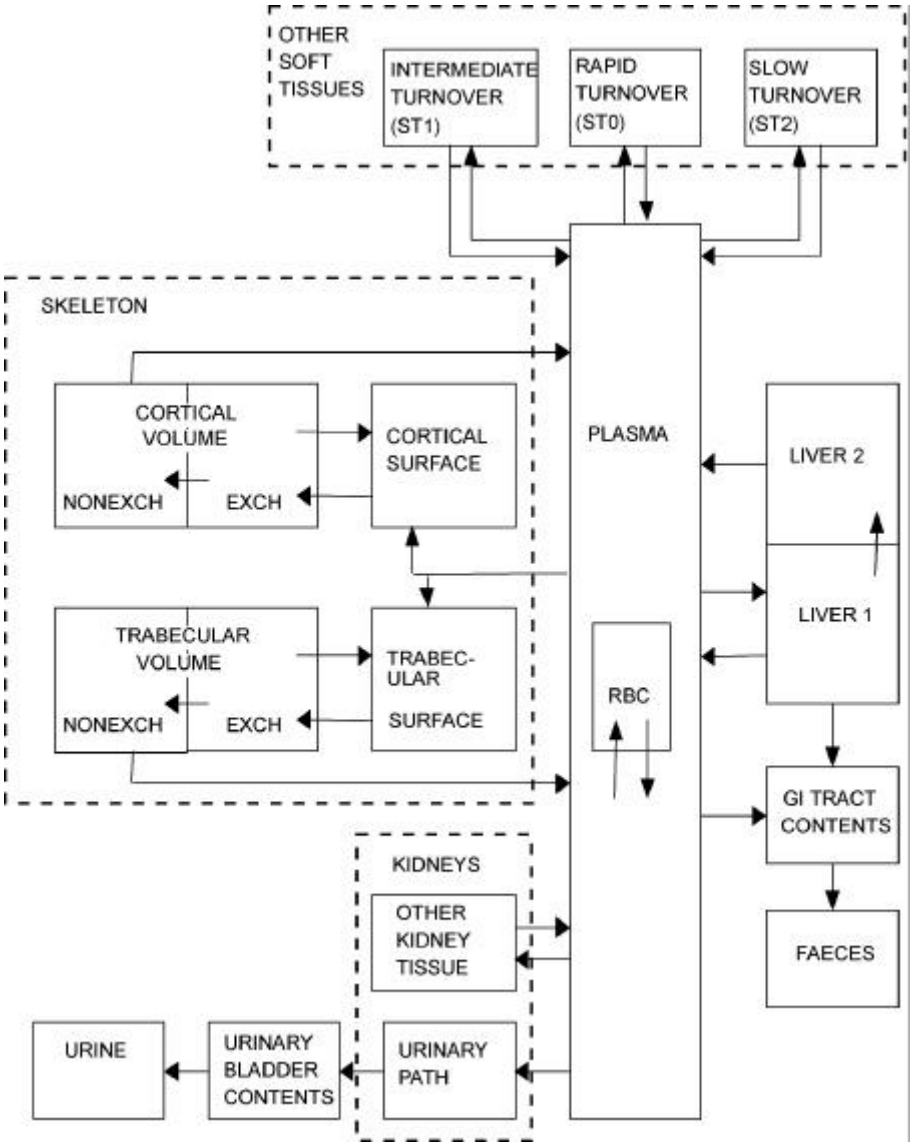

Figure A4.3 The biokinetic model for uranium.

A4.3 The model for the gastrointestinal tract.

ICRP recommended in Publication 30 (ICRP-30, 1979) the use of compartmental models to calculate the distribution of radioactive transformations in the body. Each organ is modelled as one or more compartments. For purposes of dose calculation, material is usually taken to be uniformly distributed throughout the organ. Transfers between compartments are assumed to obey first order kinetics. For the gastrointestinal (GI) tract the model has four compartments (see Figure A4.4).

Table A4.3 Retention and daily excretion following an acute unit intake (1 Bq or 1 μg) of DU directly into blood.

Time (days)	Faeces (Bq per day)	Urine (Bq per day)	Liver	Kidneys	Bone	Soft tissues	Whole Body
1	1.68×10^{-3}	6.45×10^{-1}	1.40×10^{-2}	1.12×10^{-1}	1.43×10^{-1}	7.06×10^{-2}	3.53×10^{-1}
3	9.47×10^{-4}	1.80×10^{-2}	1.20×10^{-2}	9.48×10^{-2}	1.31×10^{-1}	6.65×10^{-2}	3.10×10^{-1}
10	3.73×10^{-5}	9.43×10^{-3}	7.21×10^{-3}	5.27×10^{-2}	1.04×10^{-1}	5.63×10^{-2}	2.22×10^{-1}
30	1.11×10^{-5}	2.39×10^{-3}	2.41×10^{-3}	1.08×10^{-2}	8.08×10^{-2}	3.30×10^{-2}	1.27×10^{-1}
100	2.30×10^{-6}	3.51×10^{-4}	1.36×10^{-3}	1.26×10^{-3}	5.61×10^{-2}	7.60×10^{-3}	6.63×10^{-2}
1000	5.37×10^{-8}	8.09×10^{-6}	1.12×10^{-3}	4.51×10^{-4}	2.95×10^{-2}	3.79×10^{-3}	3.49×10^{-2}
10000	5.41×10^{-9}	8.15×10^{-7}	2.13×10^{-4}	1.80×10^{-5}	8.03×10^{-3}	3.26×10^{-3}	1.15×10^{-2}

A4.3.1 Stomach

It is assumed that no absorption takes place from the stomach and that material passes on to the next compartment with a mean residence time of one hour.

A4.3.2 Small intestine

The mean residence time is taken to be four hours. This is the compartment from which absorption takes place. It is normal to quantify absorption by using the f_1 value. f_1 is the fraction of material reaching body fluids following ingestion.

$$f_1 = \frac{l_B}{l_B + l_{SI}}$$

λ_B = rate constant for transfer to body fluids

λ_{SI} = rate constant for transfer from small intestine to upper large intestine.

It is worth noting that the small intestine is alkaline. This means that elements which hydrolyse, notably the actinides (but not uranium), are usually in an insoluble form and are not readily absorbed (i.e. have a low f_1 value).

A4.3.3 Upper large intestine

The mean residence time is taken to be 13 hours. In practice water is absorbed from the gut content in the upper large intestine. However, it is not necessary to model this since tritiated water is taken to be homogeneously distributed across all soft tissue.

A4.3.4 Lower large intestine

The mean residence time is taken to be 24 hours. It is important to realize that the lower large intestine may be the most heavily irradiated organ if the gut uptake factor is low. This will particularly be the case for materials emitting relatively non-penetrating radiation with a short physical half-life.

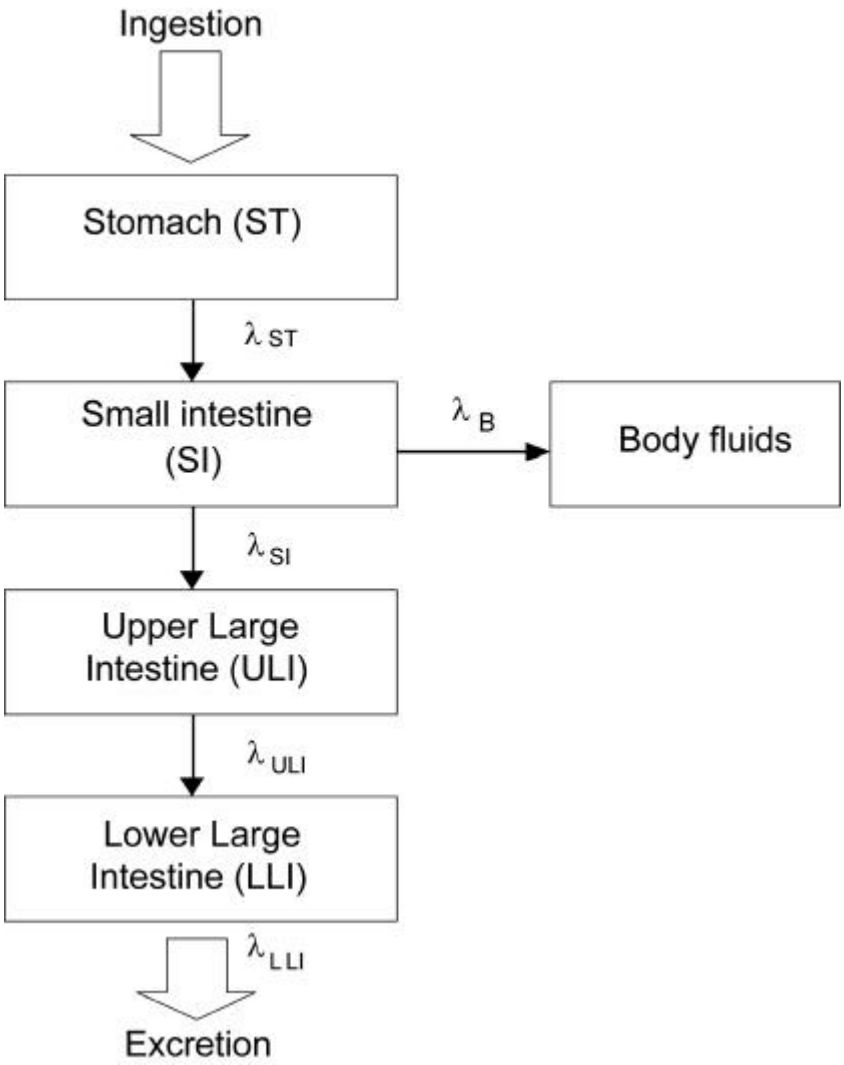

Figure A4.4 Mathematical model used to describe the kinetics of radionuclides in the gastrointestinal tract.

Annex 5 Chemical toxicity of uranium: Occupational exposure standards after inhalation and the impact of ICRP biokinetic models.

A5.1 Soluble uranium compounds

In order to appreciate the rationale behind the currently recommended exposure limits for uranium based on chemical toxicity, it is necessary to examine the developments and changes that have occurred during the past 40 years. (These are summarized in Table A5.1)

Table A5.1 Changes and developments in recommended occupational exposure limits for uranium based on chemical toxicity.

Year	Source	$(MPC)_a$/TLV mg/m^3	Daily limit mg
1940s/50s	Various	0.02–0.05	
1957	ACGIH	0.05	
1959	ICRP–2/ NCRP 2	0.2[a]	1.5
1964	ICRP–6	0.2[b]	2.5[c]
1968	ICRP–10	0.2[b]	2.5[c]
1968	ACGIH	0.2	1.5[d]
1969	Dept. Employment, UK	0.2	1.5[d]
1979	ICRP–30	N/A	N/A[e]
1980	HSE, UK	0.2	2.0[f]
1980	OJEC		2.5[c]
1988	ICRP–54	0.2	2.0[f]
1989	OSHA	0.05	0.5[f]
1994	ICRP–68	N/A[e]	N/A[e]
1995	ICRP–71	N/A[e]	N/A[e]
1996	OJEC	N/A[e]	N/A[e]
1996	ACGIH	0.2	2.0[f]
1997	HSE, UK	0.2	2.0[f]
1997	ICRP–78	N/A[e]	N/A[e]
2000	NIOSH	0.2	2.0[f]

a Value derived from the daily intake assuming a breathing rate of 6.9 m^3 per 8 h working day; value correlates with $(MPC)_a$ of 7×10^{-11} µCi/cm^3 (see Table A5.2)
b Value correlates with $(MPC)_a$ of 7×10^{-11} µCi/cm^3 (see Table A5.2)
c Value based on short- term exposure rule
d Not listed but based on an average breathing rate of 6.9 m^3 per 8 h working day
e Chemical toxicity not addressed
f Value not listed but based on a breathing rate of 9.6 m^3 per 8 h working day

A5.1.1 Initial recommendations

During the late 1940s and 1950s, various recommendations on exposure limits were made as a consequence of discussions at international conferences (Spoor and Hursh 1973). The values for maximum airborne concentrations, based on toxicity data from animal studies conducted at the time, ranged from 25 µg/m^3 to 50 µg/m^3 (Spoor and Hursh, 1973). The latter value referred to as the Threshold Limit Value (TLV), was subsequently endorsed by the American Conference of Governmental Industrial Hygienists (ACGIH) in 1957 and the recommendations published in 1960 (Spoor and Hursh, 1973; NCRP, 1959; ACGIH, 1960). The current limits on exposure stem mainly from discussions between Committees II of ICRP and NCRP (National Committee on Radiological Protection) in 1959 (Spoor and Hursh, 1973; NCRP, 1959; ICRP, 1959). In formulating these limits, it was considered that the renal concentration of uranium

that could be safely tolerated by man was 3 µg/g (Spoor and Hursh 1973). This concentration was not listed as such by ICRP-2 (1959), but could be derived from three other listed values. These were, for a dose of 50 mSv/y which was the recommended limit at the time, the maximum permissible content of uranium in the total body with the kidney considered the critical organ (so called q value), namely 5×10^{-3} µCi (185 Bq); the fraction of the uranium in the kidneys relative to that in the total body (so called f_2 value); and a kidney mass of 300 g (Spoor and Hursh 1973, ICRP 1959). At that time, the specific activity of natural uranium was considered to be 0.33 µCi/g (12.2 kBq/g) Spoor and Hursh, 1973; ICRP-2, 1959). Hence the permissible concentration in the kidney was calculated to be (Spoor and Hursh, 1973)

$$\frac{5\times10^{-3}}{0.33} \times \frac{0.065}{300} = 3.3\times10^{-6}\ \text{g/g} = 3.3\ \mu\text{g/g}$$

However, the evidence available from animal studies showed that mild to moderate kidney damage occurred in a variety of animal species at concentrations up to an order of magnitude lower than this. It has been suggested, therefore, that the Committees of ICRP and NCRP were less influenced in the choice of a safe kidney concentration by the animal data than by the concern that the calculation reflected the experience of many years of occupational exposure (Spoor and Hursh, 1973). This experience had shown no evidence of kidney malfunction in workers even at exposure levels in excess of those derived using the kidney concentration above. The procedure adopted by NCRP Committee II for deriving the exposure limit is summarized in Table A5.2.

Table A5.2 Derivation of the permissible daily intake and $(MPC)_a$ for soluble natural uranium by NCRP Committee II (Spoor and Hursh, 1973).

Assumption	Source
A maximum permissible concentration of 3µg uranium per gram kidney	Animal experiment results; Committee judgement decision.
An average kidney mass of 300 g	Standard Man (ICRP-2, 1959)
An effective half-life of 15 days for uranium in the kidney, i.e. 0.0462 × the kidney content is excreted per day	Animal experiments: Human data
That 2.8% of the uranium inhaled was deposited in the kidney (f_a as denoted by ICRP-NCRP)	The lung model (ICRP Publication 2, 1959) specifies that the 25% deposited in the pulmonary lung is absorbed into the body for soluble compounds). The 50% deposited in the upper respiratory tract is transferred to the gut and because of the negligible absorption of uranium can be neglected. Of the systemic uranium, 78% is rapidly excreted and the remaining 22% is divided equally between bone and kidney. $f_a = 0.25 \times 0.11 = 0.028$
That a worker breathes in an average of 6.9×10^6 cm^3 air per working day	Standard Man (ICRP-2, 1959)

The calculation of the maximum permissible concentration in air $(MPC)_a$ for soluble uranium is as follows:

- The maximum permissible daily kidney input equals the daily rate of loss from the kidney when the burden is the maximum permissible = 0.0462×900 = 41.5 µg.
- The corresponding lung daily input = 41.5/0.028 = 1480 µg.

Therefore, the $(MPC)_a = 1480/6.9\times10^6 = 2.1\times10^4$ g/cm^3.

This limit has been expressed (ICRP-2, 1959; ICRP-6, 1962) in terms of µCi per cm^3 air using the special curie unit used for natural uranium, 0.33 µCi/g. Accordingly, 210 µg natural uranium per m^3 air converts to 7×10^{-11} µCi/cm^3, which is the value, cited in the above references.

This corresponded to a daily intake limit of 1.48 mg based on a breathing rate of 6.9 m^3 per working day and a maximum permissible concentration in air (MPC_a) of 0.21-mg/m^3.

A5.1.2 Subsequent developments.

In 1964, ICRP recommended in Publication 6 that the inhalation of soluble uranium of any isotopic composition should not exceed 2.5 mg in any one day (ICRP-6 1964). This recommendation was re-affirmed in ICRP-10, (1968). In the same year, the ACGIH increased the TLV from 0.05 mg/m^3 to 0.2 mg/m^3, presumably to be consistent with the earlier recommendation of NCRP and ICRP (ACGIH, 1968). It is noteworthy that in the UK, the revised value of the TLV was adopted by the Department of Employment in 1969 (DEP, 1969), and has remained in force ever since (HSE, 2000).

The chemical toxicity of uranium was not considered by ICRP-30 (1979), but the previous recommendation in Publication 10 in 1968 (ICRP-10, 1968) was incorporated into European legislation in 1980 (OJEC, 1980) with the statement 'In view of the chemical toxicity of water soluble compounds of uranium inhalation and ingestion should not exceed 2.5 mg and 150 mg respectively in any one day regardless of isotopic composition'. In hindsight, the choice of the phrase 'water soluble' was unfortunate since some uranium compounds such as the trioxide, tetrafluoride and tributylphosphate which have low aqueous solubility are rapidly absorbed into the blood after deposition in the lungs (Stradling et al., 1989; Stradling and Moody, 1995; Pellow et al., 1997; Ansoborlo et al., 2001).

The chemical solubility of uranium was considered again by ICRP in Publication 54 published in 1988 (ICRP-54, 1988). The advice is unequivocal, and states that 'For soluble forms of depleted, natural and low enriched uranium, the limit on intake is determined by consideration of chemical toxicity. Annual Limits on Intake are entirely inappropriate for such materials'. The proposed daily limit of 2 mg is based on an airborne concentration of 0.2 mg/m^3 and a breathing rate of 1.2 m^3/h or 9.6 m^3 for a 8 h working day. These values are mutually incompatible when compared with the original procedure used for deriving exposure limits (see Table A5.2). In other words, an increase in the default value for the breathing rate from 6.9 m^3/d to 9.6 m^3/d should decrease the airborne concentration to 0.15 mg/m^3 whilst the daily limit should remain unchanged at 1.48 mg. Interestingly, in 1989 (OSHA, 1989), the Occupational Safety and Health Administration (OSHA) in the United States recommended a Permissible Exposure Limit (PEL) of 0.05 mg/m^3 for soluble compounds. This is equivalent to 0.5 mg/d on the basis of a breathing rate of 9.6 m^3/d.

Despite the pronouncement on chemical toxicity in ICRP Publication 54 (ICRP 1988), the subject was not addressed in Publication 60 in 1991 (ICRP-60, 1991b), nor Publication 68 in 1994 (ICRP-68, 1994b) which were intended to give advice on radiation dose only. As a consequence, advice on exposure limits based on chemical toxicity has not been included in the latest EURATOM directive (OJEC, 1996) and the International Basic Standards for Protection Against Ionizing Radiation (BSS, 1996). Nevertheless the dose coefficients (doses per unit intake, Sv/Bq) for different isotopes included in these documents are invaluable, since workers, particularly in the nuclear

industries, are potentially exposed to a mixture of radionuclides which require the committed effective dose to be assessed. However, it remains a matter of concern that the nephrotoxicity of uranium could be overlooked if the above publications alone were used to assess the health consequences of exposure to uranium compounds. Fortunately, this potential difficulty has been discussed more recently in ICRP Publication 78 (ICRP-78, 1997).

At present there is still widespread acceptance that the occupational exposure limit for soluble uranium compounds is 0.2 mg/m^3 (ACGIH 2000, NIOSH 2000, HSE 2000).

A5.1.3 The nephrotoxicity of uranium.

It is not the purpose of this section to review the nephrotoxicity of uranium. This is dealt with in Chapter 8. However there are issues relating to the basis of the normally accepted threshold concentration of uranium in the kidneys that need to be addressed.

If the current definition for the specific activity of uranium (0.68 μCi/g) and dose limit of 20 mSv/y were used in the original calculations, the permissible kidney concentration would be 0.6 μg/g, and the daily limit on intake 0.3 mg (see section A4.1). The kidney concentration would be reduced still further if the amount specified for the initial deposition in ICRP Publication 69 (ICRP-69, 1995a), 12%, was used instead of the original value of 6.5%. Together, all these factors suggest that the permissible concentration in the kidneys should be about 0.3 μg/g rather than 3 μg/g. It is noteworthy that a review of the uranium concentrations in the kidneys of animals after exposure to soluble uranium compounds for up to one year indicated that mild to moderate damage occurred in the range 0.3-3 μg/g.

The threshold concentration of 3 μg/g has also been challenged in two comprehensive review articles in which it is also claimed that this value has neither been supported by unequivocal human data, nor by studies with laboratory animals (Leggett 1989, Diamond 1998). The authors concluded that it would be prudent to lower this long-standing guidance level by at least three-fold until more is known about the physiological effects of low concentrations of uranium in the kidneys, particularly after chronic exposure. More recent animal studies would appear to support such a reduction. Recent studies by Gilman et al (1998a, b, c) with rabbits also support a reduction in kidney concentration. Moreover, it has been noted that the urinary excretion of uranium is impaired at kidney concentrations below 1 μg/g (Hodgson et al 2000), presumably as a consequence of nephro-toxicological effects. In contrast, a recent American National Standard (ANS, 1996) has re-affirmed the 3 μg/g kidney concentration limit as a basis for designing and interpreting bioassay programs. However, since the biokinetic model used for assessing this historical value is now obsolete, and the 3μg/g concentration value has been rigorously challenged, this approach has to be considered doubtful.

In conclusion, it should be stressed that a reduction in the acceptable kidney concentration does not imply a similar reduction in the value of the airborne concentration permitted in the workplace. The current ICRP physiological models show that substantially greater amounts of uranium need to be inhaled to result in the same kidney concentration as predicted by the original models (Stradling et al., 1998). The net effect is that the permitted airborne concentration of 0.2 mg/m^3 will in fact be conservative. This issue is also discussed in Chapter 9.

Table A5.3 Uranium concentration in the kidney after exposure of one year to inhalation of soluble uranium compounds[a] (from Spoor and Hursh 1973).

Uranium dust concentration µg/m^3	Compound	Dogs		Rats		Rabbits[b]		
		No.	µg/g	No.	µg/g	No.	µg/g	N
2000	$UO_2(NO_3)_2.6H_2O$	5	1.7 (1.2-2.3)[c]	24	5.6 (1.9-11.3)	3	1.4 (0.8-2.2)	1
250	$UO_2(NO_3)_2.6H_2O$	15	1.0 (0.1-1.9)	23	1.6 (0.1-4.4)	5	0.9 (0.4-1.9)	1
200	UF_6	10	0.4 (0.0-0.7)	23	2.7 (0.0-5.8)	7	0.3 (0.0-0.8)	-
200	UCl_4	13	0.2 (0.0-0.5)	12	0.4 (0.1-1.9)	10	0.4 (0.2-0.6)	1
150	$UO_2(NO_3)_2.6H_2O$	11	0.5 (0.2-1.0)	23	1.4 (0.6-3.3)	-	-	-
50	UF_6	12	0.3 (0.0-0.5)	26	0.9 (0.1-2.0)	-	-	1
50	UCl_4	15	0.2 (0.9-0.5)	7	0.4 (0.1-0.9)	-	-	-
40	$UO_2(NO_3)_2.6H_2O$	17	0.4 (0.1-1.0)	25	0.4 (0.1-2.0)	-	-	-

[a] Data compiled from Hodge et al. (1953)

[b] Exposure period was 7-9 months

[c] Range of values in parenthes

A5.2 Insoluble uranium compounds.

The methodology for deriving the so-called maximum permissible concentration in air, MPC_a, for insoluble natural uranium is described in more detail elsewhere (Spoor and Hursh, 1973). This methodology is similar to that described for soluble uranium in that it is derived from the maximum permissible lung burden (8.9×10^{-3} μCi) recommended in ICRP-2 (1959) for an annual dose limit to this tissue of 15 rem, and converts radioactivity to mass at equilibrium conditions in the lung using a simplistic metabolic model and the specific activity of the 'special curie' for uranium.

Essentially the calculation proceeded as follows (Spoor and Hursh, 1973),:

Maximum permissible lung burden for annual dose of 15 rem $=8.9\times10^{-3}$ μCi.

At equilibrium, the rate of loss from the lungs using a clearance half-time of 120 d (ICRP-2, 1959) will be

$0.693/120\ \text{d} \times 8.9\times10^{-3}\ \mu\text{Ci} = 5.1\times10^{-5}\ \mu\text{Ci/d}$

On the assumption that the fraction of the inhaled material which is deposited in the lungs is 0.125 and the average breathing rate is $6.9\times10\ \text{cm}^3/\text{d}$ (ICRP-2, 1959), the

$(MPC_a) = 5.1\times10^{-5}\ \mu\text{Ci per d} / [6.9\times10^{6}\ \text{cm}^3\ \text{per d} \times 0.125] = 6\times10^{-11}\ \mu\text{Ci/cm}^3$

This is the value listed in ICRP-2 (1959) for a 40-hour week, 50 week year.

Based on the definition of the special curie (specific activity of natural uranium 0.33-μCi/g), this concentration converts to 0.18 mg/m^3, rounded to 0.2 mg/m^3. If the currently accepted breathing rate of 1.2 m^3/h were used, then the annual intake based on a 40 hour week, 50 week year would be 480 mg.

Based on current dose limits and biokinetic models, discussed in Chapter 10, and the annual limits on intake for insoluble uranium listed for natural and depleted uranium in Table 10.3 of that chapter, it would seem prudent to reduce this value by four-fold and two-fold respectively.

However, the value of 0.2 mg/m^3 is still used in current recommendations of the American Conference of Governmental and Industrial Hygienists (ACGIH, 2000), and the US National Institute for Occupational Health (NIOSH, 2000). The value is also legally binding in France (FRA, 1988). In the UK, the Health and Safety Executive recommend values for soluble uranium compounds only (HSE, 2000).

Annex 6 Methods for chemical and isotopic analysis in support of public health standards and environmental investigations.

Methods for the determination of uranium in environmental materials such as soils and drinking water are diverse and over the past 20 years improvements in analytical techniques have considerably improved our knowledge of environmental levels (e.g. Toole et al., 1997). Techniques for the analysis of uranium can be divided into three distinctive groups.

A6.1 Non-nuclear instrumental techniques

This group of analytical techniques include inductively coupled plasma–mass spectrometry (ICP-MS), x-ray fluorescence analysis (XRF), thermal ionization mass spectrometry (TIMS) and electron microprobe analysis (EPMA) (Gill, 1997; Van Loon and Barefoot, 1989). Of these techniques the most robust and sensitive technique for the analysis of uranium in a wide variety of environmental matrices is ICP-MS. Typical detection limits for this technique in ideal matrices for uranium are in the order of 1 ng to 5 ng per litre (sample less than 10 cm^3 in volume) (Taylor et al., 1998). The speed and versatility of this technique has led the nuclear industry to use it for the analysis of uranium and plutonium in urine during routine monitoring (Ejnik et al., 2000).

XRF is a useful robust technique particularly in solid matrices such as soils and foodstuffs where detection limits in the range of 1mg/kg are commonly achievable. Although XRF cannot be used to differentiate various isotopes of uranium, its portable derivatives enable the analysis of uranium in the field to a detection limit of 50 mg/kg. Such methods greatly facilitate the identification and prioritisation of sampling strategies in the field.

Until recently TIMS was the preferred method for the determination of uranium isotopic ratios in environmental samples because of its unrivalled sensitivity, accuracy and precision. However, it is a particularly slow technique limiting its application to large-scale environmental surveys. Recently, the advent of magnetic sector and multi-collector ICP-MS offers similar accuracy and precision to TIMS but considerable advantages over this technique in terms of sample throughput and ease of use (Halliday et al., 1998).

For spatial analysis of uranium within samples, EPMA may be used with a resolution of 5 µm or better. However, the detection limit of this technique is rather poor (1000 mg/kg) and it is not possible to determine isotopic ratios using this technique. If high sensitivity spatial analysis of uranium-series isotopes is to be undertaken coupled techniques such as laser ablation ICP-MS or laser ablation multi-collector - ICP-MS offer the ability to determine mg/kg levels of uranium at a spatial resolution of 20 µm to 100 µm.

A6.2 Nuclear instrumental techniques

Prior to the advent of ICP-MS, nuclear based analytical techniques such as alpha spectrometry, gamma spectrometry, neutron activation analysis and fission track analysis (Gill, 1997; Ivanovich and Harmon, 1982; IAEA, 1989b) were routinely used

for the determination of uranium in a wide range of materials. However, each of these techniques either requires extensive sample preparation or pre-concentration to determine uranium at environmental levels, mainly because of the long half-life of ^{238}U. For this reason, the use of these techniques for the quantitative determination of uranium at environmental concentrations has generally dwindled over the past decade in favour of other methods. The use of these techniques in the field is practically limited to gamma ray spectroscopy, although some workers have used various forms of hand held proportional counters, ionization counters and GM tubes to detect surface contamination via the emission of beta particles of ^{234}Th, ^{234}Pa and ^{231}Th which 'in-grow' rapidly from pure uranium. The use of alpha and beta detectors in the field is generally inhibited by the relatively rapid absorption of alpha and beta particles by environmental media (i.e. soil). Because of this such detectors are best used for identifying surface contamination or metallic fragments of DU.

With respect to the use of gamma spectroscopy, the lack of a sufficiently high-energy, high-yield gamma emission by ^{238}U, the main constituent of DU, significantly reduces the effectiveness of this technique for the field identification, and survey, of areas impacted by DU. Gamma ray line intensities for typical samples of DU are reported in Moss (1985). The most abundant line being associated with the 1.001 MeV gamma ray from ^{234m}Pa with an absolute abundance of 103.7 photons/second /gram of ^{238}U.

A relatively portable (truck mounted) non-destructive field technique for the measurement of $^{238}U/^{238}U$ in depleted to moderately enriched uranium has been reported by Balagna and Cowan (1977) by comparing the ratio of the number of fission fragments produced during thermal and epithermal/fast fission of samples of uranium ore. Whilst accurate results were obtained, the technique requires a ^{252}Cf source (requiring associated radiological protection measures) and relatively high concentrations of total uranium (Ca. 20% U_3O_8).

Although time-consuming, fission track analysis does have the advantage of high spatial resolution (much less than 1 mm) with some degree of sensitivity (a few mg/kg) for natural uranium. Although the use of this technique for DU may require modification of irradiation conditions due to the relatively low abundance of ^{235}U.

Previous regulations concerning exposure to radioactivity in food and water have centred upon use of total alpha and total beta activity as a preliminary screening tool. However, the use of total alpha activity as a screening tool for DU, at levels close to those present in the natural environment, is severely hindered by the long half-life (and hence low specific activity) of ^{238}U, coupled to the often high levels of matrix elements in many, even ashed, foodstuffs and waters, particularly from arid and semi-arid environments.

A recent report by Haslip et al., (2000) describes a study undertaken to examine the capabilities of commercial radiation detection equipment for the detection of DU on the battlefield. The work involved some spectroscopic studies of DU munitions, and detection trials with a variety of DU sources, from large spheres to low-activity area sources. It was shown that while commercial equipment can detect alpha, beta, and gamma emission by uranium sources, beta detection is by far the preferred method to be used for contamination surveys. For example the sensitivity of the Eberlines ABP-100 alpha-beta probe (in beta mode) for DU is approximately 0.5 Bq/cm^2 where the contamination is over a large area. However, because the attenuation of beta radiation

by tissue is so great, the efficacy of this detector for detecting shards of DU embedded in wounds is much poorer. The report concluded that whilst such devices may be sufficient for detecting DU contamination on vehicles, it is probably insufficient for DU screening of wounds.

Figure A6.1 Typical hand held alpha-beta detector assembly.

A6.3 Other chemical techniques

Analytical techniques based on the complexation and subsequent spectrometric determination of uranium, such as fluorescence spectrometry have been employed (Van Loon and Barefoot, 1989). In particular, these have been used for the determination of uranium in waters and ores, and for the identification of uranium ore deposits in mineral exploration programmes. Unfortunately these techniques do not differentiate between the various isotopes of uranium and therefore cannot be used to infer the presence or absence of DU. Additionally these techniques are often subject to serious interference from the presence of other forms of contamination such as copper, molybdenum and naturally occurring dissolved organic compounds. However, they have been used to some effect to look at complexation mechanisms of uranium and can be used to provide information on chemical speciation.

Glossary

absorbed dose, radiation The energy absorbed per unit mass. The special name is the Gray (Gy = 1 joule per kilogram). The historical unit is the rad (100 ergs/gram). The conversion is 100 rad = 1 Gy.

absorption type (F (fast), M (moderate), and S (slow) clearance) A classification scheme for inhaled material according to its rate of clearance from the pulmonary region of the lungs to the blood. The practical transport rates to the gastrointestinal tract and lymph nodes are the same for all three of the absorption types.

activity The number of nuclear transformations occurring in a given quantity of material per unit time (see becquerel and curie).

activity median aerodynamic diameter (AMAD) The diameter of a unit-density sphere with the same terminal settling velocity in air as that of the aerosol particle whose activity is the median for the entire aerosol.

alpha particle A positively charged particle ejected spontaneously from the nuclei of some radioactive elements. It is identical to a helium nucleus, but of nuclear origin. It comprizes two neutrons and two protons and has a mass number of 4 and an electrostatic charge of +2. On capturing two electrons it forms an atom of helium indistiguishable from any other helium atom.

anamnesis A patient's account of his or her medical history.

anthropogenic Man-made or derived from man’s activities.

aquifer Underground body of water, hosted in permeable rocks such as sandstone or fractured igneous rocks or in unconsolidated sands and gravels.

atom The smallest particle of an element that cannot be divided or broken up by chemical means. It consists of a central core called the *nucleus,* which contains *protons* and *neutrons* and one of more outer shells of *electrons*.

atomic mass (u) The mass of a neutral atom of a nuclide, usually expressed in terms of ‘atomic mass units.’ The ‘atomic mass unit’ is one-twelfth the mass of one neutral atom of carbon-12; equivalent to 1.6604×10^{-24} g.

atomic number The number of protons in the nucleus of a neutral atom of a nuclide (Symbol: Z).

atomic weight The weighted mean of the masses of the neutral atoms of an element expressed in atomic mass units.

background concentration The concentration (or level) of a substance characteristic of a particular medium (e.g. soil, water, rock etc.) in an area or region arising from both natural sources and non-natural diffuse sources such as atmospheric deposition. (after definition for background concentration soil, ISO 11074-1:1996)

background radiation The amount of radiation to which a member of the general population is exposed from natural sources, such as terrestrial radiation from naturally occurring radionuclides in the soil, cosmic radiation originating from outer space, and naturally occurring radionuclides deposited in the human body.

becquerel (Bq) The International System of Units unit of activity and equals that quantity of radioactive material in which one transformation (disintegration) occurs per second (1 Bq = 1 disintegration per second = 2.7×10^{-11} Ci or 27 pCi).

beta particle Charged particle emitted from the nucleus of an atom. A beta particle has a mass and charge equal in magnitude to that of the electron. The charge may be either +1 (for a positron; also generically called a beta particle) or –1 (for a negatron).

biological half-life The time required for a biological system, such as that of a human, to eliminate by natural process half of the amount of a substance (such as a chemical substance or radioactive material) that has entered it.

carcinogen A chemical or substance capable of inducing cancer.

carcinoma Malignant neoplasm composed of epithelial cells, regardless of their derivation.

charged particle An ion or an elementary particle carrying a positive or negative charge.

collective dose The sum of the individual doses received in a given period of time by a specified population from exposure to a specified source of radiation.

committed effective dose E(t) Following an intake into the body of a radioactive material there is a period during which the material gives rise to an effective dose. The committed effective dose is the time interval of the effective dose rate. If the time interval is not specified it is implied that the value is 50 or 70 years, as defined by the regulator or assessor.

committed equivalent dose H_t(t) Following an intake into the body of a radioactive material, there is a period of time during which the material gives rise to an equivalent dose. The committed equivalent dose is the time integral of the equivalent dose rate, and is applied to the year the radioactive material entered the body. If the time interval is not specified it is implied that the value is 50 or 70 years as defined by the regulator or assessor.

complex / complexation A compound / process in which molecules or ions form coordinate bonds to a metal atom or ion.

cosmic rays High-energy particulate and electromagnetic radiation, which originate outside the Earth's atmosphere (e.g. protons, pions, muons, neutrinos etc.).

curie (Ci) A unit of radioactivity. One curie equals that quantity of radioactive material in which there are 3.7×10^{10} nuclear transformations per second (1 Ci = 3.7×10^{10} disintegrations per second = 3.7×10^{10} Bq). The activity of 1 gram of radium is approximately 1 Ci.

decay, radioactive Transformation of the nucleus of an unstable nuclide by spontaneous emission of charged particles and/or photons (see Disintegration).

decay chain or decay series A sequence of radioactive decays (transformations) beginning with one nucleus. The initial nucleus decays into a secondary nucleus 'or progeny nucleus' that differs from the first by whatever particles were emitted during the decay. If further decays take place, the subsequent nuclei are also usually called progeny.

decay product A new isotope formed as a result of radioactive decay. A nuclide resulting from the radioactive transformation of a radionuclide, formed either directly or as the result of successive transformations in a radioactive series. A decay product may be either radioactive or stable.

depleted uranium (DU) Uranium having a percentage of uranium-235 less than the naturally occurring distribution of U-235 found in natural uranium (less than 0.711 weight per cent U-235).

developmental toxicity The occurrence of adverse effects on the developing organism that may result from exposure to a chemical or radiation prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

disintegration constant The fraction of the number of atoms of a radioactive nuclide which decay in unit time; ? (Greek lambda; equal to 0.693/radioactive half-life) is the symbol for the decay constant in the equation $N = N_o e^{-\lambda t}$, where N_o is the initial number of atoms present, and N is the number of atoms present after some time (t) (see Decay Constant).

disintegration, nuclear A spontaneous nuclear transformation (radioactivity) characterized by the emission of energy and/or mass from the nucleus. When large numbers of nuclei are involved, the process is characterized by a definite half-life (see Transformation, Nuclear).

dose assessment An estimate of the radiation dose to an individual or a population group usually by means of predictive modeling techniques, sometimes supplemented by the results of measurement.

dose, effective The equivalent dose (H_t) multiplied by a tissue-weighting factor, w_t, with the special name sievert (Sv). The tissue-weighting factor represents the contribution of the organ or tissue to the total cancer detriment due to the effect resulting from uniform irradiation of the body. $E = (w_r)(w_t)(D)$, the sum of the weighted equivalent doses in all the tissues and organs in the body. It is given by $E = W_t H_t$ where W_t is the weighting factor for tissue T.

dose, equivalent (H_t) The absorbed dose D multiplied by a radiation-weighting factor W_r to account for the different qualities of radiation (alpha, beta, and gamma) in terms of potential effect. The special name is the sievert (Sv). The present weighting factors are as follows: alpha radiation is $w_r = 20$; beta and gamma radiation $w_r = 1$. The dose equivalent expresses all radiation on a common risk scale. (The unit of equivalent dose is the rem. In SI units, the equivalent dose is the sievert, which equals 100 rem.)

dose, pharmacological A general term denoting the quantity (mass) of a substance introduced into the body. For special purposes it must be appropriately qualified.

dose, radiation absorbed The energy imparted to matter by ionizing radiation per unit mass of irradiated material at the place of interest. The unit of absorbed dose is the rad. One rad equals 100 ergs per gram. In SI Units, the absorbed dose is the gray, which is 1 J/kg, so 100 rads = 1 Gray (see rad). Absorbed dose rate is the absorbed dose per unit time.

dose, radiation cumulative The total dose resulting from repeated or continuous exposures to radiation.

dose rate Absorbed dose delivered per unit time.

dosimetry Quantification of radiation doses to individuals or populations resulting from specified exposures.

electron volt A unit of energy equivalent to the energy gained by an electron in passing through a potential difference of one volt. Larger multiple units of the electron volt are frequently used: keV for thousand or kilo electron volts, MeV for million or mega electron volts.

embryotoxicity and fetotoxicity Any toxic effect on the conceptus as a result of prenatal exposure to a chemical or to radiation; the distinguishing feature between the two terms is the stage of development during which the insult occurred. The terms, as used here, include malformations and variations, altered growth, and in utero death.

enrichment, isotopic An isotopic separation process by which the relative abundance of the isotope element of interest is altered, thus producing a form of the element that has been enriched in one or more isotopes and depleted in others. In uranium enrichment, the percentage of uranium-235 in natural uranium may be increased from 0.7% to greater than 90% in a gaseous diffusion, thermal diffusion, centrifugation, mass spectrometric or laser separation process based on the different thermal velocities, mass differences or any other suitable property of the constituents of natural uranium (^{234}U, ^{235}U, ^{238}U).

equilibrium, radioactive In a radioactive series, the state that prevails when the ratios between the activities of two or more successive members of the series remains constant.

exposure (chemical) Contact of an organism with a chemical or physical agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g. skin, lungs, gut) and is available for absorption.

exposure (radiation) Being exposed to ionizing radiation or to a radioactive material. Exposure is quantified as the amount of radioactive material available at the exchange boundaries of the organism (e.g. the gut, skin, lungs) and available for absorption, as well as exposure to penetrating radiation from outside the body.

gamma ray, penetrating Short-wavelength electromagnetic radiation of nuclear origin.

genetic effect of radiation or substance Inheritable change, chiefly mutations, produced by the absorption of ionizing radiation by germ cells.

half-life, radioactive Time required for a radioactive substance to lose 50% of its activity by decay. Each radionuclide has a unique half-life.

harm Harm to health of living organisms or other interfaces with ecological systems of which they form part. In the case of humans some definitions include harm to property. Note that harm is not just a matter of exceeding action or trigger levels, but is that determined by a structured source-pathway-receptor analysis and the effects on a given target (i.e. not just exposure)

immunologic toxicity The occurrence of adverse effects on the immune system that may result from exposure to environmental agents, such as chemicals.

in vitro Isolated from the living organism and artificially maintained, as in a test tube.

in vivo Occurring within the living organism.

ion Atomic particle, charged atom, or chemical radical bearing a net electrical charge, either negative or positive.

ionization The process by which a neutral atom or molecule acquires a positive or negative charge.

ionization path (Track)--The trail of ion pairs produced by ionizing radiation in its passage through matter.

ionizing radiation Any radiation capable of displacing electrons from atoms or molecules, thereby producing ions. Examples: alpha, beta, gamma, X-rays, and neutrons.

isotopes Nuclides having the same number of protons in their nuclei, and hence the same atomic number, but differing in the number of neutrons and therefore in the mass number. Almost identical physical properties exist between isotopes of a particular element. The term should not be used as a synonym for nuclide.

land contamination **T**he presence of a substance or component that is not present naturally that does not necessarily cause harm (ISO 11074-1:1996)

mass numbers (A) The number of nucleons (protons and neutrons) in the nucleus of an atom.

natural background concentration The concentration or level of a substance that is derived solely from natural sources (i.e. of geological origin) after ISO 11074-1:1996.

neutron Elementary nuclear particle with no electric charge.

nuclide A species of atom characterized by the constitution of its nucleus. The nuclear constitution is specified by the number of protons (Z), number of neutrons (N), and energy content, or, alternatively, by the atomic number (Z), mass number A = (N+Z), and atomic mass. To be regarded as a distinct nuclide, the atom must be capable of existing for a measurable time.

power, stopping A measure of the ability of a material to absorb energy from an ionizing particle passing through it; the greater the stopping power, the greater the energy absorbing ability (see Linear Energy Transfer).

progeny The decay product or products resulting after a radioactive decay or a series of radioactive decays. The progeny can be stable but can also be radioactive, and the chain continues until a stable nuclide is formed.

proton Elementary nuclear particle with a positive electric charge

radiation The emission and propagation of energy through space or through a material medium in the form of waves (e.g. the emission and propagation of electromagnetic waves or of sound and elastic waves). The term radiation or radiant energy, when unqualified, usually refers to electromagnetic radiation. Such radiation is commonly classified according to frequency, as microwaves, infrared, visible (light), ultraviolet, X-rays and gamma rays (see Photon) and, by extension, corpuscular emission, such as alpha and beta radiation, neutrons, or rays of mixed or unknown type, such as cosmic radiation.

radiation, background See Background Radiation.

radiation, external Radiation from a source outside the body.

radiation, internal Radiation from a source within the body (as a result of deposition of radionuclides in body tissues).

radiation, ionizing Any electromagnetic or particulate radiation capable of producing ions, directly or indirectly, in its passage through matter (see Radiation).

radioactivity Spontaneous nuclear transformations that result in the formation of new elements. These transformations are accomplished by emission of particles from the nucleus or by the capture of an orbital electron. Each of these reactions may or may not be accompanied by a gamma photon.

radioactive decay constant (l) The fraction of the number of atoms of a radioactive nuclide that decay in unit time (see Disintegration Constant).

radioactivity, natural The property of radioactivity exhibited by more than 50 naturally occurring radionuclides.

radio-isotopes An unstable isotope of an element that decays or disintegrates spontaneously, emitting radiation. Approximately 5000 natural and artificial radio-isotopes have been identified.

radionuclide A radio-isotope or radioactive nuclide characterized by the constitution of its nucleus.

rem (rem) A non-SI unit of equivalent dose. The equivalent dose in rem is numerically equal to the absorbed dose in rad multiplied by the quality factor (radiation-weighting factor) (1 rem = 0.01 sievert).

reproductive toxicity The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical or to radiation. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The

manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

secular equilibrium If the original radionuclide has a very much longer half-life than its progeny (so there is not appreciable change in its amount in the time interval required for later products to attain equilibrium) then, after equilibrium is reached, equal numbers of atoms of all members of the series disintegrate in unit time. This condition is never exactly attained but is essentially established in such a case as radium and its series to ^{210}Pb. The half-life of radium is about 1600 years, of radon, approximately 3.82 days, and of each of the subsequent members, a few minutes. After about a month, essentially the equilibrium amount of radon is present; then (and for a long time) all members of the series disintegrate the same number of atoms per unit time. At this time, the activity of the progeny equals the activity of the original radionuclide.

SI units The International System of Units as defined by the General Conference of Weights and Measures in 1960. These units are generally based on the meter/kilogram/second units, with special quantities for radiation including the becquerel, gray, and sievert.

sievert (Sv) The SI unit of any of the quantities expressed as equivalent or effective dose. The equivalent dose in sieverts is equal to the absorbed dose, in grays, multiplied by the radiation-weighting factor (1 Sv = 100 rem). The effective dose is the equivalent dose multiplied by the tissue-weighting factor.

soil The upper layer of the Earth's crust composed of mineral parts, organic substance, water, air and living matter (ISO 11074-1, 1996).

soil function Soil functions describe the significance of soils to man and the environment (ISO 11074-1, 1996). Important soil functions include: control of substance and energy cycles as compartment of ecosystems; basis for the life of plants, animals and man; carrier of genetic reservoir; basis for the stability of buildings; basis for the production of agricultural products; buffer inhibiting movement of water, contaminants or other agents into groundwater; reservoir of archaeological remains; reservoir of paleoecological remains.

soil pollutant A substance or agent present in the soil which due to its properties, amount or concentration causes adverse impacts on (i.e. harm to) soil functions or soil use (ISO 11074-1, 1996).

sorption Generic terms covering sorbing by physical or chemical processes or both. The term does not specifically differentiate between absorption or adsorption.

specific-activity Radioactivity per gramme of a radionuclide. It may be calculated from the formula ($N_A\lambda$/MW). Where N_A is the number of atoms in one mole of a material (Avogadro's constant, 6.023×10^{23}), 8 ?is the radioactive decay constant and MW is the atomic weight of a given isotope. The specific activity for a substance containing a mixture of isotopes may be obtained using the equation:

Specific activity of mixture = the sum of?$N_A\ \lambda_i$ / $MW_i\ F_i$
Where F_i is the fractional abundance (by weight) of the given isotope (i) in the mixture. Additionally, where an isotope has more than one decay mode the

specific activity for a given mode of decay may be obtained by multiplying the specific activity for all decay modes by the relative fractional abundance of a given decay mode, ?8 is the radioactive decay constant.

speciation A broad generic term describing the various chemical forms an element can take (e.g. the bicarbonate ion HCO_3^- is a species of carbon)

spontaneous fission A relatively rare radioactive process in which fission (splitting of the original radionuclide into two radionuclides of approximately equal mass) occurs spontaneously amongst heavy nuclides such as ^{238}U.

stable isotope A non-radioactive isotope of an element.

stochastic Calculated or modelled according to the laws of probability

teratogen Any chemical or radiation that causes birth defects.

threshold limit value (TLV) The maximum concentration of a substance to which most workers can be exposed without adverse effect. TLV is a term used exclusively by the American Conference of Governmental Industrial Hygienists (ACGIH). Other terms used to express the same concept are the MAC (maximum allowable concentration) and the OSHA equivalent PEL (permissible exposure limits).

tolerable intake An estimate of the daily intake of a substance which can occur over a lifetime without appreciable health risk**.**

transformation, nuclear The process by which a nuclide is transformed into a different nuclide by absorbing or emitting a particle.

X-rays Penetrating electromagnetic radiations whose wave lengths are very much shorter than those of visible light. They are usually produced by bombarding a metallic target with fast electrons in a high vacuum. X-rays (called characteristic X-rays) are also produced when an orbital electron falls from a high energy level to a low energy level.

Bibliography

Abdelouas A., Lutze W., and Nuttall E. (1998) Chemical reactions of uranium in groundwater at a mill tailings site. *Journal of Contaminant Hydrology* 34(4), 343–361.

ACGIH (1960) American Conference of Government Industrial Hygienists. Threshold limit values for 1960. *Archives Environment and Health* 1,140–144

ACGIH (1968) American Conference of Government Industrial Hygienists. Threshold limit values in airborne contamination for 1968, recommended and intended values. St Louis, Missouri, ACGIH.

ACGIH (1993) American Conference of Government Industrial Hygienists. Threshold limit values for chemical substances and physical agents and biological exposure indices, Cincinnati, Ohio, ACGIH.

ACGIH (1996) American Conference of Government Industrial Hygienists. Threshold limit values for chemical substances and physical agents and biological exposure indices, Cincinnati, Ohio, ACGIH.

ACGIH (2000) American Conference of Government Industrial Hygienists. Threshold limiting values for chemical substances and physical agents and biological exposure indices, *Technical Affairs Office ACGIH*, Cincinnati, Ohio, USA.

AEPI (1995) Health and environmental consequences of DU use in the US army. *US Army Environmental Policy Institute*, Technical Report.

Akcay H. (1998) Aqueous speciation and pH effect on the sorption behaviour of uranium by montmorillonite. *Journal of Radioanalytical and Nuclear Chemistry* 237(1–2), 133–137.

Alloway B. J. (1995) Heavy metals in soil. *Blackie Academic and Professional*, Second Edition, Glasgow, UK.

ANS (1996) Bioassay programs for uranium. Report BPS N.13.22, 1995. McLean VA: *Health Physics* Society: ANS.

ANSI (1995) Bioassay programs for uranium, *American National Standards Institute*, ANSI/HPSN13.22-1995.

Ansoborlo E., Guilmette R. A., Hoover M. D., Chazel V., Houpert P., and Hengé–Napoli M. H. (1998) Application of in vitro dissolution tests to different uranium compounds and comparison with in vivo data. *Radiation Protection Dosimetry*, 79, 33–37.

Ansoborlo E., Chazal V., Bailey M., and Stradling N. (2001) Review of the physico–chemical properties, biokinetics and dose coefficients of uranium compounds handled during nuclear fuel fabrication in France. *Health Physics*. (in press)

Archer V., Wagoner J., and Lundin F. (1973a). Lung cancer among uranium miners in the United States. *Health Physics* 25, 351–371.

Archer V., Wagoner J., and Lundin F. J. (1973b) Cancer mortality among uranium mill workers. *Journal Occupational Medicine (United States)* 15, 11–14.

ATSDR (1990) Toxicological profile for uranium. Agency for Toxic Substances and Disease Registry, Report TP-90–29, Atlanta, USA.

ATSDR (1999) Toxicological profile for uranium (an update). Agency for Toxic Substances and Disease Registry, Atlanta, USA, September 1999.

Bailey M. R. (1993) New ICRP human respiratory tract model. *Radiological Protection Bulletin*. 144, 22–29.

Bailey M. R. (1994) The new ICRP model for the respiratory tract. *Radiation Protection Dosimetry* 53, 107–114.

Banks D., Burke S. P., and Gray C. G. (1997) Hydrochemistry of coal mine drainage and other ferruginous waters in north Derbyshire and south Yorkshire, UK. *Quaterly Journal of Engineering Geology* 30, 257–280.

Banks D., Reimann C., Royset O., and Skarphagen H. (1995) Natural concentrations of major and trace elements in some Norwegian bedrock groundwaters. *Applied Geochemistry* 10, 1–16.

Balagna, J. P., and Cowan, G. A. (1977) A non–destructive field measurement of the ratio of $^{235}U/^{238}U$ in depleted to moderately enriched uranium, *IAEA Report* TC/119/34, Vienna.

Barltrop D. (1966) The Prevalence of Pica. *American Journal of Diseases of Children* 112, 116–123.

Basham I. R., Milodowski A. E., Hyslop E. K., and Pearce J. M. (1989) The location of uranium in source rocks and sites of secondary deposition at the Needles Eye natural analogue site, Dumfries and Galloway. *British Geological Survey*, Technical Series Report, WE/89/13.

Bhattacharyya M. H., Breitenstein, B. D., Métivier, H., Muggenburg, B. A., Stradling, G. N., and Volf, V. (1992) Guidebook for the Treatment of Accidental Internal Radionuclide Contamination of workers, Eds GB Gerber and RG-Thomas, *Radiation Protection Dosimetry*, Vol 41, No 1, 1992, EUR Report, 14320 EN.

Batson V. L., Bertsch P. M., and Herbert B. E. (1996) Transport of anthropogenic uranium from sediments to surface waters during episodic storm events. *Journal of Environmental Quality* 25(5), 1129–1137.

Bauer H. D. (1997) Potential dust exposures in underground mines of the former Wismut Ltd. during the early phase of uranium mining after the second world war. *Gefahrstoffe Reinhaltung Der Luft* 57(9), 349–354.

BBC News. (2000) Search for 747 uranium. In *BBC News Online*, www.bbc.co.uk.

Becker N. M., and Vanta E. B. (1995) Hydrologic transport of DU associated with open air dynamic range testing at Los Alamos National Laboratory, New Mexico, and Elgin Air Force Base, Florida. *Los Alamos National Laboratory*, Report LA–UR–95–1213.

Benes P., Kratzer K., Vlckova S., and Sebestova E. (1998) Adsorption of uranium on clay and the effect of humic substances. *Radiochimica Acta* 82, 367–373.

Benson K. A., and Schneider L. M. (1998) Female rats implanted with DU pellets fail to exhibit signs of nephrotoxicity. *FASEB Journal* 12(5), 767.

Bentley K. W., Stockwell D. R., Britt K. A., and Kerr C. B. (1985) Transient proteinuria and aminoaciduria in rodents following uranium intoxication. *Bulletin of Environmental Contamination and Toxicology* 34, 407–416.

Bernhard G., Geipel G., Brendler V., and Nitsche H. (1998) Uranium speciation in waters of different uranium mining areas. *Journal of Alloys and Compounds* 271, 201–205.

Bertelli L., Puerta A., Wrenn M. E., Lipsztein J. L., Moody J. C., Stradling G. N., Hodgson A., and Fell T. P. (1998) Specific absorption parameters for uranium octoxide and dioxide: comparison between biokinetics of uranium in workers derived from human data and those predicted from animal studies. *Radiation Protection Dosimetry* 79(1–4), 87–90.

Bhat R. K. (2000) Review of transuranics in depleted uranium armours. Tank-automotive and armaments command and army material command, Project officer Bhat, R. K, Official communication (and associated annex documentation) to Commander US Tank and Automotive Command (AMSTA-CM-PS) Warren, MI 48397, January 19th, 2000.

Bicknell J. (1975) Pica a childhood symptom. *Butterworths*, London, UK.

Boback, M. W. (1995) A review of uranium excretion and clinical urinalysis data in accidental exposure cases. In Conference Proceedings on *Occupational Health Experience with Uranium*, Arlington,Virginia, 28–30 April 1995. Document ERDA 93. Springfield Virginia: National Technical Information Service.

Bomben A. M., Equillor H. E., and Oliveira A. A. (1996) Ra–226 and natural uranium in Argentinean bottled mineral waters. *Radiation Protection Dosimetry* 67(3), 221–224.

Bou–Rabee (1995) Estimating the concentration of Uranium in some environmental samples in Kuwait after the 1991 Gulf War. *Applied Radiation Isotopes*, 46, 217–220.

Brajnik D., Krizman M., Kobal I., and Stegnar P. (1988) Sources of technologically enhanced natural radioactivity and their impact in Slovenia (Yugoslavia). *Radiation Protection Dosimetry*, Vol 24, 551–554.

British Geological Survey. (1992) Regional geochemistry of the Lake District and adjacent areas. *British Geological Survey*, Keyworth, Nottingham, UK.

British Geological Survey. (1997) Regional geochemistry of part of North–West England and North–Wales. *British Geological Survey*, Keyworth, Nottingham, UK.

British Geological Survey. (2000) World mineral statistics 1994–98: production: exports: imports. *British Geological Survey*, Keyworth, Nottingham, UK.

Brookins D. G. (1988) *Eh–pH diagrams for geochemistry*. Springer–Verlag, Berlin, Germany.

Brown D. P., and Bloom T. (1987) Mortality among uranium enrichment workers, *Report to National Institute for Occupational Safety and Health*, Cincinnati, Ohio. NTIS PB87–188991.

BSS (1996) Basic Safety Standards. International basic safety standards for protection against ionizing radiation and for the safety of radiation sources. *IAEA, Basic Safety Standard* 115, STI/Pub/996, IAEA, Vienna, 353 pp.

Burns P. C., and Finch R. (1999) Uranium: mineralogy, geochemistry and the environment. *Mineralogical Society of America*, 38, Washington DC.

Cachoir C., Gallien J. P., and Trocellier P. (1996) Chemical durability of uranium dioxide stored in geologic medium: Uranium remobilisation. *Annales De Chimie–Science Des Materiaux* 21(8), 567–592.

Calabrese E. J. (1989) How much soil do young children ingest: an epidemiological study. *Regulatory Toxicology and Pharmacology* 10, 113–123.

Calabrese E. J., Stanek E. J., and Gilbert C. E. (1990) Adult soil ingestion estimates. in *Petroleum contaminated soils 3, School of public health, Environmental Health Sciences Programme* (editors: P. T. Kostecke and E. J. Calabrese), 349–356. Lewis, Amherst, USA.

Calabrese E. J., Stanek E. J., and Gilbert C. E. (1991) Evidence of soil pica behaviour and quantification of soil ingested. *Human and Experimental Toxicology* 10, 245–249.

Cardis E., and Richardson D. (2000) Invited editorial: Health effects of radiation exposure at uranium processing facilities. *Journal of Radiological Protection* 20(2),95-7.

CFR 20. (1991) Standards for protection against radiation, appendix B. Code of Federal Regulations (USA), 10 part 20.

Chandler S. Z., Ibrahim, S. A., and Campbell, J. G. (2000) Comparison of scintillation detection efficiencies of depleted uranium (DU) in wounds. *Journal of Radioanalytical and Nuclear Chemistry*, 243 (2), 451–457.

Checkoway H., Pearce N., Crawford–Brown D. J., and Cragle D. L. (1988) Radiation doses and cause–specific mortality among workers at a nuclear materials fabrication plant. *American Journal of Epidemiology* 127(2), 255-266.

Cheng Y. L., Lin J. Y., and Hao X. H. (1993) Trace uranium determination in beverages and mineral water using fission track techniques. *Nuclear tracks and radiation measurement* 22(1–4), 853–855.

CHPPM (2000) Depleted Uranium, Human exposure assessment and health risk characterisation. Health Risk Assessment Consultation No. 26–MF–7555–00D, *Centre for Health Promotion and Preventative Medicine* Aberdeen, Md, USA.

Chu A., and Chu G. (1975) Oriental cloisonne and other enamels: a guide to collecting and repairing. *Crown Publishers, Inc.*, New York, USA.

Clulow F. V., Dave N. K., Lim T. P., and Avadhanula R. (1998) Radionuclides (lead–210, polonium–210, thorium–230, and thorium–232) and thorium and uranium in water, sediments, and fish from lakes near the city of Elliot Lake, Ontario, Canada. *Environmental Pollution* 99(2), 199–213.

Conrad J. W. (1973) Ceramic formulas: the complete compendium. *MacMillian Publishing Company.* New York, USA.

Cooper M. (1957) *Pica*. Thomas, Springfield, Illinois, USA.

Cooper, J. R., Stradling, G. N., Snith, H., and Ham, S. E. (1982) The behaviour of U-233 oxide and uranyl-233 nitrate in rats. *International Journal of Radiation Biology*, 41, 421-433.

Cross F., Filipy R., and Loscutoff S. (1981a) Histopathologic, morphometric and physiologic investigation of lungs of dogs exposed to uranium ore dust. *International Conference: Hazards Mineral Control. Measures and Medical Aspects*, 228–235.

Cross F., Palmer R., and Busch R. (1981b) Development of lesions in Syrian golden hamsters following exposure to radon daughters and uranium ore dust. *Health Physics* 41, 135–153.

Danesi M. E. (1990) Kinetic energy penetrator long–term strategy study. *US Army Armament, Munitions, and Chemical Command*, Picatinny Arsenal, NJ, USA.

Dang H. S., Pullat V. R., and Pillai K. C. (1992) Determining the normal concentration of uranium in urine and applications of the data to biokinetics. *Health Physics* 62, 562–566.

Darby S. C., Whitley E., and Howe G. R. (1995) Radon and cancers other than lung cancer in underground miners: a collaborative analysis of 11 studies. *Journal of the National Cancer Institute* 87, 378–384.

Davies S. (1990) Quantitative estimates of soil ingestion in normal children, between the ages of 2 and 7 years: Population–based estimates using aluminium, silicon and titanium as soil tracer elements. *Archives of Environmental Health* 45, 112–122.

De Carvalho H. H., Martins J. B., Medeiros E. L., and Tavares O. A. P. (1982) Decay constant for the spontaneous fission process in uranium–238. *Nuclear Instruments and Methods* 197, 417–426.

De Vivo B., Ippolito F., Capaldi G., and Simpson P. R. (1984) Uranium geochemistry, mineralogy, geology, exploration and resources. *Institution of Mining and Metallurgy*, London, UK.

deCamargo I. M. C.. and Mazzilli B. (1996) Determination of uranium and thorium isotopes in mineral spring waters. *Journal of Radioanalytical and Nuclear Chemistry–Letters* 212(4), 251–258.

DEP (1968) Department of Employment and Productivity. HM Factory Inspectorate. Dust and Fumes in Factory Atmospheres 1968. New Series No. 8. London: HMSO.

DEP (1969) Department of Employment and Productivity. HM Factory Inspectorate Dust and Fumes in Factory Atmospheres 1968. New Series No. 8. London: HMSO: DEP (1969).

DeRey B. M., Lanfranchi H. E., and Cabrini R. L. (1983) Percutaneous absorption of uranium compounds. *Environmental Research* 30, 480–491.

DeRey B. M., Lanfranchi H. E., and Cabrini R. L. (1984) Deposition pattern and toxicity of sub–cutaneously implanted uranium dioxide in rats. *Health Physics* 46, 688–692.

Diamond, G. L. (1998) Biological consequences of exposure to soluble forms of natural uranium. *Radiation Protection Dosimetry* 26, 23–33.

Dipino R. K., Kabat M. H., and Kane R. L. (1998) A comparison of 5 measures of premorbid intellectual functioning in a sample of patients injured with DU. *Archives of Clinical Neuropsychology* 13(1), 23–24.

Dominico P. A., and Swartz F. W. (1990) Physical and Chemical Hydrogeology, John Wiley and Sons, Singapore.

Domingo J. (1989a) The developmental toxicity of uranium in mice. *Toxicology* 55(1–2), 143–152.

Domingo J. (1989b) Evaluation of the perinatal and postnatal effects of uranium in mice upon oral administration. *Archives of Environmental Health* 44(6), 395–398.

Domingo J., Llobet J., and Thomas J. (1987) Acute toxicity of uranium in rats and mice. *Bulletin for Environmental Contamination and Toxicology* 39, 168–174.

Duff M. C., and Amrhein C. (1996) Uranium(VI) adsorption on goethite and soil in carbonate solutions. *Soil Science Society of America Journal* 60(5), 1393–1400.

Dupree E.A., Cragle D.L., McLain R.W., Crawford-Brown D.J., and Teta M.J. (1987) Mortality among workers at a uranium processing facility, the Linde Air Products Company Ceramics Plant, 1943-49. *Scandinavian Journal of Work Environment and Health* 13,100-107

Dupree E.A., Watkins J.P., Ingle J.N., Wallace P.W., West C.M., and Tankersley W.G. (1995) Uranium dust exposure and lung cancer risk in four uranium processing operations. *Epidemiology* 6(4), 370-275.

Dupree–Ellis E., Watkins J., Ingle J. N., and Phillips J. (2000) External radiation exposure and mortality in a cohort of uranium processing workers. *American Journal of Epidemiology* 125, 91–95.

Durakovic` A. (1999) Medical effects of internal contamination with uranium. *Croatian Medical Journal* 40(1), 49–66.

Durbin P. W. (1984) Metabolic models of uranium. *Biokinetics of Uranium in Man*, Richland, Washington (1984), NTIS, Springfield, Va, USA. Report USUR–05, HEHF–47.

Dushenkov S., Vasudev D., Kapulnik Y., Gleba D., Fleisher D., Ting K.C., and Ensley B. (1997) Removal of uranium from water using terrestrial plants. *Environmental Science and Technology* 31(12), 3468–3474.

Dygert H. (1949) *Pharmacology and toxicology of uranium compounds.* McGraw–Hill Book Inc., New York, 603–675.

Ebbs S. D., Norvell W.A., and Kochian L.V. (1998) The effect of acidification and chelating agents on the solubilisation of uranium from contaminated soil. *Journal of Environmental Quality* 27(6), 1486–1494.

Ebinger M. H. (1998) Depleted uranium risk assessment for Jefferson proving ground: Updated risk estimates for human health and ecosystem receptors. *Los Alamos National Laboratory*, Report LA–UR–98–5053.

Ebinger M. H., Essington E. H., Gladney E. S., Newman B. D., and Reynolds C. L. (1990) Long–term fate of DU at Aberdeen and Yuma proving grounds. Final Report, Phase I: Geochemical transport and modelling. *Los Alamos National Laboratory*, Report LA–11790–MS.

Ebinger M. H., and Hansen W. R. (1994) Depleted uranium human health risk assessment, Jefferson proving ground, Indiana. *Los Alamos National Laboratory*, LA–UR–94–1809.

Ebinger M. H., Kennedy P. L., Myers O. B., Clements W., Bestgen H. T., and Beckman R. J. (1996) Long–term fate of DU at Aberdeen and Yuma proving grounds, Phase II: Human health and ecological risk assessment. *Los Alamos National Laboratory*, Report LA–13156–MS.

Ebinger M. H., and Oxenburg T. P. (1997) Modeling exposure to DU in support of decommissioning at Jefferson Proving Ground, Indiana, *National Technical Information Service*, Report LA–UR–96–3907.

EC (1996) *Council Directive* 96/29/EURATOM of 13 May 1996, Laying Down the Basic Safety Standards for the Protection of the Health of Workers and the General Public Against the Dangers Arising from Ionizing Radiation.

Edison A. F. (1994) The effect of solubility on inhaled uranium compound clearance: a review. *Health Physics* 67, 1-14.

Edmunds W. M., Cook J. M., Kinniburgh D. G., Miles D. L., and Trafford J. M. (1989) Trace–element occurrence in British groundwaters, *British Geological Survey*, Report SD/89/3 Keyworth, Nottingham, UK. 424.

Eisenbud M., and Quigley J. A. (1956) Industrial hygiene of uranium processing. *AMA Arch Indust Health* 12, 12–22.

Ejnik J. W., Carmichael A. J., Hamilton M. M., McDiarmid M., Squibb K., Boyd P., and Tardiff W. (2000) Determination of the isotopic composition of uranium in urine by inductively coupled plasma mass spectrometry. *Health Physics* 78, 143–146.

Elder J. C., and Tinkle M. C. (1980) Oxidation of DU penetrators and aerosol dispersal at high temperatures. *Los Alamos Scientific Laboratory*, Report LA–8610–MS.

Elless M. P., and Lee S. Y. (1998) Uranium solubility of carbonate–rich uranium–contaminated soils. *Water Air and Soil Pollution* 107(1–4), 147–162.

Elless M. P., Timpson M. E., and Lee S. Y. (1997) Concentration of uranium particulates from soils using a novel density–separation technique. *Soil Science Society of America Journal* 61(2), 626–631.

Elless M. P., Armstrong A. Q., and Lee S. Y. (1997) Characterisation and solubility measurements of uranium contaminated soils to support risk assessment. *Health Physics*, 72(5), 716–726.

Erickson R. L. (1990) A review of the environmental behavior of uranium derived from DU alloy penetrators. *Pacific North West Laboratory*, Report PNL–7213, Richland, Washington, USA.

Faure, G. (1986) *Principals of Isotope Geology*, Second Edition, John Wiley and Sons, Canada, 589 pp.

FEMP (1997) Fernald Environment Management Project. Technical basis for internal dosimetry at the Fernald environmental management project, revision 3, *FEMP*, USA.

Ferguson C., Darmendrail D., Frier K., Jensen B. K., Jensen J., Kasamas H., Urzelai A., and Vegter J. (1998) Risk Assessment for Contaminated Sites in Europe. *LQM Press*, Nottingham, UK.

Fetter S., and von Hippel F. J. (1999) The hazard posed by DU munitions, *Science and Global Security* 8(2), 125-161.

Fisenne I. M., Perry P. M., Decker K. M., and Keller H. K. (1987) The Daily Intake of 234,235,238U, 228,230,232Th, and 226,228Ra by New York City Residents, *Health Physics* 53, 357–363.

Fisenne, I. M. (1993) Long lived radionuclides in the environment, in food, and in humans, *Fifth International Symposium on the Natural Radiation Environment, tutorial sessions*, Report EUR 14411, EN, Commission of the European Communities, ISSN 1018–5593, 1993.

Fisher D. R., Kathern R. L., and Swint M. J. (1991) Modified biokinetic model for uranium from analysis of acute exposure to UF_6. *Health Physics* 60, 335–342.

Fisher D.R., Swint M. J., and Kathren R.L. (1990) Evaluation of health effects in equoyah Fuels corporation workers from accidental exposure to uranium hexafluoride, U.S. Nuclear Regulatory commission, Washington D.C. NUREG/CR–5566 PNL–7328.

FRA (1988) Cahier de notes documentaires, Les valeurs limites d'exposition professionnelle aux substances dangereuses en France.

Franzen D., Sackman A., Oale R., and Chopin M. (1988) Analytical results report for ambient air and residential characterisation at Prospect Square, Park City, Utah. Report prepared for US EPA Hazardous Site Evaluation Division, Washington, USA.

Fulco C. E., Liverman C. T., and Sox H. C. (editors) (2000) *Gulf War and Health*. Vol. 1. Depleted uranium, sarin, pyridostigmine bromide, vaccines. National Academy Press, Washington DC. http://books.nap.edu/catalog/9953.html.

Gallacher T. D. (1994) Radiation safety officer, senior manager, corporate radiation health protection, The Boeing Company. Letter to P.A. Schofield, Oak ridge National Laboratory. 'Boeing use of DU counterweights in aircraft' September, 1994.

Gallien J. P., Trocellier P., and Toulhoat P. (1996) Leaching of uranium dioxide under controlled redox conditions. *Journal of Trace and Microprobe Techniques* 14(2), 343–352.

Gedeon R., Smith B., Amro H., Jawadeh J., and S. K. (1994) Natural radioisotopes in groundwaters from the Amman–Zarka basin Jordan. Hydrochemical and regulatory implications. *Application of Tracers in Arid Zone Hydrology.* IAHS Publication 232.

Geissler P. W., Mwaniki D. L., Thiongo F., and Friis H. (1997) Geophagy among school children in Western Kenya. *Tropical Medicine and International Health* 2(7), 624–630.

Geissler P. W., Mwaniki D. L., Thiongo F., Michaelsen K. F., and Friis H. (1998) Geophagy, iron status and anaemia among primary school children in Western Kenya. *Tropical Medicine and International Health* 3(7), 529–534.

Gibson R. (1994) Zinc nutrition in developing countries. *Nutrition Research Reviews* 7, 151–173.

Gilbert E.S., Cragle D.L., and Wiggs L.D. (1993) Updated analyses of combined mortality data for workers at the Hanford site, Oak Ridge National Laboratory and Rocky Flats Weapons Plant. *Radiation Research* 136, 408-421

Gill R. (1997) Modern Analytical Geochemistry. *Addison Wesley Longman Ltd*, London, UK.

Gilman A. P., Villeneuve D. C., Securs V. E., Yagminas A. P., Tracy B. L., Quinn J. M., Valli V. E., Wiles R. J., and Moss M. A. (1998a) Uranyl nitrate: 28–day and 91–day toxicity studies in the Sprague–Dawley rat. *Toxicological Science* 41(1), 117–128.

Gilman A. P., Villeneuve D. C., Securs V. E., Yagminas A. P., Tracy B. L., Quinn J. M., Valli V. E., Wiles R. J., and Moss M. A. (1998b) Uranyl nitrate: 91–day toxicity studies in the New Zealand white rabbit. *Toxicological Science* 41(1), 129–137.

Gilman A.P., and Moss M. A., Villeneuve D. C., Secours V. E., Yagminas A. P., Tracy B. L., Quinn J. M ., Long G., Valli V. E. (1998c) Uranyl nitrate: 91–day exposure and recovery studies in the male New Zealand white rabbit. *Toxicological Science* 41(1), 138–51.

Golden M. H. and Golden G. (1981) Trace elements. *British Medical Bulletin* 37, 31–36.

Goodman D.R. (1985) Nephrotoxicity. Toxic effects in the kidneys. In: *Industrial toxicology, safety and health application in the work place*. Williams P.L and Burson J.L., editors. Van Nostrand Reinhold, New York, N.Y.

Gough M. (1991) Human exposures from dioxin in soil–a meeting report. *Journal of Toxicology and Environmental Health* 32, 205–245.

Guibal E., Roussy J., and LeCloirec P. (1996) Photo–chemical reaction of uranium with glucosamine, acetylglucosamine and related polymers: Chitin and chitosan. *Water Sa* 22(1), 19–26.

Hadjimichael O.C., Ostfeld A.M., D'Atri D.A., and Brubaker R.E. (1983) Mortality and cancer incidence experience of employees in a nuclear fuels fabrication plant. *Journal of Occupational Medicine* 25, 48-61.

Halliday A. N., Lee D. C., Christensen J. N., Rehkamper M., Yi W., Luo X. Z., Hall C. M., Ballentine C. J., Pettke T., and Stirling C. (1998) Applications of multiple collector–ICPMS to cosmochemistry, geochemistry, and paleoceanography. *Geochim. Cosmochim Act 62*, 919–940.

Hamilton E. I. (1972) The concentration of uranium in man and his diet. *Health Physics* 22, 149–153.

Harduin J. C., Royer P. H., and Piechowsky J. (1994) Uptake and urinary excretion of uranium after oral administration in man. *Radiation Protection and Dosimetry* 53, 245–248.

Harley J. H. (1988) Naturally occurring sources of radioactive contamination. Radionuclides in the food chain. In *International life sciences institute monographs* (ed. M. W. Carter), pp. 58–71. Springer–Verlag, Heidelberg, Germany.

Harley N., Foulkes E., Hilborne L., Hudson A., and Anthony C. (1999a) A review of the scientific literature as it pertains to Gulf war illnesses. RAND, *National Defence Research Institute*, Washington, USA.

Harley N. H., Foulkes E. C., Hilbourne L. H., Hudson A., and Anthony C. R. (1999b) A review of the scientific literature as it pertains to Gulf War illnesses. Volume 7: Depleted uranium. RAND, *National Defence Research Institute*, Washington, USA.

Harwell (2001) Harwell Scientifics Ltd., Harwell, Oxon. UK. (2001). Personal Communication.

Haschke J. M. (1998) Corrosion of uranium in air and water vapour: consequences for environmental dispersal. *Journal of Alloys and Compounds* 278(1–2), 149–160.

Haslip D. S., Cousins, T., Estan, D., and Jones, T. (2000) Field Detection of Depleted Uranium Final Report of Tasking W28476KR00Z (DSSPM), *Defence Research Establishment, Ottawa (Ontario),* Report No: DREO TM-2000-049. (NTIS order no: ADA384540INZ).

Hengé–Napoli M. H, Stradling G. N and Taylor D. M (2000) Decorporation of radionuclides from the human body, (editors: M. H. Henge– Napoli, G. N. Stradling and D. M. Taylor), *Radiation Protection Dosimetry* (Special Issue) Vol. 87, No 1, 2000, EUR Report 19330).

Herranz M., Abelairas A., and Legarda F. (1997) Uranium contents and associated effective doses in drinking water from Biscay (Spain). *Applied Radiation and Isotopes* 48(6), 857–861.

Herranz M., Abelairas A., and Legarda F. (1999) Uranium contents in raw waters from Biscay (Spain). *Applied Radiation and Isotopes* 51(2), 203–208.

Hodge H. C., Stannard J. N., and Hursh J. B. (1973) *Handbook of Experimental Pharmacology.* Springer–Verlag, Berlin, Germany.

Hodgson A., Moody J. C., Stradling G. N., Bailey M. R., and Birchall A. (2000) Application of the ICRP Respiratory Tract Model to uranium compounds produced during the manufacture of nuclear fuel. Report M–1156. *Chilton: National Radiological Protection Board*.

Hooker P. J., Ivanovich M., Milodowski A. E., Ball T. K., Dawes A., and Read D. (1989) Uranium migration at the South Terras mine, Cornwall, *British Geological Survey*, Technical Report, WE/89/13, Keyworth, Nottingham, UK.

Hooper F. J., Squibb K. S., Siegel E. L., McPhaul K., and Keogh J. P. (1999) Elevated urine uranium excretion by soldiers with retained uranium shrapnel. *Health Physics* 77(5), 512–519.

HSE (1980) Health and Safety Executive. Threshold Limit Values 1980. Guidance Note 15/80. London :HMSO.

HSE (1997) Health and Safety Executive. Occupational Exposure Limits 1996. Document EH 40/97.London: HMSO.

HSE (2000) Health and Safety Executive. *EH40/2000 Occupational Health Exposure Limits 2000.* Sudbury, Suffolk: HSE Books.

Hurtgen C. (2001) *Journal of Radioanalytical and Nuclear Chemistry,* Vol 248 (in press).

IAEA (1982) International Atomic Energy Agency Generic models and parameters for assessing the environmental transfer of radionuclides from routine releases. *IAEA Safety Series,* 57, IAEA Vienna.

IAEA (1989a) International Atomic Energy Agency. The application of the principles for limiting releases of radioactive effluents in the case of the mining and milling of radioactive ores. *IAEA Safety Series,* 90, IAEA, Vienna.

IAEA (1989b) International Atomic Energy Agency. Measurement of radionuclides in food and the environment: A guidebook. *IAEA Technical Reports Series,* 295. IAEA, Vienna.

IAEA (1994) International Atomic Energy Agency. Handbook of parameter values for the prediction of radionuclide transfer in temperate environments. *IAEA Technical Reports Series* 364, IAEA, Vienna.

IARC (1988) International Agency for Research on Cancer. *IARC Monographs on the evaluation of carcinogenic risks to humans,* Vol. 43. Man–made mineral fibres and radon., IARC, Lyons, France.

IARC (2001) International Agency for Research on Cancer *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans,* Vol.78. Some Internally Deposited Radionuclides., IARC, Lyon, France (in press).

ICRP-2 (1959) International Commission on Radiological Protection. Report of Committee II on permissible dose for internal radiation. *ICRP Publication 2*. Oxford: Pergamon Press.

ICRP-6 (1964) Recommendations of the International Commission on Radiological Protection. *ICRP Publication 6.* Oxford: Pergamon Press.

ICRP-10 (1968) International Commission on Radiological Protection. Report of committee IV on evaluation of radiation doses to body tissues from internal contamination due to occupational exposure. *ICRP Publication 10*. Oxford: Pergamon Press.

ICRP-23 (1975) International Commission on Radiological Protection. Report of the task group on reference man. *ICRP Publication 23*. Oxford: Pergamon Press.

ICRP-26 (1977) Recommendations of the International Commission on Radiological Protection. ICRP Publication 26, *Annals of the ICRP,* Vol 1(3).

ICRP-29 (1978) International Commission on Radiological Protection. Radionuclide releases into the environment: assessment of doses to man. ICRP Publication 29, *Annals of the ICRP*, Vol 2(1).

ICRP-30 (1979) International Commission on Radiological Protection. Limits for intakes of radionuclides by workers. ICRP Publication 30, *Annals of the ICRP*, Vol 4(3–4).

ICRP-54 (1988) International Commission on Radiological Protection. Individual Monitoring for Intakes of Radionuclides by Workers: Design and Interpretation. ICRP Publication 54, *Annals of the ICRP 19* (1-3).

ICRP-59 (1991a) International Commission on Radiological Protection. The Biological Basis for Dose Limitation in the Skin, ICRP Publication 59, *Annals of the ICRP 20* (1).

ICRP-60 (1991b) International Commission on Radiological Protection. Recommendations of the International Commission on Radiological Protection. ICRP Publication 60, *Annals of the ICRP*, Vol 21(1–3).

ICRP-67 (1993) International Commission on Radiological Protection. Age–dependent doses to members of the public from intake of radionuclides, ICRP Publication 67, *Annals of the ICRP*, Part 2. 23(3/4), 1–167

ICRP-66 (1994a) International Commission on Radiological Protection. Human respiratory tract model for radiological protection, ICRP Publication 66, *Annals of the ICRP*, Vol24(1–3).

ICRP-68 (1994b) International Commission on Radiological Protection. Dose Coefficients for Intakes of Radionuclides by Workers, ICRP Publication 68, *Annals of the ICRP* 24(4).

ICRP-69 (1995a) International Commission on Radiological Protection. Age–dependent Doses to Members of the Public from Intake of Radionuclides: Part 3 Ingestion Dose Coefficients, ICRP Publication 69, *Annals of the ICRP*, Vol. 25(1).

ICRP-71 (1995b) International Commission on Radiological Protection. Age–dependent Doses to Members of the Public from Intake of Radionuclides: Part 4 Inhalation Dose Coefficients, ICRP Publication 71, *Annals of the ICRP*, Vol 25(3–4).

ICRP-72 (1996) International Commission on Radiological Protection. Age dependent doses to the members of the public from intake of radionuclides: Part 5. ICRP Publication 72, *Annals of the ICRP*, Vol 25(2).

ICRP-78 (1997) International Commission on Radiological Protection. Individual monitoring for internal exposure of workers: Replacement of ICRP Publication 54, ICRP Publication 78. *Annals of the ICRP*, 27 (3-4).

ICRP-82 (1999) International Commission on Radiological Protection. Protection of the public in situations of prolonged radiation exposure, ICRP Publication 82, *Annals of the ICRP*, Vol 29(1–2).

ICRP (2001) International Commission on Radiological Protection. Guide for the practical application of the ICRP human respiratory tract model (in press).

Ivanovich M., and Harmon R. S. (1982) *Uranium–series disequilibrium: applications to environmental problems.* Oxford University Press, New York, USA.

Jacob P., Prohl G., Schneider K., and Vob J. U. (1997) Machbarkeitsstudie zur verknupfung der bewertung radiologischer und chemisch–toxischer wirkungen von altasten, pp. 145. Umweltbundesampt.

Jackson S. (1966) Creatinine in urine as an index of urinary excretion rate. *Health Physics* 12, 843-850.

Jain G. S., and Aery N. C. (1997) Effect of uranium additions on certain biochemical constituents and uranium accumulation in wheat. *Biologia* 52(4), 599–604.

Jette S. J. (1990) Aerosolization of the M29A1 and XM900E1 rounds fired against hard targets. *Pacific Northwest Laboratory*, Report PNL–7452.

Kadunas V., Budavicius R., Gregorauskiene V., Katinas V., Kliaugiene E., Radzevicius A., and TaraÓkevicius R. (1999) *Geochemical Atlas of Lithuania.* Geologijos Institutas, Vilnius, Lithuania.

Kahlos H., and Asikainen M. (1980) Internal radiation doses from radioactivity of drinking water in Finland. *Health Physics* 39, 108–111.

Kathren R. L., McInroy J. F., Moore R. H., and Dietert S. E. (1989) Uranium in the tissues of an occupationally exposed individual. *Health Physics* 57, 17–21.

Kathren R. L., and Moore R. H. (1986) Acute accidental inhalation of U: a 38 year follow–up. *Health Physics* 51(5), 609–619.

Kaye G. W. C., and Laby T. H. (1993) *Tables of physical and chemical constants*. Longman Group Ltd, 15th edition, Essex, UK.

Keenan R. E., Sauer M. M., Lawrence F. H., and Crawford D. W. (1989) Examination of potential risks from exposure to dioxin in sludge used to reclaim abandoned strip mines. In *The Risk Assessment of Environmental Hazards: A text book of case studies* (editor: D. J. Paustenbauch). John Wiley, New York, USA. pp. 935–998.

Kimborough R. D., Falk H., Stehr P., and Fries G. (1984) Health implications of 2,3,7,8 – tetrachlorodibenzeno–p–dioxin (TCDD) contamination of residential soil. *Journal of Toxicology and Envronmental Health* 14, 47–93.

Kirby A. (2000) Crashed jet contained DU. In *BBC News Online.*

Labrot F., Ribera D., SaintDenis M., and Narbonne J. F. (1996) In vitro and in vivo studies of potential bio–markers of lead and uranium contamination: Lipid peroxidation, acetylcholinesterase, catalase and glutathione peroxidase activities in three non–mammalian species. *Biomarkers* 1(1), 21–28.

Lacey E. P. (1990) Broadening the Perspective of Pica: Literature review. *Public Health Reports* 105, 29–35.

Landa E. R., and Councell T. B. (1992) Leaching of uranium from glass and ceramic foodware and decorative items. *Health Physics* 63, 343–348.

Lane R.C., McCormick W.B., Jeff'eries S.J., and Denyluk, P. (1985) Use of six–element arrays of hyperpure germanium detectors for internal actinide contamination. In *Assessment of Radioactive Contamination in Man* 79–91, IAEA Vienna.

Langmuir D (1978) Uranium solution–mineral equilibria at low temperatures with applications to sedimentary ore deposits. *Geochimica et Cosmochimica Acta* 42, 547–569.

Lapham S. C., Millard J. B., and Samet J. M. (1989) Health implications of radionuclide levels of cattle raised near U mining and milling facilities in Ambrosia Lake, New Mexico. *Health Physics* 36, 327–330.

Leach L., Gelein R., and Panner B. (1984) The acute toxicity of the hydrolysis products of uranium hexafluoride (UF6) when inhaled by the rat and guinea pig. Final report. ISS K/SUB–81–9039–3. US National Technical Information Service, Document DE84011539.

Leach L., Maynard E., and Hodge C. (1970) A five–year inhalation study with natural uranium dioxide dust. *Health Physics* 18, 599–612.

Leach L., Yuile C., and Hodge H. (1973) A five–year inhalation study with natural uranium dioxide (UO2) dust. Post–exposure retention and biologic effects in the monkey, dog and rat. *Health Physics* 25, 239–258.

Lederer C. M., Hollander J. M., and Perlman I. (1978) *Table of Isotopes*. John Wiley, New York, USA.

Ledvina R., Kolar L., and Frana J. (1996) Uranium, thorium and some other elements in topsoils of the Trebon region from the aspect of production contamination. *Rostlinna Vyroba* 42(2), 73–78.

Leggett R. W. (1989) The behaviour and chemical toxicity of U in the kidney: a reassessment. *Health Physics* 57(3), 365–383.

Leggett R. W. (1992) A generic age-specific biokinetic marker for calcium like elements, *Radiation Protection Dosimetry* 41(2-4), 183-198.

Leggett R. W (1994) Basis for ICRP's Age-specific model for uranium, *Health Physics* 67(6), 589-610.

Leggett R. W., and Harrison J. D. (1995) Fractional absorption of ingested uranium in humans *Health Physics* 68, 484–498

Lin R., Wu L., and Lee C. (1993) Cytogenetic toxicity of uranyl nitrate in Chinese hamster ovary cells. *Mutation Research* 319, 197–203.

Linsalata P. (1994) Uranium and thorium decay series radionuclides in human and animal foodchains, *Journal of Environmental Quality* 23, 633–642.

Liolios T.E. (2000) Assessing the risk from the DU weapons used in operation allied force. *Science and Global Security* 8(2), 163–181.

Loomis D.P., and Wolf S.H. (1997) Mortality of workers at a nuclear materials production plant at Oak Ridge, Tennessee, 1947-1990. *American Journal of Industrial Medicine* 29(2), 131-141.

Lopez R., Diaz Sylvester P. L., Ubios, A. M., and Cabrini, R. L. (2000) Percutaneous toxicity of uranyl nitrate: its effect in terms of exposure area and time. *Health Physics* 78, 434–437.

Lu S., and Zhao F.Y. (1990) Nephrotoxic limit and annual limit on intake for natural U. *Health Physics* 58(5), 619-23.

MAFF (1999) Radioactivity in food and the environment, 1998 RIFE–4, *Ministry of Agriculture, Fisheries and Food*, London, UK, 176pp.

Malenchenko A., Barkun N., and Guseva G. (1978) Effect of uranium on the induction and course of experimental autoimmune ochitis and thyroiditis. *Journal of Hygiene, Epidemiology, Microbiology and Immunology* 22(3), 268-277.

Mao Y., Desmeules M., Schaubel D., Berube D., Dyck R., Brule D., and Thomas B. (1995) Inorganic components of drinking water and micro–albuminuria. *Environmental Research*, 71, 135–140.

Martin P., Hancock G. J., Johnson A., and Murray A. S. (1998) Natural series radionuclides in traditional north Australian Aboriginal foods. *Journal of Environmental Radioactivity* 40, 37–58.

Maynard E., Down W., and Hodge H. (1953) Oral toxicity of uranium compounds. In *Pharmacology and toxicology of uranium compounds* (editor: Voeglin C). McGraw–Hill, New York, USA. pp 309–376.

Maynard E., and Hodge H. (1949) Studies of the toxicity of various uranium compounds when fed to experimental animals. In Pharmacology and toxicology of uranium compounds (Ed. Voeglin C), *McGraw–Hill*, New York, USA. pp. 309–376.

McConnell M. A., Ramanujam V. M. S., Alcock N. W., Gabehart G. J., and Au W. W. (1998) Distribution of uranium–238 in environmental samples from a residential area impacted by mining and milling activities. *Environmental Toxicology and Chemistry* 17(5), 841–850.

McDiarmid M.A., Hooper, F.J., Squibb, K., and McPhaul, K (1999) The utility for spot collection for urinary uranium determination in depleted uranium exposed gulf war veterans, *Health Physics* 77(3), 261-264.

McDiarmid M. A., Keogh J. P., Hooper F. J., McPhaul K., Squibb K., Kane R., DiPino R., Kabat M., Kaup B., Anderson L., Hoover D., Brown L., Hamilton M., Jacobson–Kram D., Burrows B., and Walsh M. (2000) Health effects of DU on exposed Gulf War veterans. *Environmental Research*, 82 168–80.

McDonald–Taylor C. K., Bhatnagar M. K., Gilman A., Yagminas A., and Singh A. (1992) Uranyl nitrate–induced glomerular basement membrane alterations in rabbits: a quantitative analysis. *Bulletin of Environmental Contamination and Toxicology* 48(3), 367–373.

McDonald–Taylor C. K., Singh A., and Gilman A. (1997) Uranyl nitrate–induced proximal tubule alterations in rabbits: a quantitative analysis. *Journal of Toxicologic Pathology* 25(4), 381–389.

McGeoghegan D., and Binks K. (2000) The mortality and cancer morbidity experience of workers at the Springfields uranium production facility, 1946–95. *Journal of Radiological Protection* 20, 111–137.

Medley D. W., Kathren R. L., and Miller A. G. (1994) Diurnal urinary volume and uranium output in uranium workers and unexposed controls. *Health Physics* 67, 122–130.

Meyer M. C., and McLendon T. (1997) Phytotoxicity of DU on three grasses characteristic of different successional stages. *Journal of Environmental Quality*, 26 748–752.

Meyer K. R., Voilleque P. G., Schmidt D. W., Rope S. K., Killough G. G., Shleien B, Moore R. E., Case M. J., and Till J. E. (1996) Overview of the Fernald dosimetry reconstruction project and source term estimates for 1951–1988. *Health Physics 71*(4), 425–437.

Miller A. C., Blakely W. F., Livengood D., Whittaker T., Xu J., Ejnik J. W., Hamilton M. M., Parlette E., St John T., Gerstenberg H. M., and Hsu H. (1998a) Transformation of human osteoblast cells to the tumorogenic phenotype by DU–urynal chloride. *Environmental Health Perspectives* 106(8), 465–471.

Miller A. C., Fuciarelli A. F., Jackson W. E., Ejnik E. J., Emond C., Strocko S., Hogan J., Page N., and Pellmar T. (1998b) Urinary and serum mutagenicity studies with rats implanted with DU or tantalum pellets. *Mutagenesis* 13(6), 643–648.

Miller, W. M., Alexander, W.R., Chapman, N.A., McKinley, I.G and Smellie, J.A.T. (2000) Geological disposal of radioactive wastes and natural analogues. Pergamon Waste Management Series, Volume 2, Oxford, UK.

Mirto H., Henge-Napoli M. H., Gibert, R., Ansoborlo E., Fourneir, M., and Cambar, J (1999) Intracellular behaviour of uranium (VI) on renal epithelial cell in culture (LLC-PK1): Influence of uranium speciation. *Toxicological Letters* 104, 249-256.

Mishima J., Parkhurst M. A., Scherpelz R. I., and Hadlock D. E. (1985) Potential behaviour of DU penetrators under shipping and bulk storage accident conditions. *Pacific Northwest Laboratory*, Report PNL–5415.

Misund A., Frengstad B., Siewers U., and Reimann C. (1999) Variation of 66 elements in European bottled mineral waters. *The Science of the Total Environment* 243/244, 21–41.

Moody, J. C., Birchall, A., Stradling, G. N., Britcher, A., Battersby, W. P. (1997) Biokinetics of recycled uranium trioxide and implications for human exposure. *Annals of Occupational Hygiene* 41 (sup 1) 104–110 (1997).

Morgan H., Smart G. A., and Sherlock J. C. (1988) Intakes of metals. *The Science of the Total Environment* 75, 71-100.

Morrow P., Gelein R., Beiter H., Scott J., Picano J., and Yuile C. (1982) Inhalation and intravenous studies of UF6/UO2F2 in dogs. *Health Physics* 43(6), 859–73.

Moss, C.E. (1985) Gamma–ray line intensities for DU. *Los Alamos National Laboratory*, Report No. LA-UR-85-1602 (Dec 85).

Moss M. A. (1985) Chronic low level uranium exposure via drinking water–clinical investigations in Nova Scotia. Unpublished MSc Thesis, Dalehouse University.

Moss M. A., McCurdy R. F., Dooley K. C., Givener M. L., Dymond L. C., Slater J. M., and Courneya M. M. (1983) Uranium in drinking water – report on clinical studies in Nova Scotia. In *Chemical toxicology and clinical chemistry of metals* (editors: S. Brown and J. Savory), Academic Press, London, 149–152.

Muir W. R., Young J. S., Benes C. M., Benjamin D. M., Howay J., Lobo L., Pappas C., Walentowicz R., and Schaum J. (1995) Risk*Assistant for Windows: Scientific Guide. *The Hampshire Research Institute Inc.*, USA.

Murphy B. L., Toole A. P., and Bergstrom P. D. (1989) Health risk assessment for arsenic contaminated soil. *Environmental Geochemistry and Health* 11, 163–169.

NCRP (1959) Recommendations of the maximum permissible body burdens and maximum permissible concentrations of radionuclides in air and water for occupational exposure. *NBS Handbook 69.* Washington DC: US Government Printing Office.

NCRP (1975) Natural background radiation in the United States. *National Council on Radiological Protection*, Report 45, Bethesda, MD, USA.

NCRP (1984) National Council on Radiation Protection and Measurements. Exposures from the uranium series with emphasis on radon and its daughter. NCRP Report No 77, Bethesda, Maryland, USA.

NCRP (1999) National Council on Radiation Protection and Measurements. Recommended screening limits for contaminated surface soil and review of factors relevant to site-specific studies, NCRP Report No. 129, Bethesda, Maryland, USA.

NIOSH (1994) National Institute for Occupational Safety and Health. *Pocket guide to chemical hazards.* US Department of Health and Human Services, Public Health Service, Centres for Disease Control and Prevention.

NIOSH (2000) National Institute for Occupational Safety and Health. *Pocket guide to chemical hazards.* US Department of Health and Human Services, Public Health Service, Centres for Disease Control and Prevention.

Noël A., Goengrich A., and Louis J–P. (1988) Céramiques dentaires et radioactivité. *Radioprotection*, Vol. 23, No. 3, 291–300.

NGWRC (2001) National Gulf War Resource Centre, Inc. (http://www.ngwrc.org/Dulink).

Nozaki T. (1970) Neutron activation analysis of uranium in human bone, drinking water and daily diet. *Journal of radioanalytical chemistry*, 6, 33–40.

NRC (1988) National Research Council Health. Effects of Radon and Other Internally Deposited Alpha-Emitters. Committee on the Biological Effects of Ionizing Radiation, BEIR IV. National Academy Press: Washington, D.C., USA.

NRC (1999) National Research Council. *Health effects of exposure to radon: BEIR VI.* National Academy Press, Washington DC, USA.

NRC (2000) United States Nuclear Regulatory Commission Title 10 of the Code of Federal Regulations. USNRC, Document 10 CFR 110.2

NUREG (1999) Systematic radiological assessment of exemptions for source and by product materials. Draft Report for Comment, *U.S. Nuclear Regulatory Commission*, Report 1717, December 1999.

OJEC 1980 *Official Journal of the European Communities* L246, Vol. 23, 17 Sept 1980, Luxembourg, Office for Official Publications of the European Communities (1980).

OJEC 1996 *Official Journal of the European Communities* LI59 Vol. 39, 29 June 1996. Luxembourg: Office for Official Publications of the European Communities (1996).

OMEE (1996) Monitoring data for uranium for the period 1990–1995. Ontario Ministry of Environment and Energy, *Ontario Drinking Water Surveillance Programme.* Ontario, Canada.

Orcutt J. A. (1949) The toxicology of compounds of uranium following application to the skin. In *Pharmacology and toxicology of uranium compounds* (editors: C. Voegtlin and H. C. Hodge), MCGraw–Hill, New York, USA. pp. 377-422.

Ortega A. (1989) Evaluation of the oral toxicity of uranium in a four–week drinking water study in rats. *Bulletin of Environmental Contamination and Toxicology* 42, 935–941.

OSHA (1989) Occupational Safety and Health Administration. US Department of Labor. Air contaminant: permissible exposure limits. Title 29, Code of Federal Regulations, Part 1910.1000, OSHA 3112. Washington DC:, OSHA, US Department of Labour.

Othman I.,and Yassine T. (1996) Natural radioactivity of drinking water in the southern and middle parts of Syria. *Environment International* 22(S1), S355–S359.

Oxenberg T. P., Saunders F. M., Rosson R. R., and Kahn B. (1999) Environmental monitoring to assess mobilisation and transport of DU in soils and water. *Health Physics* 76(6s), 179.

Ozmen M., and Yurekli M. (1998) Subacute toxicity of uranyl acetate in Swiss–Albino mice. *Environmental Toxicology and Pharmacology* 6(2), 111 – 115.

Palmer, H. E., and Rieksts, G. A. (1985) High–purity planar germanium detectors for *in–vivo* measurement of uranium and transuranic radionuclides. In *Assessment of radioactive contamination in man*. pp93–106. Vienna: IAEA (1985).

Parkhurst, D. L., and Appello C. A. J. (1999) Users Guide To PHREEQC: A computer programme for speciation, Batch Reaction, One dimensional Transport and Inverse Geochemical calculations, *USGS Water Resources Investigations*, Report 99–4259, 309pp.

Parker R. L. (1988) Fear of flying. *Nature* 336, 719.

Pastenbach D. J., Shu H. P., and Murray F. J. (1986) A critical examination of assumptions used in risk assessments of dioxin contaminated soils. *Regultory Toxicology and Pharmacology* 6, 284–307.

Patrick M. A., and Cornette (1977) Morphological characteristics of Particulate material formed from high velocity impact of DU projectiles with armour targets. *US Air Force Armament Laboratory* (ARATL), Report No. TR-78-117.

Pavlakis N., Pollock C. A., McLean G., and Bartrop R. (1996) Deliberate overdose of uranium: toxicity and treatment. *Nephron* 72(2), 313–317.

Pellmar T. C., Hogan J. B., Benson K. A., and Landauer M. R. (1997) Health risk assessment of embedded DU: behaviour, psychology and histology – 6 month time point. *AFFRI Special Report* 97–4.

Pellmar T. C., Fuciarelli A. F., Ejnik J. W., Hamilton M., Hogan J., Strocko S., Emond C., Mottaz H. M., and Landauer M. R. (1999a) Distribution of uranium in rats implanted with DU pellets. *Toxicological Sciences* 49, 29–39.

Pellmar T. C., Keyser D. O., Emery C., and Hogan J. B. (1999b) Electrophysiological changes in hippocampal slices isolated from rats embedded with DU fragments. *Neurotoxicology* 20(5), 785–92.

Pellow P. G., Stradling, G. N., Hodgson, A., Fell, T. P., Phipps, A. and Ellender, M. (1997) Absorption kinetics of tri–n–butylphosphate in the rat: implications for occupational exposure. *Journal of Radioanalytical and Nuclear Chemistry* 226 (1–2), 93–98 (1997).

Perez D. V., Saldanha M. F. D., Moreira J. C., and Vaitsman D. S. (1998) Total concentration of uranium and thorium in some Brazilian soils. *Pesquisa Agropecuaria Brasileira* 33(8), 1417–1423.

Plant J. A., Simpson P. R., and Smith B. (1999) Uranium ore deposits – products of the radioactive earth. In Uranium: Mineralogy, Geochemistry and the Environment, Vol. 38 (editors: P. C. Burns and R. Finch), pp. 255–319. *Mineralogical Society of America*, Washington, DC, USA.

Plant J. A., Baldock J. W., and Smith B. (1996) The role of geochemistry in environmental and epidemiological studies in developing countries. In Environmental Geochemistry and Health, Vol. 113 (editors: R. Fuge and J.D. Appleton), 7–22. *Geological Society Special Publication*, London, UK.

Polednak A., and Frome E. (1981) Mortality among men employed between 1943 and 1947 at a uranium–processing plant. *Journal of Occupational Medicine* 23, 168–178.

Pomroy, C., and Malm H. (1985) High–purity germanium detectors for *in–vivo* measurement of uranium and thorium. In *Assessment of radioactive contamination in man*. 69–78,Vienna: IAEA.

Porcelli D., Andersson P. S., Wasserburg G. J., Ingri J., and Baskaran M. (1997) The importance of colloids and mires for the transport of uranium isotopes through the Kalix river watershed and Baltic Sea. *Geochimica Et Cosmochimica Acta* 61(19), 4095–4113.

Pozzani U. (1949) *Pharmacology and toxicology of uranium compounds.* McGraw–Hill Book Company, Inc., New York, USA.

Pramauro E., Prevot A. B., Zelano V., Gulmini M., and Viscardi G. (1996) Selective recovery of uranium(VI) from aqueous acid solutions using micellar ultrafiltration. *Analyst* 121(10), 1401–1405.

Price E. W. (1990) *Podoconiosis: non–filarial elephantiasis*. Oxford Medical Books, Oxford, UK.

Priest N. D. (2001) Toxicity of depleted uranium, *The Lancet* 357, 244–246.

Puls R. (1990) Mineral levels in animal health, diagnostic data. *Sherpa International*, Clearbrook, BC, Canada.

Raff O., and Wilken R. D. (1999) Removal of dissolved uranium by nanofiltration. *Desalination* 122(2–3), 147–150.

Rao S. S. and Balakrishna Bhat T. (1997) Depleted uranium penetrators – hazards and safety. *Defence Science Journal* 47(1), 97–105.

Reimann C., Äyräs M., Chekushin V., Bogatyrev I., Boyd R., Caritat P. D., Dutter R., Finne T. E., Halleraker J. H., Jæger Ø., Kashulina G., Lehto O., Niskavaara H., Pavlov V., Räisänen M. L., Strand T., and Volden T. (1998) Environmental geochemical atlas of the central Barents region. *Geological Survey of Norway*, Trondheim, Norway.

Reimann C., Hall G. E. M., Siewers U., Bjorvaten K., Morland G., Skarphagen H., and Strand T. (1996) Radon, fluoride and 62 elements as determined by ICP–MS in 145 Norwegian hard rock groundwater samples. *The Science of the Total Environment* 192(1–19).

Ribera D, Labrot F, Tisnerat G., and Narbonne J. F. (1996) Uranium in the environment: Occurrence, transfer and biological effects. *Reviews of Environmental Contamination and Toxicology* 146, 53–89.

Rich B. L. (1988). Health physics manual of good practices at uranium facilities. U.S. Department of Energy, EGG-2530 UC–41, pp. 2–17 through 2–24.

Ritz B. (1999) Radiation exposure and cancer mortality in uranium processing workers. *Epidemiology* 10(5), 531-8.

Ritz B., Morgenstern H., Froines J., and Batts Young B. (1999) Effects of exposure to external ionizing radiation on cancer mortality in nuclear workers monitored for radiation at Rocketdyne/Atomics International. *American Journal of Industrial Medicine* 35, 21-31.

Ritz B., Morgenstern H., Crawford–Brown D., and Young B. (2000) The effects of internal radiation exposure on cancer mortality in nuclear workers at Rocketdyne/Atomics International. *Environmental Health Perspectives* 108, 743–751.

Roberts E. (1949) *Pharmacology and Toxicology of uranium compounds.* McGraw–Hill Book Company, Inc., New York, USA.

Roth P., Werner E., and Paretke H. G. (2001) Research into uranium excretion of uranium : Verification of protective measures in the German KFOR army contingent. GSF Report 3/01. GSF Institut für Strahlenschutz, Neuherberg, Germany, January 2001.

Rothermel J. (1949) *Uranium tetrachloride: pharmacology and toxicology of uranium compounds.* McGraw–Hill Book Company, New York, USA.

Rothstein A. (1949) *Pharmacology and toxicology of uranium compounds.* McGraw–Hill. New York, 548–648.

Ruby M. V., Davis A., Schoof R., Eberle S., and Sellstone C. M. (1996) Estimation of lead and arsenic bioavailability using a physiologically based extraction test. *Environmental Science & Technology* 30(2), 422–430.

Russell J. J., Kathren R. L., and Dietert S. E. (1996) A histological kidney study of uranium and non–uranium workers. *Health Physics* 70(4), 466–72.

Saccomanno G. (1982) The contribution of uranium miners to lung cancer histogenesis. *Recent Results in Cancer Research* 82, 43–52.

SAIC (1990) Kinetic energy penetrator environmental and health considerations. SAIC, Report 2, Volume 2.

Sairenji E., Söremark R., and Noguchi K. (1982) Uranium content in porcelain denture teeth and in porcelain powders for ceramic crowns, *Acta odontologica Scandinavia* 40(5), 333–339.

Salonen, L (1988) Natural radionuclides in groundwater in Finland, *Radiation Protection Dosimetry* 24(1/4), 163-166.

Scripsick, R. C., Crist, K. C., Tillery, M. I., Soderholm, S. C. (1985b) Differences in *in-vitro* dissolution properties of settled and airborne uranium material. In *Occupational Radiation Safety in Mining* (editor: Stocker, H.) Canadian Nuclear Association pp. 255-260. http://lib-www.lanl.gov/la-pubs/00374828.pdf.

Scripsick, R. C., Crist, K. C., Tillery, M. I., Soderholm, S. C., Rothenberg, S. J. (1985a) Preliminary Study of Uranium Oxide Dissolution in Simulated Lung Fluid. LA-10268-MS, *Los Alamos National Laboratory*. http://lib-www.lanl.gov/la-pubs/00318819.pdf.

Sevcova M. (1978) Problems concerning skin diseases affecting uranium industry workers. *Cesk Dernatol* 51(5), 318–322.

Simon S. L. (1998) Soil Ingestion by Humans: A Review of History, Data, and Aetiology with Application to Risk Assessment of Radioactively Contaminated Soil, *Health Physics* 74(6), 647–672.

Singh A. K., Kumar A., Jojo P. J., and Prasad R. (1998) Microanalyses of uranium in Indian soil samples using solid state nuclear track detection technique. *Journal of Radioanalytical and Nuclear Chemistry* 238(1–2), 21–24.

Singh J., Singh L., and Singh S. (1995) High U contents observed in some drinking waters of Punjab, India. *Journal of Environmental Radioactivity* 26(3), 217–222.

Singh N. (1990) Daily uranium intake in Utah residents from food and drinking water. *Health Physics* 59(3), 333-337.

Singh P., Rana N. P. S., Azam A., Naqvi A. H., and Srivastava D. S. (1996) Levels of Uranium in Waters from some Indian cities determined by fission track analysis. *Radiation Measurements* 26(5), 683–687.

Smith B., Breward N., Crawford M. B., Galimaka D., Mushiri S. M., and Reeder S. (1996) The environmental geochemistry of aluminium in tropical terrain's and its implications to health. *Geological Society Special Publication* 113, 141–153. London, UK.

Smith B., Chenery S. R. N., Cook J. M., Styles M. T., Tiberindwa J. V., Hampton C., Freers J., Rutakinggirwa M., Sserunjogi L., Tomkins A., and Brown C. J. (1998) Geochemical and environmental factors controlling exposure to cerium and magnesium in Uganda. *Journal of Geochemical Exploration* 65(1), 1–15.

Smith B., Powell A. E., Milodowski. A. E., Hards V. L., Hutchins M. G., Amro. A., Gedeon R., Kilani S., S.M S., and Galt V. (2000) Identification, investigation and remediation of groundwater containing elevated levels of uranium–series radionuclides: a case study from the Eastern Mediterranean. *In: Proceedings of the third international conference on the Geology of the Eastern Mediterranean* (editors: Panayides, I., Xenophontos, C., and Malpas, J), Nicosia, Cyprus.

Smith B., Powell J. H., Gedeon R., Amro H., and Bradley A. D. (1995) Naturally occurring uranium pollution in Jordan. *Mining and Environmental Management* (March, 1995).

Smith D. D., and Black S. C. (1975) Actinide concentrations in tissues from cattle grazing near the Rocky Flats Plant. *Report ISS NERC*–LV–539–36.

Sontag W. (1986) Multi–compartment kinetic models for the metabolism of americium, plutonium and uranium in rats. *Human Toxicology* 5, 163–173.

Spencer H. S., Osis D., Fisenne I. M., Perry P., and Harley N. H. (1990) Measured intake and excretion patterns of naturally occurring ^{238}U and calcium in humans. *Radiation Research* 24, 90–95.

Spiegel C. (1949) *Pharmacology and Toxicology of uranium compounds.* McGraw–Hill Book Company, New York, USA.

Spoor N. L., and Hursh J. B. (1973) Chapter 5, Protection Criteria. In *Uranium, Plutonium Transplutonic Elements* (editors: Hodge, H.C., Stannard, J.N., Hursh, J.B) pp241–270. New York: Springer–Verlag.

SSI (2000) Use of depleted uranium in military conflicts and possible impacts on health and environment. *Swedish Radiation Protection Institute Newsletter* 8 (Dec 2000), 1–8.

Starmet (2001) www.starmet.com

Stayner L.T., Meinhardt T., Lemen R., Bayliss D., Herrick R., Reeve G.R., Smith A.B., and Halperin W. (1985) A retrospective cohort mortality study of a phosphate fertilizer production facility. *Archives of Environmental Health* 40, 133-38.

Steele M. J., Beck B. D., Murphy B. L., and Strauss H. S. (1990) Assessing the contribution from lead in mining wastes to blood lead. *Regulatory Toxicology and Phamacology* 11, 158–190.

Steiger R.H., and Jager E. (1977) Submission on geochronology: Convention on the use of decay constants in geo and cosmochronology. *Earth Planet Science Letters* 36, 359–362.

Stokinger H. E., Baxter R. C., and Dygert H. P. (1953) Toxicity following inhalation for 1 and 2 years. In: *Pharmacology and Toxicology of Uranium compounds* Vols 3 & 4, (editors: Voegtlin C., and Hodge H. C), McGraw–Hill, New York.

Stradling G. N., Stather J. W., Price A., and Cooke N. (1989) Limits on intake and the interpretation of monitoring data for workers exposed to industrial uranium bearing dusts. *Radiation Protection Dosimetry* 26, 83–87.

Stradling G. N., and Moody J. C. (1995) Use of animal studies for assessing intakes of inhaled actinide bearing dusts. *Journal of Radioanalytical and Nuclear Chemistry* Articles 197, No. 2, 309–329.

Stradling G. N., Hodgson A., Moody J. C., Fell T. P., Rance E . (1997) Exposure limits and assessment of intake for inhaled soluble uranium compounds. *Report NRPB–M801. Chilton: National Radiological Protection Board.*

Stradling G. N., Hodgson A., Moody J. C., Fell T. P., and Rance, E (1998) Exposure limits and assessment of intake after inhalation of soluble forms of uranium. In: Proc. of Actinides '97, Baden–Baden, Germany, Sept 21–26 1997. *Journal of Alloys and Compounds* 271–273, 54–57.

Stather, J.W and A Karaoglou (eds) (1994). Intakes of Radionuclides. Proceedings of a Workshop held in Bath, England, 13-17 September 1993. *Radiation Protection Dosimetry* 53 (1-4)

Tannenbaum A., Silberstone H., and Kosiol J. (1951) The distribution and excretion of uranium in mice, rats and dogs. In *Pharmacology and Toxicology of Uranium Compounds* (editor: A. Tannenbaub), pp. 128–181. *McGraw-Hill*, New York.

Taylor R. N., Croudace I. W., Warwick P. E., and Dee S. J. (1998) Precise and rapid determination of U–238/U–235 and uranium concentration in soil samples using thermal ionisation mass spectrometry. *Chemical Geology* 144(1–2), 73–80.

Thomas P. A., and Gates T. E. (1999) Radionuclides in the lichen–caribou–human food chain near uranium mining operations in northern Saskatchewan, Canada. *Environmental Health Perspectives* 107(7), 527–537.

Thornton I. (1996) *Metals in the global environment: Facts and misconceptions.* International Council on Metals and the Environment.

Thun M. J., Baker C. B., Steenland K., Smith A.B., Halperin W., and Berl T. (1985) Renal Toxicity in uranium mill workers, *Scandinavian Journal of Work and Environmental Health* 11, 83–93.

Tirmarche M., Raphalen A., Allin F., Chameaud, J. and Bredon, P. (1993) Mortality of a cohort of French uranium miners exposed to relatively low radon concentrations. *British Journal of Cancer* 67, 1090-1097.

Toohey R., Palmer E., Anderson L., Berger C., Cohen N., Eisele G., Wacholz B., and Burr W. (1991) Current status of whole–body counting as a means to detect and quantify previous exposures to radioactive materials. *Health Physics* 60 (Suppl 1), 7–42.

Toole J., Adsley I., Hearn R., Wildner H., Montgomery N., Croudace I., Warwick P., and Taylor R (1997). Status of analytical techniques for the measurement of uranium isotopic signatures. Workshop on the status of measurement techniques for the identification of nuclear signatures, Geel, Belgium, 25–27 February, 1997, Report EUR 17312.

Ubios A. M., Marzorati M., and Cabrini R. L. (1997) Skin alterations induced by long–term exposure to uranium and their effect on permeability. *Health Physics* 72(5), 713–715.

Uijt de Haag P. A., Smetsers R. C., Witlox H. W., Krus H. W. and Eisenga A. H. (2000). Evaluating the risk from DU after the Boeing 747–258F crash in Amsterdam, *Journal of Hazardous Material* 76(1), 39–58.

UN (1991) *Trans–boundary air pollution, Report of the Secretary–General of the UN Conference on Environment and Development*, Third Session, Geneva, 1991. UN Topic 1 49 PC/59.

UNSCEAR (2000a) The United Nations Scientific Committee on the Effects of Atomic Radiation. UNSCEAR 2000. Report to the General Assembly.

UNSCEAR (2000b) The United Nations Scientific Committee on the Effects of Atomic Radiation. *Sources and effects of ionizing radiation*, UN, New York, Volume 1 Annex B.

UNEP/UNCHS Balkan Task Force (BTF) (1999) The potential effects on human health and the environment arising from possible use of DU during the 1999 Kosovo conflict. A preliminary assessment. United Nations Environment Programme.

UNEP (2001) Depleted uranium in Kosovo: post–conflict assessment. Report of the United Nations Environment Programme (UNEP) Scientific Mission to Kosovo, 5–19 November 2000, UNEP, Geneva.

Upton A. C. (1992) The health effects of low–level ionising radiation. *Annual Review Public Health* 13, 127–150.

US EPA (1984) United States Environment Protection Agency. Risk analysis of TCDD Contaminated Soil. US EPA, Office of Health and Environmental Assessment, Office of Research and Development, Report EPA/620/8–84/031, Washington, DC, USA.

US EPA (1985) United States Environment Protection Agency. Drinking water criteria document for uranium, EPA Report PB86–241049, Washington DC, USA

US EPA (1986) United States Environment Protection Agency. Washington, DC. Environmental Radiation Data, Report 42, April–June 1985. NTIS PB166311.

US EPA (1989) United States Environment Protection Agency. Exposure Factors Handbook. Office of Health and Environmental Assessment, Report EPA/600/8–89/043, Washington, DC, USA.

US EPA (1990) United States Environment Protection Agency. Occurrence and exposure assessment for uranium in public drinking water supplies. Report prepared by Wade Miller Associates, Inc for the office of Drinking Water. US EPA, Washington, DC, USA, 26 April 1990 (EPA Contract No. 68–03–3514).

US EPA (1991) United States Environment Protection Agency. Review of RSC Analysis. Report prepared by Wade Miller Associates, Inc for the US Environmental Protection Agency, Washington, DC, 9th May 1991 (follow–up to US EPA, 1990).

US EPA (1994) United States Environment Protection Agency. Environmental radiation data report 74, April-June 1993. National air and Radiation environmental laboratory, EPA 402-R-93-093.

US EPA (1995a) United States Environment Protection Agency. Drinking water criteria document for uranium. EPA PB86-241049.

US EPA (1995b) United States Environment Protection Agency. Groundwater standards for remedial actions at inactive uranium processing sites, US EPA Report 60 FR 2854 (1995) US Government Printing Office, Washington, USA.

US EPA (1999) United States Environment Protection Agency. Understanding variation in partition coefficient, K_d values, Volume I and Volume II, Office of radiation and indoor air, EPA 402-R-99-004A&B.

US EPA (2000a) United States Environment Protection Agency. Radionuclides (Uranium, Radium and Radon). In *United Air Toxics Website, Office of Air Quality Planning and Standards*, http://www.epa.gov/ttnuatw1/hltef/radionuc.html.

US EPA (2000b) United States Environment Protection Agency. Soil Screening Guidance: Technical Background Document, Office of Emergency and Remedial Response, Washington, DC, OSWER Directive 9355.4–16. EPA/540–R–00–006, NTIS PB2000 963306.

van der Steen J. (1999) The main issues concerning NORM in the Netherlands. *Natural Radiation and NORM*, IBC Energy and Safety Division, Conference December, 1999, London, UK, Report EZ1106.

Van Etten D. M., and Purtymun W. D. (1994) Depleted uranium investigation at missile impact sites in White Sands missile range. Los Alamos National Laboratory, Report LA–12675–MS.

Van Loon J. C., and Barefoot R. R. (1989) *Analytical methods for geochemical exploration.* Academic Press, Inc., London, UK.

van Wijnen J. H., Clausing P., and Brunekreef B. (1990) Estimated soil ingestion by children. *Environmental Research* 51, 147–162.

Varani F. T., Jelinek R. T., and Correll R. J. (1987) Occurrence and treatment of uranium in point of use systems in Colorado, USA. *NWWA Conference, Radon in Groundwater* 535–546.

VHI (1997) *Veterinary Environmental Guide.* Veterinary Health Inspectorate, The Netherlands.

Virk H. S. (1997) Uranium and radon surveys in Western Himalaya. *Current Science* 73(6), 536–538.

Voegtlin C., and Hodge H. C. (1949) *Pharmacology and toxicology of uranium Compounds.* McGraw–Hill, New York

Voegtlin C., and Hodge H. C. (1953) *Pharmacology and toxicology of uranium Compounds*, Vols 3 and 4, McGraw-Hill, New York.

Waxweiler R.J., Archer V.E., Roscoe R.J., Watanabe A., and Thun M.J. Mortality patterns among a retrospective cohort of uranium mill workers. In: Epidemiology Applied to Health Physics. Proceedings of the Sixteenth Midyear Topical Meeting of the Health Physics Society (CONF-830101), Albuquerque, New Mexico. January 9-13, 1983.

Wedeen R. P. (1992) Renal diseases of occupational origin, Occupational Medicine 7, 449.

Weigel F. (1983) Uranium and Uranium compounds. In (editor: Grayson, M) *Kirk–Othmer Encyclopaedia of Chemical Technology*, Vol 23, third edition, John Wiley and Sons, New York, 502–547.

WHO (1987) World Health Organisation IPCS Principals for the safety assessment of food additives and contaminants in food, Environmental Health Criteria 70, WHO, Geneva, Switzerland

WHO (1993) World Health Organisation. Guidelines for drinking–water quality. Volume 1. Recommendations. WHO, Geneva, Switzerland.

WHO (1994) World Health Organisation. Assessing Human Health Risks of Chemicals: Derivation of Guidance Values for Health–based Exposure Limits, Environmental Health Criteria 170, WHO, Geneva, Switzerland.

WHO (1997) World Health Organisation. Food consumption and exposure assessment of chemicals. Report No: WHO/FSF/FOS/97.5, WHO, Geneva, Switzerland.

WHO (1998a) World Health Organisation. Guidelines for drinking–water quality. Addendum to Volume 1. Recommendations. WHO, Geneva, Switzerland.

WHO (1998b) World Health Organisation. Guidelines for drinking–Water Quality. Addendum to Volume 2. Health criteria and other supporting information WHO, Geneva, Switzerland.

WHO (1998c) World Health Organisation. Gems/food regional diets. Report No: WHO/FSF/FOS/98.3, WHO, Geneva, Switzerland.

WHO (1998d). World Health Organisation. Derived intervention levels for radionuclides in food. WHO, Geneva, Switzerland.

Wrenn M.E., Durbin P., and Willis D. (1985) Metabolism of ingested uranium and radium. *Health Physics* 48, 601-633.

Wrenn M.E., Lipsztein J., and Bertelli L. (1988) Pharmacokinetic models relevant to the toxicity and metabolic function for uranium in humans and animals. *DOE Report*. DOE/NV/10574-2.

Yamamoto T, Masuda K., and Nukada K. (1971) Studies on environmental contamination by uranium. 6. Uranium in total diet and human urine from non-occupationally exposed persons in Okayama Prefecture (1969-1971). *Journal Radiation Research* 13(1), 5

Yusko J. G. (1999) NORM and Metals Recycling in the United States. *Natural Radiation and NORM.* IBC Energy and Safety Division, Conference December 1999, London, UK, Report EZ1106.

Zajic V. S. (1999) Review of radioactivity, military use and health effects of DU. http://members.tripod.com/vzajic

Zamora M. L., Tracy B. L., Zielinski J. M., Meyerhof D. P., and Moss M. A. (1998) Chronic ingestion of uranium in drinking water: A study of kidney bioeffects in humans. *Toxicological Sciences* 43(1), 68-77.

Ziegler J. L. (1993) Endemic Kaposis sarcome in Africa and local volcanic soils. *The Lancet* November, 1348-1351.

Zielinski R. A., AsherBolinder S., Meier A. L., Johnson C. A., and Szabo B. J. (1997) Natural or fertilizer-derived uranium in irrigation drainage: A case study in southeastern Colorado, USA. *Applied Geochemistry* 12(1), 9-21.