THE ROYAL SOCIETY

The health hazards of depleted uranium munitions Part I

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Part 1

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Preparation of this report

This report has been endorsed by the Council of the Royal Society. It has been prepared by the Royal Society working group on the health hazards of depleted uranium munitions.

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Summary

There has been a substantial amount of public discussion on the health effects of the use of depleted uranium (DU), especially on the battlefield. The Royal Society therefore convened an independent expert Working Group to review the present state of scientific knowledge about the health and environmental effects of DU, in order to inform public debate.

This is the first of two reports. It deals with the amounts of DU to which soldiers could be exposed on the battlefield, the risks from radiation, and what we know from epidemiological studies. We consider past and potential future exposures, the most likely exposures, and the 'worst-case' exposures that cannot be excluded. Our second report, to be published later this year, will address the risks from toxic poisoning and environmental issues including risks to civilian populations.

The group has consulted widely. It has focused on what is known scientifically about aspects that are relevant to health and has not considered the merits of using DU in munitions. Nor does this report analyse Gulf War syndrome, which has been the subject of other reports.

DU is a toxic and weakly radioactive heavy metal that may have adverse consequences to human health, particularly if it enters the body through inhalation, ingestion or wounding. On the battlefield it is used in kinetic energy weapons designed to penetrate the armour of tanks and other vehicles. On impact substantial amounts of DU may be dispersed as particles that can be inhaled and as shrapnel. Our approach has been to estimate the typical levels of exposure on the battlefield over a wide range of scenarios, and the worst-case exposures that individuals are unlikely to exceed. From these we calculate the potential health risks from radiation. We have also considered epidemiological studies of occupational exposures to uranium in other situations as an independent source of information on the risks of inhaling DU particles, although we recognise that the parallels may not be precise.

Based on our own estimates of intakes of DU, we have drawn the following conclusions:

a) Except in extreme circumstances any extra risks of developing fatal cancers as a result of radiation from internal exposure to DU arising from battlefield conditions are likely to be so small that they would not be detectable above the general risk of dying from cancer over a normal lifetime.
b) The greatest exposures will apply only to a very small fraction of the soldiers in a theatre of war, for example those who survive in a vehicle struck by a DU penetrator. In such circumstances, and assuming the most unfavourable conditions, the lifetime risk of death from lung cancer is unlikely to exceed twice that in the general population.
c) Any extra risks of death from leukaemia, or other cancers, as a result of exposure to DU are estimated to be substantially lower than the risks of death from lung cancer. Under all likely exposure scenarios the extra lifetime risks of fatal leukaemia are predicted to be too small to be observable.
d) Many soldiers on a battlefield may be exposed to small amounts of DU and the risks of cancer from such exposures are predicted to be very low. Even if our estimates of risk for these conditions are one hundred times too low, it is unlikely that any excess of fatal cancer would be detected within a cohort of 10,000 soldiers followed over 50 years.
e) Epidemiological studies complement assessments of actual exposures and radiation risks. Although epidemiological studies of occupational exposure to uranium are not sensitive enough to detect small increases in overall risks of cancer, they nevertheless tend to confirm our calculations of the risks derived from estimates of actual exposures to DU.

These are our main conclusions. But there are still uncertainties that need to be resolved, particularly in the estimates of DU intakes that could occur in different situations on the battlefield. Most of these uncertainties arise as a consequence of the paucity of good experimental data on the amounts of DU that may be inhaled within and close to tanks struck by a DU penetrator, and the almost complete lack of any measurements of DU in urine samples taken soon after exposure to a DU impact aerosol. It is likely that the greatest exposure to radiation resulting from inhaled DU particles will be to the lungs, but making worst-case assumptions, predicted radiation doses to the thoracic lymph nodes are about ten times higher than those to the lungs. The widely held view that lymph nodes have a low sensitivity to radiation regarding carcinogenesis has been challenged by others. We have therefore identified a number of areas where further research is necessary, and a number of other actions that would help in assessing further the hazards that may arise from the use of DU in munitions. This work would also be relevant to the assessment of toxicological risks. Our main recommendations for future research include:

- better estimates of the levels of DU, and the properties of DU aerosols, resulting from test firing under realistic conditions into heavy-armour tanks;
- experimental information on intakes of DU from resuspension of depleted uranium dust in contaminated vehicles;
- the development and validation of models to enable

DU exposures to be predicted in a wide range of circumstances;
- new independent assessments of the resultant risks, particularly from high exposures, when further experimental data are available;
- long-term *in vivo* studies of the dissolution of DU oxides;
- a detailed review of the effects of radiation from radioactive particles in lymph nodes, including any possible carcinogenic effects.

Our other recommended actions include:

- the identification of any UK veterans with high level exposures, and their invitation to participate in an independent evaluation programme;
- the publication of protocols for improved and expeditious monitoring of the health of soldiers subjected to high level exposures in any future conflict.

In conclusion, this first report indicates that the radiological risks from the use of DU in munitions are for the most part low, but that for small numbers of soldiers there might be circumstances in which risks are higher, and it is for this reason that further work should be undertaken to clarify their extent.

The Royal Society welcomes comment on this report. Comments should be sent in the first instance to the Director Science Policy, The Royal Society, 6 Carlton House Terrace, London SW1Y 5 AG; e-mail depleteduranium@royalsoc.ac.uk.

1 Background

1.1 Introduction

There has in recent years been much public discussion about the effects of using depleted uranium (DU) for a variety of purposes, especially on the battlefield. DU munitions were used both in the Gulf War in 1991 and more recently in Bosnia and Kosovo. Persistent reports of illnesses suffered by combatants in both theatres have drawn attention to the possibility that these may be the result of exposure to DU. Battlefields are not healthy places, and there are many other potential sources of harm, but the use of DU has attracted particular notice as there are legitimate concerns about the possible health consequences of using a radioactive and chemically toxic material for munitions.

The public interest in DU has highlighted controversy and scientific uncertainty. A good deal is known scientifically about DU; it is also clear that a good deal remains to be learned. The Royal Society therefore set up an independent and expert working group to review the present state of knowledge of the hazards to health from DU, with particular emphasis on its use in munitions, and to identify areas where further research should be undertaken. Our focus has been exclusively on reviewing the science; we have not taken a position on the merits or otherwise of utilising this material. Nor have we sought to assess any possible links between DU and the illnesses of Gulf War veterans, a subject that has recently been considered by another independent review (Fulco et al 2000).

This is the first of two reports. It covers the amounts of DU to which soldiers could be exposed, the radiological effects of DU and what we have learned from epidemiological studies. A second report, expected to be published later this year, will cover the health effects of DU related to its properties as a toxic heavy metal, and the environmental impact of DU, including long-term exposures of the local population resulting from its use in munitions.

We have consulted widely in the preparation of this report, and wish to continue this process. We therefore welcome your comments. They should be sent in the first instance to the Director Science Policy, The Royal Society, 6 Carlton House Terrace, London SW1Y 5AG; e-mail depleteduranium@royalsoc.ac.uk

1.2 Conduct of the study

Members of the group were chosen to provide the necessary breadth of scientific expertise, so that it could speak with authority on the complex technical issues facing it.

The terms of reference of the working group were as follows.

- To assess the exposure to depleted uranium and its oxidation products that military personnel are likely to experience by various routes (eg ingested, inhaled or embedded)
- To relate these exposures to the known chemical and radioactive toxicities of depleted uranium and its oxidation products
- To assess the likely health effects of these exposures
- To estimate the exposure, doses and possible health effects for the general population during, and shortly after, the use of depleted uranium munitions
- To estimate the longer term consequences for health of environmental contamination with depleted uranium and its oxidation products
- To identify areas where research is required to address the consequences for health and the environment of the use of depleted uranium munitions in warfare
- To consider the possible hazards associated with the use of depleted uranium as ballast on aircraft

This report is based on evidence from several sources. We have studied the very extensive literature on DU, wherever possible concentrating on what has been published in peer-reviewed scientific journals. Where that is not possible, for example when addressing the properties of the airborne particles likely to be formed from the battlefield use of DU, we have drawn on original laboratory reports. We have referenced all documented sources at appropriate places in the text.

We also issued a public call for evidence and sought the advice and opinions of a broad cross-section of individuals and organisations with an interest in the use of DU and its potential consequences. We invited some to present their views in person, including representatives of the veterans' groups and of organisations concerned about the hazards of low levels of radiation. We entered into correspondence with others with whom we were unable to meet in person. All of these are identified in the Acknowledgements. We are most grateful to those who took the trouble to respond.

The whole Working Group met a total of eight times. Subgroups were formed to work on particular themes; the work of three of these subgroups forms appendices 1 – 3. More detailed supporting papers in the form of annexes to these appendices are available over the internet as set out in the contents. We are grateful to a number of individuals who contributed to these reports and they are also identified in the Acknowledgements. The Group as a whole prepared the main report, which summarises the findings of these appendices.

This report has been reviewed and endorsed by the Council of the Royal Society.

1.3 Uranium and depleted uranium

Uranium occurs naturally within the environment and is widely dispersed in the earth's crust, rocks and soils at a level of about 2-4 parts per million by weight. It is an extremely dense metal (at 19 grams per cubic centimetre, about twice as heavy per unit volume as lead) and is toxic. Natural uranium exists in three different forms (isotopes), all of which are radioactive. The two most abundant isotopes, uranium-235 (0.72%) and uranium-238 (99.27%), have radioactive half-lives of about 7×10^8 and 4.4×10^9 years, respectively. The half-life is the time taken for the activity of any amount of a radioactive material to reduce to half its original value; highly radioactive materials can have half-lives measured in days or less (for example radon gas that accumulates within houses has a half-life of 3.82 days). Uranium-235 and uranium-238 are primordial - they were present when the earth was formed. The third isotope, uranium-234, is present at a level of about 0.0055% in natural uranium and is produced during the radioactive decay of uranium-238 (ie it is a decay product of uranium-238). It is more radioactive than uranium-235 and uranium-238 with a half-life of about 2.4×10^5 years. All three isotopes of natural uranium have long half-lives and natural uranium is therefore classified by the International Atomic Energy Agency in the lowest hazard class for radioactive materials as a low specific activity material.

We are all exposed naturally to uranium, its isotopes and decay products. These substances are present to some extent in all drinking waters, foodstuffs and air. However, as with many natural substances, there is a considerable variation in natural levels. For example, the concentration of uranium in natural groundwaters and surface waters commonly used for drinking water range from less than 0.02 micrograms per litre to more than 1 milligram (mg) per litre (WHO 1998a, b). The exact concentration is dependent upon the concentration of uranium in the local rocks, its ease of weathering and its mobility. Seawater contains relatively high concentrations of uranium (typically 3.3 micrograms per litre).

The average daily intake of uranium in food and drink is about 1 microgram per day, but most daily intakes within a country span an order of magnitude (UNSCEAR 2000), and studies in the USA, Canada and Finland have highlighted areas in which uranium intakes of hundreds of micrograms per day occur from some private water supplies (USEPA 1990, Moss et al 1983, Salonen 1988). Intakes of uranium in some private supplies in Jordan, before they were closed, potentially exceeded 3,000 micrograms (3 mg) per day (Gedeon et al 1994, Smith et al 2000).

Particles containing natural uranium can also be inhaled from the environment and this has been measured in lung and lymph nodes obtained from autopsies (Wrenn et al 1985, Kathren et al 1993). Individual particles containing uranium can be identified by sensitive autoradiographic techniques in lung and lymph nodes obtained from autopsy (Henshaw et al 1988).

Uranium is used as fuel in nuclear power plants. Most reactors require fuel that is enriched in uranium-235 from its normal level of 0.72% to about 3%. DU is a by-product of this enrichment process and contains less uranium-235 (about 0.2%), and less uranium-234, than natural uranium. As a consequence DU is about 40% less radioactive than natural uranium although there is no difference in the chemical behaviour or toxicity. Thus studies of the toxic effects of uranium as a poisonous metal can be directly applied to DU. However, any predicted radiological effects of natural uranium (of similar bioavailability) on health would be expected to be slightly less for the same mass of DU.

Since uranium has been mined and processed for use in nuclear reactors for several decades, DU is both abundant and cheap. Because of its high density and metallurgical properties it has found a number of commercial uses in, for example, radiation shielding, rotors, flywheels and counterbalances for control surfaces in aircraft.

When alloyed with small amounts of titanium (about 0.75%), DU is also used in the military sphere as a kinetic energy 'penetrator' in munition rounds designed to pierce the heavy armour of modern battle tanks. Such munitions are typically in the form of a long rod of DU held within a 'sabot' which is discarded after the round is fired. They carry no explosive charge but the large energy of motion (kinetic energy) of the very dense DU penetrator, travelling at speeds of up to 1.8 kilometres per second, is sufficient to punch a hole in the complex armour of a modern battle tank. Unlike kinetic energy penetrators made of tungsten alloys, which blunt on impact with heavy armour, DU penetrators undergo self-sharpening on impact which contributes to their superior penetrative ability.

DU penetrators are considered to be more effective than those made of tungsten alloys. Furthermore, on impact with a hard target (such as a tank) the penetrator may generate a cloud of DU dust within the struck vehicle that ignites spontaneously creating a fire that increases the damage to the target[1].

Sheets of DU, encapsulated in steel plate, are also incorporated into the armour system of some tanks, eg

[1] For brevity, the term DU is applied to the solid metal from which munitions are made, and also the oxides that are formed by slow corrosion of the bulk metal or spontaneous combustion of the finely divided metal. The chemical and physical properties of DU are very dependent on the oxidation state and whether the material is a continuous bulk solid or is finely divided. Since solubility depends on such factors, the distribution and retention in the body of inhaled or ingested DU, and thus its health effects, are very dependent on its chemical and physical states. In this report the form of the DU should be clear from the context.

the Heavy-Armour variant of the Abrams M1A1. The use of DU in such armour has been clearly demonstrated to increase significantly the protection offered to tank crews when attacked by conventional armour-piercing weapons (OSAGWI 2000).

DU munitions were first used in the Gulf War of 1991 following concerns that tungsten penetrators might not be sufficiently effective at destroying Soviet-built T72 Iraqi tanks. DU munitions are used as armour-piercing rounds for the main armament of modern battle tanks (eg the British Challenger II and the American M1A1 Abrams); the large calibre DU rounds (100 or 120 millimetre) fired by these tanks have penetrator rods with DU masses of about 4-5 kilograms. During the Gulf War approximately 9,500 of these DU rounds were fired by US tanks and 88 by UK tanks (about 44,000kg of DU). There are no reports of large calibre DU rounds being fired in the conflicts in the Balkans.

Small calibre DU rounds (20 millimetre) are also available to the UK and US navies, although these are being phased out, and are not believed to have been deployed in any military conflict, excepting a friendly fire incident in the Gulf War involving the USS Jarrett and USS Missouri. Small calibre DU rounds (30 millimetre) are used by the GAU-8 Gatling gun of US A-10 Warthog tank-busting aircraft. These rounds contain about 275 grams of DU in an aluminium fairing and are fired in short bursts of 200-300 rounds (typically a mix of 1 non-DU tracer round to every 5 DU rounds). About 780,000 of these 30 mm DU rounds (214,000kg) are believed to have been fired in the Gulf War. Many of these miss their intended target and may penetrate some distance into the ground. Although large calibre DU rounds were not used in the Balkans, about 10,000 30mm rounds (2750kg) were fired from US A-10 Warthog tank-busting aircraft in Bosnia during 1994-95, and about 31,000 (8500kg) in Kosovo in 1999. The total amount of DU in munitions fired during the Gulf War was about 258,000kg, vastly greater than the total of about 11,250kg used in the two Balkans conflicts. Much of this material remains in the ground. DU munitions were not used by either Iraqi or Serbian forces.

1.4 Radiation from depleted uranium

The radiation resulting from the decay of DU is predominantly alpha-particles, but also beta-particles and gamma-rays. Alpha-particles (helium nuclei consisting of two protons and two neutrons) are relatively massive, travel only short distances (about 30 microns in tissue for the alpha-particles from DU), and are more biologically damaging than beta-particles or gamma-rays. However, they cannot penetrate the dead external layers over most of the skin and thus pose a minimal direct external irradiation risk. Alpha-particles produced from radioactive material within tissues are hazardous, and cells traversed by an alpha-particle may be killed, or may sustain genetic damage that very rarely can lead to a sequence of events that ultimately results in cancer. This type of radiation is often referred to as high linear energy transfer (*High-LET*) radiation.

Beta-particles are highly energetic electrons and those emitted from DU travel much longer distances (up to about one centimetre in tissues) than alpha-particles, while gamma-rays (short wave-length electromagnetic radiation) are extremely penetrating. Exposure to both beta-particles and gamma-rays therefore occurs when DU is handled and exposure to gamma-rays will occur at a considerable distance from DU. However, the biological damage caused by the passage of beta-particles or gamma-rays through cells is much less than that caused by alpha-particles. The radiation from both beta-particles and gamma-rays is referred to as low linear energy transfer (*Low-LET*) radiation.

More details of the composition and radioactive properties of DU can be found in appendix 1, section 1.1.

The DU used in munitions (and tank armour) has been reported to contain trace amounts of americium, neptunium, plutonium, technetium, and uranium-236. This could arise if some of the DU has been obtained from the reprocessing of irradiated uranium from nuclear reactors as well as from natural uranium (OSAGWI 2000), or if it had become contaminated by passing through equipment used for reprocessing uranium from reactors (Brown 2000). Some of these contaminants in DU are highly radioactive and highly toxic (eg plutonium), but they appear to be present in only trace amounts, and it has been calculated that together they contribute less than 1% to the total radiation dose from DU (OSAGWI 2000). Our independent calculations from the data of Bhat (2001) support this view (annexe D). However, independent analyses of the levels of these contaminants in DU munitions are required to confirm that the reported levels are not significantly exceeded in other batches of penetrators. The United Nations Environmental Program (UNEP) Scientific Mission to Kosovo has detected uranium-236 and plutonium in DU penetrators recovered from sites in Kosovo. Their independent analyses confirm that only trace amounts of these contaminating radioactive materials are present in the DU penetrators from the 30 millimetre rounds fired in Kosovo (UNEP 2001).

1.5 Depleted uranium munitions on the battlefield

DU penetrators may miss the target, hit a 'soft' target or hit a 'hard' target. Those striking a soft target (eg a lorry, or lightly armoured vehicle) may well pass straight

through. The effect of hitting a hard target (such as the heavy armour of a battle tank or perhaps a large rock) is to disperse a substantial amount of the DU penetrator in the form of an aerosol of the metal and its combustion products (uranium oxides). Some of this dust may be inhaled or ingested by any surviving combatants within the struck vehicle, or by those who enter the vehicle to rescue any survivors. DU may also be inhaled or ingested when materials deposited in a struck vehicle are resuspended by soldiers or civilians entering the vehicle at a later time. Lower levels of exposure will occur at a distance owing to the spread of DU-containing aerosols. The consequences for health of inhaling small particles of this weakly radioactive metal are a major focus of this report. However, uranium is also a toxic metal that has been particularly associated with effects on the kidney. The levels of uranium that might occur in the kidney after inhalation or ingestion are estimated and their possible effects on health examined in our second report.

A further important potential hazard arises on the battlefield when fragments of a DU penetrator that has impacted or pierced a tank cause shrapnel wounds. Small pieces of shrapnel embedded deep within tissues can be difficult or hazardous to remove and a cohort of US soldiers are being studied who have embedded DU fragments resulting from 'friendly fire' incidents in the Gulf War (annexe B). The radiation from these embedded fragments, and the uranium released by their slow dissolution, results in potential radiological and chemical hazards.

Finally, there can be direct irradiation from DU penetrators, either to soldiers who handle them, or the crews of tanks loaded with DU munitions, but also to civilians who return to the area and come in contact with intact penetrators, or fragment of penetrators, left on the battlefield.

1.6 Depleted uranium in aircraft

Ingots of DU are present in some older aircraft as counterweights. Concerns about exposure to aerosols of DU have been raised in connection with fires following crashes at Amsterdam and Stansted. We have not assessed the risks to health from these specific accidents (a detailed assessment of the former crash has been published; Uijt de Haag 2000), but we have considered the risks from aerosols of DU released in fires, which are relevant to both military situations and aircraft crashes.

2 Estimating exposure to depleted uranium on the battlefield

2.1 Intakes and risks

In order to evaluate the possible effect of DU on the people who may come into contact with it, all of the main routes to exposure need to be assessed, and the levels of exposure estimated, along with the possible consequences to health from both the radioactive nature and chemical toxicity of DU.

We have undertaken four main assessments:

1 intakes of DU (ie the exposure to DU) on the battlefield;
2 consequent risks from the radioactivity of DU;
3 consequent risks from the chemical toxicity of uranium;
4 environmental consequences of the use of DU on the battlefield, including long-term exposures to the local population.

This report summarises the results from the first two assessments. The full details are given in the technical reports that form appendix 1 and appendix 2 of this main report and in annexes to the appendices. The annexes are available on the Society's website. The assessment of the risks from the chemical toxicity of DU and of the effects on the environment will be published in our second report.

There has been extensive occupational exposure to uranium, as uranium ores have been mined and uranium separated from the ores, processed, and milled for about 50 years. The final part of this report examines whether information about the safety of exposures to uranium can be obtained from these long-term studies of the health of workers who have been occupationally exposed to uranium (appendix 3 and associated annexes).

Evaluating the risks to health requires estimates of the possible range of exposures to DU that occur on the battlefield. Unfortunately the inherent characteristics of the battlefield are such that measurements are essentially impossible. So far as we are aware, only one group of soldiers was measured for DU contamination immediately after exposure to DU in the Gulf War and no others until about one year after exposure (OSAGWI 2000, Brown 2000). We have therefore tried to estimate the likely levels of exposure for a variety of scenarios covering a wide range of reasonable possibilities, and to calculate the consequent risks to health. Our approaches and results are summarised below, and described in detail in appendix 1 and the associated annexes.

2.2 Battlefield exposure scenarios

To provide independent views on the intakes of DU that are likely on the battlefield, we have made detailed assessments for a set of specific scenarios considered to be representative of the main exposures to DU. In each scenario we have assessed only the dominant exposure pathways.

For short-term exposures to DU it seemed reasonable to follow the approach used in the report from the US Department of Defense 'Office of the Special Assistant for Gulf War Illnesses' (OSAGWI 2000) and to address three generic situations, as follows. Note however that OSAGWI (2000) is concerned exclusively with exposures of US personnel involved in the Gulf War, whereas our assessment is intended to be a more general analysis of exposures in conflicts where DU munitions are, or may be, deployed. There are likely to be considerable differences in exposures in different conflicts: this is evident from the differences in the deployment of DU weapons, in the extent of the exposure of civilians compared to soldiers, and in the Gulf War compared to the conflicts in Bosnia.

Level I exposures include those to personnel in a vehicle that is struck by a DU penetrator, or to personnel entering a struck vehicle immediately, typically to assist injured comrades. It is assumed that exposure is dominated by inhalation of the aerosol generated by the impact and by embedded DU shrapnel fragments (the latter topic is considered in annexe B).

Level II exposures generally occur after combat, typically working in or on contaminated vehicles to carry out repairs etc. It is assumed that exposure is dominated by inhalation from resuspension of DU deposited within the contaminated vehicle and by ingestion (hand to mouth transfer from contaminated surfaces within vehicles).

Level III includes all other exposures, for example during combat being downwind of impact(s) or fire(s), or brief entry into contaminated vehicles. The following types of Level III exposures have been considered:

- the DU within an aerosol plume from an impact;
- the DU from an aerosol plume from a fire;
- resuspension of deposited DU within a contaminated vehicle;
- resuspension from contaminated ground;
- ingestion of DU by hand to mouth transfer in contaminated vehicles.

It is assumed that exposures resulting from entry into contaminated vehicles are similar, with the main difference being in the duration of exposure: typically this might be

for a few hours for Level II and a few minutes for Level III. Therefore the amounts of DU inhaled per hour have been estimated and can be converted into total intakes by considering the length of the exposure. Level III exposures by inhalation of the plume from munition impacts or fires involving DU could include both soldiers and civilians.

The long-term exposures to civilians from resuspension of DU in contaminated soil, transfer to the food chain, and to water supplies, will form part of the environmental risk assessment, to be included in our second report.

We made two assessments of DU intakes for each scenario:

(i) A '*central estimate*', intended to be a central, representative value, based on likely values of all parameters that determine the intake according to the information available, or where information is lacking, values that are unlikely to underestimate the exposures greatly. The central estimate is intended to be representative of the average individual within the group (or population) of people exposed in that situation.

(ii) For individuals in each group values could be greater than (or less than) the central estimate. We calculated a '*worst-case*' estimate using values at the upper end of the likely range, but not extreme theoretical possibilities. The aim is that it is unlikely that the value for any individual would exceed the worst-case. Thus the worst-case should not be applied to the whole group to estimate, for example, the number of excess cancers that might be induced. One aim of the worst-case assessments is to try to prioritise further investigation. If even the worst-case assessment for a scenario leads to small exposures, then there is little need to investigate it more closely. If, however, the worst-case assessment for a scenario leads to significant exposures, it does not necessarily mean that such high exposures have occurred, or are likely to occur in a future battlefield, but that they might have occurred, or might occur in future conflicts, and further information and assessment are needed.

Details of the methods used and assumptions made in estimating the intakes of DU are provided in appendix 1.

2.3 Radiation doses

Once a radioactive material is inside the human body, it will irradiate the tissues surrounding the place where it is deposited (for example in the lungs), and generally it will in time move to other tissues or organs (for example the kidneys), and irradiate the surrounding tissues there. A *biokinetic model* describes how a radioactive (or non-radioactive) material moves around a biological system such as a human body. A biokinetic model is based on our current knowledge about the processes involved and describes what happens to the material in terms of mathematical equations. Using it, we can estimate how much of the material that entered the body at the time of the intake is present in each organ at any time afterwards.

Another type of model (a *dosimetric model*) is then applied to calculate how much of the energy released in each type of radiation (eg alpha- or beta-particles) is absorbed in each tissue or organ, and hence the radiation doses to the different organs. Rather than have a single biokinetic model for the whole human body, it has been found convenient to have separate models to describe the *respiratory tract*, the *gastro-intestinal tract*, and the *systemic* behaviour of uranium, ie its organ distribution, retention and excretion after it is absorbed into blood. The most relevant of these for intakes of DU is the respiratory tract model, which is described in annexe A.

The spread of the radioactive material around the body results in radiation doses to all parts of the body, but as different materials, introduced by different routes, concentrate more in some tissues or organs than others, the radiation doses to the different tissues and organs vary. The estimated intakes of DU, and the predicted levels in various tissues, are therefore used to estimate the radiation absorbed doses to the individual organs (the *organ absorbed dose*). Some types of radiation are considered to be more damaging than others and the organ absorbed doses are converted to *equivalent doses* (measured in sieverts, Sv) which takes into account the judgements of the International Commission on Radiological Protection (ICRP) on the relative effectiveness of different types of radiation at causing cancer[2]. Radiation such as beta-particles and gamma-rays is given a weighting of one, whereas the weighting of the more damaging alpha-particles is set to 20.

Different tissues and organs vary in their sensitivity to radiation; the same equivalent dose may result in a substantial risk of cancer if it occurs to one organ but little risk if it occurs to another organ. Therefore, once the individual organ absorbed doses have been converted to the equivalent doses, the relative

[2] The amount of energy that ionising radiation deposits in a unit mass of human tissue (or other matter) is called the absorbed dose, and is expressed in a unit called the gray (Gy) or, for smaller doses, the milligray (mGy; 1000 mGy = 1 Gy). 1 Gy = 1 joule per kilogram. The equivalent dose is a measure that takes into account the differing extent of the damage to biological tissues by the same absorbed dose from different types of ionising radiation, and is measured in sieverts (Sv) or, for smaller doses, in millisieverts (mSv; 1000 mSv = 1Sv) or microsieverts (μSv; 1000 μSv = 1mSv). One Gy of absorbed dose from alpha-irradiation would therefore give a higher equivalent dose than 1 Gy from beta-irradiation, as the latter is less damaging to cells.

sensitivity to radiation of the different tissues and organs is then taken into account. The equivalent doses to the individual organs are therefore converted into the *effective dose* (measured in Sv or mSv) for the whole body by using other weighting factors based on the ICRP's judgement of the relative contribution of that organ to the total damage (cancers and a small contribution from hereditary effects) that results from the uniform irradiation of the whole body by an equivalent amount of penetrating low-LET radiation.

The overall risk (the *whole body risk*) of cancer is the sum of the risks of cancer to the different organs and tissues (the *organ risks*). However, in many cases, the whole body risk is dominated by the risk to one particular organ in which the radioactive material becomes concentrated. For example, iodine becomes almost exclusively concentrated in the thyroid and the whole body risk from an exposure to radioactive iodine would largely be the cancer risk to the thyroid. In other cases, the tissue or organ that receives the highest equivalent dose may be rather insensitive to radiation, and the risk of cancer may be dominated by an organ that receives a lower equivalent dose but is more prone to developing cancer when exposed to radiation.

With these models the following can be calculated for any defined intake of DU:

- The central estimate and worst-case for radiation doses to the organs from the different types of irradiation (alpha-particles, beta-particles, gamma-rays), the effective dose and the uptake to blood. All these quantities are calculated over the 50 years after the intake of DU to evaluate the long-term risks. Worst-cases for radiation dose tend to be higher when the rate at which the inhaled DU particles dissolves is low; the potential for chemical toxicity will be higher when this rate is high. However, uncertainties in the estimates of the intakes of DU are generally likely to be much greater than uncertainties in the effective radiation doses received per unit of intake.

- The maximum uranium kidney concentrations, which are used in our second report to assess risks from the chemical toxicity of DU.

The estimated intakes of DU (in mg) for the different Level I, II and III scenarios, and the corresponding estimated radiation doses (in mSv) are shown in table 1.

In calculating radiation doses resulting from the intakes in the different scenarios, account is taken of the route of intake (inhalation or ingestion), the estimated size distribution, density, and dissolution rate of the particles, and the level of exercise of the subject. Details are given in appendix 1. The committed effective dose (mSv) from intake of 1 mg DU oxide is nearly 1000 times lower for ingestion than for inhalation. There are several reasons for this: the residence time for particles passing through the gut is much shorter than for particles deposited deep in the lungs; it is calculated that a much smaller fraction of the radiation from alpha- particles reaches sensitive cells; and a smaller fraction of the intake is absorbed into blood and deposited in other organs such as kidney and bone. For the inhalation scenarios, the committed effective dose from intake of 1 mg DU oxide varies by about a factor of two between the different types of exposure.

The central estimate radiation doses are less than 0.1 mSv, except for the Level I inhalation exposure, which is about 20 mSv. 20 mSv corresponds to the current annual dose limit for workers in the UK and about nine times the average dose from natural background radiation in the UK (2.24 mSv). It also corresponds to twice the annual dose from radon and its decay products at the 'Action Level' at which householders in the UK are advised to consider remedial measures to reduce exposure (NRPB 1990).

However, there are very large uncertainties associated with major parts of the assessment. These uncertainties stem partly from limited availability of information (in particular, reliable estimates of the intakes of DU from penetrator impacts), and partly from the inherent variability of the processes involved. The intake of material by a person will depend on the amount of DU involved, details of the event (impact or fire), the interaction of the person with the dispersed material, and the location and duration of exposure. For all of these there are wide ranges of possible values. There are also uncertainties in some of the values that need to be included in the biokinetic models, such as the rate at which the particles of DU dissolve.

The worst-case estimates are much higher than the corresponding central estimates, reflecting the large uncertainties in the values of some of the key parameters. Worst-case estimates of intakes are typically two to three orders of magnitude higher than the corresponding central estimates. Worst-case estimates of dose also used worst-case estimates of dose per mg,which were between about three and ten times higher than the corresponding central estimates. Nevertheless, only in a few cases are estimated worst-case radiation doses greater than 20 mSv. These are Level I inhalation exposures and both Level II and Level III inhalation exposures to resuspended dust inside vehicles. The worst-case doses for Level I and Level II inhalation exposures are, however, substantial at around 1 Sv.

Table 1. Summary of estimated intakes (mg) and effective doses (mSv) for the different battlefield scenarios. Doses above 20 mSv are highlighted in bold

Scenario	Central estimate		Worst-case	
	Intake mg	Dose mSv	Intake mg	Dose mSv
Level I inhalation of impact aerosol	250	**22**	5000	**1100**
Level II inhalation of resuspension aerosol within contaminated vehicle	10	0.5	2000	**440**
Level II ingestion within contaminated vehicle	5	0.0005	500	0.3
Level III inhalation of resuspension aerosol within contaminated vehicle	1	0.05	200	**44**
Level III ingestion within contaminated vehicle	0.5	0.00005	50	0.03
Level III inhalation of plume from impacts	0.07	0.004	5	2.8
Level III inhalation of plume from fires	0.05	0.004	2	1.2
Level III inhalation of resuspension from ground	0.8	0.03	80	18

2.4 External exposure to radiation from depleted uranium

It is relatively straightforward to determine external irradiation doses from DU, as they can be easily calculated and can be directly measured. A sheet of paper would block the alpha-particles from DU, and even if DU is in direct contact with skin, the alpha-particles will not penetrate the inert outer skin layer over most of the body. Beta-particles provide exposure when penetrators are handled, which can be reduced by protective gloves. Exposure to gamma-rays will also occur during handling of penetrators and will be the main direct external exposure from unfired DU rounds (eg to crews in tanks loaded with DU munitions). Civilians, and particularly children attracted by fragments of penetrators, are also at risk of external exposure from both beta-particles and gamma-rays.

Fetter and von Hippel (1999) carried out the only comprehensive assessment of the hazards from DU munitions that has been published in a peer-reviewed journal. Furthermore, they provide clear information about the methods and assumptions made. According to Fetter and von Hippel (1999): "The theoretical maximum whole body gamma-irradiation dose rate from external exposure to DU is 2.5 millirem per hour. Dose rates in this range might be experienced by a person surrounded by DU munitions". One millirem corresponds to 0.01 mSv, hence the maximum possible dose rate is 0.025 mSv per hour, or 0.6 mSv per day. The highest external exposures likely to arise in practice are in a vehicle fitted with DU armour and carrying DU ammunition. According to DRPS (1993), measurements demonstrated that personnel would need to be in a fully DU loaded tank for 1500 hours to reach the then current whole body occupational dose limit of 50 mSv.

We are not aware of any independent direct exposure measurements, but according to US Army measurements cited by Harley et al (1999), the whole-body dose rate in or near a tank fully loaded with DU munitions is less than 0.002 mSv per hour. Thus driving such a tank for 1000 hours gives a dose similar to the typical annual dose from all sources of ionising radiation (on average, 2.6 mSv per year in the UK).

Fetter and von Hippel (1999) estimated that the dose rate to a person standing on ground uniformly contaminated with 1 tonne DU per square kilometre, (ie 1 gram per square metre of ground, which they estimate to be a reasonable upper-bound for a battlefield area) would be 0.01 mSv per year, which is one-tenth of the dose-rate from the uranium naturally present in soil.

The dose-rate to skin from direct contact with DU is much higher, at about 2.5 mSv per hour, mostly from beta-particles (Fetter and von Hippel 1999). Skin is relatively insensitive to radiation, so that even continuous contact (keeping a piece of DU in a pocket or wearing it as jewellery) is unlikely to produce a radiation burn or other short-term effect. Such effects require doses of a few sieverts (Sv), delivered over a short time, but at 2.5 mSv per hour, the DU would need to be in contact for weeks to give such doses. There would, however, be expected to be an increased risk of skin cancer and such contact should be avoided. Direct skin contact with a DU penetrator for 400 hours (about 17 days) would increase the risk of skin cancer (fatal and non-fatal) by perhaps 40% (UNSCEAR 2000).

2.5 Internal exposure to depleted uranium

Embedded fragments of DU shrapnel could give rise to high exposures, but there appears to be little information available to assess the hazard, other than the follow-up study on the US friendly fire victims (annexe B), and related ongoing animal experiments (Pellmar et al 1999, Hahn et al 2000).

Inhalation seems likely to give rise to the most widespread uncontrolled exposures on the battlefield, and could give rise to high exposures close to impacts by DU penetrators. We have therefore made a detailed assessment of the likely intakes of DU by the inhalation route. The extent to which there are suspended particles in the air after an impact will depend on their sizes, on the way they are dispersed by the initiating events, and on local conditions. The weather may be important, since rain will tend to speed particle precipitation, and resuspension will be easier from dry ground than from wet. How far the particles reach within the respiratory system will depend on their size. Some of the particles may be very small, with diameters less than a micron, such as those formed by nucleation and growth from a vapour. These may penetrate deep into the lungs. Fragmentation products will usually be bigger than a few microns. The coarser particles formed by fragmentation will not penetrate deep into the lungs and will be cleared by mucous flow and swallowed. The description of suspended particles as an aerosol usually includes both the very fine particles and those coarser particles which are far less likely to penetrate deep into lung. There is only limited information available on these important size distributions.

There are a number of ways in which the amounts of DU inhaled on the battlefield can be assessed, along with the resulting radiation doses to the organs:

- measurements have been made of the properties (concentration in air, size distribution, solubility, chemical composition) of the particles formed when a DU penetrator hits armour plate or when uranium is exposed to fire;
- calculations can be made of how the particles disperse in air, how much is inhaled, and how much is deposited in the lungs and is subsequently retained in the various organs of the body;
- some measurements have been made on personnel exposed to DU during the Gulf War, which can be used to estimate how much they might have inhaled.

A simple alternative approach to assessing the likely upper limit, which can be applied to intakes of weakly radioactive materials such as DU, is to consider the feasibility of inhaling the large mass of dust required to give a substantial radiation dose. This puts the whole assessment into perspective.

Such an approach was taken by the United Nations Environment Programme Depleted Uranium Desk Assessment Group (UNEP 1999, Snihs and Åkerblom 2000). They noted in relation to acute exposures that: "An instantaneous intake by breathing of more than 1 gram dust is unendurable, and assuming 10% DU, means a maximum intake of 100 mg DU". Inhalation of 1 mg DU would typically lead to an effective dose of the order of 0.1 mSv and hence, if we accept that breathing more than one gram of dust is "unendurable", it is unlikely that anyone would incur an effective dose greater than 10 mSv from acute inhalation of DU. Similar reasoning can be applied to protracted intakes. It is very unlikely that any dust except very close to a point of impact would be more than 10% DU. An air concentration of 100 mg per cubic metre would be noticeably dusty, and normal breathing rates are of the order of 1 cubic metre per hour. Hence to inhale 100 mg DU, someone would need to inhale dusty air that was heavily contaminated with DU for 10 hours. Hence, according to the assumptions used by UNEP, it is unlikely that anyone would incur a dose greater than 10 mSv from inhalation of DU as a result of battlefield use or a fire involving DU. This may be a reasonable assumption in most situations, including Kosovo where only small calibre DU rounds were used.

However, it is possible that larger amounts of DU, with correspondingly higher radiation doses, could be inhaled in exceptional circumstances, in particular within a heavy-armour tank pierced by a large calibre DU penetrator. It seems quite possible that the airborne dust within such a struck vehicle could be considerably more than 10% DU. It is also not clear whether breathing 1 gram of dust would be "unendurable" for soldiers confined, possibly trapped, within a vehicle immediately after an impact. The worst-case Level I inhalation intake of 5 grams DU is based on breathing air carrying 50 grams of dust per cubic metre at 50 litres per minute for one minute, followed by 5 grams of dust per cubic metre at 50 litres per minute for 10 minutes (annexe C). Breathing irritants can induce a vagal-mediated laryngeal reflex that severely constrains breathing (and thus intake). Irritancy is affected by particle size as well as chemical composition and temperature, and so is hard to estimate. Studies with rodents show that air carrying non-irritant dust at 5 grams per cubic metre can be breathed for several hours (OECD 1981), but further information would be required to determine whether air with 5 grams per cubic metre of the DU-containing dust could be breathed without inducing the reflex. The reflex is clearly much more likely to be induced at a concentration of 50 grams per cubic metre, so that the 50% of the worst-case intake that assumes this concentration might be unlikely. However, it does seem likely that worst-case intakes of more than 1 gram DU are possible.

3 Radiation and cancer

3.1 The International Commission on Radiological Protection (ICRP) models

The ICRP models allow one to predict, from data on the exposure to radiation, the overall risk of cancer, and the risk of individual types of cancer. The detailed assessment of the risks of cancer for various Level I, Level II and Level III exposures, and a more complete description of the assumptions and methods of the ICRP approach, are given in appendix 2.

The ICRP model of radiation risks has been developed and refined over many years and is an internationally accepted method for estimating the risks of fatal cancers from irradiation. It has been adopted and used by most radiation protection professionals and organisations around the world, and forms the basis of legally enforced standards in most countries, including member states of the EU. The ICRP model has largely been developed by using the observed cancer rates in the survivors of the atomic bombs in Japan where there were high dose, high dose-rate, exposures to penetrating external radiation (mainly gamma-rays). The risks from these exposures have been extrapolated to low doses, and low dose-rates, and to other types of radiation such as alpha-irradiation.

Clearly this procedure involves many assumptions and approximations. One of the most important is the linear no-threshold (LNT) assumption, which implies that any dose, however small, carries some risk and that the risk is directly proportional to dose for low-level exposures. There are grounds for believing that the LNT model is more secure for high-LET (alpha-particle irradiation) than for low-LET irradiation (for example gamma-rays) since the dose is delivered in the form of a relatively small number of densely ionising tracks through only a small proportion of the cells within the exposed organ. Another important assumption is that DU, being an alpha-particle emitter, should be expected to be carcinogenic along with other similar radionuclides that are known to be. However according to the International Agency for Research on Cancer (IARC), the direct evidence for the induction of cancer by inhaled or ingested natural uranium is weak, so that for DU is likely to be even weaker (IARC 2001).

3.2 Radiation as a cause of cancer

It is generally accepted that cancers arise in tissue stem cells when their daughter cells are transformed into cells with an ability to divide in an uncontrolled manner. Cancers arise from a normal cell by a series of rare mutational events, or other changes, one or more of which can be induced by radiation. Cancer is therefore the end result of a multistage process and, following exposure to radiation, typically takes many years to appear because a sequence of several rare chance events has to occur. The requirement for a number of rare chance events also explains the variability in the period between radiation exposure and the appearance of cancer among individuals. Cancers involving different tissues have different gestation periods. For example, cancers arising from radiation normally take longer to appear for lung cancer and sarcomas than for leukaemia. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 2000) has recently completed a global review of radiation-induced cancers. This includes an assessment of the cancer risks for different organs resulting from exposure to alpha-particle irradiation; a summary is included in appendix 2.

There is good evidence that alpha-particle irradiation induces cancers in lung, bone and liver and some evidence for increased risk of leukaemia; there is relatively little direct evidence for an increased risk in the case of cancer of the kidney or lymphoma or myeloma. There is no direct evidence that inhaled or ingested DU or natural uranium causes cancer in humans, although internal exposure to all alpha-irradiation is expected to cause cancer but the risk becomes increasingly small at low doses. The lack of evidence of carcinogenicity of uranium (and DU) is probably due to the difficulties of detecting statistically significant excesses of cancers because of the lack of large cohorts that have sufficiently high exposures to this weakly radioactive material without having other exposures, for example to external radiation. In contrast, there is good evidence of excess lung cancers in long-term studies of large cohorts of miners (mainly uranium miners) who have been exposed to high doses of internal alpha-irradiation from the highly radioactive radon gas and its decay products that accumulate in some mines.

3.3 Estimates of radiological risk of fatal cancers

This section presents the estimated radiation risks of fatal cancer that result from a selection of the Level I, II and III exposure scenarios. These estimates are obtained by multiplying together the intakes of DU, the radiation doses for individual tissues and organs for a unit intake of DU, and the ICRP's 'nominal probability coefficients' for individual tissues or organs or for whole body risk. These coefficients adjust the cancer risks to each tissue from a particular dose of radiation to reflect their differing sensitivities to radiation-induced cancers. They are based on cancer risks from radiation for a 'world population' and vary for many types of cancers between different populations. In each case this leads to a central estimate and a worst-case increased (or excess) lifetime risk of fatal cancer per individual in the exposed

population. Note that ICRP gives a probability coefficient for an organ if there is sufficient evidence to calculate one. These are based mainly on the Japanese survivors of the atomic bombs, who were whole-body irradiated; all organs received similar doses. Thus the lack of a probability coefficient implies no significant excess of cancer has been observed, and that the risk is considered to be lower than in the organs for which one is given.

A risk of 1 per hundred (0.01 or 10^{-2}) from the radiological effects of exposure to DU implies that, on average, over the life of an individual, their chance of dying of cancer is increased by 1%. Therefore, if the normal lifetime risk of dying of cancer were 25%, the 1% excess risk would increase the overall risk to 26%. The risk can also be applied to a population, in which case 1 individual out of a hundred who received the radiation exposure corresponding to a 1% excess risk would be expected to die of cancer attributed to the exposure (of course many of the hundred would die of cancer unrelated to the radiation exposure as cancer is such a prominent cause of death). The lifetime excess risks of fatal cancer are provided for the whole body (ie the overall risk of any fatal cancer), and for individual fatal cancers (eg of the lung or colon) in the following three sections.

3.4 Risks from Level I inhalation of impact aerosols

The central estimate and worst-case risks for fatal cancers and for total health detriment (which are typically slightly higher as they include some allowance for non-fatal cancers and a small assumed hereditary risk) are shown in table 2 for the estimated intakes of 250 mg (central estimate) and 5,000 mg (worst-case) of DU. Note that the worst-case doses use not only the worst-case intakes, but also the worst-case doses per unit intake, so the differences in risk may be more than the factor of 20 differences in the intakes. The doses in mSv to the most exposed tissues and organs are also shown. The assumptions about hereditary damage are based on animal experiments as these effects have not been clearly documented in the children of the survivors of the atomic bombs in Japan.

For all individual tissues or organs the equivalent dose (and therefore the risk) is dominated by alpha-particle irradiation. For both the central estimate and the worst-case, the greatest risk is to the lungs. The estimated risks to other organs are much less than 1% of these lung risks.

For the central estimate, the excess risk of fatal lung cancer is about 1 per thousand; excess risks of other cancers are less than 5 per million. For the worst-case, the risk of lung cancer is 6.5 per hundred. The next greatest risks for the worst-case estimate are to red bone marrow and bone surfaces (potentially leading to leukaemia and osteosarcoma), each of which are about 5 per hundred thousand.

The most notable radiation doses are to the lung, and also to the associated lymph nodes. The risks of irradiation of the latter are very uncertain because of a lack of evidence, and ICRP (1991) has not provided estimates of their susceptibility to radiation-induced cancer. The very high dose of 91 Sv to the thoracic lymph nodes in the worst-case scenario is due almost entirely to alpha-particles, and this corresponds to approximately 9 alpha-particle traversals per 'cell location' during the extended time of the irradiation (perhaps as long as a lifetime). Individual lymphocytes are unlikely to survive even one or two alpha-particle traversals, but in a lymph node the killed cells would normally be replaced by circulation. The possibility exists over the extended time course of exposure for some lymphocytes, and lymphoid and haemopoietic precursor cells, to survive alpha-particle traversals while circulating through these lymph nodes. The high accumulated equivalent dose in thoracic lymph nodes corresponds to 0.09 Gy per year from alpha-particles, and this is equivalent to less than 1 Gy per year of low-LET irradiation per year for organ damage. Hence, some accumulated organ injury might be experienced at the very highest dose levels. Indeed, in experiments in dogs where alpha-emitting particles lodged in the lymph nodes delivered accumulated alpha-particle doses ranging from 1 - 4,000 Gy, the observed effects were atrophy and fibrosis of the lymph nodes (Muggenburg et al 1999).

Lymph node cancers were not observed in dogs by Muggenburg et al (1999), suggesting that the high doses to the thoracic and extra thoracic lymph nodes from the worst-case Level I exposures to DU are unlikely to lead to cancers of these tissues (Hahn et al 1999a). However, any lymphocytes, and lymphoid and haemopoietic precursor cells, surviving alpha-particle traversals while passing through lymph nodes could contribute slightly to the overall risk of leukaemia. This phenomenon might have contributed to the small, largely myeloid, leukaemia incidence observed in, for example, patients exposed to Thorotrast (a thorium compound once used as a diagnostic radiation contrast medium) where alpha-particle emitting particulates were retained in lymph nodes and elsewhere.

3.5 Risks from Level II intakes from entry into vehicles

These exposure scenarios include both inhalation and ingestion from entry into contaminated vehicles. In many cases Level II exposures can be minimised by suitable precautions, including protective clothing. The calculated risks of fatal cancer and total health detriment are given in table 3 for the estimated DU intakes from the central estimate and worst-case scenarios.

Table 2. Risks from Level I inhalation of impact aerosols

	Central estimate		Worst-case	
DU intake	250 mg		5,000 mg	
	Risk of fatal cancer	Total detriment	Risk of fatal cancer	Total detriment
Whole body	$8.8 \times 10^{-4*}$	1.2×10^{-3}	4.4×10^{-2}	$6.2. \times 10^{-2}$
Individual tissues				
Bladder	9.6×10^{-7}	9.6×10^{-7}	1.0×10^{-5}	1.0×10^{-5}
Red bone marrow	4.9×10^{-6}	1.0×10^{-5}	5.2×10^{-5}	1.1×10^{-4}
Bone surfaces	4.7×10^{-6}	7.1×10^{-6}	5.0×10^{-5}	7.5×10^{-5}
Breast	6.5×10^{-7}	1.2×10^{-6}	6.7×10^{-6}	1.2×10^{-5}
Colon	3.1×10^{-6}	3.7×10^{-6}	3.4×10^{-5}	4.1×10^{-5}
Liver	1.9×10^{-6}	2.0×10^{-6}	2.0×10^{-5}	2.2×10^{-5}
Lungs	1.2×10^{-3}	1.1×10^{-3}	6.5×10^{-2}	6.1×10^{-2}
Oesophagus	9.6×10^{-7}	7.6×10^{-7}	1.0×10^{-5}	8.0×10^{-6}
Ovary	3.2×10^{-7}	4.8×10^{-7}	3.3×10^{-6}	5.0×10^{-6}
Skin	8.0×10^{-8}	1.2×10^{-7}	8.3×10^{-7}	7.5×10^{-6}
Stomach	3.5×10^{-6}	3.2×10^{-6}	3.7×10^{-5}	3.4×10^{-5}
Thyroid	2.4×10^{-7}	4.8×10^{-7}	2.5×10^{-6}	5.0×10^{-6}
Tissues with highest equivalent doses				
Lungs	178	mSv	9,500	mSv[1]
Extra thoracic airways	53	mSv	2,400	mSv
Extra thoracic lymph nodes	12	mSv	4,150	mSv
Thoracic lymph nodes	150	mSv	91,000	mSv[2]
Bone surfaces	12	mSv	125	mSv
Kidney	4.3	mSv	45	mSv
Liver	1.6	mSv	17	mSv
Red bone marrow	1.2	mSv	13	mSv
Effective dose	22	mSv	1,100	mSv

* See footnote to table 2 in appendix 2.
[1] This lung dose is due to 0.47 Gy from alpha-particles and 0.09 Gy from low-LET radiation, delivered over >50 years.
[2] This thoracic lymph node dose is due to 4.6 Gy from alpha-particles and 0.39 Gy from low-LET radiation, delivered over >50 years.

For all individual tissues or organs the equivalent dose (and therefore the risk) is dominated by alpha-particle irradiation. The alpha-particles are responsible for >89% of the equivalent dose in all cases, and in most cases >98%.

For both the central estimate and the worst-case, the greatest risk is for lung cancer. The estimated risks of cancers to other organs are less than 1% of the lung risk. For the central estimate, the risk of lung cancer is 2.5 per hundred thousand; the excess risk of other cancers is less than 1 per ten million. For the worst-case, the risk of lung cancer is 2.4 per hundred. The next greatest risks for the worst-case are for bone marrow (leukaemia), bone cancer, colon cancer and stomach cancer, and are all roughly 10 per million.

The lower part of Table 3 gives the tissues or organs that receive the greatest equivalent doses of radiation. Most notable in the worst-case are doses to lung (3.6 Sv), thoracic lymph nodes (38 Sv) and extra-thoracic lymph nodes (1.1 Sv); these doses are dominated by the intakes from inhalation.

3.6 Risks from Level III intakes from smoke plumes

This exposure scenario includes inhalation of smoke plumes both from DU impacts and fires. A table of risks is not shown as they are all very low, but can be found in table 4 of appendix 2. For both the central estimate, and the worse case, the greatest risk amongst the tissues or

Table 3. Risks from Level II intakes (inhalation + ingestion) from entry into contaminated vehicles

	Central estimate		Worst-case	
DU intake	10 mg (inhalation) + 5 mg (ingestion)		2000 mg (inhalation) + 500 mg (ingestion)	
	Risk of fatal cancer	Detriment	Risk of fatal cancer	Detriment
Whole body	$2.1 \times 10^{-5*}$	2.9×10^{-5}	1.8×10^{-2}	$2.5. \times 10^{-2}$
Individual tissues				
Bladder	1.4×10^{-8}	1.4×10^{-8}	2.5×10^{-6}	2.5×10^{-6}
Red bone marrow	7.0×10^{-8}	1.5×10^{-7}	1.3×10^{-5}	2.7×10^{-5}
Bone surfaces	6.6×10^{-8}	9.9×10^{-8}	1.2×10^{-5}	1.8×10^{-5}
Breast	9.0×10^{-9}	1.6×10^{-8}	1.7×10^{-6}	3.1×10^{-6}
Colon	6.4×10^{-8}	7.7×10^{-8}	1.1×10^{-5}	1.3×10^{-5}
Liver	2.7×10^{-8}	2.9×10^{-8}	5.0×10^{-6}	5.5×10^{-6}
Lungs	2.5×10^{-5}	2.4×10^{-5}	2.4×10^{-2}	2.3×10^{-2}
Oesophagus	1.4×10^{-8}	1.1×10^{-8}	2.5×10^{-6}	2.0×10^{-6}
Ovary	4.5×10^{-9}	6.8×10^{-9}	8.3×10^{-7}	2.1×10^{-6}
Skin	1.1×10^{-9}	1.7×10^{-9}	2.1×10^{-7}	3.1×10^{-7}
Stomach	5.2×10^{-8}	4.7×10^{-8}	9.3×10^{-6}	8.4×10^{-6}
Thyroid	3.4×10^{-9}	6.8×10^{-9}	6.2×10^{-7}	1.2×10^{-6}
Tissues with highest equivalent doses				
Lungs	3.7	mSv	3,600	mSv[1]
Extra thoracic airways	3.7	mSv	620	mSv
Extra thoracic lymph nodes	0.83	mSv	1,100	mSv
Thoracic lymph nodes	3.4	mSv	38,000	mSv[2]
Bone surfaces	0.16	mSv	31	mSv
Kidney	0.062	mSv	12	mSv
Liver	0.023	mSv	4.2	mSv
Red bone marrow	0.017	mSv	3.2	mSv
Effective dose	0.52	mSv	440	mSv

* See footnote to table 2 in appendix 2.
[1] This lung dose is due to 0.18 Gy from alpha-particles and 0.04 Gy from low-LET radiation, delivered over >50 years.
[2] This thoracic lymph node dose is due to 1.9 Gy from alpha-particles and 0.16 Gy from low-LET radiation, delivered over >50 years.

organs that are considered by the ICRP models is to the lungs. The estimated risk to any other tissue or organ is much less than 1% of the lung risk. For the central estimate, the risk of lung cancer is about 4 per ten million. For the worst-case, the risk of lung cancer is about 2 per ten thousand.

The estimated equivalent doses to tissues and organs from Level III exposures are mostly very much less than the average annual equivalent doses received from natural background radiation (in the UK ~1 mSv to all tissues or organs, apart from the lung which is assigned ~10 mSv from radon and its short-lived progeny, giving a total effective dose of 2.24 mSv per year). Notable exceptions for the worst-case DU exposures are lung (28 mSv) and thoracic lymph nodes (280 mSv).

3.7 Comparison with natural background radiation

The average annual effective dose from all sources of ionising radiation to members of the UK population has been estimated to be about 2.6 mSv (Hughes 1999). The majority of this (ie 2.24 mSv) is from natural background sources, with the balance being due to medical (0.37 mSv), occupational (0.006 mSv), fallout (0.004 mSv), disposals (0.003 mSv) and consumer products (0.001 mSv).

Tables 4 and 5 compare the annual effective doses from natural radiation to the total effective doses received from intakes of DU from Level I, II and III scenarios. The highest central estimate exposure from Level I scenarios, from which most of the committed effective dose is

Table 4. Average annual effective dose from natural sources

Cosmic rays	0.32	mSv/year
Terrestrial gamma rays	0.35	mSv/year
Internal long-lived radionuclides	0.27	mSv/year
Internal radon and short-lived progeny	1.20	mSv/year
Internal thoron and short-lived progeny	0.10	mSv/year
Total	2.24	mSv/year

Table 5. Estimated effective doses from DU exposure scenarios

	Central estimate		Worst-case	
Level I exposure	22	mSv	1,100	mSv
Level II exposure	0.52	mSv	440	mSv
Level III exposure to smoke plumes	0.007	mSv	4.0	mSv
(Level III exposure to all pathways)	(0.09	mSv)	(66	mSv)

received within the first year, corresponds to about ten times the average received in one year from natural sources of radiation. The Level II and III scenarios give central estimate doses that are lower than the annual natural dose. The exposure from the Level I worst-case exposure is about five hundred times the annual natural dose, whereas the worst-case Level II and Level III exposures give, respectively, doses that are two hundred times, and twice, the normal annual levels of radiation.

3.8 Chemical versus radiological action of depleted uranium

Our analysis above assesses only the radiological risk from DU, following standard internationally accepted methods. We have not included chemical effects from DU, either in isolation, or in combination with the radiation. Toxic chemical effects of uranium on the kidney and other organs will be included in our second report. However, the potential for effects (including cancer) resulting from the chemical action of uranium at the DNA and cellular levels is considered here.

Experiments with a human osteoblast-like cell line *in vitro* have shown that soluble or insoluble DU can induce malignant transformation of the cells (Miller et al 1998a, 2001). DU has been reported also to damage the genetic material of cells, inducing DNA strand breaks and chromosome rearrangements (Miller et al 1998a, 2000). Studies of rats with embedded DU pellets have shown changes that are associated with carcinogenesis, and the uranium-containing urine was able to produce mutations in bacterial systems designed to identify chemicals that can damage the genetic material (the Ames bacterial reversion assay; Miller et al 1998b, 2000). Although many, or all, of the above effects may be expected from the radiation exposure from DU, there are reasons to suggest that they may be chemical effects as they were very much more frequent than might be expected from the very small proportion of cells that were hit by an alpha-particle. Similar frequencies of malignant transformations are observed with the non-radioactive heavy-metal carcinogens nickel and lead (Miller et al 1998a). The other indicators of damage to the genetic material are also observed with nickel and its alloys (with tungsten and cobalt), although none is detected with the inert metal tantalum (Miller et al 2000). In the vicinity of particles or fragments of DU, cells will be subject to these putative chemical effects of DU which might increase their sensitivity to the occasional passage of alpha-particles, or the more frequent but less damaging beta-particles or gamma-rays. Further studies would be required to examine the possibility of synergy between the chemical effects and radiation effects of DU.

3.9 Embedded shrapnel

The dose and dose-rate to tissue in immediate contact with an embedded fragment of DU are extremely high. As evaluated in annexe B, tissue in contact with a piece of DU shrapnel is irradiated with alpha-particles at a dose-rate of about 1300 Gy per year (ie 3.6 Gy per day)[3]. The alpha-particle dose-rate decreases gradually away from the surface, until at about 30 micrometres distance it drops to zero because of the small ranges of the alpha-particles. In this very thin irradiated shell of tissue most of the cells would be sterilised on a daily basis. For example, this dose would correspond to about

[3] At these very large doses, to only a small volume of tissue, we have directly used the absorbed dose to the irradiated tissue (in gray; Gy), as the averaging procedure and the assumptions underlying the concept of equivalent dose (in Sv) may be less appropriate in this case.

7 alpha-particle traversals per cell nucleus, which would almost certainly kill most types of cell. This would be a continuing process if the tissue is capable of replacing the killed cells. Killing is protective as dead cells cannot become cancerous but there would be some genetically damaged cells surviving in the regions near the ends of the ranges of the alpha-particles. Cells beyond this could possibly also be damaged by 'bystander' effects (damage to unirradiated neighbouring cells that is transmitted from a directly irradiated cell) if these occur. The microenvironment of very high local tissue damage, and possibly high levels of replacement of dead cells, may well influence the potential of any mutated viable cells to become cancerous. Alternatively, and particularly in non-renewing tissues such as muscle, a fibrotic layer may develop around the DU shrapnel, as has been reported around DU implants in rats (Hahn et al 1999b). If this layer is more than about 30 micrometres thick it would shield the tissue from further alpha-particle irradiation. In addition the cells around shrapnel would be continuously bathed in dissolved uranium, leading to possible chemical effects from the heavy metal and synergy between the chemical and radiation effects.

As described in annexe B, tissue in the vicinity of a piece of DU shrapnel is also exposed to low-LET irradiation, mostly from beta-particles emitted by the short-lived decay products of the uranium. These beta-particles can travel up to about 1.1 centimetres in tissue and the average dose rate within the region around a shrapnel fragment of 1 centimetre diameter is estimated to be about 69 Gy per year. The dose rate is substantially greater than this near the surface of the shrapnel and decreases quite rapidly to near zero at a distance of 1.1 centimetres. 69 Gy per year corresponds to about 0.2 Gy per day, which implies that, on average, each cell around the shrapnel is irradiated by a few hundred separate electrons per day. Such a dose rate is unlikely to kill the cells and an accumulation of viable genetically damaged cells may be expected. Because the amount of tissue exposed to beta-particles is so very much greater than that exposed to alpha-particles, and because of the survival of the beta-exposed cells, it may be that the tissue exposed to beta-particles represents a greater long-term risk than the smaller number of surviving cells damaged by the alpha-particles. Most shrapnel is reported to be embedded in muscle but, as pointed out in annexe B, the beta-radiation from a DU fragment could extend into tissues that are more prone to the development of cancer than muscle. For example, a fragment embedded near a bone could expose a small fraction of the red bone marrow to beta-radiation. However, over the 1.1 cm range, the small dose from beta-particles would decrease with distance from the shrapnel and passage through bone would further attenuate the dose reaching the radiation-sensitive cells within the bone marrow.

In view of the various possibilities, and the variety of tissues of interest, the risk from the radiological effects of DU fragments cannot be generalised. Conventional ICRP approaches of averaging the dose throughout the tissue, with the underlying assumptions of linearity of dose response and additivity of equivalent doses, are unlikely to be helpful in assessing the risks from the large and inhomogeneous doses that arise from DU shrapnel. Implants of fragments of DU in the muscle of male Wistar rats were reported at a recent meeting to have led to significant increases in tumours (Hahn et al 2000). The authors concluded that their results indicate that DU, in sufficient dosage, reduced weight gain in the rats, caused a local tissue reaction and caused local tumours. Further evaluation of these animal studies must await their publication in a peer-reviewed journal. Studies on US soldiers with shrapnel have so far detected no major health consequences attributable to the retained DU (McDiarmid et al 2000).

4 Epidemiological evidence

4.1 Epidemiological estimates of risk and ICRP risk estimates from alpha-irradiation

One criticism of the ICRP approach to cancer risks used here is that it is based to a considerable extent on estimating cancer risks from the high dose, high dose-rate, external low-LET gamma-irradiation from the atomic bombs in Japan, and there are many assumptions and uncertainties in extrapolating from these exposures to estimate cancer risks from internal exposure to high-LET alpha-particle irradiation.

Lung exposures provide the only case to date where there has been sufficient human data for both low-LET and high-LET radiation to allow a direct comparison of the two general approaches to the estimation of cancer risk for internal alpha-emitters, one based on direct epidemiological observations of cohorts of uranium underground miners who were occupationally exposed to radon, and the other based on the standard ICRP dosimetric approach that we have applied. In this particular case the two estimates of the risk of lung cancer differ by a factor of about 3 (or in the range 2-5 depending on the particular conditions considered) (Birchall and James 1994, Porstendörfer and Reineking 1999, NAS 1999.) The direct estimate from the epidemiological studies of exposed miners gives the lower risk. This difference may be regarded as a close agreement in view of the very many assumptions and uncertain parameter values inherent in the two approaches.

The most extensive studies of the hazards of internalised alpha-particle emitters relate to the inhalation of radon gas (half-life of 3.8 days), and its short-lived decay products (half-lives of < 29 minutes) attached to aerosols, which provide exposure predominantly to the lung, and within the lung mainly to the bronchial tree. Inhalation of DU aerosols differs significantly as small particles can be retained deep in the lungs, and be translocated from the lung to the associated lymph nodes where they remain for many years. The accuracy of the risks calculated using ICRP models for alpha-emitting particles that remain within tissue for many years has been challenged by some groups, particularly in the context of the emissions from Sellafield and from the radioactive releases resulting from the accident at Chernobyl. We recognise some of these uncertainties in extrapolating to situations where radioactive particles or shrapnel are retained in tissue (although particles of DU are very weakly radioactive compared to the 'hot particles' of plutonium and other highly radioactive alpha-emitting materials from Sellafield or Chernobyl). We are not, however, aware of any rigorous peer-reviewed studies that indicate any substantial under-estimation of the risk of internally-deposited radioactivity by the ICRP approach. A cautious approach would be to consider the possibility that there is an order of magnitude in the uncertainty of the ICRP estimates of risk from inhalation of DU, and that they could therefore slightly overestimate, or underestimate, these risks.

Most of the concern surrounding discharges from Sellafield has focused on the risk of leukaemia. The risk assessments for intakes of DU predict that the risk of leukaemia is far lower than the risk of lung cancer. This risk estimate is based largely on the doses to the red bone marrow and does not include any additional risk of leukaemia from the very high doses to the thoracic and extra thoracic lymph nodes predicted with worst-case assumptions. There is no convincing evidence that alpha-particle irradiation of these lymph nodes leads to any material increase in the risk of leukaemia, but this is an area of some uncertainty where more information is required. Even if the lifetime risk estimates for leukaemia were one hundred fold greater than our central estimate, we would only expect an excess risk of leukaemias of about 2 per thousand from a Level I exposure. It is therefore considered unlikely that any leukaemia would be observed among soldiers with Level I exposures.

4.2 Occupational exposure to uranium

The ICRP models that are used to estimate the excess of cancers expected after exposure to a known amount of radiation are largely derived from studies of the survivors of the atomic bombs released in Hiroshima and Nagasaki. As discussed above, it has been suggested by some that these estimates, based on high doses, and high dose-rates, of external penetrating radiation (mainly gamma-rays), may not be directly applicable to the situation that occurs when particles of alpha-emitting radioactive materials are inhaled and directly irradiate internal tissues.

We have therefore considered the epidemiological studies of the health of workers in the uranium industry. If inhalation of particles of DU was very much more likely to cause cancer than the estimates of risk we obtain from applying the ICRP models, might we not expect to see a marked excess of cancers in workers in the uranium industry, compared to that in the general population?

The health of workers who are occupationally exposed to uranium has been studied extensively. These are mainly uranium miners and workers in the nuclear industry. Studies of uranium miners have identified an increased risk of lung cancer. However, these excess

lung cancers are attributed to the inhalation of radon gas that accumulates within underground uranium mines. Radon-222 is a decay product of uranium-238; radon and its short-lived decay products form a sequence of highly radioactive alpha-emitters. Little exposure to radon gas arises from DU, so these studies are not directly relevant to the risks from uranium exposure. Workers in the nuclear industry have however been engaged in the extraction, milling and machining of uranium, in the absence of appreciable exposure to radon over a 50 year period, and the study of their health should shed some light on the long-term effects of such exposure.

There are many similarities between the exposures to uranium in industry and those that occur on the battlefield. In both situations there is the possibility of inhalation of uranium dust or aerosols, and of ingestion, with the associated risks from the retention of radioactive particles in the lung, or in associated lymph nodes, and from the toxic chemical effects of uranium. There are also likely to be important differences. For example, natural uranium is more radioactive than DU, which would make excess cancers more likely to occur than with exposure to the same mass of DU. The size distribution and chemical form of the uranium in dust or aerosols may differ, although the uranium oxides within DU aerosols are likely to be present in some stages of uranium processing**.** These data are important as they represent the most comprehensive information available relating to long-term exposures to uranium.

Our investigation has focused on fourteen major studies of the health of workers in the uranium industry, eleven relating to workers in the US and three to workers in the UK. The total number of workers involved is about 120,000 among whom 33,000 deaths have been reported. For each study, the total number of deaths from all causes, from all cancers, and from thirteen different forms of cancer, and from genitourinary disease, was compared with the expected number of deaths if the occupationally exposed workers had experienced the mortality rates of the general population. The results are set out in detail in appendix 3 and annexe I.

There are several problems in interpreting the data from these kinds of epidemiological studies; these are addressed in more detail in appendix 3. Firstly, there are few reliable data on the levels of exposure of workers, especially in the early years of uranium processing when safety standards were relatively lax and exposures due to inhalation of uranium-containing dust are likely to have been high. Monitoring of the levels of exposure is better in more recent cohorts of workers, but increased awareness of safety, and the introduction of more stringent safety legislation, have resulted in much lower levels of exposure. In addition, approximately 90% of lung cancers are caused by smoking, and good information on smoking habits is not available for any of the cohorts.

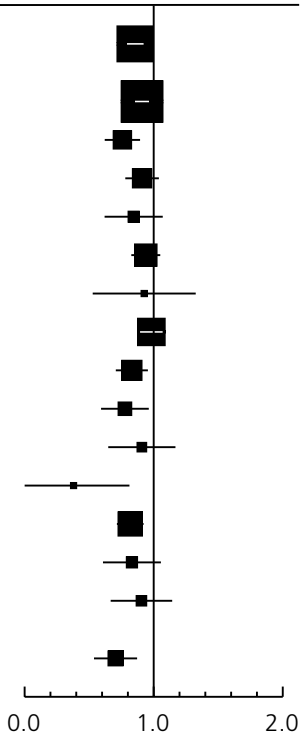
Another problem is that the risks of dying of cancer are expected to be lower in uranium workers than the general population as they are typically of a relatively high socio-economic status and would all have been healthy at the time they started employment (the 'healthy worker effect'). Thus, if the risk of dying of cancer in uranium workers is the same as that in the general population, this might be considered to be evidence of an increased risk, but whether this would be an appropriate interpretation would depend on the strength of the healthy worker effect which is difficult to quantify.

Notwithstanding these methodological issues, there is no evidence of a significant increase in deaths from any cause, or from all cancers, or individual types of cancer, or genitourinary disease (including kidney dysfunction), in the large cohorts of uranium workers whose health has been monitored, in many cases for several decades. Table 6 shows the observed numbers of deaths among the uranium workers followed in the fourteen studies (Observed; O), compared to the general population (Expected; E). In all cases the central estimate of the risk is less for the uranium workers than for the general population (the estimated ratio of observed deaths to expected deaths is less than 1). Estimates of risk from these types of studies can often be subject to considerable uncertainty, but as this analysis includes a very large number of workers (and of deaths) from the fourteen studies combined, the lack of an excess health detriment is clear. (The 95% confidence intervals shown in table 6 are the range of estimates of the observed over the expected deaths from occupational exposure to DU that have a 95% chance of including the real value of the relative risk.) These studies suffer from a lack of good data on levels of exposure to uranium, particularly by inhalation, which makes it difficult to relate the occupational exposures to those estimated for the intakes of DU on the battlefield. However, if exposure to uranium were a substantial danger, this should have been apparent from the epidemiological studies. The epidemiological studies thus provide some support for our views that exposure to DU is unlikely to increase greatly the risks of cancer.

Studies of the health of workers in the uranium industry therefore show no sign of excess deaths due to cancer or kidney disease related to inhaling or ingesting uranium. It should be stressed that epidemiological studies of this kind are not very sensitive at detecting small increases in risk. However, it is likely that a doubling in the lifetime risk of a fatal cancer would be detected by this approach.

Table 6. Summary of mortality for various causes in uranium workers compared with that expected in the general population

Cause of death [a]	Total number of deaths	O/E (95% CI)*	Estimated ratio of observed (O) to expected (E) deaths & 95% CI*
All causes	33502	0.86 (0.79-0.93)	
All cancers	7442	0.91 (0.85-0.97)	
Stomach cancer	365	0.76 (0.62-0.89)	
Colorectal cancer	728	0.91 (0.78-1.04)	
Liver cancer	123	0.84 (0.62-1.07)	
Lung cancer	2846	0.94 (0.83-1.05)	
Bone cancer	30	0.93 (0.53-1.33)	
Prostate cancer	490	0.98 (0.89-1.07)	
Bladder cancer	196	0.83 (0.71-0.96)	
Kidney cancer	151	0.78 (0.59-0.96)	
Brain cancer	223	0.91 (0.65-1.17)	
Thyroid cancer	7	0.38 (0.00-0.81)	
Non-Hodgkin lymphoma	266	0.82 (0.71-0.92)	
Hodgkin's disease	68	0.83 (0.61-1.06)	
Leukaemia	295	0.90 (0.67-1.14)	
All genito-urinary diseases	318	0.70 (0.54-0.87)	



[a] Detailed results that contribute to each cause of death shown here are given in annexe I

* Estimated ratio of observed number of deaths (O) to that expected in the general population (E), with 95% confidence intervals (CI)

4.3 Epidemiological studies of soldiers exposed to depleted uranium

Epidemiological studies require large cohorts, and such studies are underway for both UK and US veterans of the Gulf War. These studies show a small excess of deaths due to accidents but none has so far detected any excess of deaths from causes that could possibly be attributed to any radiological or chemical toxic effects of DU (Kang and Bullman 1996, MacFarlane et al 2000). Even if epidemiological studies did show an excess of deaths due to cancer, it would be very difficult to link this to DU, as there are typically multiple potentially toxic exposures in modern warfare that could contribute to any excess mortality.

The studies of the health problems, or mortality, of large cohorts of veterans are of great importance to detect any detriments to health, from whatever cause, in modern warfare. In the context of exposure to DU, the majority of soldiers within these large cohorts will have had Level III exposures to DU (if any) and, according to even our worst-case estimates of the excess risk of fatal cancers from Level III exposures, we would not expect to observe any excess mortality from cancer in a cohort of 10,000 veterans (annexe J). For example, even if the risk of fatal lung cancer were one hundred times greater than the central estimate, we would only have a one in three chance of observing a single case of lung cancer in 10,000 soldiers by age 75.

The epidemiological approach is useful when considering the health monitoring of the small number of soldiers with Level I exposures to DU. Assume 100 soldiers in a military conflict receive Level I exposure to DU. The estimated excess number of deaths among 100 soldiers from lung cancer, over their lifetime, attributed to the exposure is 0.12. The expected number of deaths from lung cancer in a group of 100 males aged 25, with a typical mix of cigarette smokers and non-smokers, is 5.85 after 50 years of follow-up. As the 100 soldiers were exposed to DU, the predicted number that die of lung cancer is 5.97 (ie 5.85 plus 0.12). Such a tiny excess of lung cancer deaths would certainly not be observable. Even if the risk from DU were one hundred times larger than this central estimate, to give a 12% lifetime excess risk of lung cancer (nearly twice that in the worst-case scenario), we would expect to see only 0.19 deaths from lung cancer in the cohort of 100 soldiers after 20 years. Only after 50 years would we see a clear

difference, as 5.8 deaths from lung cancer would be expected in the 100 non-exposed men but 17.8 deaths in the 100 soldiers with the 12% lifetime excess risk. Thus, even after long-term health surveillance of soldiers with Level I exposures there is little expectation, even under the most extreme assumptions, of seeing an excess risk of lung cancer in a cohort of 100 soldiers after 20 years. Also the smoking habits of any cohort of soldiers whose health is being monitored would need to be carefully recorded as smoking carries a substantial risk of lung cancer.

We can also consider the risk of leukaemia. There have been media reports of cases of leukaemia in soldiers or peace-keepers from the Balkans which have been attributed to exposure to DU. Peace-keepers would not be expected to have exposures to DU greater than those in the Level III scenarios. Even if the risk of leukaemia were one hundred times greater than that predicted for the central estimate by the ICRP approach for Level III exposure, there would only be an expected 0.0017 additional deaths due to leukaemia by age 75 in a group of 10,000 men. The ICRP approach therefore does not predict an observable increase in the numbers of deaths from leukaemia over that expected in the general population.

The expected number of cases of leukaemia in a cohort of 10,000 men aged 25-29 is 0.25 per year. Thus five years after the conflict in Bosnia we would expect to find approximately 1.25 cases among a group of 10,000 men of this age who had no exposure to DU or to any specific cancer-causing agent. Careful studies would be required to establish whether the observed incidence of leukaemia among peace-keepers is significantly greater than this value and, if so, whether risks other than DU might have contributed.

5 Conclusions and recommendations

Table 7. Estimates of numbers of deaths from lung cancer and leukaemia in 10,000 soldiers by age 75, and the predicted excess deaths among the 10,000 soldiers from DU exposure

	Lung cancer	Leukaemia
Number of deaths in unexposed group based on rates in the general population	585	42.4
Predicted excess deaths due to:		
Level I exposure to DU	12	0.049
Level II exposure to DU	0.25	0.00070
Level III exposure to DU from smoke plumes*	0.0037	0.000017

* Level III exposures are also assessed for entry into contaminated vehicles and inhalation of resuspended soil. Associated risks are not calculated here but can be derived from information given in the report. The greatest risk from Level III exposures is assessed to be from entry into contaminated vehicles, at one-tenth the Level II values.

5.1 Conclusions

DU is radioactive and poisonous. Exposure to sufficiently high levels might be expected to increase the incidence of some cancers, notably lung cancer, and possibly leukaemia, and may damage the kidneys. The key question is whether exposures to DU on the battlefield are such that the increased incidence of cancer, or the likelihood of kidney damage, are insignificant or are high enough to cause concern.

This is a very difficult question to answer given the lack of good quality data on some of the parameters that determine the extent of the exposure or the subsequent risk of disease. Most notable are the limited data on the levels of DU that might be inhaled in the impact aerosol within a tank pierced by a DU penetrator or that might be inhaled by soldiers entering the tank soon after the impact. There is also an almost complete absence of measurements of levels of uranium in samples of urine taken soon after exposure to DU.

The estimates of risk to soldiers from direct exposure to DU penetrators are less problematic since levels of exposure (eg radiation doses within a tank loaded with DU munitions) can be readily measured and appropriate safety guidelines implemented. A greater concern is from DU left on the battlefield as a slightly increased risk of skin cancer is expected from long exposures to fragments of DU penetrators. This is a particular concern for children attracted to these objects.

Our study provides some limits on the extent of any risk from internal exposure to radiation from DU on the battlefield. In all scenarios considered, the greatest risk is for lung cancer. The highest value from the central estimates of radiological risks is 1.2 extra deaths per thousand from lung cancer from Level I exposures. This additional lifetime risk implies that the expected number of deaths from lung cancer in 10,000 soldiers with Level I exposures would be 597 by age 75, compared to the 585 deaths expected from the rate of fatal lung cancer in the general population (table 7[4]). For those soldiers who received both shrapnel wounds and Level I exposure to an impact aerosol, the radiation risk from shrapnel, which is difficult to quantify, is added to that from inhalation. Preliminary reports, that have not yet been subjected to peer-review, of tumours in animals with implanted DU pellets raise some concern about the long-term consequences of embedded DU shrapnel.

For Level II and III scenarios the central estimate of the excess lifetime risk of fatal lung cancer is less than 3 per hundred thousand. The central estimates of excess lifetime risks of other fatal cancers, from Level I, II or III exposures, are less that 5 per million.

Perhaps the greatest uncertainty arises with Level I exposures since this scenario involves soldiers within any vehicle struck by a DU penetrator. We have therefore provided a worst-case estimate of risk which is a judgement of the highest exposure to DU that might occur for Level I. The worst-case is not expected to apply to many soldiers with Level I exposures but could occur, under the most unfavourable conditions, when a large calibre DU penetrator strikes and pierces the heavy armour of a battle tank. The excess risk of 6.5 per hundred for lung cancer should not therefore be seen as the risk for all soldiers with Level I exposures but as the highest excess lifetime risk for a soldier who is maximally exposed to DU from an impact. Thus any individual soldier who did receive the worst-case estimate of exposure would have a risk of dying of cancer by age 75 that is approximately twice that of the general population. Their worst-case excess risks of other cancers are all less than 6 per hundred thousand.

[4] For ease of comparison the predicted numbers of excess deaths in table 7 for Level I, II and III exposures are given for a cohort of 10,000 soldiers. Only a very small fraction of this number would be expected to receive Level I exposures.

Similarly, a few individuals could receive the worst-case intake of DU from Level II exposures. Their individual lifetime excess risk of lung cancer is calculated to be 2.4 per hundred; their excess risks of other cancers are all less than 2 per hundred thousand.
The highest value for the central estimate of the excess lifetime risk of leukaemia is about 5 per million, which would be expected to result in an extra 0.05 cases of leukaemia in a cohort of 10,000 soldiers with Level I exposures to DU (table 7). It has been suggested by some that the ICRP approach to calculating radiation risks underestimates the risk of leukaemia from the inhalation of radioactive particles by a factor of 100. Even if this were true, the central estimate excess lifetime risk of fatal leukaemia, for all exposure scenarios, would still be less than 1 in two thousand.

In contrast to Level I and II exposures which might be received by a few hundred soldiers, hundreds of thousands of soldiers and civilians could receive Level III exposures in tank battles close to urban centres. A substantial cancer risk from these scenarios would be of great concern. The risk assessment of Level III exposures, some of which are also relevant to peace-keepers who enter areas where DU munitions were used, give central estimates of excess cancers that in all cases are less than 3 per million. Worst-case risks do not apply to large populations and only provide the risk that might occur to an individual receiving an exposure at the top of the range of estimated intakes. The worst-case risk to the most highly exposed individual for Level III would increase their lifetime risk of lung cancer by 2.4 per thousand.

Levels of uranium in the kidney from Level I, II and III exposures are given in this report but our estimates of risk from the chemical toxicity of DU will be discussed in our second report. However, we do note here that the central estimate for Level I exposures is predicted to result in 4 micrograms of uranium per gram kidney, which is considered to be a level that could result in some disturbance of kidney function.

Associations between an exposure and disease can sometimes be established relatively quickly where the disease is previously unknown and is so distinctive that it can be unambiguously recognised. However, exposures to radioactive or poisonous materials are expected to increase the risk of diseases that already occur within the community. Determining whether this happens, and to what extent, is much more difficult and usually requires large long-term epidemiological studies.

Consider the link between cigarette smoking and lung cancer where the evidence is overwhelming as the increased risk due to smoking is so large. The development of lung cancer in both smokers and non-smokers takes many years and lung cancer consequently is rare in young people. If the incidence of lung cancer is followed in two cohorts of teenagers, one of habitual smokers and one of non-smokers, it is unlikely that any significant difference in lung cancer rates will appear after 10 years of follow-up although, by the time they are 70, the increased incidence of lung cancer in the smokers compared to the non-smokers will be very evident. Thus, even for lung cancer and smoking, where the increased risk of cancer is substantial, a highly significant association between smoking and cancer appears only after many years of monitoring the incidence of disease in large cohorts of smokers and non-smokers.

Any increased risk of lung cancer due to exposure to DU will also be very difficult to establish and, even if substantial, may take several decades to become evident. The problem is made more difficult (and perhaps impossible) by the fact that the excess risks are only expected to be significant for Level I and II exposures that typically will involve too few soldiers to detect the increased risk.

The best that can be achieved is to compare the health of large cohorts of soldiers from conflicts where DU was used with those of cohorts of soldiers who have not been exposed. These studies are being undertaken for both UK and US veterans of the Gulf War and have, so far, shown no increased mortality due to cancers or kidney disorders. However, as discussed, these studies are not very sensitive, and will not detect small increases in overall risks of lung cancer, or even relatively large increases in risks to a small subset of the most heavily exposed soldiers. Estimates of risk can be wrong, and there may be significant exposures other than DU in modern warfare, and the epidemiological studies of the health of soldiers should be continued over several decades. In terms of risks from DU, some attempt to categorise the extent of exposure (Level I, II or III) should be attempted and, as lung cancer is expected to be the greatest risk, the smoking habits of each soldier under study needs to be carefully documented.

Veterans who have become ill since returning from the Gulf War naturally reflect on whether their disease is associated with exposure to DU on the battlefield. Similarly, there have been reports in the press of cases of leukaemia in soldiers or peace-keepers who served in Bosnia and Kosovo, which are proposed to be due to exposure to DU.

This report does not directly address the causes of the illnesses reported by veterans, but it does suggest that the risks of cancer from the radioactivity associated with exposures to DU on the battlefield are likely to be very low for the great majority of soldiers who would have received Level III exposures. Similarly, peace-keepers would mostly have received exposures that are very low and it is considered very unlikely that these would give rise to any detectable increase in the incidence of

leukaemia. Determining whether diseases among veterans are linked to exposures on the battlefield (to DU or other potentially toxic agents) is very difficult and needs to be studied using rigorous epidemiological methods. For example, simply identifying veterans or peace-keepers with leukaemia provides no evidence that their disease is linked to DU (or any other battlefield exposure). It needs to be shown that the incidence of leukaemia is greater in those exposed to DU than would be expected from the underlying incidence of disease for that age group within the general population.

For governments the difficulties in establishing whether the use of DU munitions does or does not lead to a significant (or even substantial) excess risk of cancer are considerable. There is an onus on governments that wish to retain the use of DU munitions to understand the risks to their own soldiers, and to the soldiers and civilians within the countries where conflicts arise. If epidemiological studies are unlikely to provide quick and clear indications of the extent of the risk, there is a need to obtain much better estimates of the levels of exposure to DU that may occur on the battlefield, and particularly in tanks pierced by large calibre penetrators. The difficulty in estimating upper bounds for the intake of DU is the major uncertainty in assessing the extent of the radiological risks from Level I exposures, and leads to a substantial difference in the risks of lung cancer and of levels of uranium in tissues and organs (particularly the kidney which is believed to be the organ at most risk), for the central estimate and worst-case intakes. New test firings under realistic conditions are required and the detailed results of these should be made available.

A tension is likely to remain between soldiers who are convinced that their health problems are due to exposure to DU, and those who consider the risks to be small, partly because of the great difficulties in establishing rigorously whether there is a significant risk to health and in quantifying that risk. Given this uncertainty, the health of soldiers who are likely to have been exposed to DU at Level I and Level II should continue to be carefully monitored, preferably by a mechanism that is seen to be completely independent of government, with particular emphasis on sensitive tests of kidney function and tests for the continuing excretion of DU. In any future conflicts where DU munitions are deployed efforts should be made to obtain urine samples as soon as is practical, and at intervals thereafter, from at least a sample of soldiers involved in Level I and II exposures. Levels of DU in these samples will provide better information on the intakes which will complement information arising from new test firings of DU penetrators.

5.2 Recommendations

We have identified a number of areas where further research is necessary, and a number of other actions that would help in assessing the hazards that may arise from the use of DU. Most of these arise as a consequence of the paucity of good experimental data on the amounts of DU that may be inhaled within and close to tanks struck by a DU penetrator, and the size distribution and properties of the DU particles within aerosols, and the almost complete lack of any measurements of DU in urine samples taken soon after exposure to a DU aerosol.

We recommend that topics for future research include:

- further test firings under realistic conditions into heavily-armoured vehicles to provide better estimates of the levels of DU, and the properties of DU aerosols, within and released from struck vehicles;
- experimental information on resuspension from surfaces in contaminated vehicles to enable reliable assessments to be made of exposures resulting from various forms of entry into struck vehicles and enable recommendations to be made for appropriate precautions to be taken by service personnel and civilians;
- the development and validation of models to enable DU exposures to be predicted in a wide range of circumstances;
- an independent and fully resourced assessment of the risks, particularly from Level I and II exposures, ensuring that all of the data are available, including restricted material not available to us, and data from any new test firings;
- a long-term follow-up of mortality and cancer incidence in Gulf War veterans since any excess cancers may not become apparent for 30 – 40 years, if at all. For all soldiers in these studies, there should be an attempt to obtain information now on likely exposures to DU (for example Level I, II or III) and smoking habits;
- long-term *in vivo* studies of the dissolution of DU oxides formed from penetrator impacts and fires involving DU. These are needed to assess doses from inhalation prospectively, and, more importantly, to assess intakes and doses (especially lung and thoracic lymph node doses) from urine samples. Doses to thoracic lymph nodes are especially sensitive to the long-term dissolution rate of DU oxides in the lungs;
- a review of all information on the ICRP uranium systemic model as used to predict urinary excretion, especially at long times after exposure. This is used to assess intakes and hence radiation doses and uranium levels in organs from exposures. Studies, possibly involving human volunteers, to refine the model should also be considered;

- a thorough review of the effects of radiation from radioactive particles retained in lymph nodes including any possible carcinogenic effects.

We also recommend that the following further actions be undertaken:

- the identification of any UK veterans who received Level II exposure to DU (and confirmation that the UK has no veterans that received Level I exposure) and their invitation to participate in an independent evaluation programme, which would include levels of DU in urine;
- the publication by Government of plans for improved and expeditious monitoring of the health of soldiers in any future conflict. Where DU munitions are deployed, Government should ensure in future that, via occupational health, urine samples are taken from soldiers exposed to Level I and II intakes, as soon after exposure as practical, and at intervals thereafter. Levels of DU in urine should be measured, where appropriate. Arrangements should include timely complementary measurements to detect characteristic photon emissions from uranium in the thorax using the most sensitive *in vivo* monitoring equipment available. If uranium is detected, appropriate follow-up measurements should be made, and if sufficient uranium is present, consideration should be given to assessment of its distribution within the thorax, between the lungs, lymph nodes and ribs;
- wherever possible, and with appropriate consent, the performance of independent detailed autopsies on soldiers with significant potential exposure to DU, to characterise DU deposition, particularly in the lung, lymph nodes, kidney and bone, and signs of abnormal histopathology of lymph nodes or kidney;
- the publication of further independent information on the isotopic composition of DU in munition stocks, including levels of uranium-236, transuranics and fission products;
- it should be incumbent on nations using DU munitions in future conflicts to advise the local population of the potential dangers of handling fragments of penetrators.

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7 Glossary of abbreviations

DRPS	Defence Radiological Protection Service
DU	Depleted Uranium
EU	European Union
Gy	Gray, the amount of energy deposited by ionising radiation per unit mass of tissue or other matter, expressed in joules per kilogram
High-LET	High Linear Energy Transfer
IARC	International Agency for Research on Cancer
ICRP	International Commission on Radiological Protection
LNT	Linear No Threshold
Low-LET	Low Linear Energy Transfer
NAS	National Academy of Science, USA
NRPB	National Radiological Protection Board
OECD	Organisation for Economic Cooperation and Development
OSAGWI	Office of the Special Assistant to the Deputy Secretary of Defense for Gulf War Illnesses, USA
Sv	Sievert, the equivalent dose taking into account the differing extent of damage to biological tissues by the same absorbed dose from differing types of radiation compared to low-LET radiation
UNEP	United Nations Environmental Programme
UNSCEAR	United Nations Scientific Committee on the Effects of Atomic Radiation
WHO	World Health Organisation

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Head, Gulf Veterans' Illness Unit

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Dr Michael Birt, Defence Evaluation and Research Agency Farnborough
Depleted uranium kinetic energy penetrators

Mr Richard Bramhall, The Low Level Radiation Campaign

Surgeon Commander Dr David Brown, Ministry of Defence
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Dr John Marriage, AWE Aldermaston
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Appendix 1: Exposures arising from the use of depleted uranium on the battlefield
Michael Bailey and Clive Marsh

1 Introduction

The objective of this assessment was to provide central and upper estimates of radiation doses and of systemic uptake of uranium, from exposures arising from the use of DU on the battlefield. These estimates were then to be used to assess the radiation and chemical hazards associated with the exposures. Note that in keeping with the terminology used in radiological protection, *intake* refers to the amount of material (mass or activity) that enters the body through the nose, mouth or a wound, and *uptake* refers to the amount of material that is absorbed into the blood.

For both situations and hazards there are three main exposure pathways:

- inhalation: breathing in airborne particles containing DU;
- ingestion: taking in DU via the mouth through hand to mouth transfer or consuming contaminated food. However, in this assessment, only intakes by the former route are assessed;
- entry through a wound or via intact skin.

For ionising radiation an additional exposure pathway arises from direct irradiation resulting from the emission of radiation (notably gamma-rays and beta-particles) during the decay of the uranium isotopes present or of the radioactive nuclides formed when they decay.

Such assessments have two main stages:

- assessment of the exposures, ie the amounts of DU present in the environment with the potential to enter the body, together with characteristics which determine how much enters the body, and its subsequent behaviour. It is emphasised, however, that assessments of intake were made without any expert knowledge of the battlefield situations, and were inevitably largely based on reviewing the available literature. Assumptions are therefore clearly stated and references given to data used;
- calculation of the radiation doses to organs, and of uptake of uranium into the bloodstream, and hence deposition in other organs (particularly kidneys) resulting from the exposures.

1.1 Composition of DU and radiation emitted from isotopes present

Naturally occurring uranium exists in the form of three isotopes, uranium-234 (^{234}U), ^{235}U and ^{238}U, all of which undergo radioactive decay. The composition by mass of natural uranium is given in column 2 of table 1. Almost all of the mass of the uranium is in the form of the ^{238}U isotope. ^{238}U and ^{235}U are primordial radionuclides, ie they were present in the material from which the earth was formed. ^{234}U, which has a much shorter half-life, is present because it is formed when ^{238}U decays, ie it is a 'decay product' of ^{238}U. Each radioactive isotope (radionuclide) undergoes nuclear transformations (decays) at a characteristic rate. Thus if we have N atoms of the radionuclide, the average number of transformations per second is given by $N\lambda$, where λ is the radioactive decay constant for that radionuclide. The 'activity' of a quantity of radioactive material is the average number of decays that take place per second, and is measured in becquerels (Bq). (1 Bq = 1 transformation per second.) The half-life of a radionuclide ($t_{\frac{1}{2}}$) is the time taken for the activity of any amount of the radionuclide to decay to half its original value. It can be shown that $t_{\frac{1}{2}} = \ln(2)/\lambda$. ^{234}U and ^{235}U have much shorter half-lives than ^{238}U (table 1, column 3).

Table 1 shows how the composition of natural uranium, expressed in terms of the activities of the uranium isotopes, is related to the mass fractions of the isotopes. The number of atoms in 1 gram of a radionuclide is given by N_A/A, where N_A is Avogadro's number (6.022×10^{23}) and A is the atomic mass of the radionuclide. Hence the activity of unit mass (in this case 1 gram) of a radionuclide (that is, its 'specific activity' SpA) is related to its half-life by the following expression:

$$\text{SpA (Bq g}^{-1}) = (\ln(2) \times N_A) / (t_{\frac{1}{2}} \times A) \qquad (1)$$

Thus, radionuclides with shorter half-lives have higher specific activities (table 1, column 4). The activity of each uranium isotope in 1 milligram (mg) of uranium (table 1, column 5) can be determined by multiplying the specific activity of the radionuclide by its mass fraction. Because a shorter half-life radionuclide has a higher specific activity, the fractions of ^{234}U and ^{235}U in natural uranium measured by activity (table 1, column 6) are larger than the mass fractions.

DU is a by-product of the process of enrichment in which the concentration of ^{235}U is increased. Uranium is described as 'depleted' if the mass concentration of ^{235}U is less than 0.711%, but generally it is lower and the DU used by the US Department of Defence has a ^{235}U concentration less than 0.3% (AEPI 1995). A typical composition of DU is given in table 2. If the DU contains uranium obtained by reprocessing material that has been used for fuel in a nuclear reactor, rather than from uranium ore, it will also contain some of the isotope ^{236}U

Table 1. Composition of natural uranium

Isotope	Mass fraction[a] (%)	Half-life (years)[b]	Specific[c] activity (Bq g^{-1})	Activity[d] in 1 mg natural uranium (Bq)	Activity fraction (%)
^{234}U	0.0055 ± 0.0005	2.444×10^5	2.31×10^8	12.35	49
^{235}U	0.7200 ± 0.0012	7.038×10^8	8.00×10^4	0.58	2
^{238}U	99.2745 ± 0.0015	4.468×10^9	1.24×10^4	12.35	49
Total				25.3	

a Brown and Firestone (1986)
b ICRP (1986)
c Calculated using equation (1)
d The activities of ^{238}U and ^{234}U in natural uranium are equal when in equilibrium. The activity of ^{234}U has therefore been taken to be equal to that of ^{238}U. The value corresponds to a mass fraction of 0.0053%, which is well within the range quoted by Brown and Firestone (1986).

(table 2). (^{236}U is an alpha-particle emitter with a half-life of 2.3 x 10^7 years. It decays to Thorium-232 (^{232}Th), which has a half-life of 1.4 x 10^{10} years.) Activities of the uranium isotopes in 1 mg of DU, and activity fractions, are given in table 2. There may also be trace amounts of plutonium, neptunium, americium, and fission products in DU used in munitions or in armour. These were reported in OSAGWI (2000, section III.B.1 and tab C, see annexe E). Recent information (Bhat 2000) was used to assess the contribution that these might make to radiation doses (annexe D).

When ^{238}U decays, the disintegrating nucleus emits an alpha particle and some photons, and forms another radionuclide, ^{234}Th. This in turn decays to protactinium-234m (^{234m}Pa). This process is shown schematically in figure 1. There are another 10 members of this decay chain beyond ^{230}Th, including radium-226 and radon-222, the final member being a stable isotope of lead. During chemical purification (separation) of uranium from its ore, all the decay products are removed, except ^{234}U. Following separation, the ^{238}U and ^{234}U continue to decay, and the decay products 'grow' back in. ^{234}Th and ^{234m}Pa have short half-lives and soon return to their equilibrium values (where the rate of formation from ^{238}U equals the rate of decay). However, ^{230}Th has a long half-life and so grows in very slowly. Hence in processed uranium (natural or depleted) the activities of ^{230}Th and subsequent members of the chain are negligible. For uranium in its natural state, the ^{238}U and ^{234}U isotopes are in equilibrium and their activity fractions are the same (table 1). However, equilibrium conditions do not always exist, even for natural uranium in the environment (eg in groundwater). The various alpha, beta and gamma emissions resulting from the decay of the radionuclides shown in figure 1 other than ^{230}Th are summarised in table 3.

^{235}U is part of a different decay chain (figure 2). Following separation, ^{231}Th grows in rapidly and so almost always accompanies ^{235}U. For separated uranium the chain effectively ends at ^{231}Pa, which grows in slowly from the decay of ^{231}Th. There are another 9 members of this decay chain beyond ^{231}Pa, the final member again being a stable isotope of lead. The various alpha, beta and gamma emissions resulting from the decay of ^{235}U and ^{231}Th are summarised in table 4.

Table 2. Composition of depleted uranium

Isotope	Mass fraction[a] (%)	Half-life (years)[b]	Specific[c] activity (Bq/g)	Activity in 1 mg depleted uranium (Bq)	Activity fraction (%)
^{234}U	0.001	2.444×10^5	2.31×10^8	2.31	15.53
^{235}U	0.20	7.038×10^8	8.00×10^4	0.16	1.07
^{236}U	0.0003	2.341×10^7	2.40×10^6	0.007	0.05
^{238}U	99.8	4.468×10^9	1.24×10^4	12.42	83.35
Total				14.9	

a AEPI (1995)
b ICRP (1986)
c Calculated using equation (1)

Table 3. Uranium-238 decay series (truncated)[a,b]

Nuclide	Half-life	Radiation	Energy (keV)	Yield (%)
Uranium-238	4.47 x 10^9 years	α	4038	0.078
			4151	20.9
			4198	79.0
		γ	49.6	0.064
Thorium-234	24.1 days	β	86.3	2.9
			106.3	7.6
			106.7	19.2
			199.1	70.3
		γ	63.3	4.8
			83.3	0.079
			92.4	2.8
			92.8	2.8
			112.8	0.28
Protactinium-234m	1.17 minutes	β	1224.4	1.0
			1459.0	0.69
			2268.9	98.2
		γ	258.2	0.073
			742.8	0.080
			766.4	0.294
			1001.0	0.837
Protactinium-234[c]	6.70 hours	β	471.6	0.053
Uranium-234	2.46 x 10^5 years	α	4603.5	0.20
			4722.4	28.4
			4774.6	71.4

a Chu et al (1999)
b Only emissions with yields greater than 0.05% are shown
c The yields for ^{234}Pa have been multiplied by the isomeric transition (IT) branching ratio (figure 1 ie 0.16%)

Table 4. Uranium-235 decay series (truncated)[a,b]

Nuclide	Half-life	Radiation	Energy (keV)	Yield (%)
Uranium-235	7.04 x 10^8 years	α	4214.7	5.7
			4366.1	17
			4397.8	55
			4414	2.1
			4502	1.7
			4556	4.2
			4596.4	5.0
		γ	109.2	1.5
			143.8	11.0
			163.4	5.1
			185.7	57.2
			202.1	1.1
			205.3	5.0
Thorium-231	25.52 hours	β	25.6	14.5
			84.2	6.6
		γ	142.2	2.7
			206	12.8
			215.3	1.3
			287.2	12
			288.1	37
			305.3	35

a Chu et al (1999)
b Only emissions with yields greater than 1% are shown

Figure 1. The uranium-238 decay series (truncated)

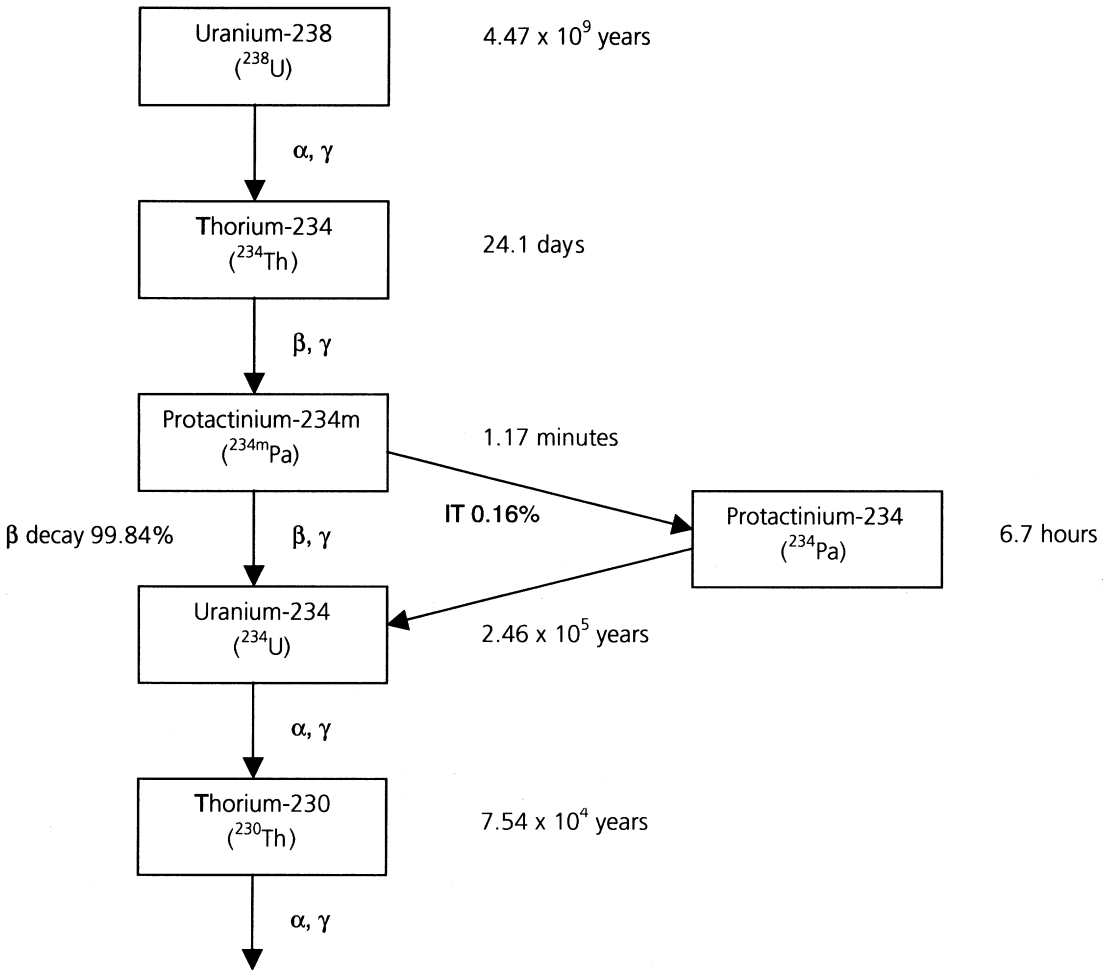
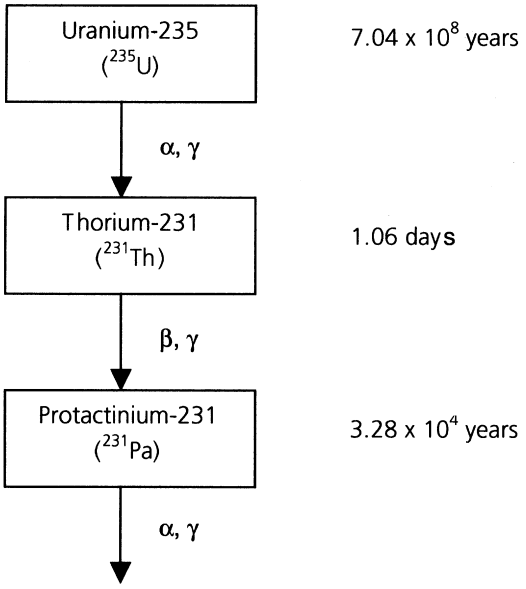


Figure 2. The uranium-235 series (truncated)



1.2 Assessments of exposure

Assessments of exposure require estimates to be made of various factors according to the route of exposure:

Inhalation:

- the concentration of DU in the air multiplied by the time for which it is present in the air (time-integrated air concentration);
- the properties of the cloud of airborne particles (aerosol) formed, especially the size distribution and density, which determine the fraction of the inhaled aerosol deposited in each part of the respiratory tract (for further details see annexe A);.
- the dissolution characteristics of the particles deposited in the respiratory tract (*in vivo* solubility), which determines transfer to blood and deposition in other organs (for further details see annexe A);
- knowledge of the chemical composition may also be of use in predicting dissolution, by using existing information about the *in vivo* dissolution of individual uranium compounds, notably U_3O_8 and UO_2.

Ingestion:

- the concentration of DU on surfaces with which personnel might come in contact;
- the rate of transfer from surfaces to hand and on to the mouth;
- the dissolution characteristics of the particles in the gastro-intestinal tract (*in vivo* solubility).

Entry through a wound:

- the amount of DU remaining in wound after treatment;
- the dissolution characteristics of the DU in the wound (*in vivo* solubility) before and after treatment.

Direct irradiation:

- the amount of DU in the environment;
- the distance between the DU and the subject;
- the time spent near the DU (occupancy).

1.3 Calculation of uranium biokinetics and radiation doses

To enable radiation doses to organs (and hence risks) to be estimated for a wide range of exposure conditions, 'biokinetic models' have been developed which describe the various processes involved in terms of sets of equations. Complementary 'dosimetric' models are used to calculate the amounts of energy deposited in the organs from the radioactive decay of the material present. Models for intakes of radioactive materials (including uranium compounds) by inhalation and ingestion have been developed and refined over many years. The International Commission on Radiological Protection (ICRP) has promoted the development of such models, and the models that it has adopted are used by most radiation protection professionals and organisations around the world, and similarly form the basis of legally enforced standards in most countries, including member states of the EU. The most relevant models, those for the respiratory tract, and for the behaviour of uranium that has entered the blood, were published about 5 years ago (ICRP 1994a, 1995a). They are therefore reasonably up-to-date with respect to the state of knowledge of the subject. Nevertheless, they have been published long enough for any major discrepancies with current knowledge to have been identified and brought to the attention of the radiation protection community and the ICRP, through the literature or relevant conferences. The current ICRP models (section 2.2 and annexe A) have therefore been applied here.

In order to assess radiation doses to organs, the first part of the process is to calculate the amount of material present in each organ as a function of time after intake. Hence, although developed for assessing radiation doses, the models can be applied to determine the distribution by mass, and hence the mass concentration of an element in an organ as a function of time after intake, and have been used here for that purpose.To enable the models to be used for assessments under standard or reference conditions, ICRP provides reference values of all the parameters used by the models. ICRP does, however, recommend that in appropriate circumstances, and where available, specific information should be used in preference to the reference or 'default' values. It has recently developed guidance on practical application of the respiratory tract model (Bailey et al 1998, ICRP 2001). Typically such information could relate to the size distribution and density of an aerosol, the *in vivo* dissolution rate of the inhaled material, and the physical activity of the subject during exposure. Such information has been applied here, where it was considered that the available information was adequate to justify its use.

2 Approach

2.1 Assessment of exposures

A number of reviews and assessments have been published, especially with regard to exposures in the Gulf War. They were consulted principally to obtain an overview of the likely scenarios, and to obtain references to source documents, ie first hand reports of relevant observations, measurements or calculations. Relevant aspects of these reviews and assessments are summarised in annexes E and F respectively. With the objective of conducting an *independent* assessment, emphasis is given here to source documents describing studies of the aerosol produced from the impact of DU projectiles, and studies on uranium (depleted or natural)

subjected to heat or fire, in preference to secondary documents (review articles or previous assessments). Those source documents that were obtained are summarised in annexes G and H. However, many of the source documents cited were not obtainable through the normal library request channels, because their distribution was restricted. After a number of such requests were unsuccessful, requests were not made for similar reports that were identified later. Those reports that were not obtained are identified in the list of references (section 6). This review of the literature provided much of the information (experimental and test data) and modelling approaches to assess intakes, and specific information to use with the ICRP biokinetic models to calculate doses and uptake to blood per unit intake. The various exposure pathways are addressed next. In the rest of this document, emphasis is placed on the inhalation route.

2.2 Exposure pathways

Inhalation
From the literature review it was confirmed that the main route of exposure to consider was inhalation. Aerosols containing DU may be formed by the impact of a DU projectile (especially with a 'hard' target, such as armour plate), in a fire involving DU, or through resuspension of particles deposited on the ground or other surfaces by activities that cause disturbance. Such aerosols may well be inadvertently inhaled by personnel close to the source or downwind of it unless respiratory protection is worn.

Ingestion
In the battlefield scenarios considered here, ingestion could occur through contact with contaminated surfaces or objects and subsequent hand-to-mouth contact. This is readily avoidable by occupational health and personal hygiene practices. Moreover, since the fraction of ingested uranium that is absorbed into blood is so small, large amounts of DU need to be ingested to give rise to a significant uptake to blood. Nevertheless, assessments are made of this exposure route.

Wounds
The main concern about entry through wounds relates to embedded fragments of DU shrapnel.
Contamination of an open wound by DU dust is possible, but it is relatively straightforward to remove the bulk of such contamination by washing etc, and it is unlikely that the residual mass would be as great as might be the case for a shrapnel wound. Contaminated wounds are therefore not considered further. Shrapnel wounds are most likely to arise from the impact nearby of a DU projectile. Clearly every such impact is a unique event, so it would be very difficult to predict the frequency and extent of any DU shrapnel wounds. The best guide currently available may be experience gained during the Gulf War. In that conflict, only the US (and to a much smaller extent UK) forces used DU weapons. A number (about 20) of US personnel who were victims of 'friendly fire' incidents are known to have embedded fragments of DU shrapnel, and are participating in a follow-up study (McDiarmid et al 2000, Hooper et al 1999, OSAGWI 2000 tab P). It is not clear how representative these might be of soldiers in the other forces involved in actions in which DU was used. So far as we know, no UK personnel have embedded DU shrapnel. Presumably large numbers of Iraqi personnel sustained injuries from DU shrapnel and have embedded fragments, but we know of no figures or follow-up studies on them. The range of size of the fragments in the US personnel is up to about 20 mm (Hooper et al 1999). Personnel who received shrapnel wounds will probably also have been exposed to DU aerosols. Doses resulting from embedded shrapnel fragments are considered in annexe B.

External exposure
Direct irradiation is relatively straightforward to determine on the basis of calculations and to confirm by measurements, and hence control. The alpha-particles have energies of 4.0– 4.8 MeV, and so a range of slightly over 30 μm in tissue (ICRU 1993). Hence they would be blocked by a sheet of paper, and even if the DU were in direct contact with skin, they would not penetrate the inert outer layer, over most of the body. Beta-particles have longer ranges, of the order of a centimetre in tissue (depending on their energy), while the gamma-rays are much more penetrating. The most important gamma-ray emission from DU is the 1.0 MeV photon produced in 0.6% of decays of ^{234m}Pa (a decay product of ^{238}U, table 2). According to Fetter and von Hippel (1999): "The theoretical maximum whole body gamma dose rate from external exposure to DU is 2.5 millirem per hour. Dose rates in this range might be experienced by a person surrounded by DU munitions". One millirem (mrem) corresponds to 0.01 millisievert (mSv), hence the maximum possible dose rate is 0.025 mSv per hour or 0.6 mSv per day. The highest exposures likely to arise in practice are in a vehicle fitted with DU armour and carrying DU ammunition. According to DRPS (1993), measurements demonstrated that personnel would need to be in a fully DU loaded tank for 1500 hours to reach the then current whole body dose limit of 50 mSv. We are not aware of any independent exposure measurements, but according to U.S. Army measurements cited by Harley et al (1999), the whole-body dose rate in or near a tank fully loaded with DU munitions is less than 0.002 mSv per hour. Thus driving such a tank for 1000 hours gives a dose similar to the annual dose from natural background radiation (on average 2.24 mSv per year in the UK).

According to Fetter and von Hippel (1999) the dose rate to a person standing on flat ground uniformly contaminated with 1 tonne DU per square kilometer (ie 1 gram per m^2, which they estimate to be a reasonable

upper bound for a battlefield area) would be 1 mrem (0.01 mSv) per year, which is one-tenth of the dose-rate from the uranium naturally present in soil. (Note that the dose rate from one gram of uranium naturally present in soil will be considerably greater than that from one gram of processed natural uranium or DU at the same depth, because the extraction of uranium removes many of the decay products, which also emit gamma-rays.)

The dose-rate to skin from direct contact with DU is much higher, at about 2.5 mSv per hour mostly from beta-particles (Fetter and von Hippel 1999). It is easily reduced by wearing gloves, or if the DU is encased in some other material. Furthermore, skin is relatively insensitive to radiation, so that even continuous contact (keeping a piece of DU in a pocket or wearing it as jewellery) is unlikely to produce a radiation burn or other short term effect. Such effects require doses of a few sieverts (Sv), delivered over a short time, but at 2.5 mSv per hour, the DU would need to be in contact for weeks to give such doses. There would, however, be expected to be a small increase in the risk of skin cancer.

Assessment of inhalation exposure

Inhalation seems likely to give rise to the most widespread uncontrolled exposures, and could give rise to high exposures close to impacts by DU projectiles. Embedded fragments of DU shrapnel could also give rise to high exposures, but there appears to be little information available to assess the hazard, other than the follow-up study on the US friendly fire victims (annexe B). Effort was therefore focused on the inhalation route. For this there are three potential approaches to assessing the exposures:

- measurements of the characteristics of DU aerosols formed from impacts of DU projectiles and from effects of combustion on uranium;
- calculations of aerosol formation and dispersion from impacts of DU projectiles and fires involving DU;
- measurements on personnel exposed to uranium or DU in such situations (monitoring).

Assessments of intakes by inhalation and ingestion are described in annexe C. An alternative approach to assessing the likely upper limit that is relevant to intakes of low specific activity materials such as DU is to consider the feasibility of inhaling the large mass of dust required to give a significant dose. This is considered here first as it puts the whole assessment into perspective.

Such an approach was taken by the United Nations Environment Programme 'Depleted Uranium Desk Assessment Group' (UNEP 1999, Snihs and Åkerblom 2000). They noted in relation to acute exposures that: "An instantaneous intake by breathing of more than 1 gram dust is unendurable, and assuming 10% DU means a maximum intake of 100 mg DU". Inhalation of 1 mg DU would typically lead to a (committed effective) dose of the order of 0.1 mSv (UNEP 1999, Snihs and Åkerblom 2000, section 3), and hence it is unlikely that anyone would incur a dose greater than 10 mSv from acute inhalation of DU. Similar reasoning can be applied to protracted intakes. It is very unlikely that any dust except very close to a point of impact would be more than 10% DU. An air concentration of 100 mg m^{-3} would be noticeably and unpleasantly dusty, and normal breathing rates are of the order of 1 m^3 per hour. Hence to inhale 100 mg DU, someone would need to inhale dusty air that was heavily contaminated with DU for 10 hours. Hence, except in exceptional circumstances, it is unlikely that anyone would incur a dose greater than 10 mSv from inhalation of DU as a result of battlefield use or a fire involving DU.

Battlefield Exposure scenarios

To make the assessment manageable, a set of specific scenarios was defined to be representative of exposures. In each only the exposure pathways considered to dominate were assessed. For short-term exposures it seemed reasonable to follow the approach used in OSAGWI (2000) and address three generic situations as follows. Note however that OSAGWI (2000) is concerned exclusively with exposures of US personnel involved in the Gulf War, whereas our assessment is intended to be more general.

- Level I exposures include those to personnel in a vehicle that is struck by a DU penetrator, or personnel entering a struck vehicle immediately, typically to assist injured comrades. Assume that exposure is dominated by inhalation of aerosol generated by the impact and embedded DU shrapnel fragments (considered separately in annexe B).
- Level II exposures generally occur after combat, typically working in or on contaminated vehicles to carry out repairs etc. Assume that exposure is dominated by inhalation from resuspension within the contaminated vehicle and by ingestion (hand to mouth transfer from contaminated surfaces within vehicles).
- Level III includes all others, eg during combat being downwind of impact(s) or fire(s), brief entry to contaminated vehicles. Consider inhalation of all four types of aerosols:
  – plume from impact;
  – plume from fire;
  – resuspension within contaminated vehicle;
  – resuspension from contaminated ground, and ingestion by hand to mouth transfer in contaminated vehicles.

It was assumed that exposures resulting from entry to contaminated vehicles were similar, with the main difference being in the duration of exposure, typically a few hours for Level II and a few minutes for Level III.

Therefore exposure rates (mg h^{-1}) were estimated. Level III exposures by inhalation of the plume from munition impacts or fires could here include members of the public.

For each scenario, two assessments were made:

- a 'central estimate' which is intended to be a central, representative value, using likely values of parameters according to the information available, and where information is lacking, values that are unlikely to underestimate exposures greatly. The central estimate is intended to be representative of the average for the group (or population) of people exposed in that situation. However, the term average is not used, because the central estimates are not based on statistical analyses of data. It is recognised that for individuals in each group values could be greater than (or less than) the central estimate;
- a 'worst-case' which uses values at the upper end of the likely range, but not extreme theoretical possibilities. The aim is that it is unlikely that the value for any individual would exceed the worst-case. Thus the worst-case should not be applied to the whole group to estimate, for example, the number of excess cancers that might be induced. One aim of the worst-case assessments is to try to prioritise further investigation. If even the worst-case assessment for a scenario leads to small exposures, then there is little need to investigate it more closely. If, however, the worst-case assessment for a scenario leads to significant exposures, it does not necessarily mean that such high exposures have occurred, or are likely to occur in a future battlefield, but that further information is needed.

2.3 Assessment of radiation doses and of uptake of uranium to blood

The current system of radiation protection

The following is based on the system of protection proposed by the ICRP in its most recent recommendations (ICRP 1991), and applied in subsequent ICRP publications relating to intakes of radioactive materials (ICRP 1993, 1994a, 1994b, 1995a, 1995b, 1997, 2001).

Absorbed dose
The risk of harm resulting from irradiation of tissue by ionising radiation (such as alpha-particles, beta-particles and gamma-rays) is generally considered to be related to the 'absorbed dose', which is the amount of energy absorbed per unit mass of tissue. It is expressed in grays, symbol Gy. One Gy is equal to one joule per kilogram. The 'organ dose' is taken to be the average absorbed dose in the organ or tissue of interest. For some organs, eg liver, kidneys, the organ dose calculated is the average dose to the whole organ, ie it is considered that the radiation sensitive cells are distributed throughout the whole organ. In other cases, notably in the parts of the gastro-intestinal (GI) tract and respiratory tract, sensitive cells are identified, and the average dose to these is calculated, ie the average dose to the tissue at a specified range of depths from a specified organ surface.

Equivalent dose
For the same absorbed dose to a tissue or organ, some types of ionising radiation, notably alpha-particles, are more harmful than others. To take account of this, the absorbed dose is 'weighted', which means that it is multiplied by a factor (the radiation weighting factor, symbol w_R), to give the 'equivalent dose'. For gamma-rays, X-rays and beta-particles the factor is set to 1, while for alpha-particles it is set to 20. Equivalent dose is expressed in sieverts, symbol Sv, but since this is rather large, the millisievert (mSv), which is one thousandth of a sievert, and the microsievert (μSv) which is one thousandth of a millisievert, are more commonly used. Equivalent dose provides a measure of the risk of harm resulting from irradiation of a particular tissue or organ.

Effective dose
The risk of harm per millisievert is not the same for the various tissues of the body; for example it is lower for the liver than for the lungs. To take account of this while obtaining an estimate of the overall risk of harm to a person, the equivalent dose to each important organ or tissue is multiplied by a factor (the tissue weighting factor, symbol w_T, table 5). This factor represents the risk of harm per millisievert to the tissue, compared to the risk of harm per millisievert to the whole body. The sum of the weighted tissue equivalent doses is called the 'effective dose' (or 'effective whole-body dose'), and is also expressed in Sv or mSv. Effective dose provides a measure of the risk of harm resulting from irradiation of a person taking account of different types of radiation, and different doses to different organs. It is therefore a useful standardised measure of the risks from exposures to radiation, especially those resulting from intakes of radionuclides, which will often result in very different doses to different tissues. The primary standards of radiation protection (limits and constraints), are mainly expressed in terms of effective dose.

Doses from intakes of radionuclides
When a person is irradiated with gamma-rays or X-rays from a source outside the body (external), the radiation dose is received only while the person is in the presence of the source. However, after radionuclides are inhaled or ingested they remain in the body for some time, and continue to irradiate tissues. How long this goes on for depends upon the route of intake, the chemical form, and the radioactive half-lives of the radionuclides. For example, when technetium-99m is administered for medical diagnosis, nearly all the dose is received within a day, because its half-life is only six hours. However, the

Table 5. Tissue weighting factors[a] (based on table 1 of ICRP publication 71, ICRP 1995b)

Organ or tissue (*T*)	Tissue weighting factor w_T	Organ or tissue (*T*)	Tissue weighting factor w_T
Gonads[b]	0.20	Liver	0.05
Bone marrow (red)	0.12	Oesophagus	0.05
Colon[c]	0.12	Thyroid	0.05
Lungs[d]	0.12	Skin	0.01
Stomach	0.12	Bone surface	0.01
Bladder	0.05	Remainder[e,f]	0.05
Breast	0.05		

a The values have been developed from a reference population of equal numbers of both sexes and a wide range of ages. In the definition of effective dose they apply to workers, to the whole population, and to either sex (ICRP publication 60, 1991).
b Dose coefficients are calculated for both the ovaries and testes, the higher of which is multiplied by the gonad tissue weighting factor for the calculation of effective dose.
c The colon w_T is applied to the mass average of the equivalent doses H_{ULI}, and H_{LLI}, in the walls of the upper large intestine (ULI) and lower large intestine (LLI) of the GI tract (ICRP publication 30, 1979).
d Thoracic airways.
e For purposes of calculation, the remainder is composed of the following ten additional tissues and organs: adrenals, brain, extrathoracic airways, small intestine, kidneys, muscle, pancreas, spleen, thymus and uterus. The extrathoracic airways described in the Human Respiratory Tract Model (ICRP publication 66) were added to the list of remainder tissues in ICRP publication 68. Their masses are given in table 11 of ICRP publication 71.
f Whenever the most exposed remainder tissue or organ receives the highest committed equivalent dose of all organs, a weighting factor of 0.025 (half of remainder) is applied to that tissue or organ and 0.025 (half of remainder) to the mass-averaged committed equivalent dose in the rest of the remainder tissues and organs (ICRP 1994b).

radioactive half-life of ^{238}U is 4,500 million years and, as a heavy metal, it is retained in tissues such as bone for many years. When ^{238}U is inhaled it may therefore continue to irradiate tissues, albeit at a decreasing rate, over the rest of the person's life. Thus although the intake may occur over a short time - a few breaths or a meal - the person is *committed* to receiving a dose for a long time to come. The committed dose (equivalent or effective) is the dose that is expected to be received in a stated period after the intake, usually taken to be 50 years for workers, or up to age 70 for members of the public.

Dose coefficients

A 'dose coefficient' is the equivalent dose (in sieverts) to an organ (or the effective dose) that results from an intake of unit activity (1 becquerel, Bq) of a radionuclide. Since this assessment deals with intakes of radionuclides, these will be committed doses.
ICRP publications 68 and 72 (ICRP 1994b, 1996) give effective dose coefficients for workers and members of the public respectively. The dose coefficients given in ICRP publications 68 and 72 have been adopted in the International Basic Safety Standards (IAEA 1996) and in the Euratom Directive (EC 1996). Organ dose coefficients are given for ingestion and inhalation of uranium compounds by members of the public in ICRP publications 69 and 71 respectively (ICRP 1995a, 1995b). Organ dose coefficients for workers are given in the ICRP Database of Dose Coefficients (ICRP 1998). These dose coefficients thus provide internationally recognised, standard conversion factors to relate intakes of radionuclides to effective doses, and so to risks of harm. They are widely used for purposes such as planning, demonstrating compliance, and environmental impact assessment.

Biokinetic models

In order to calculate committed doses and hence dose coefficients, we have to take account of how the radionuclide behaves after it has entered the body, and this is the role of biokinetic models. Once a radioactive material is inside the human body, it will irradiate the tissues surrounding the place where it is deposited (eg in the lungs), but generally it will in time move to other tissues or organs (eg the kidneys), and irradiate the surrounding tissues there. A biokinetic model describes how a radionuclide moves around a biological system such as a human body. A model is therefore a summary of current knowledge about the processes involved. However, the model describes what happens to the material in terms of mathematical equations. Using it, we can calculate how much of the material that entered the body at the time of the intake is present in each organ at any time afterwards. Generally, the model first describes how the atoms would behave if they were not radioactive, so it actually describes the behaviour of a stable isotope of the element. Radioactive decay is then taken into account to determine the amount of activity present in each organ at any time; the shorter the half-life, the more rapidly the activity decreases. A complicating factor, which also has to be taken into account, is that when some radionuclides decay, the atoms into which they are transformed are themselves radioactive, but are atoms of a different element, and so may behave differently in the body. This is the case for uranium, but the decay products generally only

make small contributions to the effective dose compared to the uranium itself.

One major use of biokinetic models is in calculating committed doses. The biokinetic model is used to calculate how many nuclear transformations (radioactive decays) take place in each organ in a given time after intake. A *dosimetric model* is then applied to calculate how much of the energy released in each type of radiation (eg alpha- or beta-particles) is absorbed in each tissue or organ, and hence the committed doses to the organs. The organ in which the radionuclide is located is known as the 'source organ', and that in which the energy is absorbed is known as the 'target organ'. The dosimetric model takes account of energy absorbed in the source organ itself ('self-irradiation'), which predominates for non-penetrating radiation such as alpha- and beta-particles, and also energy absorbed in other organs ('cross-fire') which can be important for penetrating radiation such as gamma-rays.

The other major use is in the interpretation of measurements of activity present in the body or excreted in urine and/or faeces (known as 'individual monitoring' or 'bioassay'). The biokinetic models are first used to calculate, for a given intake, how much activity is present in the organ measured, or excreted at the specified time after intake. By comparing this with the measured value, the intake can be estimated.

Thus the models may be used 'prospectively', ie in assessments of current, future or hypothetical exposures, or 'retrospectively', ie in assessments of past exposures based on measurements of activity in the body or excreted in urine or faeces. Rather than have a single biokinetic model for the whole human body, it has been found convenient to have separate respiratory tract, GI tract, and systemic models.

Respiratory tract model
A respiratory tract model describes the processes involved in the entry of radionuclides into the body by inhalation, including:

- how much air is breathed in through the nose and mouth;
- how much of the radioactive material in the inhaled air deposits in each part of the respiratory tract (eg the nose or lungs);
- how quickly the radionuclides that have deposited are cleared, either by being carried in mucus to the throat where they are swallowed, or by being absorbed into the bloodstream.

These three stages form the biokinetic model of the respiratory tract. The current ICRP Human Respiratory Tract Model for Radiological Protection (HRTM) is used in the assessments made here. It is described in full in ICRP publication 66 (ICRP 1994a), and a brief description is given in annexe A. The HRTM also includes a dosimetric model to calculate doses to each part of the respiratory tract from inhaled radionuclides. A single respiratory tract model is used for all inhaled radionuclides, but parameter values are changed to reflect predicted differences in behaviour according to the chemical composition of the inhaled material etc. Radionuclides that are cleared from the respiratory tract are then handled by the GI tract or appropriate systemic model, according to whether they are swallowed or absorbed into the bloodstream.

GI tract models
A GI tract model describes the processes involved in the entry of radionuclides into the body by ingestion. The biokinetic model accounts for the time it takes swallowed material to pass through each part of the GI tract, such as the stomach, and what fractions of the swallowed radionuclides are absorbed into the bloodstream, where they are handled by the appropriate systemic model. (The rest is excreted in faeces in due course.) The GI tract model currently used by ICRP comes from ICRP publication 30 (ICRP 1979), and a brief description is given in annexe A. Like the respiratory tract model, a single GI tract model is used for all ingested radionuclides, with only the fraction absorbed into the bloodstream, the so-called 'f_1 value' changing according to the chemical composition of the ingested material. The ICRP publication 30 GI tract model also includes a dosimetric model to calculate doses to each part of the GI tract from radionuclides passing through it.

Systemic models
Systemic models describe the behaviour of radionuclides after they enter the bloodstream (or systemic circulation). Generally, a separate systemic model has been developed for each element, or group of similar elements. The systemic model describes how long the element remains in the circulation, how much goes to each important organ, or is excreted in urine or faeces, and how long it remains in each organ. The behaviour varies greatly between elements: some, like hydrogen, are uniformly distributed; many heavy metals are retained in liver and bone; while iodine concentrates in the thyroid. Up-to-date systemic models which take account of the age of the person have been developed for the 31 most important elements; the current systemic model for uranium is described in detail in ICRP publication 69, and a brief description is given in annexe A.

Implementation of the biokinetic models in computer codes

Two computer codes previously developed at the National Radiological Protection Board (NRPB), and which implement the current ICRP biokinetic and dosimetric models, were used to calculate doses etc per

unit intake: PLEIADES and LUDEP. In addition, fractional uptake to blood and concentrations in kidney were calculated using a commercially available software tool MODELMAKER.

PLEIADES
PLEIADES (Program for Linear Age-dependent Dosimetry) is an NRPB program for calculating dose coefficients for internal emitters. It is currently based on a DEC Alpha platform and uses an eigenvalue method of solution. PLEIADES implements all current ICRP models: systemic, respiratory tract and gastrointestinal tract. PLEIADES also implements the ICRP's full age-dependent methodology described in ICRP publication 67 and the treatment of independent kinetics for decay products detailed in ICRP publication 71. PLEIADES is one of the computer codes used by the ICRP Task Group on Dose Calculations (DOCAL) for the calculation of the dose coefficients published in the various key ICRP publications (eg ICRP 1994b, 1995a,b, 1998) and in the Euratom Directive (EC 1996) and the IAEA Basic Safety Standards (IAEA 1996). Quality Assurance for PLEIADES has been provided for many years by intercomparison of results with those calculated by other DOCAL laboratories and by LUDEP. PLEIADES was used here to calculate doses from intakes.

LUDEP
LUDEP (Lung Dose Evaluation Program) is a personal computer program that implements the HRTM Version 2 (Jarvis et al 1996) and is commercially available from NRPB (current version 2.07). It implements the HRTM, but has the previous (ICRP publication 30, ICRP 1979) systemic model for uranium rather than the current (ICRP publication 69, ICRP 1995a) model. However, an in-house modified version (2.81) was available to the authors in which the ICRP publication 69 uranium model is implemented. LUDEP is only applicable to adults and does not implement independent kinetics for decay products. However, it has been shown that for the uranium isotopes considered here, the error introduced by giving the decay products the same biokinetics as the uranium parent is small. LUDEP is more flexible than PLEIADES in that parameter values can easily be changed to investigate the effect on doses. A suitably modified version (with additional software) can be run in batch mode for sensitivity analyses, such as the graphs of deposition and dose as a function of particle size included in annexes A and D and in ICRP (2001).

MODELMAKER
(Family Genetix Ltd, formerly Cherwell Scientific). The software allows compartment models to be implemented quickly on a personal computer, parameter values to be changed easily, and results to be presented in graphical or tabular form as the model is solved.

Outputs from calculations of radiation doses and of uptake of uranium to blood

The required outputs for unit intake of 1 mg DU oxide are as follows:

- doses to organs (high-LET absorbed dose, low-LET absorbed dose, equivalent dose) and effective dose; uptake to blood. All these quantities are the amounts committed to 50 years after intake to evaluate long-term risks. Some examples are calculated of doses to 1, 5 and 10 years after intake (to assess the likelihood that exposures in Kosovo, Bosnia or the Gulf respectively could have produced effects by now);
- maximum uranium kidney concentration;
- central estimate and worst-case for radiation, and central estimate and worst-case for chemical toxicity (see *Battlefield exposure scenarios* in section 2). Worst-cases are different for radiation effects and chemical toxicity: radiation doses from inhaled uranium tend to be higher when solubility (dissolution rate) is low; potential for chemical toxicity will be higher when solubility (dissolution rate) is high.

3 Results

3.1 Introduction
As noted above the assessment is made more manageable by separating it into two steps:

- assessments of intakes, ie amounts entering the body, which can generally be masses or activities; in this case the mass in milligrams (mg) is appropriate. For some scenarios intake rates (mg per unit time) are assessed, which can be multiplied by an appropriate estimate of exposure time;
- assessment of the outcome of unit intake, which can then be scaled by multiplying by the estimated intakes in each scenario.

Assessments of intakes are described in annexe C. Detailed tables of organ doses are given in annexe D.

3.2 Intakes in each battlefield scenario

Level I

Details are given in annexe C, section C2. Estimated intakes are summarised in table 6.

Table 6. Summary of Level I intakes (mg) from inhalation of aerosol

	Central estimate	Worst-case
Inhalation	250	5000

It seems unlikely that an individual would be involved in more than one such incident, so the values in table 6 are taken to be the intakes for Level I.

Level II

Details are given in annexe C, section C3. Estimated intakes are summarised in table 7. We have no statistical data for assessing how many hours personnel might spend working in contaminated vehicles without taking precautions to avoid contamination. For the purposes of this assessment, assume 10 hours as a central estimate and 100 hours as a worst-case. Taking these with the central estimate and worst-case exposure rates respectively gives the exposures listed in table 8.

Level III

Entry into contaminated vehicles
Exposure rates for entry into vehicles are as for Level II (table 7). We have no statistical data for assessing how many hours personnel might spend visiting contaminated vehicles without taking precautions to avoid contamination. For the purposes of this assessment, assume 1 hour as a central estimate and 10 hours as a worst-case. Taking these with the central estimate and worst-case exposure rates gives the exposures listed in table 9.

Downwind of impacts and fires
Details are given in annexe C, section C4. Estimated intakes for a person breathing at 1.2 $m^3 h^{-1}$ (3.3×10^{-4} $m^3 s^{-1}$ corresponding to ICRP 'light work'), directly downwind of a release of 1 kg DU, from impacts and fires (from table C11) are given in table 10.

We have no statistical data for assessing how many impacts or fires, or the masses of DU involved, to which personnel might be exposed at various distances during a battle, in order to assess the total exposure from such events. For the purposes of this assessment, consider a hypothetical soldier downwind of a number of impacts and fires. The numerical values chosen here are inevitably somewhat arbitrary, but it is reasonable to assume that the soldier will be exposed to smoke from a few impacts and fires nearby, and to more at greater distances. As a central estimate assume exposure to impacts and fires, which release a total of 1 kg DU at 100 m upwind of the soldier, 10 kg DU at 1 km upwind and 100 kg DU at 10 km upwind. For a worst-case, take 10 times these amounts. Taking these with the central estimate and worst-case exposures per kg (table 10) gives the exposures listed in table 11.

Table 7. Summary of Level II intake rates (mg h^{-1}) for entry into contaminated vehicles

	Central estimate	Worst-case
Inhalation	1	20
Ingestion	0.5	5

Table 8. Summary of Level II intakes (mg) for entry into contaminated vehicles

	Central estimate	Worst-case
Inhalation	10	2000
Ingestion	5	500

Table 9. Summary of Level III intakes (mg) for entry into contaminated vehicles

	Central estimate	Worst-case
Inhalation	1	200
Ingestion	0.5	50

Table 10. Central estimate and worst-case estimates of DU inhaled (mg) by an individual directly downwind from release of 1 kg respirable DU generated by explosive impacts or fires

	Central estimate		Worst-case	
Distance (km)	Impacts	Fire	Impacts	Fire
0.1	0.022	0.013	0.20	0.054
1	0.0032	0.0021	0.026	0.013
10	0.00014	0.00011	0.00032	0.00016

Table 11. Summary of Level III intakes (mg) downwind of impacts and fires

	Central estimate	Worst-case
Impacts Distance (km)		
0.1	0.022	2.0
1	0.032	2.6
10	0.014	0.32
Total	**0.068**	**4.9**
Fires Distance (km)		
0.1	0.013	0.54
1	0.021	1.3
10	0.011	0.16
Total	**0.045**	**2.0**

Resuspension of soil

Estimated intakes from resuspended soil are assessed in annexe C, section C4.2. It is assumed that there is an initial deposition of 1 g DU m^{-2}. It is assumed that soldiers spend 28 days in the area (24 hours per day), and estimates of intake are provided for each of the four weeks. However, since resuspension rates decrease rapidly with time after deposition, the duration is not an important factor. Two approaches are used: the 'resuspension factor' and the 'dust loading approach', but the former is considered preferable over this time scale, and the results based on that approach are used here. The central estimate is based on UK-like conditions, and the worst-case is based on arid, dusty conditions. It is recognised that there is considerable uncertainty (two to three orders of magnitude) in rates of resuspension, and therefore the worst-case estimate was multiplied by a factor of 10.

Total intakes over the 28 days are given in table 12. For calculation of doses etc, the conservative assumption is made that the entire intake occurs at one time. Furthermore, since most of the intake will occur during the working day, parameter values appropriate to work activities are used to calculate doses etc per unit intake. However, since the resuspension rate decreases rapidly with time this will not lead to gross overestimates.

Level III summary

Level III intakes are summarised in table 13, in some cases rounded from previous values.

3.3 Parameter values to determine doses per unit intake in each scenario

Summary of results from literature review

The following input parameter values were based mainly on the reports of studies on aerosols formed from impacts and from combustion of DU (annexes G and H).

Size distribution

Measurements are available on aerosols formed immediately after impact: AMAD (activity median aerodynamic diameter, see annexe A section A2.3)

Table 12. Summary of Level III intakes (mg) from inhalation of resuspended soil in 28 days after deposition of 1 gram DU m^{-2}

	Central estimate	Worst-case
Inhalation	0.8	80

Table 13. Summary of Level III intakes (mg)

	Central estimate	Worst-case
Vehicle entry: inhalation (table 9)	1	200
Vehicle entry: ingestion (table 9)	0.5	50
Inhalation downwind of impacts (table 11)	0.07	5
Inhalation downwind of fires (table 11)	0.05	2
Inhalation of resuspended soil (table 12)	0.8	80

~2 µm, GSD (geometric standard deviation) ~13 (unusually wide). For other situations (later times or greater distances from impact, aerosols from combustion, or resuspended aerosols) there is insufficient information to justify any change from the default parameter values (workplace AMAD = 5 µm, GSD = 2.5; environment AMAD = 1 µm, GSD = 2.5).

Density

The chemical form of particles formed from impacts is a mixture of U_3O_8 and UO_2 (and perhaps amorphous uranium oxide), but predominantly U_3O_8. The chemical form from combustion is almost entirely U_3O_8. Hence a density based on these was used when the aerosol is mainly DU oxide. The density of UO_2 = 10.96 g cm^{-3} and that of U_3O_8 = 8.38 g cm^{-3} (Lide and Frederikse 1996). To determine the specific activity of the DU oxide, U_3O_8 was assumed. Hence 1 mg DU oxide = 0.85 mg U (uranium content of U_3O_8 =3x238/(3x238+8x16) = 0.848). (For UO_2 it would be 0.88 mg). Thus a central estimate of 9 g cm^{-3} was used and a worst-case of 11 g cm^{-3}. It is recognised that since many of the particles will be agglomerates, the effective particle density could well be less than the bulk density of U_3O_8 assumed. This may result in some overestimation of doses etc, because deposition tends to increase with density (annexe A).

The ICRP publication 66 default density (3.0 g cm^{-3}) was used when DU is a minor component of the aerosol, eg for resuspended soil.

Shape factor

Patrick and Cornette (1982) (annexe G) studied the morphological characteristics of particles formed from impacts of DU projectiles on an armour target. They found that most were spherical or ellipsoidal but with highly convoluted surfaces, and extensively fractured. Many ultrafine particles (<0.1 µm) were on the surface of larger particles. Hence, there is no justification for changing from the default value of 1.5, which applies to compact irregular particles.

Dissolution

Results of *in vitro* tests on material collected from DU impacts and combustion were used to define the fraction that dissolves rapidly. For aerosols formed from impacts (annexe G, table G1) a typical value is 0.3 with a range of 0.1 – 0.5. For aerosols formed from combustion (annexe H, table H1) a typical value is 0.05 with a range of 0.05 – 0.1.

Results of *in vivo* experiments on U_3O_8 and UO_2 (annexe A, table A5) were used to assess the rapid dissolution

Table 14 Parameter values for Level I inhalation of impact aerosol. Except where indicated, parameter values are only given where they differ from those used in ICRP publication 68 (default) to calculate dose coefficients for workers for UO_2 and U_3O_8

Parameter	Central estimate	Worst-case (radiation)	Worst-case (chemical toxicity)
Subject exercise level, type	Heavy exercise	Heavy exercise, habitual mouth breather	Heavy exercise, habitual mouth breather
AMAD[1], µm	2 (table G2)	0.8 (lowest reported in table G2)	0.8 (lowest reported in table G2)
GSD[2]	13 (table G2, in particular Chambers et al 1982)	18 (highest reported in table G2)	18 (highest reported in table G2)
Density ρ, g cm^{-3}	9 (UO_2 and U_3O_8 are ~11.0 and ~8.4 respectively. Mainly U_3O_8)	11 (UO_2)	11 (UO_2)
Shape factor, χ	1.5 (default)	1.5 (default)	1.5 (default)
Rapid dissolution fraction, f_r	0.3 (typical of table G3)	0.1 (lowest from table G3)	0.5 (highest from table G3)
Rapid dissolution rate, s_r, d^{-1}	1 (typical of U compounds, table A5)	0.4 (low value for U compounds, table A5)	14 (high value for U compounds, table A5)
Slow dissolution rate, s_s, d^{-1}	0.001 (typical of U_3O_8 *in vivo*, table A5)	0.0001 (as type S, see table A3)	0.0015 (highest for U_3O_8 or UO_2 *in vivo*, table A5)
Gut uptake factor, f_1	0.002 (default for type S)	0.02 (default for types M and F)	0.02 (default for types M and F)

1 AMAD: Activity median aerodynamic diameter; see annexe A section A2.3
2 GSD: Geometric standard deviation; see annexe A section A2.3

Table 15. Parameter values for Level II or III inhalation of resuspension aerosol (impact or combustion) within vehicle. Except where indicated, parameter values are only given where they differ from those used in ICRP publication 68 to calculate dose coefficients for workers for UO_2 and U_3O_8. Justification given only where also different from table 14

Parameter	Central estimate	Worst-case (radiation)	Worst-case (chemical toxicity)
Subject exercise level, type of breathing	Heavy work (7 h light exercise + 1 h heavy exercise, ICRP publication 66, table 6)	Heavy exercise, habitual mouth breather	Heavy exercise, habitual mouth breather
AMAD, μm	5 (default workplace)	1 (resuspension aerosols normally 'coarse', >1 μm)	1 (resuspension aerosols normally 'coarse', >1 μm)
GSD	2.5 (default)	4 (upper bound for aerosol from single source)	4 (upper bound for aerosol from single source)
Density ρ, g cm^{-3}	9	11	11
Rapid dissolution fraction, f_r	0.2 (mid-way between typical values, table 14 for impacts and table 18 for combustion)	0.005 (lowest for combustion in table 18)	0.5 (highest for impact in table 14)
Rapid dissolution rate, s_r, d^{-1}	1	0.4	14
Slow dissolution rate, s_s, d^{-1}	0.001	0.0001	0.0015
Gut uptake factor, f_1	0.002	0.02	0.02

Table 16. Parameter values for Level II or III ingestion (hand to mouth transfer in contaminated vehicles)

Parameter	Central estimate	Worst-case (radiation)	Worst-case (chemical toxicity)
Gut uptake factor, f_1	0.002 (ICRP publication 68 default for UO_2 and U_3O_8)	0.02 (ICRP publication 68 default for U compounds except UO_2, U_3O_8 and most tetravalent compounds)	0.02 (ICRP publication 68 default for U compounds except UO_2, U_3O_8 and most tetravalent compounds)

rate ($1 d^{-1}$) and the central estimate of the slow dissolution rate ($0.001 d^{-1}$). It is recognised however that the dissolution rate of these compounds is likely to depend on the process of formation, size distribution etc. Furthermore, the material is not pure uranium, but typically contains 0.75% titanium, and in the course of an impact material from the target (mainly iron) will be incorporated in the aerosol. For the worst-case estimate for radiation dose, it was noted that very long-term lung retention has occasionally been observed in follow-up studies of humans occupationally exposed to uranium compounds, and a slow dissolution rate of $0.0001 d^{-1}$, as for default type S (see annexe A) was assumed.

To make the assessment manageable, it was assumed that the characteristics of resuspended aerosols are the same, whether the material originated from impact or fire. The other parameter values selected relate mainly to the subject and exercise level.

The following exposure situations were considered and the parameter values selected are given in tables 14 to 19:

- Level I inhalation of impact aerosol (close to the impact in space and time) (table 14);
- Level II or III inhalation of resuspension aerosol (impact or combustion) within vehicles (table 15);
- Level II or III ingestion (hand to mouth transfer in contaminated vehicles) (table 16);
- Level III inhalation of plume of smoke from impact (table 17);
- Level III inhalation of plume of smoke from DU combustion (table 18);
- Level III inhalation by resuspension of dust from ground contaminated by impact or smoke from DU combustion (table 19).

3.4 Doses per unit intake

Annexe D gives detailed tables of organ doses (absorbed and equivalent) for each scenario and for a typical combination of uranium isotopes in DU (table 1). Because of the current interest in the dose that lymph nodes receive from inhalation of DU oxides, doses to thoracic and extrathoracic lymph nodes (LN_{TH} and LN_{ET}) are also given, although these are treated by ICRP as regions of the respiratory tract (extrathoracic and

Table 17. Parameter values for Level III inhalation of plume of smoke from impact. Except where indicated, parameter values are only given where they differ from those used in ICRP publication 68 to calculate dose coefficients for workers for UO_2 and U_3O_8. Justification given only where also different from table 14

Parameter	Central estimate	Worst-case (radiation)	Worst-case (chemical toxicity)
Subject exercise level, type of breathing	Light work (2.5 h sitting + 5.5 h light exercise, ICRP publication 68 default)	Heavy exercise, habitual mouth breather	Heavy exercise, habitual mouth breather
AMAD, μm	1 (default for environment, ICRP publication 71)	0.03 (maximum, see fig. D1)	0.06 (maximum, see fig. D4)
GSD	2.5 (default)	1.04 (default for AMAD and ρ)	1.12 (default for AMAD and ρ)
Density ρ, g cm^{-3}	9	11	11
Rapid dissolution fraction, f_r	0.3	0.1	0.5
Rapid dissolution rate, s_r, d^{-1}	1	0.4	14
Slow dissolution rate, s_s, d^{-1}	0.001	0.0001	0.0015
Gut uptake factor, f_1	0.002	0.02	0.02

Table 18. Parameter values for Level III inhalation of plume of smoke from DU combustion. Except where indicated, parameter values are only given where they differ from those used in ICRP publication 68 to calculate dose coefficients for workers for UO_2 and U_3O_8. Justification given only where also different from table 14

Parameter	Central estimate	Worst-case (radiation)	Worst-case (chemical toxicity)
Subject exercise level, type of breathing	Light work (2.5 h sitting + 5.5 h light exercise, ICRP publication 68 default)	Heavy exercise, habitual mouth breather	Heavy exercise, habitual mouth breather
AMAD, μm	1 (default for environment, ICRP publication 71)	0.03 (maximum, see fig. D2)	0.07 (maximum, see fig. D5)
GSD	2.5 (default)	1.04 (default for AMAD and ρ)	1.16 (default for AMAD and ρ)
Density ρ, g cm^{-3}	9	11	11
Rapid dissolution fraction, f_r	0.05 (typical of table H4)	0.005 (lowest in table H4)	0.1 (highest in table H4)
Rapid dissolution rate, s_r, d^{-1}	1	0.4	14
Slow dissolution rate, s_s, d^{-1}	0.001	0.0001	0.0015
Gut uptake factor, f_1	0.002	0.02	0.02

thoracic airways respectively), not as individual organs. A summary of effective doses per unit intake is presented here (table 20).

The doses given in table 20 are committed effective doses, integrated to 50 years after intake. To illustrate how doses build up with time, and to give an overview of individual tissue doses, tables 21 and 22 give committed doses to 1, 5, 10 and 50 years after intake, and a selection of organ doses. (Doses to all organs for these cases are given in annexe D.) As representative of intakes by inhalation, Level II or III intakes by inhalation of resuspension aerosol within contaminated vehicles (table 15) are considered in table 21. There is not much difference in the pattern of organ doses between the various inhalation scenarios (except between the central estimate and worst-case for each). This scenario has the potential to lead to relatively high exposures and involves a mixture of impact and combustion aerosols.

The highest organ doses are generally to the lungs, extrathoracic (ET) airways, bone surfaces, and kidneys. Doses to the thoracic lymph nodes (LN_{TH}) and extrathoracic lymph nodes (LN_{ET}) are given, because they are relatively high, and doses to red bone marrow, because of current interest in leukaemia, for which it is generally considered to be the target tissue. For both the central estimate and worst-case, just over half of the (50-year) committed effective dose is received in the first year. Most of the lung dose comes in the first year, but other doses, particularly those to lymph nodes and red bone marrow, build up more slowly.

As representative of intakes by ingestion, Level II or III intakes by hand to mouth transfer within contaminated vehicles (table 16) are considered in table 22. In this case, the highest doses are to GI tract organs: ULI (upper large intestine), LLI (lower large intestine) (the colon is a combination of ULI and LLI, see table 5). The highest doses to other organs are to liver and kidneys, and again doses to red bone marrow are included. Most of the

Table 19. Parameter values for Level III inhalation by resuspension from ground contaminated by impact or smoke from DU combustion. Except where indicated, parameter values are only given where they differ from those used in ICRP publication 68 to calculate dose coefficients for workers for UO_2 and U_3O_8. Justification given only where also different from table 14.

Parameter	Central estimate	Worst-case (radiation)	Worst-case (chemical toxicity)
Subject exercise level, type of breathing	Light work (2.5 h sitting + 5.5 h light exercise,ICRP publication 68 default)	Heavy exercise, habitual mouth breather	Heavy exercise, habitual mouth breather
AMAD, µm	5 (typical of resuspension aerosols, close to source, Dorrian 1997)	1 (resuspension aerosols normally 'coarse', >1 µm)	1 (resuspension aerosols normally 'coarse', >1 µm)
GSD	2.5 (default)	4 (upper bound for aerosol from single source)	4 (upper bound for aerosol from single source)
Density ρ, g cm^{-3}	3.0 (default, DU oxide minor component of soil)	11	11
Rapid dissolution fraction, f_r	0.2 (mid-way between typical values, table 1 for impacts and table 5 for combustion)	0.005 (lowest for combustion in table 5)	0.5 (highest for impact in table 1)
Rapid dissolution rate, s_r, d^{-1}	1	0.4	14
Slow dissolution rate, s_s, d^{-1}	0.001	0.0001	0.0015

Table 20. Effective doses (µSv) from intakes of 1 mg DU oxide[a,b]

Scenario	Table	Central estimate	Worst-case
Level I inhalation of impact aerosol	14	88	220
Level II or III inhalation of resuspension aerosol within contaminated vehicle	15	52	220
Level II or III ingestion within contaminated vehicle	16	0.097	0.58
Level III inhalation of plume from impact	17	55	560
Level III inhalation of plume from fire	18	71	610
Level III inhalation of resuspension from ground[c]	19	37	220

a 1 mg DU oxide = 0.848 mg DU (14.9 Bq mg^{-1}, table 2) = 12.6 Bq uranium isotopes
b For comparison, effective doses for default type M and type S (5 µm AMAD inhaled by a reference worker, as used to calculate dose coefficients in ICRP publication 68 are 20 and 72 µSv, respectively).
c Doses calculated for an acute intake of the total amount inhaled over 28 days

Table 21. Committed doses (µSv) to organs following Level II or III intake by inhalation of 1 mg DU resuspension aerosol within contaminated vehicles (table 15)

	Central estimate				Worst-case			
Time after intake, years	1	5	10	50	1	5	10	50
Tissue								
Bone surfaces	2.6	6.5	9.3	16	1.2	3.6	6.0	13
Kidney	2.3	3.9	4.9	6.0	1.0	2.3	3.3	4.9
Red bone marrow	0.30	0.78	1.1	1.7	0.14	0.44	0.71	1.4
Extrathoracic airways	190	360	370	370	100	260	310	310
Lungs	320	350	370	370	1100	1500	1600	1800
LN_{ET}	7.8	57	78	83	4.2	65	160	550
LN_{TH}	67	260	330	340	230	1800	4100	19,000
Effective dose	**39**	**51**	**51**	**52**	**130**	**170**	**200**	**220**

Table 22. Committed doses (μSv) to organs following Level II or III intake by ingestion of 1 mg DU (hand-to-mouth transfer) within contaminated vehicles (table 16)

	Central estimate				Worst-case			
Time after intake, years	1	5	10	50	1	5	10	50
Tissue								
Bone surfaces	0.21	0.39	0.54	0.91	2.1	3.9	5.4	9.1
Kidney	0.17	0.23	0.28	0.33	1.7	2.3	2.8	3.3
Red bone marrow	0.024	0.048	0.062	0.096	0.24	0.48	0.62	0.96
Liver	0.011	0.039	0.065	0.12	0.11	0.39	0.65	1.2
ULI	0.20	0.20	0.20	0.23	0.21	0.24	0.27	0.50
LLI	0.57	0.59	0.60	0.62	0.60	0.62	0.65	0.89
Colon	0.35	0.37	0.38	0.40	0.38	0.40	0.43	0.67
Effective dose	**0.053**	**0.060**	**0.068**	**0.097**	**0.12**	**0.20**	**0.27**	**0.58**

doses to the GI tract organs are received within one year, reflecting the short residence time, while doses to organs resulting from absorption to blood and subsequent deposition accumulate more slowly.

3.5 Estimated total doses in each scenario

Table 23 gives a summary of the estimated intakes for each scenario (from tables 6, 8, and 13) and the estimated effective doses per unit intake (1 mg DU oxide) from table 20. The central estimate and worst-case intakes are multiplied by the central estimate and worst-case doses per unit intake, respectively, to produce the central estimate and worst-case doses. Doses over 20 mSv are highlighted in bold type; 20 mSv corresponds to the current annual dose limit for workers in the UK and about nine times the average dose from natural background radiation in the UK (2.24 mSv). It also corresponds to twice the annual dose from radon and its decay products at the Action Level (200 Bq radon-222 m^{-3}) at which householders in the UK are advised to consider remedial measures to reduce exposure (NRPB 1990).

Central estimates of doses are small, <0.1 mSv in all cases except for the Level I exposure, at about 20 mSv. Worst-case estimates are much higher than the corresponding central estimates, two to four orders of magnitude higher, reflecting the large uncertainties in major parts of the assessment: the amounts of material dispersed; resuspension factors etc. Nevertheless, only in a few cases are they greater than 20 mSv: Level I inhalation exposures and both Level II and Level III inhalation exposures to resuspended dust inside vehicles. The worst-case doses for Level I and Level II inhalation exposures are substantial at around 1 Sv.

3.6 Uptake to blood and maximum kidney concentration per unit intake

Annexe D gives details, including information on the time course of uptake. A summary of the fraction of intake absorbed into blood (the *uptake*) by 50 years after intake is given in table 24. For the inhalation scenarios, central estimates range from 3% to 10% of the intake; worst-case estimates range up to about 50%. For the ingestion scenario, the central estimate is 0.2% of the intake, and the worst-case estimate is 2%. Because some standards for exposure to uranium are based on the concentration in the kidneys the maximum concentration of uranium in the kidneys

Table 23. Summary of estimated intakes (mg) and effective doses (mSv) in battlefield scenarios. Doses above 20 mSv are highlighted in bold

Scenario	Central estimate			Worst-case		
	Intake, mg[a]	mSv per mg[b]	mSv	Intake, mg[a]	mSv per mg[b]	mSv
Level I inhalation of impact aerosol	250	0.088	**22**	5000	0.22	**1100**
Level II inhalation of resuspension aerosol within contaminated vehicle	10	0.052	0.5	2000	0.22	**440**
Level II ingestion within contaminated vehicle	5	0.000097	0.0005	500	0.00058	0.3
Level III inhalation of resuspension aerosol within contaminated vehicle	1	0.052	0.05	200	0.22	**44**
Level III ingestion within contaminated vehicle	0.5	0.000097	0.00005	50	0.00058	0.03
Level III inhalation of plume from impacts	0.07	0.055	0.004	5	0.56	2.8
Level III inhalation of plume from fires	0.05	0.071	0.004	2	0.61	1.2
Level III inhalation of resuspension from ground	0.8	0.037	0.03	80	0.22	18

a From tables 6, 8, and 13
b From table 20, converted from μSv to mSv

Table 24. Fraction of intake (%) absorbed into blood by 50 years after intake

Scenario	Table	Central estimate	Worst-case
Level I inhalation of impact aerosol	14	10.2	22
Level II or III inhalation of resuspension aerosol within contaminated vehicle	15	3.6	20
Level II or III ingestion within contaminated vehicle	16	0.20	2.0
Level III inhalation of plume from impacts	17	8.7	53
Level III inhalation of plume from fires	18	6.1	34
Level III inhalation of resuspension from ground[a]	19	3.3	20

a Calculated for an acute intake of the total amount inhaled over 28 days

Table 25. Maximum concentration of uranium in kidney ($^{max}[U]_k$, µg U per gram kidney) following intake of 1 mg DU oxide

Scenario	Table	Central estimate	Worst-case
Level I inhalation of impact aerosol	14	0.016	0.08
Level II or III inhalation of resuspension aerosol within contaminated vehicle	15	0.0047	0.048
Level II or III ingestion within contaminated vehicle	16	0.0006	0.006
Level III inhalation of plume from impacts	17	0.013	0.12
Level III inhalation of plume from fires	18	0.0024	0.025
Level III inhalation of resuspension from ground[a]	19	0.004	0.048

a Calculated for an acute intake of the total amount inhaled over 28 days

($^{max}[U]_k$, (µg U per gram kidney) has also been calculated. A summary of values of $^{max}[U]_k$ following intake of 1 mg DU oxide is given in table 25.

3.7 Estimated total uptake to blood and maximum kidney concentration in each scenario

Table 26 gives a summary of the estimated intakes for each scenario (from tables 6, 8, and 13) and the estimated uptake to blood per unit intake (1 mg DU oxide) from table 24. The central estimate and worst-case intakes are multiplied by the central estimate and worst-case uptakes per unit intake, respectively, to produce the central estimate and worst-case uptakes.

Table 27 gives a summary of the estimated maximum kidney concentrations in each scenario derived in the same way as the uptakes above. UNSCEAR (2000, table 19, page 128) gives a reference value for the level of uranium in the kidney resulting from intakes in diet etc of 30 mBq $^{238}U\ kg^{-1}$ (0.0025 (µg U per gram kidney). Harley et al (1999, table 2.2) give a range of 0.00011–0.00254 µg U per gram kidney. Maximum kidney concentrations over 3 µg U per gram kidney are highlighted in bold type as this level has been used as the basis for occupational exposure limits (Leggett 1989, Harley et al 1999). All central estimates lead to concentrations well below this level, except for the Level I inhalation, which is just above it.

Table 26. Summary of amounts (mg) absorbed into blood in battlefield scenarios

Scenario	Central estimate			Worst-case		
	Intake, mg[a]	% uptake[b]	Uptake, mg	Intake, mg[a]	% uptake[b]	Uptake, mg
Level I inhalation of impact aerosol	250	10.2	26	5000	22	1100
Level II inhalation of resuspension aerosol within contaminated vehicle	10	3.6	0.4	2000	20	400
Level II ingestion within contaminated vehicle	5	0.20	0.01	500	2.0	10
Level III inhalation of resuspension aerosol within contaminated vehicle	1	3.6	0.04	200	20	40
Level III ingestion within contaminated vehicle	0.5	0.20	0.001	50	2.0	1
Level III inhalation of plume from impacts	0.07	8.7	0.006	5	53	3
Level III inhalation of plume from fires	0.05	6.1	0.003	2	34	0.7
Level III inhalation of resuspension from ground[a]	0.8	3.3	0.03	80	20	16

a From tables 6, 8, and 13
b From table 24

Table 27. Summary of maximum concentrations of uranium in kidney ($^{max}[U]_k$, µg U per gram kidney) following intakes in battlefield scenarios. Values above 3 µg U per gram kidney are highlighted in bold

Scenario	Central estimate			Worst-case		
	Intake, mg[a]	$^{max}[U]_k$ per mg[b]	$^{max}[U]_k$	Intake, mg[a]	$^{max}[U]_k$ per mg[b]	$^{max}[U]_k$
Level I inhalation of impact aerosol	250	0.016	**4**	5000	0.08	**400**
Level II inhalation of resuspension aerosol within contaminated vehicle	10	0.0047	0.05	2000	0.048	**96**
Level II ingestion within contaminated vehicle	5	0.0006	0.003	500	0.006	**3**
Level III inhalation of resuspension aerosol within contaminated vehicle	1	0.0047	0.005	200	0.048	**10**
Level III ingestion within contaminated vehicle	0.5	0.0006	0.0003	50	0.006	0.3
Level III inhalation of plume from impacts	0.07	0.013	0.0009	5	0.12	0.6
Level III inhalation of plume from fires	0.05	0.0024	0.00012	2	0.025	0.05
Level III inhalation of resuspension from ground[a]	0.8	0.004	0.003	80	0.048	**4**

a From tables 6, 8, and 13
b From table 25

Similar to the results for doses (section 3.5), worst-case estimates of maximum kidney concentration are much higher than the corresponding central estimates: two to four orders of magnitude higher, reflecting the large uncertainties in assessments of the amounts of material dispersed, resuspension factors etc. Worst-case levels exceed 3 µg U per gram kidney in the same scenarios as those in which worst-case doses exceed 20 mSv: Level I inhalation exposures and both Level II and Level III inhalation exposures to resuspended dust inside vehicles. However, the worst-case estimated levels also exceed 3 µg U per gram kidney for Level II ingestion inside contaminated vehicles and Level III inhalation from resuspended soil.

4 Conclusions

This assessment indicated that, in most of the battlefield situations considered here, intakes of DU are likely to be small, less than 1 mg. Higher intakes were estimated for some situations: of the order of 10 mg for personnel working on contaminated vehicles without taking precautions (Level II exposures); and of the order of 100 mg for personnel in or close to vehicles when they are struck by DU penetrators (Level I exposures). Central estimates of doses are correspondingly small, <1 mSv, except for the Level I inhalation exposure, at about 20 mSv. Similarly, central estimates of maximum kidney concentrations are well below 3 µg U per gram kidney in all situations considered, except for the Level I inhalation exposure, which is just above it.

However, there are very large uncertainties associated with major parts of the assessment: the amounts of material dispersed, resuspension factors etc. These uncertainties stem partly from limited availability of information, and partly from the inherent variability of the processes involved. The sources of the exposures are impacts of DU penetrators and fires involving DU. The intake of material by a person will depend on the amount of DU involved, details of the event (impact or fire), the interaction of the person with the dispersed material and the location and duration of exposure. For all of these there are wide ranges of possible values. A detailed analysis of such events would be needed to derive reliable probability distributions. There are also large uncertainties in parameter values associated with the processes that have to be modelled, such as resuspension factors and the *in vivo* dissolution rate of the particles formed.

The worst-case estimates are much higher than the corresponding central estimates, reflecting the large uncertainties. Worst-case estimates of intakes are typically two to three orders of magnitude higher than the corresponding central estimates. Worst-case estimates of radiation doses and uptake to blood per unit intake are typically one order of magnitude higher than the corresponding central estimates. Combining these, worst-case estimates of doses and uptake to blood in the various situations are typically two to four orders of magnitude higher than the corresponding central estimates. Nevertheless, only in a few cases are estimated worst-case doses greater than 20 mSv: Level I inhalation exposures and both Level II and Level III inhalation exposures to resuspended dust inside vehicles. The worst-case doses for Level I and Level II inhalation exposures are, however, substantial at around 1 Sv. Worst-case estimated kidney concentrations exceed 3 µg U per gram kidney in the same situations as those in which worst-case doses exceed 20 mSv, and also for Level II ingestion inside contaminated vehicles and Level III inhalation from resuspended soil.

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* No request made

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Appendix 2: Radiological risks of depleted uranium
Dudley Goodhead and Jolyon Hendry

1 ICRP risk estimation for exposures to depleted uranium

The standard international approach to estimating health risks from ionising radiation, including internally deposited radionuclides, follows the recommendations of the International Commission for Radiological Protection (ICRP) (ICRP 1991). This approach for radiological protection is based on:

- an estimation of the physical absorbed dose in gray (Gy) to individual organs in the body from the individual types of radiation. The ICRP have developed a series of biokinetic models to calculate radiation doses to organs from intakes of radionuclides (see appendix 1);
- the conversion of these to the equivalent dose (in sievert, Sv) to the individual organs, by application of a specified radiation weighting factor (w_R) for each individual type of radiation, before summation of the doses. ICRP (1991) specifies that $w_R = 1$ for gamma-rays and other radiations of low linear-energy transfer (LET), and that $w_R = 20$ for alpha-particles of high LET. These values are based on the ICRP's judgements of the relative effectiveness for cancer induction of the different radiation types per unit of absorbed dose;
- the combination of the equivalent doses to individual organs into an effective dose (Sv) for the whole body, by applying a tissue weighting factor (w_T) to the equivalent dose to each organ, before summation of the doses. ICRP (1991) specifies fixed values of w_T, that vary from 0.01 to 0.20 for individual organs, based on their judgements of the relative contribution of that organ or tissue to the total health detriment due to stochastic effects (ie cancer and hereditary effects) resulting from uniform irradiation of the whole body by penetrating low-LET radiation;
- the use of nominal probability coefficients for stochastic effects per individual per unit effective dose, and also the component nominal probability coefficients for individual tissues or organs, to include fatal cancers, detriment from non-fatal cancers, and severe hereditary disorders. The coefficients for individual organs are correspondingly lower than the whole-body coefficients, approximately by the respective tissue weighting factors (w_T). ICRP (1991) includes tables of risk probability coefficients for low-level exposures for a population of working age (table 1), and for a general population, taking into account age dependence. These rounded values of risk coefficients have been recommended by ICRP from their evaluations of excess cancers that have been observed in epidemiological studies of exposed populations, particularly the survivors of the atomic bombs in Japan and certain medical exposures. These data are predominantly from high dose, high dose-rate, exposures to penetrating external low-LET radiation, such as the gamma-ray exposures from the atomic bombs;
- the extrapolation of these human data to low doses and low dose-rates using statistical modelling, theoretical assumptions and experimental data from laboratory systems. For this purpose a dose- and dose-rate-effectiveness factor (DDREF) for low-LET radiation has been defined and given the value of 2 by ICRP (1991);
- the extrapolation to other radiation types, and to non-uniform exposures, by means of radiation-weighting factors (w_R) and tissue-weighting factors (w_T), as above;
- the assumption of a linear no-threshold (LNT) dose response at low doses and low dose-rates. This assumption implies that any dose, however small, carries some risk and that the risk for low-level exposures (including those where risks cannot be directly measured) is directly proportional to dose;
- the assumption that all forms of ionising radiation are carcinogenic provided that some dose is delivered to relevant tissues;
- the inclusion of some additional risk for the hereditary effects via the germ line. ICRP (1991) assigns this a risk probability coefficient of 0.8% Sv^{-1} for a working population, as compared to the corresponding cancer risk probability coefficient of 4.8% Sv^{-1}.

2 The linear no-threshold assumption

This type of risk-assessment clearly includes many assumptions and approximations, which have been derived by ICRP from the limited human and experimental information for application in radiological protection. One major assumption is the linear no-threshold (LNT) dose response.

LNT may be best justified on the basis that radiation carcinogenesis is a stochastic process that results predominantly from a rare radiation event in the multistage sequence of events leading to cancer, such as a specific gene mutation, occurring in a single cell, from the ionising track of the radiation.

LNT has been challenged from both directions, as either overestimating or underestimating the risks of low levels

Table 1. Nominal probability coefficients for individual tissues and organs of a worker population and assigned values of tissue weighting factor (w_T) (ICRP 1991)

Tissue or organ[3]	Probability of fatal cancer[2] ($10^{-2}Sv^{-1}$)	Aggregated detriment[2] ($10^{-2}Sv^{-1}$)	Tissue weighting factor (w_T)
Bladder	0.24	0.24	0.05
Red bone marrow	0.40	0.83	0.12
Bone surface	0.04	0.06	0.01
Breast	0.16	0.29	0.05
Colon	0.68	0.82	0.12
Liver	0.12	0.13	0.05
Lung	0.68	0.64	0.12
Oesophagus	0.24	0.19	0.05
Ovary	0.08	0.12	See gonads[1]
Skin	0.02	0.03	0.01
Stomach	0.88	0.80	0.12
Thyroid	0.06	0.12	0.05
Remainder	0.40	0.47	0.05
Total	4.00	4.74	
	Probability of severe hereditary disorder ($10^{-2}Sv^{-1}$)	Hereditary detriment ($10^{-2}Sv^{-1}$)	
Gonads[1]	0.6	0.80	0.20[1]
Grand Total (rounded)		5.6	1.00

[1] Gonads $w_T = 0.20$ includes cancer in ovary. The coefficients for hereditary effects are based on data from animals (mostly mice) due to lack of evidence in humans.
[2] These nominal coefficients are intended to be representative of lifetime risks (or detriment) for members of a 'world' population, with worker age distribution and equal number of both sexes, as described by the ICRP (1991).
[3] A specific coefficient for kidney is not provided by the ICRP (1991) because it is regarded as having relatively low radiation sensitivity for cancer induction. Together with other unlisted radiogenic tissues and organs, the kidney is included as part of the 'remainder'.

of radiation, under general or specific conditions. However it remains the ICRP recommended approach based on scientific and practical grounds.

Justification of the LNT model may be more secure for high-LET alpha-particle irradiation than for low-LET irradiation, because in the former case the dose is delivered in the form of a relatively small number of densely ionising tracks through only a small proportion of the cell population in the exposed organ. A single alpha-particle through a cell nucleus delivers a high dose (typically ~0.3 Gy) to that nucleus and it is clear from experimental data that this can lead to one or more of a large variety ofchanges to the cell, including gene mutations, visible chromosome rearrangements, induced genomic instability or destruction of the cell. The probability that one of these should, by chance, be a viable cancer-initiating change, might be expected to increase in direct proportion to the small proportion of cells that are thus exposed, and hence lead to an LNT dose response (ie proportional to the number of alpha-decays). Such a rationale was considered by the National Academy of Sciences BEIR VI Committee as supporting evidence for selecting a linear no-threshold relationship between exposure and risk of lung cancer for the relatively low exposures at issue for domestic indoor radon (NAS 1999). The magnitude of the risk was estimated from linear fits to epidemiological data from the more heavily radon-exposed miners, with emphasis on the lower range of exposures and exposure rates. At these levels the alpha-particle decays are far apart in space and time (NAS 1999).

This simple rationale based on single isolated alpha-particles and their effects, may require modification due to longer-range biological processes or local tissue/dose inhomogeneities. 'Bystander' effects have been observed in cellular systems at low or moderate doses of radiation, including from alpha-particles, whereby unirradiated cells in the population receive molecular signals, via diffusible factors or cell-cell contact, that extend the sphere of influence from the irradiated cells (Lehnert and Goodwin 1997, Azzam et al 1998, Little 2000). Such processes may have the potential to distort

dose-responses in either direction. For example, they can increase radiation damage as a single alpha-particle can damage both an irradiated cell and surrounding unirradiated cells, or they could reduce the effects by inducing a homeostatic response, or an adaptive radioresistance, in the tissue micro-environment. Additionally, regions of high local dose due to a non-homogeneous distribution of the alpha-emitting radionuclides (eg those from DU particles or DU shrapnel) could affect local tissue viability and alter cell kinetics, thereby enhancing, or diminishing, the carcinogenic potential of the individual particles. These factors are unlikely to apply equally in all tissues.

3 DU as a radiation carcinogen

According to the ICRP approach, DU in the body, being a source of ionising radiation, should be expected to be carcinogenic, in common with other radionuclides from the natural environment or elsewhere. Because of the very short range of the alpha-particles emitted by DU the risk should be confined predominantly to those organs which contain DU and the risk should be approximately proportional to dose down to the very lowest levels, for which the doses and risks are extremely small. There is also a smaller contribution to dose from more penetrating beta- and gamma-rays, which can extend to other organs.

4 IARC evaluations of Group 1 carcinogens

The International Agency for Research on Cancer (IARC) has evaluated a variety of different ionising radiations in terms of standardised categorisation criteria for carcinogens.

X-radiation and gamma-radiation (low-LET) and neutrons (high-LET) were categorized by IARC (2000) as carcinogenic to humans (Group 1).

IARC (2001) evaluated a variety of individual radionuclides and mixtures when internally deposited in the body. This led to inclusion of global evaluations of two broad categories of internally deposited radionuclides, namely, (i) that internally deposited radionuclides that emit alpha-particles are carcinogenic to humans (Group 1), and (ii) that internally deposited radionuclides that emit beta-particles are carcinogenic to humans (Group 1).

In reaching these conclusions, the IARC Working Groups considered the available evidence on human carcinogenicity, animal carcinogenicity, genetic evidence, and other supporting evidence from a wide range of studies of individual radionuclides and mixtures.

The final evaluation that alpha-emitting radionuclides in general are Group 1 carcinogens, included the following considerations:

- Alpha-particles emitted by radionuclides, irrespective of their source, produce the same pattern of secondary ionisations, and the same pattern of localised damage to biological molecules, including DNA. These effects, observed in *in vitro* systems, include DNA double-strand breaks, chromosomal aberrations, gene mutations, and cell transformation.
- All radionuclides that emit alpha-particles and that have been adequately studied, including ^{222}radon and its decay products, have been shown at sufficient doses to cause cancer in humans and in experimental animals.
- Alpha-particles emitted by radionuclides, irrespective of their source, have been shown to cause chromosomal aberrations in circulating lymphocytes and gene mutations in humans *in vivo*.
- The evidence from studies in humans and experimental animals suggests that similar doses to the same tissues - for example lung cells or bone surfaces - from alpha-particles emitted during the decay of different radionuclides produce the same types of non-neoplastic effects and cancers.

For a number of individual alpha-emitting radionuclides, or mixtures, the IARC concluded that there is sufficient evidence in humans of carcinogenicity and that for a larger number there is sufficient evidence of carcinogenicity in animals.

There was judged to be inadequate evidence in humans for the carcinogenicity of natural uranium internally deposited in the body. In experimental animals there was limited evidence of carcinogenicity for natural uranium and inadequate evidence for ^{233}uranium (an alpha-emitter; half-life of 6×10^{-5} years) and its decay products.

Previously IARC (1999) had evaluated the carcinogenicity of surgical implants and other foreign bodies, particularly those introduced through a puncture in the skin such as bullets, pellets, shrapnel and shell fragments. These are generally non-radioactive, with the exception of DU, and radiation aspects were not explicitly considered. They concluded that there was inadequate evidence in experimental animals for the carcinogenicity of implants of DU and, in their overall evaluations, that implanted foreign bodies of DU are not classifiable as to their carcingenicity in humans (Group 3).

In summary, it may be concluded that internalised DU is expected to be able to cause cancer in humans, but there is little direct human evidence to support this, presumably because of the limited numbers of highly-exposed people and limited information on the actual

doses received. For comparison, the BEIR VI analysis of lung cancer risk for radon exposure was based on a total of about 68,000 exposed miners (from 11 epidemiological studies) who had received a wide range of accumulated lung doses (from <10 mSv to >10,000 mSv), with individual quantitative estimates of cumulative exposure and many years follow-up.

5 Radiation-induced cancer: latency and dose-responses

It is generally accepted that cancers arise in tissue stem cells, or in daughter cells that are transformed into immortalised cells with an unlimited division capacity and a low differentiation probability. These are all target cells for carcinogenesis. Following an initiating event in a normal cell, cancers arise following a sequence of subsequent mutational events, and the whole sequence is called the multistage theory of carcinogenesis (Fearon and Vogelstein 1990). The random nature of these events explains in part the variation in latency periods between individuals. There is a variation in latency between tissues, since the number and duration of the stages of cancer development are tissue-dependent, as well as age-dependent. The latter is due partly to the more rapid proliferation of cells in young growing tissues compared to the replacement-only feature of most adult tissues. Cancers arising after ionising radiation usually take several years or more to develop, because of the rare occurrence of the several mutational events which are all needed finally to result in overt malignancy. These events may be associated in some cases with the phenomenon of 'genomic instability', and the development of mutations in tumour suppressor genes such as p53, which then favour the development of malignancies (Ullrich and Ponnaiya 1998). Hence latency periods are usually of the order of years, and are longer for epithelial (eg lung cancers) and mesenchymal tumours (eg sarcomas), than for haemopoietic malignancies (eg leukaemias).

There have been reports in the media of previously fit soldiers developing leukaemia within 2 - 3 years of serving in areas where DU munitions were employed. These reports need to be substantiated, and the incidence compared with that for comparable unexposed populations. The best documented evidence for latency periods for leukaemia induction by radiation is for the Japanese A-bomb survivors (Ichimaru and Ishimaru 1975, UNSCEAR 1988, 1994, 2000). The latency was shorter for younger individuals and for higher dose levels. For acute and chronic leukaemias, and for the ages of young soldiers, the incidence rose from a few years after irradiation to a peak at 5-10 years and then declined. Other sources of evidence for the time course of leukaemia induction include, for example, the treatment of ankylosing spondylitis with x-rays (Darby et al 1987), and the Thorotrast-injected patients (see below, leukaemia), giving broadly similar time courses. Also, there are the leukaemias arising in cancer patients given radiotherapy and/or chemotherapy for other types of primary cancer (Ng et al 2000), where evidence was quoted for the appearance of these second malignancies from shortly after treatment onwards. However, it should be noted that these were occurring after high treatment doses which also damage the milieu where the target cells reside, and this may affect the development of the disease. Hence, if indeed it is shown that radiation does induce leukaemia in 1-2 years in fit people, at rates above that for unexposed populations, further cases might be expected to arise in subsequent years.

A global epidemiological review and re-evaluation of radiation-induced cancer has recently been completed by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 2000). The organ-specific cancer risks from intakes of alpha-emitting radionuclides are summarised below with emphasis on the main tissues that are believed to be exposed to DU:

- Lymphoma and Myeloma: For non-Hodgkin's lymphoma, a relative risk of 2.5 was reported for over 2000 patients given thorotrast (a thorium compound) as a diagnostic radiation contrast medium, but no analysis was made in relation to the level of exposure (van Kaick et al 1999). For ankylosing spondylitis patients given ^{224}Ra (Wick et al 1999), the number of cases was too small to permit detailed inferences. With radon-exposed uranium miners (Darby et al 1995), the total number of deaths was less than expected from controls, and there was no evidence of a dose-response relationship. Overall, there was considered to be little evidence for an association between internal high-LET irradiation (or internal or external low-LET irradiation) and the risk of non-Hodgkin's lymphoma. There is no evidence for an association of Hodgkin's lymphoma with irradiation, either for external or internal exposures (thorotrast patients and radon-exposed miners). Regarding multiple myeloma, there is very limited information on risks from internal high-LET irradiation. There are studies on patients given thorotrast (Andersson et al 1995, van Kaick et al 1999), and radon-exposed uranium miners (Darby et al 1995). Some studies have suggested an elevated risk, but based on small numbers of cases.

- Leukaemia: Dose-related increases in leukaemia risk, in particular myeloid leukaemia, have been found among patients with large exposures to thorotrast in series in Germany (van Kaick et al 1999), Denmark (Andersson et al 1993, 1995), Portugal (dos Santos Silva et al 1999), and Japan (Mori and Kido 1999). There is less evidence for elevated risks among patients injected with radium or in the radium dial painters

and, as discussed above and in appendix 3, there is no evidence of increased risk from uranium.

- Lung cancer: There is substantial information from cohorts of radon-exposed uranium miners. The risk appears to increase linearly with cumulative exposure (Lubin and Boice 1997). Data for domestic radon exposures are consistent with the miner data (Lubin and Boice 1997). Workers at the Mayak plant in Russia had an elevated risk from high lung doses from plutonium (Tokarskaya et al 1995, Koshurnikova et al 1996, 1997).

- Bone tumours: Most of the information on bone tumour risks and internal high-LET irradiation comes from studies of intakes of radium for the treatment of ankylosing spondylitis (Nekolla et al 2000) and other diseases, and from studies of radium dial painters (UNSCEAR 1994). There is strong evidence that large intakes of radium induce bone sarcomas. The excess absolute risk decreases with increasing time since exposure, and with age at exposure, and the effect of exposure rate is small at doses below around 10 Gy. The data are consistent with a linear dose-response over a range up to more than 100 Gy accumulated dose, although there is uncertainty in extrapolating the findings down to doses of a few gray (UNSCEAR 2000).

- Kidneys: There is little specific evidence for kidney tumours being induced in man by radiation (Dupree-Ellis et al 2000, UNSCEAR 2000). A recent report indicates that high doses of ^{233}U (as well as the alpha-particle-emitting actinides ^{239}Pu and ^{241}Am) can cause kidney tumours in mice (Ellender et al 2001). However, the relative contributions of the radiation and the chemical properties of ^{233}U in tumour induction is unknown.

- Liver: Studies of thorotrast-exposed patients in different series consistently show increased risks of liver cancer from alpha-radiation exposure, in a dose-dependent fashion (van Kaick et al 1999).

6 Effects of alpha-particles

Alpha-particles emitted from radionuclides have very short ranges; the 4.2 MeV and 4.4 MeV alpha-particles from ^{238}U and ^{235}U have ranges in tissue of about 30 μm (ICRU 1993). Therefore, they have the capability of traversing only a few cells in their path and the main carcinogenic potential of alpha-emitters should depend greatly on their distribution within the body and their location near to critical cells within tissues. The alpha-particle ranges in pure uranium are less than 10 μm, so there will be substantial self-absorption within aerosol particles or fragments of larger size.

A single alpha-particle traversing a cell delivers a large absorbed dose to that cell (typically 0.1-0.5 Gy, or 2-10 Sv if converted to equivalent dose with $w_R = 20$). This has a high probability of causing very substantial DNA damage in the cell including many complex double-strand breaks. A single alpha-particle (or a few) has been shown to be capable of inducing essentially any cellular effect that is known from ionising radiation. This includes inactivation of the cell (death by apoptosis or necrosis, or loss of proliferative ability), gene mutations, visible chromosome aberrations, genomic instability and malignant transformation. These include the stochastic events that are commonly assumed to be the primary radiation step in the multistage carcinogenic process. With regard to the carcinogenic potential of that cell, inactivation is protective because the cell is then unable to express any long-term genetic defects. Different cell types differ considerably in their ability to survive the passage of a single alpha-particle; experimentally available values show that there is variation from essentially zero to greater than 80% survival, with haemopoietic stem cell being towards the lower end of this range (Goodhead 1999). On this basis alone, it should be expected that the relative biological effectiveness of alpha-particles for carcinogenesis (relative to low-LET radiations such as gamma-rays) might vary greatly between tissues and therefore, in some cases at least, deviate considerably from the value of 20 assumed in radiological protection by the use of a universal value of w_R for all tissues.

Information on bystander effects in unirradiated cells of the tissue is not yet sufficiently clear to allow quantitative estimation of their role in enhancing or reducing carcinogenic potential.

Because the alpha-particle energy deposition is concentrated into a small number of densely ionising tracks, most of the cells in the tissue will receive no alpha-particle irradiation at all, except at extremely high average doses. For example, for alpha-particle irradiation providing an accumulated equivalent dose of 100 mSv to a tissue containing cells of diameter 7 μm (e.g. haemopoietic cells), on average about 1% of these cells in the tissue are ever irradiated by an alpha-particle because of the very limited number of alpha-particles.

In the case of ^{238}U or ^{235}U the tissue will also receive some dose from low-LET beta-particles and gamma-rays. Although this is only a small proportion of the equivalent dose (say 2% in a given situation) this corresponds to a much higher proportion of the absorbed dose (~40%) and might mean that many, or all, of the cells have received some direct irradiation by the passage of electron(s). In the above example (102 mSv total dose), the majority of the cells will have received some direct irradiation, by one or more electrons.

7 Quantitative risk estimates for alpha-particle emitters

For most tissues, the ICRP-recommended risk probability coefficients are based on data from human exposures to external low-LET irradiation. These are applied to internal high-LET alpha-emissions by estimating the tissue absorbed doses from biokinetic models and multiplying by $w_R = 20$ to obtain the equivalent doses.

In a few tissues of relatively low radiation sensitivity, for which there are insufficient low-LET data but some quantitative human data after internal alpha-particle irradiation, the primary risk coefficients are based on these alpha-particle data. This is the situation for bone cancers and liver cancers (ICRP 1991). (In these cases the low-LET risk coefficients are derived in reverse, implicitly dependent on the biokinetic evaluation of the alpha-emitter and the value of w_R.)

In the unique case of lung cancers due to radon and its short-lived decay products, from which alpha-decays predominate, the ICRP recommends that the risks are estimated directly from the extensive studies of radon-exposed miners, and in terms of exposure rather than by detailed evaluation of internal dose. To bring this into accord with the general system of radiological protection in terms of effective dose and equivalent dose, ICRP has provided a 'dose conversion convention' to convert these exposures to equivalent doses to lung so as to yield the same risk coefficients as are recommended from the external low-LET radiation data (ICRP 1994).

Lung provides the only case to date where there has been sufficient human data for both low- and high-LET radiation to allow a direct comparison of the two general approaches to risk estimation for internal alpha-emitters, one based on direct epidemiological observations of exposed cohorts of workers, and the other based on the standard ICRP dosimetric approach, including biokinetic models and radiation weighting factors. In this particular case the two estimates differ by a factor of about 3 (or in the range 2-5 depending on the particular conditions considered) (Birchall and James 1994, Porstendörfer and Reineking 1999, NAS 1999). Note that the direct estimate from the epidemiology of exposed miners gives the lower risk. ICRP applies this lower risk and reconciles it with the dosimetric approach by selection of the value of the 'dose conversion convention'.

This difference of about factor 3 may be regarded as a close agreement in view of the very many assumptions and uncertain parameter values inherent in the two approaches. It has been suggested that the difference is unlikely to be reconcilable solely on the basis of uncertainties in parameters of the ICRP biokinetic lung model (Birchall and James 1994). Additional uncertainties arise from the epidemiological analyses and from the general methods of extrapolation from the A-bomb data to the particular alpha-particle exposures to the lung. These include dose and dose-rate assumptions and the radiation weighting factor as applicable specifically to carcinogenesis by these lung exposures. The uncertain influence of cell killing by the individual alpha-particle could alone be sufficient to introduce an uncertainty of at least factor 3 in the true relative radiobiological effectiveness for alpha-particle carcinogenesis in the lung.

For the case of uranium exposures, as with most other situations of internal exposure, detailed evaluations are not available of the combined uncertainties that might result from the overall exposure/dose/risk assessment. It might be reasonable to suggest an order of magnitude uncertainty either way in the final risk estimates, but this suggestion would not have any firm foundation.

The most extensive studies of the hazards of internalised alpha-particle emitters relate to the inhalation of radon gas (half-life of 3.8 days), and its short-lived decay products (half-lives of < 29 minutes) attached to aerosols, which will provide exposure predominantly to the lung. Inhalation of DU aerosols differs significantly as small particles can be retained deep in the lungs, and can be translocated from the lung to the associated lymph nodes where they remain for many years. The accuracy of the risks calculated using ICRP models for alpha-emitting particles that remain within tissue for many years have been challenged by some groups, particularly in the context of the emissions from nuclear power stations and from the radioactive releases resulting from the accident at Chernobyl. As mentioned in the previous paragraph, we recognise some of these uncertainties in extrapolating to situations where radioactive particles or shrapnel are retained in tissue and would suggest that the best estimates of risk from the ICRP approach that we use could underestimate, or overestimate, risk by an order of magnitude.

In principle epidemiological studies on humans exposed to uranium may be able to provide some limits of confidence to the risk estimates for DU obtained by application of the standard ICRP methodology. For example:

a) Are any of the epidemiological studies of uranium workers significantly discordant with the quantitative risk estimates derived for the DU exposures using ICRP methodology, bearing in mind the differences in exposure conditions?

b) Can any of the these epidemiological studies provide meaningful quantitative upper limits to what the actual risks may be for relevant exposure conditions?

These epidemiological studies are reviewed in appendix 3.

8 Estimated radiological risk from the DU-exposure scenarios

This section presents the estimated radiological risks of fatal cancer that result from a selection of the specific exposure scenarios listed in appendix 1. These estimates are obtained as the product of the intakes of DU, the doses per unit intake for individual tissues and organs (both from appendix 1), and the ICRP's nominal probability coefficients for stochastic effects for individual tissues or organs or for whole body risk (table 1). These coefficients are based on a 'world population' and will vary for many types of cancers between different populations. In each case this leads to a 'central estimate' and a 'worst-case' risk per individual in the exposed population. A risk of 1 per hundred (0.01) from the radiological effects of exposure to DU implies that, on average, over the life of an individual, their chance of dying of cancer is increased by 1%.

8.1 Level I inhalation of impact aerosols

The risks of these exposure scenarios use the intakes of table 6 and the parameter values of table 14 of appendix 1, and the doses per unit intake of table D1 in annexe D. The consequent estimates of risk are shown in table 2.

Table 2. Risks from Level I inhalation of impact aerosols

	Central estimate		Worst-case	
DU intake	250 mg		5,000 mg	
	Risk of fatal cancer	Total detriment	Risk of fatal cancer	Total detriment
Whole body	8.8×10^{-4}*	1.2×10^{-3}	4.4×10^{-2}	$6.2.\times10^{-2}$
Individual tissues				
Bladder	9.6×10^{-7}	9.6×10^{-7}	1.0×10^{-5}	1.0×10^{-5}
Red bone marrow	4.9×10^{-6}	1.0×10^{-5}	5.2×10^{-5}	1.1×10^{-4}
Bone surfaces	4.7×10^{-6}	7.1×10^{-6}	5.0×10^{-5}	7.5×10^{-5}
Breast	6.5×10^{-7}	1.2×10^{-6}	6.7×10^{-6}	1.2×10^{-5}
Colon	3.1×10^{-6}	3.7×10^{-6}	3.4×10^{-5}	4.1×10^{-5}
Liver	1.9×10^{-6}	2.0×10^{-6}	2.0×10^{-5}	2.2×10^{-5}
Lungs	1.2×10^{-3}	1.1×10^{-3}	6.5×10^{-2}	6.1×10^{-2}
Oesophagus	9.6×10^{-7}	7.6×10^{-7}	1.0×10^{-5}	8.0×10^{-6}
Ovary	3.2×10^{-7}	4.8×10^{-7}	3.3×10^{-6}	5.0×10^{-6}
Skin	8.0×10^{-8}	1.2×10^{-7}	8.3×10^{-7}	7.5×10^{-6}
Stomach	3.5×10^{-6}	3.2×10^{-6}	3.7×10^{-5}	3.4×10^{-5}
Thyroid	2.4×10^{-7}	4.8×10^{-7}	2.5×10^{-6}	5.0×10^{-6}
Tissues with highest equivalent doses				
Lungs	178 mSv		9,500	mSv[1]
Extra thoracic airways	53 mSv		2,400	mSv
Extra thoracic lymph nodes	12 mSv		4,150	mSv
Thoracic lymph nodes	150 mSv		91,000	mSv[2]
Bone surfaces	12 mSv		125	mSv
Kidney	4.3 mSv		45	mSv
Liver	1.6 mSv		17	mSv
Red bone marrow	1.2 mSv		13	mSv
Effective dose	22 mSv		1,100	mSv

* In this and subsequent tables, the estimates of whole-body risk of fatal cancer are less than the estimates for lung alone. Such a possibility is recognized by ICRP when the distribution of equivalent dose in the body is non-uniform. The discrepancy arises from the approximations and rounded values of w_T that are used to define effective dose, and hence whole-body risk, which includes allowance for detriment for non-fatal cancers and hereditary conditions (see table 1). ICRP (1991) states that "the difference between the two methods will not be significant because the individual tissue coefficients are not known with sufficient accuracy".

[1] This lung dose is due to 0.47 Gy from alpha-particles and 0.09 Gy from low-LET radiation, delivered over >50 years.

[2] This thoracic lymph node dose is due to 4.6 Gy from alpha-particles and 0.39 Gy from low-LET radiation, delivered over >50 years.

From these data the following comments can be made.

a) For all individual tissues or organs the equivalent dose (and therefore risk) is dominated by high-LET radiation (alpha-particles). The alpha-particles are responsible for >92% of the equivalent dose in all cases, and in most cases for >98%. In terms of absorbed dose, alpha-particles are responsible for >40% in all cases and >70% in most cases.

b) For both the central estimate and the worst-case, the greatest risk amongst the ICRP-specified tissues or organs is to the lungs. The estimated risks to other organs are <<1% of these lung risks.

c) For the central estimate the risk to lung is about 1 per thousand and this dominates the overall whole-body risk.

d) For the worst-case the risk to lung is about 6.5 per hundred and this, too, dominates the whole-body risk. The next greatest risks are to bone marrow and bone surfaces, each of which are about 50 per million.

e) The lower part of table 2 lists the tissues or organs that receive the greatest equivalent doses. Most notable are the doses to the lung, and also some of the associated tissues for which ICRP (1991) have not provided individual risk probability coefficients. The very high dose of 91 Sv to the thoracic lymph nodes in the worst-case estimate is due almost entirely to alpha-particles (4.6 Gy absorbed dose, which corresponds to approximately 9 alpha-particles traversals per 'cell location' during the extended duration of the irradiation, perhaps throughout the person's lifetime). Low-LET radiation contributes about 0.39 Sv equivalent dose (0.39 Gy absorbed dose).

f) The risks of irradiation of the thoracic lymph nodes are very uncertain because of a lack of evidence, and ICRP (1991) has not provided estimates of their susceptibility to radiation-induced cancer. Individual lymphocytes are unlikely to survive even one or two alpha-particle traversals, but in a lymph node the killed cells would normally be replaced by circulation. The possibility exists over the extended time course of exposure for occasional lymphocytes and lymphoid/haemopoietic precursor cells to be traversed by an alpha-particle, and to survive, while circulating through these lymph nodes. The high accumulated equivalent dose in thoracic lymph nodes corresponds to 0.09 Gy per year from alpha-particles, and this is equivalent to less than 1 Gy per year of low-LET irradiation per year with regard to organ damage. Hence, some accumulated organ injury might be experienced at the very highest dose levels. Indeed, in experiments in dogs with alpha-particle emitting particles lodged in the lymph nodes, delivering accumulated alpha-particle doses ranging from 1 - 4000 Gy, there was lymphopenia, fibrosis and atrophy of the lung-associated lymph nodes, and pneumonitis (Muggenburg et al 1999). Lymph node cancers or leukaemias were not observed (Hahn et al 1999a). In one review, lymph nodes were considered unlikely as a possible site for development of lymphocytic leukaemia following radioactive particulate intakes (Baverstock and Thorne 1989). Nonetheless, it is possible that any haemopoietic progenitor cells that survive the traversal of an alpha-particle while circulating through lymph nodes could contribute to a risk of leukaemia (COMARE 1996). However, in the large US and UK cohorts of uranium workers, there was no evidence of induced lymphoid malignancies or leukaemia (Polednak and Frome 1981, McGeoghegan and Spinks 2000, appendix 3), and similarly for thorium oxide workers (Polednak et al 1983). In the patients injected for imaging purposes with Thorotrast (a colloidal suspension of thorium oxide), considerable quantities were retained in lymph nodes, as well as in liver, spleen and bone marrow. Where leukaemia was found among the Thorotrast patients, myeloid leukaemias were the dominant type, with long latency periods. From all this evidence it is considered unlikely that DU in lymph nodes would lead to lympho/haemopoietic malignancies. However, it cannot be excluded that irradiation of circulating cells in lymph nodes might contribute to the low incidence of leukaemia in the Thorotrast cases.

8.2 Level II intakes from entry into vehicles

These exposure scenarios include both inhalation and ingestion from entry into contaminated vehicles. The exposure parameter values are as table 15 (inhalation) and table 16 (ingestion) of appendix 1, the doses per unit intake are as table D2 (inhalation) and table D3 (ingestion) in annexe D, and the exposure intakes for this scenario are as in table 8 of appendix 1. The consequent estimates of risk are shown in table 3.

From these data the following comments can be made.

a) For all individual tissues or organs the equivalent dose (and therefore risk) is dominated by high-LET radiation (alpha-particles). The alpha-particles are responsible for >89% of the equivalent dose in all cases, and in most cases >98%.

b) For both the central estimate and the worst-case, the greatest risk amongst the ICRP-specified tissues or organs is for the lungs. The estimated risks to other organs are less than 1% of the lung risk.

c) For the central estimate the risk to the lung is only about 2.5 per hundred thousand and this dominates the whole body risk.

Table 3. Risks from Level II intakes (inhalation + ingestion) from entry into contaminated vehicles

	Central estimate		Worst-case	
DU intake	10 mg (inhalation) + 5 mg (ingestion)		2000 mg (inhalation) + 500 mg (ingestion)	
	Risk of fatal cancer	Detriment	Risk of fatal cancer	Detriment
Whole body	$2.1 \times 10^{-5*}$	2.9×10^{-5}	1.8×10^{-2}	$2.5. \times 10^{-2}$
Individual tissues				
Bladder	1.4×10^{-8}	1.4×10^{-8}	2.5×10^{-6}	2.5×10^{-6}
Red bone marrow	7.0×10^{-8}	1.5×10^{-7}	1.3×10^{-5}	2.7×10^{-5}
Bone surfaces	6.6×10^{-8}	9.9×10^{-8}	1.2×10^{-5}	1.8×10^{-5}
Breast	9.0×10^{-9}	1.6×10^{-8}	1.7×10^{-6}	3.1×10^{-6}
Colon	6.4×10^{-8}	7.7×10^{-8}	1.1×10^{-5}	1.3×10^{-5}
Liver	2.7×10^{-8}	2.9×10^{-8}	5.0×10^{-6}	5.5×10^{-6}
Lungs	2.5×10^{-5}	2.4×10^{-5}	2.4×10^{-2}	2.3×10^{-2}
Oesophagus	1.4×10^{-8}	1.1×10^{-8}	2.5×10^{-6}	2.0×10^{-6}
Ovary	4.5×10^{-9}	6.8×10^{-9}	8.3×10^{-7}	2.1×10^{-6}
Skin	1.1×10^{-9}	1.7×10^{-9}	2.1×10^{-7}	3.1×10^{-7}
Stomach	5.2×10^{-8}	4.7×10^{-8}	9.3×10^{-6}	8.4×10^{-6}
Thyroid	3.4×10^{-9}	6.8×10^{-9}	6.2×10^{-7}	1.2×10^{-6}
Tissues with highest equivalent doses				
Lungs	3.7	mSv	3,600	mSv[1]
Extra thoracic airways	3.7	mSv	620	mSv
Extra thoracic lymph nodes	0.83	mSv	1,100	mSv
Thoracic lymph nodes	3.4	mSv	38,000	mSv[2]
Bone surfaces	0.16	mSv	31	mSv
Kidney	0.062	mSv	12	mSv
Liver	0.023	mSv	4.2	mSv
Red bone marrow	0.017	mSv	3.2	mSv
Effective dose	0.52	mSv	440	mSv

* See footnote to table 2 in appendix 2.
[1] This lung dose is due to 0.18 Gy from alpha-particles and 0.04 Gy from low-LET radiation, delivered over >50 years.
[2] This thoracic lymph node dose is due to 1.9 Gy from alpha-particles and 0.16 Gy from low-LET radiation, delivered over >50 years.

d) For the worst-case the risk to lung is about 2.4 per hundred and this, too, dominates the whole-body risk. The next greatest risks are for bone marrow, bone surface, colon and stomach, each of which are roughly 10 per million.

e) The lower part of table 3 lists the tissues or organs that receive the greatest equivalent doses. Most notable in the worst-case are doses to lung (3.6 Sv), thoracic lymph nodes (38 Sv) and extra thoracic lymph nodes (1.1 Sv); these doses are dominated by the intakes from inhalation. As described in appendix 1, all doses are calculated as 'committed doses' up to 50 years after DU intake. From table 3.19 of appendix 1, it can be inferred that for worst-case inhalation most of the lung dose is accumulated already within the first year. In contrast, most of the thoracic lymph node dose is accumulated quite uniformly throughout the 50 years, due to localisation of the insoluble particles.

8.3 Level III intakes from smoke plumes

This exposure scenario includes inhalation of smoke plumes both from impacts and from fires. The exposure parameters are as table 17 (impact) and table 18 (fire) of appendix 1, the resulting doses per unit intake are as table D4 and table D5 in annexe D, and the intakes for this scenario are as in table 11 of appendix 1. The consequent estimates of risk are shown in table 4.

Table 4. Risks from Level III intakes from smoke plumes from impacts and fires

	Central estimate		Worst-case	
DU intake	0.068 mg (impact)+0.045 mg (fire)		4.9 mg (impact)+2.00 mg (fire)	
	Risk of fatal cancer	Detriment	Risk of fatal cancer	Detriment
Whole body	$2.7 \times 10^{-7*}$	3.9×10^{-7}	1.6×10^{-4}	2.2×10^{-4}
Individual tissues				
Bladder	3.3×10^{-10}	3.3×10^{-10}	2.4×10^{-8}	2.4×10^{-8}
Red bone marrow	1.7×10^{-9}	3.5×10^{-9}	1.2×10^{-7}	2.6×10^{-7}
Bone surfaces	1.6×10^{-9}	2.3×10^{-9}	1.2×10^{-7}	1.8×10^{-7}
Breast	2.2×10^{-10}	4.0×10^{-10}	1.6×10^{-8}	3.0×10^{-8}
Colon	1.0×10^{-9}	1.2×10^{-9}	8.4×10^{-8}	1.0×10^{-7}
Liver	6.6×10^{-10}	7.1×10^{-10}	4.7×10^{-8}	5.1×10^{-8}
Lungs	3.7×10^{-7}	3.5×10^{-7}	1.9×10^{-4}	1.8×10^{-4}
Oesophagus	3.3×10^{-10}	2.6×10^{-10}	2.4×10^{-8}	1.9×10^{-8}
Ovary	1.1×10^{-10}	1.7×10^{-10}	8.1×10^{-9}	1.2×10^{-8}
Skin	2.8×10^{-11}	4.1×10^{-11}	2.0×10^{-9}	3.0×10^{-9}
Stomach	1.2×10^{-9}	1.1×10^{-9}	9.0×10^{-8}	8.1×10^{-8}
Thyroid	8.3×10^{-11}	1.7×10^{-11}	6.1×10^{-9}	1.2×10^{-9}
Tissues with highest equivalent doses				
Lungs	0.055 mSv		28.1 mSv	
Extra thoracic airways	0.022 mSv		2.4 mSv	
Extra thoracic lymph nodes	0.0050 mSv		4.4 mSv	
Thoracic lymph nodes	0.044 mSv		279 mSv	
Bone surfaces	0.0039 mSv		0.30 mSv	
Kidney	0.0014 mSv		0.11 mSv	
Liver	0.0006 mSv		0.039 mSv	
Red bone marrow	0.0004 mSv		0.038 mSv	
Effective dose	0.0069 mSv		4.0 mSv	

* See footnote to table 2

From these data the following comments can be made.

a) For all individual tissues and organs the equivalent dose (and therefore risk) is dominated by high-LET radiation (alpha-particles).

b) For both the central estimate and the worst-case, the greatest risk amongst the ICRP-specified tissues or organs is to the lungs. The estimated risks to any other tissue or organ is <<1% of the lung risk.

c) For the central estimate the risk even to lung is about 4 per ten million, and this lung risk dominates the whole body risk.

d) For the worst-case the risk to the lung is about 2 per ten thousand and this dominates the whole body risk.

e) The lower part of table 4 lists the tissues or organs that receive the greatest equivalent dose. They are mostly very much less than the average equivalent doses received from natural background radiation (in the UK ~1 mSv to all tissues or organs, apart from the lung which is assigned ~10 mSv from radon and its short-lived progeny, giving a total effective dose of 2.24 mSv per year). Notable exceptions for the worst-case DU exposures are lung (28 mSv) and thoracic lymph nodes (280 mSv).

9 Comparison with natural background radiation

The average annual effective dose from all sources of ionising radiation to members of the UK population

has been estimated to be about 2.6 mSv (Hughes 1999). The majority of this (ie 2.24 mSv) is from natural background sources, with the balance being due to medical (0.37 mSv), occupational (0.006 mSv), fallout (0.004 mSv), disposals (0.003 mSv) and consumer products (0.001 mSv). The components of the natural background effective dose are shown in table 5 (Hughes and O'Riordan 1993, Hughes 1999) to provide a comparison with the estimates of effective doses from the various DU exposure scenarios considered in this section (summarised in table 6). In all cases the tissue or organ receiving the greatest equivalent dose (and risk) is the lung, although this is less marked for total natural radiation than for the DU scenarios.

breaks and chromosome rearrangements, as shown in alkaline-elution, sister-chromatid exchange (SCE), dicentric and micronucleus assays (Miller et al 1998a, 2000). *In vivo* studies with embedded DU pellets in animals have shown aberrant expression of oncogenes and tumour suppressor genes that are associated with carcinogenesis, and the uranium-containing urine showed mutagenic activity in the Ames bacterial reversion assay (Miller et al 1998b, 2000). Although many, or all, of the above effects may be expected from the radiation exposure from DU, there are a variety of grounds to suggest that chemical effects may predominate. In the *in vitro* transformation, and sister chromatid exchange studies, induced effects were very much more frequent than might be

Table 5. Average annual effectiveness dose from natural sources

Cosmic rays	0.32	mSv/y
Terrestrial gamma-rays	0.35	mSv/y
Internal long-lived radionuclides Of which: uranium 0.005 mSv/y other heavy elements 0.09 mSv/y	0.27	mSv/y
Radon and short-lived progeny	1.20	mSv/y
Thoron and short-lived progeny	0.10	mSv/y
	2.24	mSv/y

Table 6. Estimated effective doses from DU exposure scenarios

Scenario	Central estimate		Worst-case	
Level I exposure	22	mSv	1,100	mSv
Level II exposure	0.52	mSv	440	mSv
Level III exposure from smoke plumes	0.007	mSv	4.0	mSv

10 Chemical versus radiological action of DU

In our analysis we have estimated only the radiological risk for stochastic effects from the ionising-radiation emissions of DU, following standard international methods. Chemical effects from the DU have not been included, either in isolation, or in synergy with the radiation. Toxic chemical effects of uranium on the kidney and other organs will be considered in our second report. An additional question arises as to the potential for stochastic effects (including cancer) resulting from the chemical action of uranium at the DNA and cellular levels. Experiments with a human osteoblast-like cell line *in vitro* have shown that soluble or insoluble DU can induce neoplastic transformation of the cells (Miller et al 1998a, 2001). Isolated transformants were shown to produce tumours in athymic mice, express the *k-ras* oncogene and have altered phosphorylation of the retinoblastoma tumour suppressor gene. DU has been reported also to be genotoxic, inducing DNA strand

expected from the very small proportion of cells that were hit by an alpha-particle (~$1x10^{-5}$ cells for 10 µm DU). Similar frequencies (within a factor 2) of transformation were observed with the non-radioactive heavy-metal carcinogens nickel and lead (Miller et al 1998a). The other *in vitro* indicators of genotoxicity were also observed with nickel and its alloys (with tungsten and cobalt), but none was detected with the inert metal tantalum (Miller et al 2000). Miller et al (1998a, 2001) have speculated that genotoxicity may be due to the uranyl ions acting as a transition metal to produce free radicals via a Fenton-like reaction, especially if the ions are effectively chelated to DNA as are other metal ions. One could speculate further that the potential for synergistic effects between the radiation and chemical actions of DU would be greatest in the vicinity of particles or fragments of DU, from which essentially all the surrounding cells are chemically exposed and may thereby be sensitized to the occasional radioactive decay particle.

11 Embedded Shrapnel

The dose and dose rate to tissue in immediate contact with an embedded fragment of DU is extremely high. As evaluated in annexe B of appendix 1, tissue in contact with a piece of DU is irradiated with alpha-particles at a dose rate of about 1300 Gy per year (ie 3.6 Gy per day). The alpha-particle dose rate decreases gradually away from the surface, until at about 30 μm distance it drops to zero because of the small ranges of the alpha-particles. In this very thin irradiated shell of tissue most of the cells would be sterilized on a daily basis. For example, for cells with spherical nuclei of about 7 μm diameter, 3.6 Gy would correspond to about 7 alpha-particle traversals per cell nucleus which would almost certainly be lethal to most cell types. This would be a continuing process if the tissue is capable of replacing the cells. Apart from the lethally-irradiated cells, there should be some genetically damaged surviving cells in the lower-dose region near the end of the alpha-particle ranges. Cells beyond this, too, might be affected by bystander effects, if any, from the irradiated cells. The microenvironment of very high local tissue damage and possibly high cell turnover may well influence the carcinogenic potential of any mutated viable cells. In addition the cells would be continuously bathed by dissolved uranium, leading to possible chemical effects from the heavy metal and synergy between chemical and radiation effects. Alternatively, and particularly in non-renewing tissues such as muscle, a fibrotic layer may develop around the DU, as has been observed around DU implants in rats (Hahn et al 1999b). If this layer is more than about 30 μm thick it would shield the tissue from further alpha-particle irradiation.

As described in annexe B of appendix 1, tissue in the vicinity of a piece of DU is exposed also to low-LET irradiation, mostly from beta-particles emitted by the short-lived decay products of the uranium. This beta-exposure extends to about 1.1 cm, corresponding to the maximum range of the beta-particles. The average dose rate in this spherical shell around, say, a 1 cm diameter spherical shrapnel fragment has been estimated to be about 69 Gy per year. The dose rate will be substantially greater than this at the surface of the DU and decrease quite rapidly to near zero at 1.1 cm distance. 69 Gy per year corresponds to about 0.2 Gy per day which implies that on average each cell in the exposed volume is irradiated by a few hundred separate electrons per day. Such a dose rate is unlikely to kill the cells, so an accumulation of viable genetically-damaged cells may be expected. Because the volume of tissue exposed to beta-particles is so very much greater than that exposed to alpha-particles, and because of the high viability of the beta-exposed cells, it may well be that the beta-exposed tissue represents the greater long-term risk. As pointed out in annexe B of appendix 1 the DU fragment may also be sufficiently close to other tissue (eg red bone marrow) for the beta-radiation to reach this too.

In view of the various above possibilities, and the variety of tissues of interest, general risk expectations cannot be made for radiological effects of DU fragments. Conventional ICRP approaches of averaging the dose throughout the tissue, with the underlying assumptions of linearity of dose response and additivity of equivalent doses, are unlikely to be helpful in assessing the risks from the large and inhomogeneous doses that arise from DU shrapnel. It was reported in the abstract from a recent scientific meeting that implants of fragments of DU in the muscle of male Wistar rats have led to significant increases in tumours (Hahn et al 2000). The authors concluded that their results indicate that DU, in sufficient dosage, reduced weight gain in the rats, caused a local tissue reaction and caused local tumours.

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Appendix 3: Epidemiologal studies of uranium workers
Valerie Beral and Sarah Darby

1 Introduction

Human exposure to depleted uranium in battlefield situations is a relatively recent phenomenon and so little information is, as yet, available about the long-term effects of such exposure on the risk of cancer or other chronic illnesses. Exposure to DU-containing weapons occurred in 1991 during the Gulf War, and further exposures occurred later, during the Balkan conflicts. Most of those exposed to DU on the battlefield were soldiers and, as they are generally young adults, their risk of developing cancer in the next decade or so is relatively small. For example, among 53,000 UK veterans of the Gulf War (none of whom is known to have been heavily exposed to DU, ie Level I exposures) only 1 in 1000 - 53 in total - died from cancer during an 8-year follow up period, and this was very similar to the cancer mortality rate in a comparison group of UK armed forces personnel who were in service at the same time but did not serve in the Gulf (Macfarlane 2000). Clinical studies of 28 US Gulf War veterans who had DU-containing shrapnel embedded in their bodies showed no major untoward effects so far (McDiarmid, 2000), but the expected incidence of cancer or other chronic diseases is extremely low in such a small number of young subjects. It will, therefore, probably take decades to evaluate directly the effects of exposure to DU on cancer incidence in exposed populations.

In contrast to the lack of information on military populations exposed specifically to DU, there is a large amount of epidemiological information about populations occupationally exposed to uranium. Information on the effects of uranium exposure is derived largely from follow-up studies of uranium miners and of workers in the nuclear industry. Underground uranium miners are exposed to uranium ore during the course of their work, but they are also exposed to radon gas, a decay product of uranium that is released from the ore, which can reach high concentrations within the confines of a mine and which is known to cause lung cancer. The studies of radon-exposed miners indicate a linear relationship between radiation dose and risk of radon-induced lung cancer and demonstrate higher absolute risks for cigarette smokers than non-smokers. Recent estimates by the US National Academy of Sciences Committee on Health Risks of Exposure to Radon (1998) indicate that the lifetime risk of radon-induced lung cancer for an individual exposed to 100 mSv equivalent lung dose each year is around 1% for a lifelong non-smoker and around 10% for a cigarette smoker. Studies of lung cancer in uranium miners are therefore dominated by the effects of exposure to radon gas, and so the findings from such studies are not considered here.

Workers in the nuclear industry have, since the 1940s, been engaged in the extraction, milling and machining of uranium for the production of nuclear weapons and nuclear power. Exposure to radon gas is not a problem, although the physical and chemical form of the uranium handled varies considerably, depending on the process performed. Nevertheless, some nuclear workers have had sufficiently large intakes of uranium – whether by inhalation, ingestion, or some other way – that the study of their health should, in principle, shed some light on the long-term effects of such exposures on the incidence of cancer and other conditions.

We summarize here the results from published studies of the mortality of workers in the nuclear industry who may have been exposed to uranium while at work. Altogether about 120,000 workers have been followed for up to 50 years after first exposure, and about a quarter of them (33,000) have died. Information on the cause of death is available for these subjects, and emphasis is given here to mortality from cancer and from genito-urinary diseases (eg kidney disease), as these are the conditions considered to be most likely to be adversely affected by exposure to uranium. The limitations of these studies and the problems of interpreting the results are not inconsiderable, and these are discussed below.

It is uncertain how relevant nuclear workers' exposures to uranium are, when considering the effects of exposure to DU on the battlefield. DU is a byproduct of some of the processes which some nuclear industry workers would have performed, but in most cases workers would have been exposed to natural or enriched uranium as well, and the radiation doses, and therefore the radiological hazard to health, would be expected to be greater than that of soldiers who were exposed to DU, which has only about 60% of the radioactivity of natural uranium. However, the route of exposure and the chemical and physical properties of the aerosolised uranium in the work place are probably not the same as on the battlefield.

2 Studies included

The US National Academy of Sciences Institute of Medicine study on Health Effects Associated with Exposures During the Gulf War (2000) has reviewed in detail studies of people in the nuclear industry whose work would be likely to have involved exposure to uranium. These studies are included in our review but, in

addition, literature searches have been carried out to identify further studies, such as those published since the Institute of Medicine report. After elimination, as far as possible, of overlap between studies, 14 remained and the details of each are summarized in table 1. Three studies (McGeoghegan et al 2000a, McGeoghegan et al 2000b, Beral et al 1988) concerned workers in the UK and the remaining eleven concerned workers in the US. One study concerned workers at a phosphate production plant where the processing of phosphate ore may have resulted in human exposures to uranium (Stayner et al 1985); one study concerned workers at a plant that had previously participated in the Manhattan project, where uranium and other radionuclides were detectable in the soil and on surfaces inside and outside buildings (Teta and Ott 1988); and the remaining twelve studies involved workers in the nuclear industry. Six of the studies (Teta and Ott 1988, Cragle et al 1988, Brown and Bloom (1987), Stayner et al 1985, Waxweiler et al 1983, Hadjimichael et al 1983) had no information available on the extent of exposure to uranium received by individuals, three had only monitored external radiation doses (McGeoghegan and Binks 2000a, McGeoghegan and Binks 2000b, Dupree-Ellis et al 2000), two had external doses and also information on whether or not an individual had been monitored for internal exposure to uranium or other radionuclides (Frome et al 1997, Beral et al 1988), two had estimated internal lung doses from alpha-particles produced by uranium or other radionuclides incorporated into the body (Ritz et al 2000, Dupree et al 1987), and one had monitored external doses and estimated internal lung doses (Ritz 1999a).

3 Method of analysis

For each study, the total number of deaths observed in the entire study population, or in an appropriate subgroup, was extracted, as was the number of deaths that would have been expected if the study subjects had experienced the mortality rates of the general population from which they were drawn, taking into account the variation in mortality with age, sex and calendar period. Whenever data were available the numbers of observed (O) and expected (E) deaths were also extracted for all cancers, thirteen individual types of cancer, and genito-urinary diseases. For each cause of death the ratio of observed to expected deaths (O/E) was calculated, together with its 95% confidence interval (CI). O/E estimates the average ratio of the age-sex-calendar year specific death rates in the study population to those in the general population (Armitage and Berry 1987). Summary values of O/E from all studies combined were then calculated. A test for heterogeneity suggested that for all causes of death, all cancers, and many individual causes, O/E varied from study to study. This variation was taken into account in calculating the summary estimates (Whitehead and Whitehead 1991).

4 Results and discussion

As far as can be assessed, the 14 studies whose results are summarised here include the large majority of uranium-exposed workers in the nuclear industry of the USA or the UK over the last 50 years. Study-specific results and a summary estimate, combining the results from all 14 studies, are plotted for all causes of death (figure 1 of annexe I), for death from all cancers combined (figure 2), for death from 13 specific cancers (figures 3-15) and for death from all genito-urinary diseases (figure 16). For each cause of death the width of the confidence interval associated with the result for a particular study reflects the amount of information available from that study, so that studies with larger numbers of deaths have narrower confidence intervals than studies with smaller numbers of deaths. Summary estimates for all 16 causes of death are given in figure 17. The width of the confidence interval for each summary value reflects the amount of information available concerning that cause of death so that causes for which there are only a few deaths, such as thyroid cancer, have relatively wide confidence intervals. For every condition the summary estimate suggests that the death rate in workers exposed to uranium was similar to, or slightly lower than, that in the general populations from which they had been drawn. For some conditions the deficit reached statistical significance ($p<0.05$). Thus, the large body of available data on mortality in nuclear industry workers who may have been exposed to uranium suggests that such workers do not have higher death rates from any particular cancer, genito-urinary disease, or all causes of death combined, than those found in the general population.

For the studies where external monitored radiation dose, internal monitoring status or internal radiation dose to the lung was available most of the authors carried out subgroup and/or dose-response analyses, usually for at least 10 different types of cancer, and a number of significant positive findings were reported. For example a significant positive dose-response relationship was found between external monitored radiation dose (lagged by 10 years) and mortality from all cancers (Frome 1997, Ritz 1999a), for Hodgkin's disease and bladder cancer (McGeoghegan and Binks 2000a), kidney cancer (Dupree-Ellis 2000), and lung cancer (Frome 1997, Ritz 1999a). A significant increase of prostate cancer was found among workers monitored for exposure to uranium (Beral et al 1988), and significant dose-response relationships were found between internal lung dose and upper aerodigestive tract cancers and haematopoietic and lymphatic cancers (Ritz 2000). However, no study reported a significant dose-response relationship between internal lung dose and mortality from lung cancer.

The interpretation of the findings shown in figures 1-17 of annex I for nuclear industry workers is not, however,

straightforward. A relatively minor problem is the likely residual overlap between individuals in the 14 epidemiological studies, particularly in studies from the USA. We have tried not to include studies with known duplication, but it is difficult to know how much double counting is still going on.

Perhaps the most serious difficulty in interpreting the results shown in figures 1-17 is the so-called 'healthy worker effect'. The nuclear industry tends to employ comparatively healthy people, often drawn from the upper socio-economic groups. Because such people would, regardless of their employment, be expected to have lower mortality rates than the average, the calculated expected numbers of deaths, based on mortality rates in the general population, are likely to be overestimates. This is a well-recognised problem, but it is equally well recognised that it is impossible to know what the true expected death rate would be for any particular cause in such selected populations.

Another problem is that in studies such as these, which often require the retrieving of old records kept for other purposes, it is sometimes difficult to know exactly which individuals were exposed to uranium and what their likely exposure was. In general the largest exposures to uranium occurred during the 1940s and 1950s. At that time safety requirements were less stringent than now, record-keeping was limited, and very few individuals were tested for possible exposure to uranium or other radionuclides (individuals were generally tested for contamination only if there was an 'incident' such as a fire, large spillage, etc). Therefore, there are few reliable measurements of exposure to uranium at the time when it was relatively high. Studies restricted to workers with measured internal exposure to uranium are thus often studies of recent workers, who had relatively low exposures. Also, a relatively small proportion of a total workforce would have been heavily exposed to uranium. Many people who work in uranium processing plants or similar places do not actually handle uranium themselves - for example security officers, builders, administrators, clerical workers and cooks would have minimal exposure. Thus the mortality experience of workers who actually handled uranium may be diluted by the experience of people with little or no direct exposure.

Moreover, workers who handle uranium tend also to handle other radioactive material and chemicals, so multiple exposures to potentially hazardous substances are common in the small subgroup of nuclear industry workers with extensive exposure to uranium.

Finally, there are other known causes of cancer that need to be considered. Of particular relevance is cigarette smoking in lung cancer. The vast majority of lung cancers is caused by cigarette smoking and the lifetime risk of lung cancer varies from 0.4% for lifelong non-smokers to around 16% for cigarette smokers (Peto et al 2000). Therefore studies of lung cancer in nuclear industry workers are difficult to interpret unless information on smoking in all study subjects is also available and is taken into account in the analysis. Unfortunately such information is not available for the existing studies.

5 Summary and conclusions

Although, as yet, virtually no data are available on the long-term effects of exposure to DU in humans, extensive information is available on the mortality of workers involved in processing uranium in the nuclear industry. Some of these workers have been followed for 50 years since they were exposed. Overall the findings on 120,000 workers in uranium plants, among whom 33,000 deaths have been reported, do not suggest an excess mortality from either cancer, genito-urinary disease, or all causes combined. For the reasons discussed above it would not, however, be justified to conclude on the basis of the available data that there are definitely no long-term hazards associated with occupational exposure to uranium. What can be said is that if there were a relatively small excess of any particular condition, it would probably be undetectable, given the limitations of the existing data. However, comparatively large excesses, for example a true doubling in the risk of any of the relatively common conditions considered here, would probably be evident in the data examined, despite the methodological problems outlined above.

It is unclear how relevant the exposures of uranium nuclear workers are for DU-exposed soldiers, although it is noteworthy that some nuclear industry workers would also have been exposed to DU, albeit in a different form. There are only limited data on the levels of exposure to uranium of nuclear workers (particularly by inhalation, the pathway of most relevance to this report), and it is difficult to know how these relate to exposures to DU on the battlefield. The problems in interpreting the results of studies of uranium workers suggest that it will not be easy to establish reliably what the long-term effects of human exposure to DU are, since some of the methodological problems encountered for uranium workers are also likely to affect epidemiological studies of DU-exposed populations.

Table 1 Epidemiological studies of workers exposed to uranium

Plant, location and reference	Description of population studied	Exposure information	Total no. of workers in results presented	Total no. of deaths	Earlier references covering a similar or overlapping population
Springfields, Lancashire, UK (McGeoghegan and Binks 2000a)	Radiation workers at plant fabricating uranium fuel and uranium hexafluoride	External monitored radiation dose	13,960	3,476	—
Mallinckrodt, Missouri, USA (Dupree-Ellis et al 2000)	Workers at uranium processing plant	External monitored radiation dose	2,514	1,013	Dupree et al 1995
Rocketdyne/ Atomics International, California, USA (Ritz 2000)	Workers involved in nuclear fuel assembly and disassembly, monitored for internal exposure to uranium or mixed fissile products	International radiation dose to the lung	2,297	433	—
Capenhurst, Cheshire, UK (McGeoghegan and Binks 2000b)	Radiation workers at uranium enrichment plant	External monitored radiation dose	3,244	585	—
Fernald Feed, Ohio, USA (Ritz 1999a)	Uranium processing workers	External monitored radiation dose and internal radiation dose to the lung	4,014	1,064	Dupree et al 1995, Ritz 1999b
Four Federal nuclear plants, Tennessee, USA (Frome et al 1997)	Workers involved in uranium enrichment or nuclear materials fabrication	External monitored radiation dose and internal monitoring status	67,600*	22,724	Dupree et al 1995, Loomis et al 1996, Wing et al 1991, Frome et al 1990, Checkoway et al 1988, Checkoway et al 1985, Polednak et al 1981 Cookfair et al 1983
Linde, New York, USA (Teta and Ott 1988)	Workers at a site previously contaminated with uranium and other radionuclides	—	8,146	1,160	—
Savannah River, South Carolina, USA (Cragle et al 1988)	Workers at a nuclear fuel production plant	—	9,860	1,091	—
Atomic Weapons Establishment, Berkshire, UK (Beral et al 1988)	Workers at a weapons production plant who were monitored for exposure to uranium	External radiation dose and uranium monitoring status	3,044	137	—
Linde, New York, USA (Dupree et al 1987)	Workers at a uranium processing facility	Estimated internal lung dose	995	429	—
Portsmouth, Ohio, USA (Brown and Bloom 1987)	Workers at a uranium enrichment facility	—	5,244	483	—
Polk, Florida, USA (Stayner et al 1985)	Workers at a phosphate fertilizer production facility	—	3,160	155	—
Seven mills Rocky Mountain region, USA (Waxweiler et al 1983)	Uranium mill workers	—	2,002	533	Archer et al 1973, Wagoner et al 1964
United Nuclear, Connecticut, USA (Hadjimichael et al 1983)	Workers at a nuclear fuels fabrication plant	—	3,512	219	—

* Approximate

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