ICBUW's commentary on the Scientific Committee on Health and Environmental Risks (SCHER) Opinion on the environmental and health risks posed by depleted uranium (DU)

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Executive summary

Introduction: p4

The origins of the *Opinion on the Environmental and Health Risks Posed by Depleted Uranium* produced by the Scientific Committee on Health and Environmental Risks (hereafter the 'SCHER report') lie in European Parliament resolution P6 TA(2008)0233, adopted in May 2008. The resolution requested the European Commission to "*commission scientific studies into the use of depleted uranium in all regions where European and international military and civilian personnel have been deployed*."

Following this, the Commission was asked to investigate the potential health impact of DU weapons. The Commission mandated SCHER to produce an opinion on the issue. The broad rationale behind this was that, although several desk studies have been undertaken on this issue, a large body of peer-reviewed research had been subsequently produced, particularly on the issue of genotoxicity, which was not considered in those desk studies.

Following publication of SCHER's draft opinion it was noted that it did not include reference to a series of studies indicating that DU was genotoxic. Although the final SCHER report does make reference to these studies, it seeks to diminish their relevance. Similarly, while it was pointed out that SCHER's draft opinion did not deal in detail with recent studies on toxicity, this was not resolved in the final draft.

This commentary seeks to outline some of the flaws in the final SCHER report and explain to the reader why ICBUW does not believe that the SCHER report is a sound basis for informing EC policy on DU. It is hoped that this overview will enable the reader to appreciate the SCHER report in context and to form their own opinion on the potential risks posed by the use of DU. The commentary analyses SCHER's handling of the six areas of knowledge outlined in bold below.

SCHER's treatment of previous studies on DU: p5

SCHER's treatment of previous studies into this issue is misleading, because the relationship between these studies and their shared methodologies is not made clear. There is no discussion of any limitations or uncertainties in the approach of these studies. Most of these reports deal exclusively with radiological risk. Those that address the question of chemical toxicity are limited to only a few specific health outcomes. None of them assess the risks from the genotoxic effects of DU. SCHER also lists some studies that are only brief summaries or are derivative of other studies in the list.

Risk assessments using ICRP models and radiation protection standards: p7

Radiation risk assessments, including those cited by SCHER, use dose limits from standards that were devised for civilian nuclear programmes. These standards are not applicable to the use of DU in conflict because they depend upon institutions and safeguards that are not available during wartime. These standards are also conditional upon a society deciding that the risks from exposure to a radiation source are outweighed by the potential benefits. This can never be the case with the military use of DU, as the civilians who bear the risk and the military who gain the apparent benefit are separate and distinct parties.

Although the SCHER report suggests that there is some doubt about the cancer risk from low radiation doses, such a position is contrary to accepted scientific opinion, including that of the ICRP. It is widely accepted that low level radiation doses are carcinogenic, and this is reflected in international and European radiation safety standards. There can be little doubt that any exposure to DU carries with it a risk of developing cancer.

There are several different interconnected ICRP models for different parts of the body. While the models are based on available scientific data, there are still significant uncertainties. The characteristics of inhaled radioactive particles – their size, shape, structure and solubility – can all significantly influence the eventual dose. While some of these uncertainties are recognised by the ICRP, WHO and others, they are not discussed in the SCHER report, despite most of the expert reports cited by SCHER being based on the ICRP models. The UK government's CERRIE committee found that uncertainty in calculating radiological risk from internal emitters may be as high as two orders of magnitude.

Contamination and exposure data: p11

Data characterising DU contamination in the field is fragmentary and is skewed towards certain types of weapon. Furthermore, most field data is based on single visits to contaminated sites, often years after the use of the weapons, so little is known about changes in contamination over time. Although some US test data allows for improvements to ICRP risk modelling, it also suggests that the characteristics of contamination may vary considerably from strike to strike, further complicating risk assessment. SCHER has omitted important data on the contamination of water in Bosnia from their report.

Exposure studies on soldiers and civilians: p13

While few studies have shown elevated uranium levels in the urine of those studied, the literature is heavily skewed towards veterans, with almost no civilians having been studied in affected areas. Furthermore, most of the studies have not identified the subjects most at risk from exposure, so questions remain about their relevance. The presentation of this material by SCHER is again misleading, particularly with regard to the number of civilians who have been studied.

Extrapolating toxicological effects of DU from animal and laboratory data: p14

Although animal and laboratory evidence suggests that there are other health outcomes that may be linked to DU exposure, the SCHER report only attempts to assess the toxicological risk of kidney damage. Other health effects may occur below the levels of exposure that would cause kidney damage. Instead of acknowledging this uncertainty, the SCHER report assumes that kidney damage is the only health outcome of interest. Instead of interpreting a number of recent papers on the different toxicological or genotoxic effects of DU, the SCHER report seeks to dismiss them as irrelevant.

Epidemiology: p17

Because epidemiological data on civilian populations exposed to DU is missing from the literature, SCHER attempts to apply the results of studies on uranium miners and millers to the health risks of military DU exposure. The health effects found in uranium miners are widely thought to be linked to exposure to radon gas. However the strong link with radon exposure does not preclude potential harm from uranium exposure.

Studies on workers in uranium mills, where radon exposure is less of a problem, have not identified any statistically significant link between uranium exposure and health problems, although some show an elevated risk of some problems below the level of statistical significance. Unfortunately these studies lack any meaningful exposure data, so they should not be relied upon for information about the risks of military-origin DU. There is also reason to believe that the characteristics of particulate contamination (such as size and solubility) in uranium mills may be less hazardous.

Medical studies on US veterans exposed to DU during warfare: p18

Medical studies conducted under the auspices of the US Department of Veterans Affairs are of limited use for assessing the potential health effects of DU exposure. The lack of a single cohort, or of a control group, as well as the small number of subjects make meaningful interpretation of the results impossible. Furthermore, some matters of interest have not been properly followed up, or have even been completely omitted from reports, including incidence of tumours within the group.

Recommended risk reduction measures have not and will not be put in place: p19

While there is a significant body of literature dealing with the potential health effects of DU exposure, the picture presented by the SCHER report is misleading. While it is clear that exposure carries with it some level of risk, it is difficult to quantify it with any level of certainty. The shortcomings in the different areas identified in this commentary are such that the evidence cannot reliably be used to reinforce the assertion that the risk from exposure is negligible, as the SCHER report appears to suggest. A better approach would be to acknowledge the uncertainties and to exercise precaution, rather than assume that harm is unlikely.

Although SCHER does acknowledge that there is some potential for harm, the measures suggested to reduce it by international agencies such as UNEP, WHO, Royal Society and IAEA have generally not been followed through in states where DU weapons have been used. Furthermore, the nature of post conflict conditions ensures that they are unlikely to be in the future. As such, a sensible approach to this issue is that recommended by the European Parliament: the use of these weapons should be subject to an international moratorium and steps should be taken towards an international treaty ban. This is the only approach that is in line with radiation protection norms and a common-sense approach to environmental contamination.

Introduction

The origins of the '*Opinion on the Environmental and Health Risks Posed by Depleted Uranium*' produced by the Scientific Committee on Health and Environmental Risks (hereafter the 'SCHER report') lie in European Parliament resolutionP6 TA(2008)0233, adopted in May 2008 with the support of 94% of MEPs. The resolution called for a global moratorium on the use of depleted uranium (DU) weapons and for international negotiations towards a global ban. The resolution also requested the European Commission to "*commission scientific studies into the use of depleted uranium in all regions where European and international military and civilian personnel have been deployed.*"[1]

Following this, one of the co-sponsors of the resolution, Els De Groen MEP, wrote to the European Commission requesting that they investigate the potential health impact of DU weapons. The Commission subsequently mandated SCHER to produce an opinion on the issue, with its terms of reference based upon questions set out in Els De Groen's letter. The broad rationale behind this was that, although several desk studies have been undertaken on this issue,[2] a large body of peer-reviewed research had been subsequently produced, particularly on the issue of genotoxicity, (i.e. a substance's ability to damage the genetic materials within cells – often associated with carcinogenicity) which was not considered in those desk studies.

Following the publication of SCHER's draft opinion, a number of individuals and organisations responded, including ICBUW. Amongst the comments in ICBUW's submission, it was noted that the draft opinion did not include reference to a series of studies by the US Armed Forces Radiobiology Research Institute indicating that DU was genotoxic, and therefore could potentially cause cancer. Although the final SCHER report does make reference to these studies, it seeks to diminish their relevance.[3] Similarly, while it was pointed out that SCHER's draft opinion did not deal in detail with recent studies on toxicity, this was not resolved in the final draft.[4]

This commentary aims to outline some of the flaws in the final SCHER report and explain to the reader why ICBUW does not believe that the SCHER report is a sound basis for informing EC policy on DU. It is not a complete overview of the criticisms that could be made, nor should it be seen as a full discussion of the risks of military-origin DU and related issues. For reasons of brevity, studies of DU contamination in the environment are not dealt with in detail, and human exposure through inhalation is the primary focus.[5] It is hoped that this overview will enable the reader to appreciate the SCHER report in context and to form their own opinion on the potential risks posed by the use of DU.

DU is used in armour piercing tank shells and bullets because of its extreme density. It is both radioactive and chemically toxic, and both these factors must be taken into account when considering the risks from its use. Much of the literature on this issue takes the form of risk assessments that only address one effect, or deal with the two effects separately. Although as SCHER notes, all real-world studies on uranium describe the combined effects, the potential for a combined synergistic effect is not taken into account by risk assessments that deal solely with either radiological risk or chemical toxicity.

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[1] See http://www.bandepleteduranium.org/en/a/181.html or http://www.europarl.europa.eu/sides/getDoc.do?pubRef=-//EP//TEXT+TA+P6-TA-2008-0233+0+DOC+XML+V0//EN

[2] E.g. WHO, 2001. *Depleted uranium: Sources, Exposure and Health Effects*, Geneva: Department of Protection of the Human Environment World Health Organization. Available at: http://whqlibdoc.who.int/hq/2001/WHO_SDE_PHE_01.1.pdf

[3] SCHER, 2010. *Opinion on the Environmental and Health Risks Posed by Depleted Uranium*, p9

[4] Response of Dr Rosalie Bertel to SCHER draft opinion, kindly provided to ICBUW. This part of the SCHER report is addressed in the section on toxicity below.

[5] The reason for this is that modelling of the effects of DU exposure on humans suggests that inhalation is the pathway of most concern, in terms of possible health consequences for civilians.

Attempts to assess the risks to humans from exposure to military-origin DU, typically draw on six different bodies of knowledge. These are:

- The suite of models of the human body devised by the International Commission for Radiological Protection (ICRP) to assess radiation risks;
- Data on environmental contamination caused by the use of DU weapons, which could help with calculating exposure and possible risk;
- Assessing risk by extrapolating data from animal and laboratory experiments;
- Epidemiological studies of people exposed to uranium from other sources;
- Studies that examine uranium excretion levels in urine to identify those who may have been exposed to DU;
- Medical studies on persons known to have been exposed to DU during warfare.

The SCHER report deals with all six of these areas, although in less detail than some previous desk studies. This commentary will first discuss SCHER's treatment of these previous studies and then give a critical appraisal of SCHER's treatment of each area.

SCHER's treatment of previous studies on DU

SCHER's treatment of previous studies into this issue is misleading, because the relationship between these studies and their shared methodologies is not made clear. There is no discussion of any limitations or uncertainties in the approach of these studies. Most of these reports deal exclusively with radiological risk. Those that address the question of chemical toxicity are limited to only a few specific health outcomes. None of them assess the risks from the genotoxic effects of DU. SCHER also lists some studies that are only brief summaries or are derivative of other studies in the list.

On several occasions, the SCHER report refers to a list of expert groups: the International Atomic Energy Agency (IAEA), World Health Organisation (WHO), United Nations Environment Programme (UNEP) and the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), which have published on the DU issue.[6] The way these lists are presented by SCHER is unfortunate, as it could mislead the reader into thinking that each assessment was a separate health risk assessment based on independent primary research. Instead, most of the reports are desk studies using identical models and performing calculations based on the same body of literature. It is therefore no surprise that they reached similar conclusions. Other items cited by SCHER do not contain risk assessments of DU at all.

Of the 13 studies cited on page 17 to support SCHER's contention that "*health risks due to the chemical and radiological toxicity of DU are not expected*",[7] three are environmental audits of contaminated sites that

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[6] See SCHER p8: *"Any DU-derived radiation will remain below an effective radiation dose < 1 mSv and thus well below accepted dose-rate limits derived for radiation protection. This conclusion was confirmed by other international expert groups…and SCHER agrees with this conclusion,"* p13: *"A detailed assessment of such potential exposures has been performed in Kosovo …Serbia-Montenegro …Bosnia and Herzegovina…, Kuwait … and Iraq,"* and p17*"The conclusion is supported by all expert panels that were tasked with risk assessment for DU uses regarding the general population"*

[7] *Ibid.*

only briefly deal with health risks in short appendices (UNEP 2001, 2002, 2003),[8] and four deal solely with the issue of radiological health risks (UNEP/UNHCS, EURATOM 2001, IAEA 2003, 2009).[9]

Of the remaining six, three are discussions on radiological risk and do not deal specifically with DU at all (UNSCEAR 1993, UNSCEAR 2000a, UNSCEAR 2000b);[10] one describes a programme to build technical capacity for field monitoring in Iraq and only discusses potential health risks in passing (UNEP 2007);[11] and another only deals with DU in a section of just over 200 words that discusses an ICBUW publication and concludes that an earlier opinion does not need to be revised (EU-EURATOM 2009).[12]

Of the list, only the WHO (2001) study deals with the question of non-radiological risks. However, its consideration of chemically-induced health risks is based only on damage to the kidney. All of the studies that contain radiological risk assessments base their risk assessment upon the same dose calculation models: those developed by the ICRP and incorporated into the IAEA's International Basic Safety Standards. The table in Appendix A below includes all of the studies cited more than once in the lists provided by SCHER and shows clearly the degree to which all these risk assessments rely on the ICRP models. These models are discussed below.

The other papers referenced on page 8 on SCHER also fit this pattern. The WHO 2003 paper,[13] is a brief bullet-point summary rather than a detailed risk assessment. Durante and Pugliese,[14] use ICRP dose coefficients[15] that are derived from the same set of radiation dose models,[16] and Li et al also use the ICRP models, although with some modifications.[17]

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[8] United Nations Environment Programme, 2001. *Depleted Uranium in Kosovo: Post-Conflict Environmental Assessment*; United Nations Environment Programme, 2002. *Depleted Uranium in Serbia and Montenegro Post-Conflict Environmental Assessment in the Federal Republic of Yugoslavia*; United Nations Environment Programme, 2003. *Depleted Uranium in Bosnia and Herzegovina: Post-Conflict Environmental Assessment 2003* 3rd ed.;

[9] UNEP/UNCHS Balkans Task Force (BTF), 1999. *The potential effects on human health and the environment arising from possible use of depleted uranium during the 1999 Kosovo conflict: A preliminary assessment*; EURATOM, 2001. *The Opinion of the Group of Experts Established According to Article 31 of the Euratom Treaty - Depleted Uranium*, Luxembourg; IAEA, 2003. *Radiological conditions in areas of Kuwait with residues of depleted uranium : report by an international group of experts*, Vienna: International Atomic Energy Agency; IAEA, 2010. *Radiological Conditions In Selected Areas Of Southern Iraq With Residues of Depleted Uranium: Report by an International Group of Experts*, International Atomic Energy Agency , Vienna.

[10] UNSCEAR, 1993. *Sources and effects of ionizing radiation*: UNSCEAR 1993 report to General Assembly with scientific annexes New York; UNSCEAR, 2000a. *Sources and effects of ionizing radiation*, vol. II Effects. UNSCEAR Report to the General Assembly, with Scientific Annexes., United Nations Scientific Committee on the Effects of Atomic Radiation; UNSCEAR, 2000. *Sources and effects of ionizing radiation*, vol. I Sources. UNSCEAR Report to the General Assembly, with Scientific Annexes., United Nations Scientific Committee on the Effects of Atomic Radiation.

[11] UNEP, 2007. *Technical Report on Capacity-building for the Assessment of Depleted Uranium in Iraq*, Geneva: United Nations Environment Programme. The results from this study are analysed in the IAEA 2009 publication.

[12] EURATOM, 2009. Meeting of the Group of Experts (GoE)referred to in Article 31 of the Euratom Treaty: *Summary Report*, Luxembourg. Available at: http://ec.europa.eu/energy/nuclear/radiation_protection/doc/art31/2009_06_report.pdf. The earlier opinion in question is presumably EURATOM (2001) *op. cit.*

[13] Anon, 2003. WHO Depleted uranium Fact sheet N°257. Available at: http://www.who.int/mediacentre/factsheets/fs257/en/ [Accessed September 16, 2011].

[14] Durante, M. & Pugliese, M., 2002. *Estimates of radiological risk from depleted uranium weapons in war scenarios.* Health Physics, 82(1), pp.14-20.

[15] A dose coefficient is a figure for the radiation dose that one would expect an individual to receive upon exposure to a certain quantity of a radioactive particle through a certain route (e.g. from ingesting uranium).

[16] This paper also contains the claim that DU is found in Tomahawk missiles, now thought to be erroneous see, http://www.bandepleteduranium.org/en/a/404.html

[17] Li, W.B. et al., 2008. *Radiation dose assessment of exposure to depleted uranium*. Journal of Exposure Science and Environmental Epidemiology, 19(5), pp.502-514.

As can be seen from the above, and from appendix A below, most of the studies cited by SCHER on pages 8 and 17 do not contain extensive risk assessments, and those that do are skewed towards consideration of radiological risk using the ICRP models. That this is not made clear to the reader is unhelpful, and the impression given by the SCHER report that each study is an independent expert risk assessment is therefore misleading.

Risk assessments using ICRP models and radiation protection standards

Radiation risk assessments, including those cited by SCHER, use dose limits from standards that were devised for civilian nuclear programmes. These standards are not applicable to the use of DU in conflict because they depend upon institutions and safeguards that are not available during wartime. These standards are also conditional upon a society deciding that the risks from exposure to a radiation source are outweighed by the potential benefits. This can never be the case with the military use of DU, as the civilians who bear the risk and the military who gain the apparent benefit are separate and distinct parties.

Although the SCHER report suggests that there is some doubt about the cancer risk from low radiation doses, such a position is contrary to accepted scientific opinion, including that of the ICRP. It is widely accepted that low level radiation doses are carcinogenic, and this is reflected in international and European radiation safety standards. There can be little doubt that any exposure to DU carries with it a risk of developing cancer.

There are several different interconnected ICRP models for different parts of the body. While the models are based on available scientific data, there are still significant uncertainties. The characteristics of inhaled radioactive particles – their size, shape, structure and solubility – can all significantly influence the eventual dose. While some of these uncertainties are recognised by the ICRP, WHO and others, they are not discussed in the SCHER report, despite most of the expert reports cited by SCHER being based on the ICRP models. The UK government's CERRIE committee found that uncertainty in calculating radiological risk from internal emitters may be as high as two orders of magnitude.

Applicability of the ICRP model and the Basic Safety Standards

The models developed by the ICRP are widely used. They allow calculations to be made about the dispersal of radioactive substances around the body. The ICRP has been developing this family of models for the last 50 years,[18] and the different elements of the model are updated periodically when a sufficient body of new evidence emerges.

While the ICRP is an independent scientific group, these models and other recommendations by the ICRP form the backbone of the *International Basic Safety Standards for Protection against Ionizing Radiation and for the Safety of Radiation Sources*, or Basic Safety Standards (BSS).[19] The BSS are jointly produced by a

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[18] Miller, G. et al., 2009. *Methods used to calculate doses resulting from inhalation of Capstone depleted uranium aerosols*. Health Physics, 96(3), pp.306-327.

[19] BSS, 1996. *International basic safety standards for protection against ionizing radiation and for the safety of radiation sources*., Vienna: IAEA.

number of UN agencies and international institutions, including the IAEA.[20] They form the basis for radiation standards worldwide, including radiation protection regulations at the European level.[21]

The radiological risk assessments mentioned above, estimate the radiation dose that might be expected from exposure to a certain quantity of DU using the ICRP models. This is then typically compared to the BSS recommendations for an individual dose over a given time period. This is also the approach taken by SCHER, who state that: *"[a]ny DU-derived radiation will remain below an effective radiation dose < 1 mSv and thus well below accepted dose-rate limits for radiological protection*",[22]referring to the BSS 1mSv radiation dose limit for members of the public from all man-made radiation sources.[23]

However, it is not at all clear that the BSS is an appropriate comparison to make in the context of an uncontrolled release of DU during conflict. One of the key principles of the BSS is that a condition of any radiation risk being accepted by society is the benefits that society will accrue from the use of that radiation.[24] This is coupled with the principle that no radioactive source or practice should be allowed unless there is sufficient benefit to the society, or to an individual, to outweigh the possible risk.[25] In the case of DU in weaponry, this central condition cannot be satisfied. The risk from the use of the weapons is typically transferred onto the opposing side and on to the civilian population, who have no say in the matter. Meanwhile any perceived benefit remains solely with those who employ the weapons.

The BSS were developed for use in a civilian nuclear context. This is far removed from the military use of DU. The standards are based on a presumption that a national radiation protection infrastructure is in place. This would typically be comprised of legislation, regulatory bodies, resources and trained personnel to inspect and manage radiation risks to the population.[26] The use of DU in the context of a war where, by definition, the writ of the state does not apply within the contested territory, cannot satisfy this requirement. In practice, states recovering from conflict may wait many years before these structures are in place.[27] During this time radioactive contamination may not be managed at all.

It therefore follows that standards that depend upon such structures being in place cannot be applied. There is a strong case to be made that the military use of DU is clear breach of the BSS. Thus the guidance levels set out in the BSS should not be considered an "*acceptable*" upper limit for radiation doses attributable to DU. As the safeguards to help ameliorate risk are not in place, the logical course of action is not to introduce excess risk by introducing radioactive contamination into the environment.

The Linear No Threshold model

Radiological contamination entails excess risk at any level. This is the considered opinion of most experts in the field. Specialists believe that any radiation dose, however small, brings with it an increased risk of

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[20] These are the: Food and Agriculture Organization of the United Nations (FAO), the International Atomic Energy Agency (IAEA), the International Labour Organisation (ILO), the Nuclear Energy Agency of the Organisation for Economic Co-operation and Development (OECD/NEA), the Pan American Health Organization (PAHO) and the World Health Organization (WHO).

[21] See Mundigl, S., 2010. *Revision of the Euratom Basic Safety Standards Directive- current status*. Radiation Protection Dosimetry, 144(1-4), pp.12-16.

[22] SCHER 2010 *op. cit.* p8

[23] BSS 1995 *op. cit.* p93

[24] *"The acceptance by society of risks associated with radiation is conditional on the benefits to be gained from the use made of radiation...Purely scientific considerations, however, are only part of the basis for decisions on protection and safety, and the Standards implicitly encourage decision makers to make value judgements about the relative importance of risks of different kinds and about the balancing of risks and benefits*." Ibid. p1-2

[25] *Ibid.* p4-5

[26] *Ibid.* p7

[27] For example, there was no radiation protection legislation enacted in Republika Srpska, the Serbian entity in Bosnia and Herzegovina, from the end of hostilities in 1995 and 2007. See ICBUW, 2010. *A Question of Responsibility: Depleted Uranium Weapons in the Balkans*, Manchester, UK: International Coalition to Ban Uranium Weapons.

cancer. The risk of cancer increases proportionately with increasing dose. This relationship is known as the Linear No Threshold (LNT) model, meaning that the relationship between dose and risk is linear, and that there is no threshold below which this relationship does not hold.

SCHER seems to be out of step with established scientific thinking on this issue, as the report seeks to cast doubt on the LNT model. However the model is so widely accepted that SCHER concedes the LNT is the "*accepted standard*".[28] However, the impression given in the SCHER report that the LNT has recently fallen out of favour is not correct. While the model has been re-examined during recent years by the ICRP and others, the conclusion has been that "*a linear relationship is consistent with most of the available mechanistic and quantitative data*", and that the evidence suggests that there is no threshold.[29]

Furthermore, the WHO's International Agency for Research on Cancer (IARC) has recently classified all radioactive particles that emit alpha radiation within the body as "*carcinogenic to humans*".[30] As such, it is clear that any amount of DU within the body carries with it a risk of developing cancer. The LNT model is an integral part of the BSS and accepted by radiation protection authorities around the world, including all the UN agencies that produce the BSS, and radiation protection authorities at member state,[31] and European[32] level.

The ICRP models

The models developed by the ICRP – the Human Respiratory Tract Model,[33] the Systemic Model[34] and the Gastro-Intestinal Tract Model,[35] represent the human body as a series of interconnected compartments and provide methods for calculating how radionuclides pass through those regions of the body. Their purpose is to provide a basis for assessing the radiation dose to different body parts. Although complex, they are intentionally simplified models, designed for the purpose of creating a framework that can be replicated and used for dose assessment.[36] The models are discussed in some detail here, in part because studies based on them feature so prominently in the SCHER report, but also because some of the significant factors involved in assessing risk using the models are relevant to evaluating other types of evidence that pertain to the risks of DU exposure. A diagram of the three models is included in Appendix C.

The models are derived from data from animal experiments, knowledge of human physiology and some theoretical models of certain biological factors. They are seen by states and regulators as the best attempt by a group of experts to combine a range of data sources into a working model, which can be used to assess dose from a number of easily measurable criteria. At their most basic, these models produce a simple numerical dose coefficient, where a predicted radioactive dose can be calculated from the mass of a given radionuclide that an individual is exposed to.

The cumulative effect of the simplifications and assumptions used when constructing the models is that there are significant uncertainties when using them to calculate radiation dose. By way of an example, an

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[28] SCHER 2010 *op. cit.* p8

[29] UNSCEAR 2000a *op. cit.* p10-11, see also International Commission on Radiological Protection., 2007. *The 2007 Recommendations of the International Commission on Radiological Protection*. Annals of the ICRP, 37(2-4), pp.1-332.

[30] El Ghissassi, F. et al., 2009. *A review of human carcinogens–part D: radiation*. The Lancet Oncology, 10(8), pp.751–752

[31] See CDS1 RPD, *"Simplification and review of Radiation Protection Directives*," Directive, September 21st, 2007, http://www.hse.gov.uk/aboutus/europe/euronews/dossiers/radiationprotect.htm.

[32] See Mundigl, 2010 *op. cit.*

[33] ICRP, 1994. *Human Respiratory Tract Model for Radiological Protection*. ICRP Publication 66. Ann. ICRP 24 (1-3).

[34] See ICRP, 1995. *Age-dependent Doses to Members of the Public from Intake of Radionuclides - Part 3 Ingestion Dose Coefficients*. ICRP Publication 69. Ann. ICRP 25 (1)

[35] ICRP, 1979. *Dosimetric model for the gastrointestinal tract*. Annals of the ICRP, 2(3-4), pp.30-34. Although this model is the basis for the majority of risk assessments mentioned in this commentary, it has since been superseded by the Human Alimentary Tract Model (HATM). See: ICRP, 2006. *Human Alimentary Tract Model for Radiological Protection*. Annals of the ICRP, 36(1-2), p.i-336.

[36] ICRP 1994. *op. cit.* p6

overview of some of the uncertainties in the Human Respiratory Tract Model (HRTM) can be found in Appendix B. Of particular interest in terms of estimating the risk from DU exposure are the characteristics of the inhaled particles, some of which can significantly alter the calculated dose. This includes the average aerodynamic size of the particles, the range of aerodynamic sizes and how soluble the particles are.[37] It should also be noted that most radiation risk data is extrapolated from data from atomic bomb survivors, who were subjected to single large external radiation doses. There are also uncertainties around whether the effects from chronic low internal doses, such as would result from DU in the body, are directly equivalent. The uncertainty in assessing the risk from internal radiological emitters may be as high as two orders of magnitude.[38]

Dealing with uncertainties in the ICRP models

The purpose of the above is not to denigrate the ICRP models, but to give the reader a proper understanding of their limitations and place them within a proper scientific context. These limitations are openly acknowledged by the ICRP, who dedicate part of the publication describing the HRTM and discussing how an uncertainty analysis could be carried out. They suggest that it would give a probability distribution of radiation doses that could result from an exposure - where all the parameters used by the model were known.

A study of this type has been carried out by the UK Health Protection Authority. The purpose of the study was to investigate the uncertainty of estimating dose from excretion of uranium in the urine. This is not immediately applicable to most of the risk estimation work done on DU, where the calculation in question is the radiological dose from inhaling or ingesting a certain quantity of DU. However, the range of the uncertainty is instructive.

For measurements taken 10 days after inhalation, the highest dose estimates were 50 times as large as the lowest estimated dose. Later on, for measurements between 1,000 and 10,000 days after inhalation, the highest dose was between seven and 10 times the lowest estimated dose. In the sensitivity analysis carried out as part of the study, numerous single variables were identified where the uncertainties for that variable alone could alter the dose by 20%.[39] The number of different variables in the ICRP models increases the uncertainty. It does not appear that SCHER is familiar with this study, and if they are aware of the uncertainties in the ICRP models, they do not share this information with the reader.

The only statement made by SCHER on the certainty of models used in risk assessments is that monitoring of uranium levels in urine can be used to confirm the conclusions of exposure assessments. The study cited to substantiate this statement also relies on ICRP models for its calculations.[40] While the study does discuss using urine analysis to assess likely exposure levels, it is a conceptual error to suppose that one can use calculations of this nature to validate the model itself. To do so would require a cohort where all exposure parameters were known and where levels of DU in urine could be regularly monitored. To the best of ICBUW's knowledge, no such cohort exists anywhere in the world.

The ICRP are quite clear about the need to substitute, wherever possible, real world data in place of the standard values given in the models. For some variables, such as those governing the aerodynamic behaviour of particles in the respiratory system, this is not practically possible. However the WHO,[41] and Li

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[37] To be specific these variables are the Aerodynamic Median Activity Diameter (AMAD), Aerosol Geometric Standard Deviation (GSD) and the Type S dissolution rate, see Bailey, M. & Health Protection Agency (Great Britain);Health Protection Agency (Great Britain)., 2007. *Uncertainty analysis of the ICRP human respiratory tract model applied to interpretation of bioassay data for depleted uranium*, Didcot: Health Protection Agency. 48-49

[38] See CERRIE, 2004. *Report of the Committee Examining Radiation Risks of Internal Emitters* (CERRIE), London, Great Britain: Committee Examining Radiation Risks of Internal Emitters.

[39] Bailey et al 2007 *op. cit.*

[40] Valdes, M., 2009. *Estimating the lung burden from exposure to aerosols of depleted uranium*. Radiation Protection Dosimetry, 134(1), pp.23-29.

[41] WHO 2001 *op. cit.*

et al,[42] both substitute some of the variables related to particle solubility. Even so, there are still many uncertainties that are not addressed, as well as the inherent uncertainty in the ICRP models.

While the ICRP models may be a useful tool for estimating radiological doses that have already occurred, or where a society wishes to carry out a theoretical cost-benefit analysis of a particular use of radioactive materials, the uncertainties are such that caution should be exercised. It therefore follows that the models should not simply be used as a basis for discounting risk, as seems to be the case in SCHER.

Contamination and exposure data

Data characterising DU contamination in the field is fragmentary and is skewed towards certain types of weapon. Furthermore, most field data is based on single visits to contaminated sites, often years after the use of the weapons, so little is known about changes in contamination over time. Although some US test data allows for improvements to ICRP risk modelling, it also suggests that the characteristics of contamination may vary considerably from strike to strike, further complicating risk assessment. SCHER has omitted important data on the contamination of water in Bosnia from their report.

As data characterising post-conflict contamination is an essential element of accurate risk assessments, it is unfortunate that the information in the literature is skewed towards contamination from one type of DU weapon and largely based on environmental survey work completed years after the end of conflicts.

Since the first studies on contaminated sites, it has been clear that contamination is concentrated in ‘hot-spots’, and it is through contact with these areas that there is the greatest risk of exposure. In the report, SCHER gives an overview of some of the concentrations of uranium in samples taken at DU strike sites. However, the table on page 14 contains an oversight, as it claims that there is no data on the concentrations of DU in water at sites visited by UNEP in Bosnia.[43] This omission is puzzling, as Bosnia was the only country where isotopic analysis[44] unequivocally showed the presence of DU contamination in a drinking water sample. This result can be found in the same report that SCHER cites for soil concentration.

The most detailed data on post-conflict contamination comes from the three UNEP reports completed on contamination in the Balkans, which resulted from visits to sites between one and seven years after the end of hostilities.[45] The contamination in the Balkans was due solely to 30mm rotary cannon fire by US A-10 Thunderbolt aircraft. Although this type of contamination comprises a larger portion of DU fired by mass than any other round (where quantities are known), only the US fields DU rounds of this type. It is not clear how the nature of this contamination compares to contamination from other calibres of round fielded worldwide,[46] and to other air and land platforms. It is therefore of limited use in extrapolating to other contaminated sites.

What information there is on contamination immediately following a DU strike comes not from battlefield studies, but primarily from US weapons testing grounds. These tests are completed under idealised control conditions and, as noted by the WHO, should not be regarded as wholly representative of battlefield contamination.[47] The most detailed study of this type has been the US Capstone study,[48] which involved

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[42] Li et al. 2008 *op. cit.*

[43] SCHER 2010. *op. cit.* P14

[44] As DU is comprised of natural uranium that has had most of the isotope U235 removed, the way to ascertain if a sample of uranium contains DU is to look at the ratio of U235 to the other uranium isotopes in the sample. This is called isotopic analysis.

[45] UNEP 2001, 2002, 2003 *op. cit.*

[46] Primarily 25, 105, 120, and 125mm armour piercing rounds.

[47] WHO 2001. *op. cit.* p48

[48] M. A. Parkhurst et al., 2004. *Capstone Depleted Uranium Aerosols: Generation and Characterization*. Oak Ridge, TN: Pacific Northwest National Laboratory.

firing one, or in a few cases two large calibre DU rounds, at a tank hull that had been largely stripped of internal equipment and flammable materials. Rounds were mainly fired at 90 degree angles, passing right through the target and avoiding secondary fires from the ignition of ammunition or fuel. This ensured that the contamination would have been relatively clean. Nonetheless the results are very much of interest.

Particles were found to be more significantly varied in shape, structure and chemical composition than anticipated, giving a greater than expected variation in solubility.[49] Furthermore the study found that the type of armour and shot used in the test was not predictive of the solubility of the samples, meaning that even DU strikes in apparently similar scenarios might produce particulate DU with dissimilar characteristics.[50] The Capstone authors also found that the particle size distribution did not correlate with the uni-modal (i.e. a single bell curve peak) distribution assumed in the HRTM. Instead they found that a bi-modal distribution (i.e. a curve with two peaks) was more common,[51] although the distribution in some samples did not fit with either. The authors of the Capstone study believed that the variation between samples was due to the particles having gone through several physical processes in the strike: a liquid-drop phase, vaporization, condensation and oxidization.[52] This unpredictability has significant implications for the modelling of aerodynamic and solubility characteristics in the ICRP HRTM model.

The Capstone study used some experimental data in an adapted version of the ICRP models to run a health risk assessment. It concentrated only on the risks to military personnel, and found that in certain circumstances the soldiers could receive significant radiation doses.[53]

The UK Health Protection Agency (HPA) sensitivity study on the ICRP models showed that changing the particle size distribution and solubility of DU particles could result in significant changes to the dose calculation.[54] This means that the apparent variations between and within samples of particle size distribution and dissolution rate in the Capstone results pose serious obstacles to accurate risk evaluation.

Although the implication of the Capstone study is that the risk to civilians was likely to be considerably lower than that estimated for soldiers, there are still considerable uncertainties involved. As well as the inherent uncertainties in the ICRP risk models mentioned above, and whether contamination in actual battlefield conditions would be similar to Capstone's experimental conditions, results were only collected for two hours after the strike. How characteristics of contamination will change after a strike,[55] and the degree to which the Capstone results are analogous to the kind of contamination studied in the field are not known. This means that significant questions still remain about which particle characteristics should be used in risk assessments. Another complicating factor is the lack of knowledge about the long-term behaviour of DU in the environment, particularly in arid conditions.[56]

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[49] Ibid. p7.11

[50] Guilmette, R.A. & Cheng, Y.S., 2009. *Physicochemical characterization of Capstone depleted uranium aerosols IV: in vitro solubility analysis*. Health Physics, 96(3), pp.292-305.p303

[51] Parkhurst et al. 2004 *op.cit.*p5.49

[52] See Szrom, F. et al., 2009. *Calculating Capstone depleted uranium aerosol concentrations from beta activity measurements*. Health Physics, 96(3), p243

[53] Some scenarios estimated doses could be higher than a 600 mSv dose to the lungs. The study also showed that those attending an attacked vehicle to help the occupants might have an increased lifetime cancer risk of up to 0.01% or 1 per 1,000

[54] Bailey et al 2007 op. cit. p48-49

[55] Parkhurst et al. 2004 *op.cit.*p5.70

[56] M. Burger & H. Slotte, 2007. *United Nations Environment Programme Results Based on the Three DU Assessments in the Balkans and the Joint IAEA/UNEP Mission to Kuwait*. In A. Miller, ed. Depleted Uranium Properties, Uses, and Health Consequences.

Exposure studies on soldiers and civilians

While few studies have shown elevated uranium levels in the urine of those studied, the literature is heavily skewed towards veterans, with almost no civilians having been studied in affected areas. Furthermore, most of the studies have not identified the subjects most at risk from exposure, so questions remain about their relevance. The presentation of this material by SCHER is again misleading, particularly with regard to the number of civilians who have been studied.

When discussing "*biomonitoring studies*", which in this context refers to monitoring the level of uranium excreted in urine and whether the subject has been exposed to DU,[57] the SCHER report says the studies show that *"the incorporation of DU in soldiers serving in Iraq and in residents of Kosovo is very low".*[58] This statement is justified by reference to a table[59] where the results of biomonitoring studies are set out.

Neither the table, nor the accompanying text, make it explicit that five of the studies listed, including the two with the largest number of subjects, were actually carried out on unexposed civilians, and are presumably only included as examples of the levels of urinary excretion found in the general population.[60] One further study was carried out on residents and workers exposed to contamination by a factory that manufactured DU weapons in the US, and is presumably included in the list to demonstrate the sensitivity of biomonitoring methods.[61]

As such, only six of the papers in the table are relevant to SCHER's conclusion about the exposure of soldiers and local residents, two of which pertain to the same study.[62] These studies cover around 2500 soldiers but only 25 residents. Furthermore, two of the studies did identify soldiers who were excreting DU in their urine.[63] It should also be pointed out that in the larger two studies,[64] no real detail is provided about the process of subject selection. Without this information it is difficult to know whether these studies identified subjects who had actually been involved in incidents, or spent time at locations where they might have been exposed to DU. It may be the case that some of the subjects put themselves forward for testing and were never in danger of exposure.

However, while these studies do indicate that only a few soldiers from the groups studied were exposed to DU, it is difficult to justify the conclusions SCHER draws about the possible exposure of civilians from these

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[57] All urine contains trace amounts of uranium. The presence of DU is determined by the ratio of the different uranium isotopes.

[58] SCHER 2010 *op. cit.* p16

[59] Table 8. *Ibid.* p15

[60] UBA, 2005. *Uran und Human-Biomonitoring. Bundesgesundheitsblatt - Gesundheitsforschung - Gesundheitsschutz*, 48(7), pp.822-827; NHANES, 2005. *Third National Report on Human Exposure to Environmental Chemicals*, Atlanta, Georgia: Department of Health and Human Services Centers for Disease Control and Prevention; Al-Jundi, J., 2004. *Thorium and uranium contents in human urine: influence of age and residential area*. Journal of Environmental Radioactivity, 71(1), pp.61-70; Galletti, M. et al., 2003. *Uranium daily intake and urinary excretion: a preliminary study in Italy*. Health Physics, 85(2), pp.228-235; Karpas, Z. et al., 2005. *Urine, hair, and nails as indicators for ingestion of uranium in drinking water*. Health Physics, 88(3), pp.229-242.

[61] Parrish, R.R. et al., 2008. *Depleted uranium contamination by inhalation exposure and its detection after ~20 years: Implications for human health assessment*. Science of The Total Environment, 390(1), pp.58-68.

[62] Oeh, U. et al., 2007a *Daily uranium excretion in German peacekeeping personnel serving on the Balkans compared to ICRP model prediction*. Radiation Protection Dosimetry, 127(1-4), pp.329-332 and Oeh, U. et al., 2007b *Measurements of daily urinary uranium excretion in German peacekeeping personnel and residents of the Kosovo region to assess potential intakes of depleted uranium (DU)*. The Science of the Total Environment, 381(1-3), pp.77-87.

[63] Dorsey, C.D. et al., 2009. *Biological Monitoring for Depleted Uranium Exposure in U.S. Veterans*. Environmental Health Perspectives. Available at: http://ehp.niehs.nih.gov/docs/2009/0800413/abstract.html [Accessed September 18, 2011]; Gwiazda, R.H. et al., 2004. *Detection of depleted uranium in urine of veterans from the 1991 Gulf War.* Health Physics, 86(1), pp.12-18.

[64] Oeh et al 2007a, b. *op. cit.*; Dorsey et al. *op. cit*

studies. This is particularly true when the biomonitoring of 25 individuals is used to justify statements that imply much more extensive research.[65] The potential for these statements to mislead the reader is compounded by the fact that, while SCHER has listed the number of individuals in every other group in the biomonitoring table, they have neglected to do so for the residents. It would be easy for the reader to assume the number of Kosovo residents was comparable to the 1228 German peacekeepers studied by the same authors.

On the basis of such a small study group, it is not prudent to make any assumptions about the possible exposure of civilians. Civilians may live or work in or near contaminated areas and their exposure profile may differ greatly from soldiers who were exposed in a single incident or during a relatively short posting. It should also be noted that the study in question does not go into any detail about the selection of these 25 civilians. Some of the subjects were chosen because of their participation in an earlier study where they were (apparently incorrectly)[66] identified as having been exposed to DU. The rest are identified only by the town they reside in.

As DU contamination is known to be concentrated in relatively localised areas, biomonitoring programmes that do not seek to identify individuals who are most at risk of coming into contact with contamination are unlikely to yield fruitful results. It should also be noted that SCHER does not appear to be familiar with a study undertaken in Serbia, which did identify some civilians who tested positive for DU contamination, and where the contamination appeared to correlate with evidence of radiation damage at a genetic level.[67] Although this study did not use the most sensitive methods for determining exposure, failing to include it is unfortunate. In summary, SCHER is mistaken to draw conclusions about the exposure of civilians on the basis of the evidence it includes. And, as has been noted elsewhere,[68] there is a need for studies to determine whether the residents of contaminated areas have been, and continue to be exposed.

Extrapolating the toxicological effects of DU from animal and laboratory data

Although animal and laboratory evidence suggests that there are other health outcomes that may be linked to DU exposure, the SCHER report only attempts to assess the toxicological risk of kidney damage. Other health effects may occur below the levels of exposure that would cause kidney damage. Instead of acknowledging this uncertainty, the SCHER report assumes that kidney damage is the only health outcome of interest. Instead of interpreting a number of recent papers on the different toxicological or genotoxic effects of DU, the SCHER report seeks to dismiss them as irrelevant.

In recent years, a number of studies have shown that DU exposure has the potential to cause various changes in body function, many of which may well be linked with negative health outcomes. However, the major expert reports into the DU issue have limited themselves to investigating either radiological risk, or damage to the kidneys induced by the chemical toxicity of DU.

Although these other health outcomes are often referred to as being caused by the toxicological effects, and are briefly dealt with in that section of the SCHER report, it is quite possible that the radiological effects of

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[65] "*[B]iomonitoring for the presence of DU has been performed in military personnel and long-term Kosovo residents. Most of these studies have failed to find increased concentrations of DU in the sampled population.*" SCHER 2010. *op. cit.* p19

[66] It is thought that this error occurred due to contamination of the testing vessels. The authors of the earlier study are also co-authors of the study cited by SCHER.

[67] Milacić, S. et al., 2004. *Examination of the health status of populations from depleted-uranium-contaminated regions*. Environmental Research, 95(1), pp.2-10.

[68] B. Spratt, 2007. *Health Hazards of Depleted Uranium Munitions: Estimates of Exposures and Risks in the Gulf War, the Balkans, and Iraq*. In A. Miller, ed. Depleted Uranium Properties, Uses, and Health Consequences. pp. 121-142. p139

DU also play a role, as experimentally it is difficult to separate the two. For the purposes of brevity, these effects will be described as being purely toxicological, unless it is necessary to go into further detail.

Many of these studies have dealt with the genotoxic effects of DU (i.e. its propensity to damage the genetic material in cells),[69] other studies have indicated that DU damages nerve cells and the brain,[70] changes liver function,[71] alters bone formation[72] and can cause developmental problems in the offspring of exposed animals.[73]

Although some of these health problems may only occur at levels greater than the levels sufficient to cause kidney damage, for many of the effects, the dose required to cause an adverse effect is not yet clear. The damage to kidney function can be the result of an acute dose of DU (a single large dose at one time), and 'tolerable' intake levels are set below the level necessary to produce this effect.

There is of course no reason to suppose that the levels deemed to be safe for the kidney do not have the potential to cause other adverse health outcomes. Other health effects that have been less well studied could potentially be triggered at lower levels or could result from chronic long-term exposure to DU at levels below those that would damage kidney function. Some, including genotoxic effects, may have no threshold or 'safe' dose. Although there is insufficient evidence to determine whether this is in fact the case, developmental effects have been recorded at dose levels below those that can cause kidney damage,[74] and one study reported changes to the reproductive system at doses of 0.00039 mg/kg/day – 0.65% of the lowest LOAEL[75] dose level cited in the SCHER report.[76]

The literature dealing with chemical toxicity does not tend to use the detailed body models of the ICRP system. Instead it studies the effect of a given dose on a laboratory animal and extrapolates to the dose one might expect to give a similar effect in a human. When determining *'safe'* exposure levels, the methodology is relatively unsophisticated: the levels deemed not to be injurious during animal experimentation are translated over to humans by using a multiplication factor. This is applied to allow for the expected variation between species and between individuals. In the absence of experimental data to determine the multiplication factor, a standard factor of 100 is used.[77] There is very little information on whether children are more susceptible than adults to the effects of uranium.[78]

This approach has the benefit of helping to assess risk without the need for identifying or understanding the biological mechanisms at work – a given health outcome can be correlated with dose.[79] However, aside

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[69] See D. McClain & A. Miller, 2007. *Depleted Uranium Biological Effects: Introduction and Early in Vitro and in Vivo Studies*. In A. Miller, ed. Depleted Uranium Properties, Uses, and Health Consequences. pp. 1-20 for a good overview of the genotoxicity of DU

[70] W. Briner, 2007. *Neurotoxicology of Depleted Uranium in Adult and Developing Rodents.* In A. Miller, ed. Depleted Uranium Properties, Uses, and Health Consequences.p72

[71] Guéguen, Y. et al., 2005. *Short-term hepatic effects of depleted uranium on xenobiotic and bile acid metabolizing cytochrome P450 enzymes in the rat*. Archives of Toxicology, 80(4), pp.187-195.

[72] Fukuda, S., 2005. *Clinical diagnostic indicators of renal and bone damage in rats intramuscularly injected with depleted uranium*. Radiation Protection Dosimetry, 118(3), pp.307-314

[73] Briner, W., 2010. *The Toxicity of Depleted Uranium*. International Journal of Environmental Research and Public Health, 7(1), p306

[74] *Ibid.* P306

[75] Lowest Observed Adverse Effect Level – the dose level below which no harmful changes can be observed.

[76] Agency for Toxic Substances and Disease Registry (ATSDR) 2011. *Draft Toxicological Profile For Uranium*, Atlanta, Georgia.

[77] WHO, 2001. *Depleted uranium: Sources, Exposure and Health Effects*, Geneva: Department of Protection of the Human Environment, World Health Organization. Available at: http://whqlibdoc.who.int/hq/2001/WHO_SDE_PHE_01.1.pdf. p67-68

[78] ATSDR 2011 p230-233

[79] Although, it should be pointed out that correlating a dose with a health outcome in the experimental animal does not mean that the calculation used to extrapolate over to humans will necessarily be valid.

from the need to experiment on animals, another drawback of this approach is that only studies of final health outcomes can produce quantitative results, and it appears that it is impossible to draw any firm conclusions from studies that do not conform to this model.

This shortcoming is evident in the way the SCHER report deals with the recent literature on the toxicological effects of DU. A list of 37 studies is dismissed en masse with the explanation that some *"have focused on U and DU effects after administration of single or repeated high doses, used a short time frame of observation, or…used inappropriate routes of administration such as intraperitoneal injection."*[80]

It is extremely unsatisfactory to dismiss evidence in this way, rather than discussing the papers individually. This was pointed out to SCHER during consultation,[81] but the presentation of these papers was not substantially changed in the final Opinion. As it is not possible to identify which of the apparent shortcomings in this list pertains to which paper, the papers are presented in a table in Appendix D for the reader to assess. Although there are a number of in vitro (i.e. test tube) studies, around half of the studies are animal studies using exposure through inhalation or in drinking water. In the 13 studies that used drinking water as a vehicle for DU ingestion in laboratory rodents, dosage was low in 10 of these studies and several other studies used both low and high doses. Many of them use timescales comparable to those used to derive the WHO Tolerable Daily Intakes cited by SCHER.[82]

The other objection given is that some of the studies *"focused on selected biochemical changes without characterizing functional or pathologic consequences"*, in other words, the studies were investigating physiological changes, and it was not clear if these changes were directly linked to health problems. While this is certainly the case with a number of the studies,[83] it would surely be more appropriate for a body such as SCHER to make judgements about the potential effects of such changes, rather than to just conclude that the study had no "*relevant information"* and fail to draw any conclusions from the results.

Many of these studies deal with the issue of genotoxicity, effects at the cellular level or changes in biochemical behaviour that have not yet been directly linked to clearly identifiable *pathological consequences*. This is only to be expected in research, where the full implications of a newly discovered physiological reaction may not yet be understood. Even if these studies do not fit SCHER's criteria for risk assessment,[84] it would be prudent for the potential for health problems to be identified and a certain amount of caution exercised when estimating '*tolerable'* exposure levels. SCHER's refusal to engage with this new literature is particularly unfortunate as the desire to see such literature incorporated into assessments of the risks from DU was a major motivating factor in the SCHER report originally being commissioned.

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[80] SCHER 2010. *op cit.* p10

[81] Bertel, *op. cit.*

[82] The latest WHO drinking water standards for uranium: WHO, 2006. *Guidelines for drinking-water quality: first addendum to third edition*. Volume 1, Recommendations., Geneva: World Health Organization. The 60µg/kg/day level cited by SCHER is based on a 91-day study on rats looking at damage to the kidney with a LOAEL of 0.96 mg/L

[83] This is not true of all the studies listed. Guéguen, Y. et al., 2007. *Effect of acetaminophen administration to rats chronically exposed to depleted uranium*. Toxicology, 229(1-2), pp.62-72. is a chronic nine month study of liver and kidney damage from exposure in drinking water up to 40mg DU/Litre. This does not appear to fit any of the shortcomings identified by SCHER.

[84] See SCHER 2010, *op. cit.* p10

Epidemiology

Because epidemiological data on civilian populations exposed to DU is missing from the literature, SCHER attempts to apply the results of studies on uranium miners and millers to the health risks of military DU exposure. The health effects found in uranium miners are widely thought to be linked to exposure to radon gas. However the strong link with radon exposure does not preclude potential harm from uranium exposure.

Studies on workers in uranium mills, where radon exposure is less of a problem, have not identified any statistically significant link between uranium exposure and health problems, although some show an elevated risk of some problems below the level of statistical significance. Unfortunately these studies lack any meaningful exposure data, so they should not be relied upon for information about the risks of military-origin DU. There is also reason to believe that the characteristics of particulate contamination (such as size and solubility) in uranium mills may be less hazardous.

The SCHER report only briefly covers the subject of epidemiological studies. When discussing the radiological effects of DU, the report explains why it is very difficult to detect increases in cancer risk from low radiation doses through epidemiological studies.[85] Reference is also made to studies on uranium miners and the well documented association between exposure to radon and lung cancer in uranium miners. While in some cases attempts have been made to estimate radiation dose from long-lived radionuclides such as uranium, these appear to have been limited to radiological risk only,[86] and it remains an open question whether uranium dust in mines shares the same characteristics as DU contamination from a weapon strike.

What SCHER does not discuss is the lack of any epidemiological studies on civilians or military personnel who have been exposed to DU from weapons. This complete lack of such studies is a major argument for caution when assessing the risk of health problems from DU exposure. Although the practical problems in performing epidemiological studies in the aftermath of conflict are significant,[87] and it may not be possible to identify a cohort of sufficient size,[88] a well designed study would be considerably more powerful than much of the evidence discussed in this paper.

Another potential avenue is epidemiological studies on workers in uranium mills, processing uranium ore, where the exposure to radon is not such a significant factor. However, the picture from these studies is not clear. Although none of them have shown statistically significant increases in health problems, some studies have shown increased risk of certain health problems associated with work in uranium mills, but not a strong enough association for statistical significance.[89] Other studies have shown an increased risk of cancer in mill workers.[90]

Studies comparing miners and mill workers are complicated by a lack of exposure data, meaning that subjects can only be grouped by the type of work they were involved with. These are obviously categories where the individual exposure may vary considerably, reducing the precision of the study.[91] There may also

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[85] *Ibid.* p7

[86] Panel on Dosimetric Assumptions Affecting the Application of Radon Risk Estimates, 1991. Comparative Dosimetry of Radon in Mines and Homes, Washington D.C.: National Academy Press. p26

[87] Due to, for example, the collapse in health provision or large-scale population movements. See ICBUW 2010 *op. cit.*

[88] Institute of Medicine (U.S.);Institute.; National Academies Press (U.S.), 2008. *Epidemiologic studies of veterans exposed to depleted uranium feasibility and design issues*, Washington D.C.: National Academies Press. Stated that over >1 million DU-exposed veterans would be required for a lung cancer study

[89] See Boice Jr, J.D. et al., 2007. *Mortality among residents of Uravan, Colorado who lived near a uranium mill*, 1936–84. Journal of Radiological Protection, 27(3), pp.299-319; Boice Jr, J.D. et al., 2008. *A cohort study of uranium millers and miners of Grants, New Mexico, 1979–2005*. Journal of Radiological Protection, 28(3), pp.303-325; Dawson, S.E., Madsen, G.E. & Spykerman, B.R., 1997. *Public health issues concerning American Indian and non-Indian uranium millworkers*. Journal of Health & Social Policy, 8(3), pp.41-56.

[90] ATSDR 2011 *op. cit.* P103-104

[91] See Boice et al 2008 *op. cit.* p306-307.

have been significant changes in working practices and conditions over time, which can further complicate matters.[92]

Another question to consider is whether the type of exposure that might be found in a uranium mine or mill is actually equivalent to the exposure from the dust created by a DU-weapon strike. As is clear from the ICRP risk models and data from the Capstone tests, small changes in the characteristics of the DU particles (size, shape, solubility etc) can significantly alter both dose and the behaviour of DU within the body, and the debris from a strike can vary considerably in these characteristics. Certainly, at least one study found that only a small proportion of the dust in uranium mills was comprised of small enough particles to be inhaled into the lungs, whereas during weapons testing the proportion was always more than 50%.[93]

Medical studies on US veterans exposed to DU during warfare

Medical studies conducted under the auspices of the US Department of Veterans Affairs are of limited use for assessing the potential health effects of DU exposure. The lack of a single cohort, or of a control group, as well as the small number of subjects make meaningful interpretation of the results impossible. Furthermore, some matters of interest have not been properly followed up, or have even been completely omitted from reports, including incidence of tumours within the group.

The series of health studies on US veterans carried out under the auspices of the US Department of Veterans Affairs is one of the most commonly cited areas of evidence about the health risks of DU. However, it is somewhat problematic to claim that these studies provide *"[f]urther support for an absence of health effects of lower DU exposures"* as stated in the SCHER report. These clinical studies were carried out on a very small number of individuals, and are far too small to give statistically significant findings.

The studies cover only around 74 individuals in total,[94] and as the same individuals are not included in each periodical update, the studies cannot meaningfully track changes in the subjects over time. The studies have also come in for significant criticism for not following up findings of interest, such as the discovery of uranium in the sperm of subjects. Most troubling was the revelation by a Scientific Committee answering to the US Congress, that the studies had failed to include the incidence of two tumours within the group in the study results. When challenged, the lead author responded that the tumours were *"not included because they were not believed to be the result of DU exposure".* It hardly needs repeating that omitting information on the basis of a belief is entirely contrary to good scientific practice.[95]

Therefore, although these studies hold some interest, for example as a long-term study of urine excretion in exposed individuals, it is not appropriate to rely on them to draw wider conclusions about the health consequences of DU exposure

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[92] See Dawson et al 1997 *op. cit.*

[93] Reif, R.H. & Andrews, D.W., 1995. *Derivation and implementation of an annual limit on intake and a derived air concentration value for uranium mill tailings*. Health Physics, 68(6), pp.823-826 found that in the most dusty conditions between 86% and 100% particles had an AMAD over 10µm. In the less dusty conditions it was between 56% and 98%. By comparison, Cheng, Y.S. et al., 2009. *Physicochemical characterization of Capstone depleted uranium aerosols II: particle size distributions as a function of time*. Health Physics, 96(3), pp.266-275. found that the median AMAD for Capstone samples fell between 5 and 10µm. Particles under 10µm are considered respirable under the ICRP models.

[94] K. Squibb & M. McDiarmid, 2007. *Exposure and Health Surveillance in Gulf War Veterans Exposed to Depleted Uranium*. In A. Miller, ed. Depleted Uranium Properties, Uses, and Health Consequences. pp. 105-120.

[95] Research Advisory Committee on Gulf War Veterans' Illnesses, 2008. *Gulf War Illness and the Health of Gulf War Veterans: Scientific Findings and Recommendations*, Washington, D.C: U.S. Department of Veterans Affairs. pp. 96-99

Recommended risk reduction measures have not, and will not, be put in place

While there is a significant body of literature dealing with the potential health effects of DU exposure, the picture presented by the SCHER report is misleading. While it is clear that exposure carries with it some level of risk, it is difficult to quantify it with any level of certainty. The shortcomings in the different areas identified in this commentary are such that the evidence cannot reliably be used to reinforce the assertion that the risk from exposure is negligible, as the SCHER report appears to suggest. A better approach would be to acknowledge the uncertainties and to exercise precaution, rather than assume that harm is unlikely.

Although SCHER does acknowledge that there is some potential for harm, the measures suggested to reduce it by international agencies such as UNEP, WHO, Royal Society and IAEA have generally not been followed through in states where DU weapons have been used. Furthermore, the nature of post conflict conditions ensures that they are unlikely to be in the future. As such, a sensible approach to this issue is that recommended by the European Parliament: the use of these weapons should be subject to an international moratorium and steps should be taken towards an international treaty ban. This is the only approach that is in line with radiation protection norms and a common-sense approach to environmental contamination.

From this summary of the different spheres of research that can be used to assess the risk from DU exposure, it is clear that in many cases the interpretation of the evidence given in the SCHER report is open to challenge. Furthermore, some of the assertions made by SCHER, for example that biomonitoring studies have shown that there is little risk to civilians, cannot be substantiated.

While SCHER does not deny that in some circumstances exposure to DU could cause health problems, it states that these circumstances are quite limited, and that the risks to the general population are very low. This rather misses the point: it is clear that DU contamination presents some level of risk to civilians who may encounter contaminated land or vehicles. To diminish this risk, SCHER recommends that target vehicles and used DU ammunition should be made inaccessible to the general public.[96] This is in line with recommendations made by the WHO, and UNEP, which have both recommended that contaminated sites should be isolated.[97]

However, ICBUW's research into the fate of DU strike sites in the Balkans shows clearly that in countries recovering from conflict, oversight of contaminated areas is minimal, few records are kept and it is often impossible to ascertain what the fate of target vehicles and contaminated scrap metal has been. In some cases there is clear evidence that unwitting civilians came into close contact with this contaminated material. In one industrial site in Bosnia and Herzegovina, significant quantity of contaminated material was cleared and disposed of by hand, by workers who had no knowledge of what they were handling and therefore took no safety measures.[98]

From ICBUW's contact with ex-government officials in Iraq and demining organisations working in the field, it is clear that in the immediate aftermath of conflict, civilians will inevitably come into contact with DU contamination and risk the "*[h]igher exposures*" that SCHER mentions only in passing. As such it is unrealistic for SCHER to assume that such exposures can be avoided.

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96 SCHER 2010 *op. cit.* p.18

97 A summary of UNEP recommendations for contaminated sites in the Balkans can be found in ICBUW 2010 *op. cit.* pp 23-24

98 A longer account of this case studycan also be found in ICBUW 2010 *op. cit.* pp 9-10

By failing to critically evaluate the uncertainties in the current evidence about the risks of DU, and neglecting to interpret more recent studies into the health effects of DU, the SCHER report has failed to properly follow through its terms of reference, and should be regarded as a missed opportunity.

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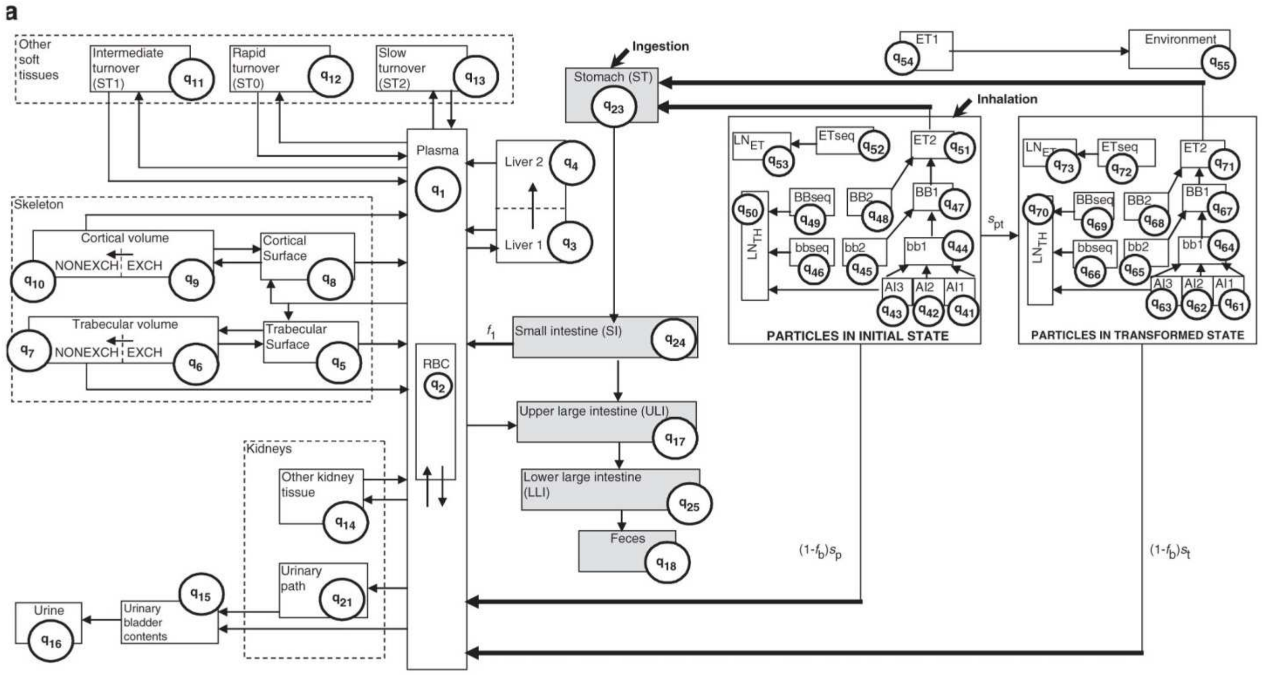
Appendix A

Page	Study	Type of DU risk assessment	Uses ICRP Models	Comments
8, 17	**EU-EURATOM, 2001**	Radiological risk.	Yes	
8, 17	**EURATOM, 2009**	No risk assessment of DU.	N/A	Discusses an ICBUW paper and concludes that earlier assessment will not be revised.
8, 13, 17	**IAEA, 2003**	Radiological risk.	Yes	
8, 13, 17	**IAEA, 2009**	Radiological risk.	Yes	Analysed results of UNEP 2007
8, 13, 17	**UNEP, 2001**	Radiological risk and toxicological risk to kidneys.	Yes	Risk assessments in an appendix only. Uses field data with dose coefficients and limits derived from other studies.
8, 17	**UNEP, 2002**	Radiological risk and toxicological risk to kidneys.	Yes	Risk assessments in an appendix only. Uses field data with dose coefficients and limits derived from other studies.
8, 13, 17	**UNEP, 2003**	Radiological risk and toxicological risk to kidneys.	Yes	Risk assessments in an appendix only. Uses field data with dose coefficients and limits derived from other studies.
8, 17	**UNEP, 2007**	Report on building technical capacity in Iraq. No health risk assessment	N/A	Results are analysed in IAEA 2009
8, 17	**UNEP/UNCHS, 1999**	Radiological risk.	Yes	Desk calculation based on no field data.
8, 17	**UNSCEAR, 1993**	No risk assessment of DU.	N/A	
8, 17	**UNSCEAR, 2000a**	No risk assessment of DU	N/A	
8, 17	**UNSCEAR, 2000b**	No risk assessment of DU.	N/A	
8, 17	**WHO, 2001**	Radiological risk and toxicological risk to kidneys only.	Yes	

Appendix B

ICRP models



Pictured are the three ICRP models(from left to right): the Systemic model, the Gastro-Intestinal Tract modeland the Human Respiratory Tract Model.[99]

Appendix C

This is an overview of some of the uncertainties and assumptions in the ICRP Human Respiratory Tract Model.[100] The HRTM has been chosen because inhalation is widely regarded as the most significant exposure pathway to DU and the HRTM is also the part of the model subject to the greatest range of uncertainty, according to analysis.[101] A diagram of the HTRM, as well as the ICRP systemic and Gastro Intestinal Tract model, is included above.

- The model is based on typical values for physiological variables, such as lung capacity, fitness, the degree to which the subject breathes through the nose etc. Different breathing rates can be used and standard values are given for different types of activity (e.g. exercise, sleeping) undertaken during the time of exposure. Although different age groups are catered for, the model is based on figures for Caucasians, although some reference values for other ethnic groups are also provided.[102]
- The size, shape, crystalline structure and chemical makeup of the particles inhaled are the major factors in determining where in the lungs the particles are deposited, the quantity deposited and what

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[99] Illustration taken from Li et al 2008 *op. cit.*

[100] ICRP 1994. *op. cit.*

[101] Harrison, J. & Health Protection Agency (Great Britain)., 2007. *Uncertainty analysis of the ICRP systemic model for uranium as applied to interpretation of bioassay data for depleted uranium*, Didcot: Health Protection Agency. pv.

[102] ICRP 1994. *op. cit.* p22

happens to the particles upon deposition. The degree to which the particles dissolve are absorbed into the bloodstream is also largely a function of these variables. The model provides for three separate dissolution speeds, and the inhaled uranium is assigned to one of these categories depending on its chemical form. Other variables affecting the dissolution are not factored into the model, though an experimentally derived value for dissolution rate can be used instead of the standard values.[103]

- The variables around aerodynamic behaviour of particles (chiefly size, shape and density) are particularly difficult to model meaningfully. The model assumes that the degree to which particles aerodynamically vary from acting like perfect spheres is quite regular – that the distribution of variability forms a standard 'normal' distribution (i.e. a 'Bell Curve'). Two standard values for the median point (the apex of the curve) are given – for workplace and public exposures. These two values are assumed to be sufficient for all materials and exposures.[104] SCHER includes a table of a range of particle sizes,[105] though it isn't clear what the source for the data is, and there is no discussion about how this variable could affect dose, or how it might affect the risk assessments cited by SCHER.

- The model assumes that particle deposition (the proportion of particles land in each part of the respiratory system) is governed entirely by aerosol size, so these assumptions about the distribution of particle size also affect deposition calculations within the model.[106] Even if the particle size distribution were to be modelled more sophisticatedly, data on aerosol deposition within the respiratory tract is incomplete and the subject of debate. The ICRP's approach to modelling deposition is a combination of empirical and theoretical models.[107]

- There may be some biological processes that are not covered by the model, because of a lack of data, or because they were considered too complex to incorporate. For example, some animal studies suggest that aerosolized uranium can cross the "nose-brain" barrier but this is not catered for in the HRTM,[108] and the brain is not modelled as a separate organ within the ICRP system at all.

- In each compartment of the HRTM, except for the front of the nasal passage, a certain amount of material (depending on the dissolution rates mentioned above), is assumed to dissolve into the bloodstream. The rest of the material is assumed to be cleared by various biological processes, so that the respiratory system will slowly clear itself of contaminants. However, when modelling these processes "major *uncertainties remain, which can only be resolved by further research*". Because of these uncertainties, assumptions and simplifications have been made in order to create a working predictive model of these systems.[109] These variables are significant because they determine the length of time DU is present in the various parts of the respiratory system. The transport of radionuclides within the lungs is also known to be affected by particle size and chemical toxicity of the materials involved,[110] however neither of these factors affect how these processes are modelled and a standard transfer rate is given regardless of particle size and material.

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103 WHO 2001 and Li et al 2008 both use different dissolution rates for their calculations, rather than the ICRP standard rates.

104 ICRP 1994 *op. cit.* 291-292

105 SCHER 2010 *op. cit.* p13

106 International Commission on Radiological Protection., 1996. *Age-dependent doses to members of the public from intake of radionuclides*, Part 4 1st ed., Oxford: Pergamon for the Commission.p11-12

107 ICRP 1994 *op. cit.* p38

108 See Research Advisory Committee 2008 *op. cit.* p91

109 ICRP 1994 op. citp65-72

110*Ibid.* p318

- The sensitivity of these parts of the respiratory system to radiation dose is not well known and ICRP state that these values cannot be assigned with any degree of confidence. As such, the values assigned to calculate radiation sensitivity within the model are that of related, but separate variables.[111]
- Beyond the scope of the HRTM, there are also uncertainties about how uranium is distributed in small quantities to organs not modelled in the ICRP Systemic model and how uranium is distributed at a cellular level.[112]

Appendix D

Short title	Dose (Qty)	Observation length	Outcome studied	Exposure	Citation
Arnault et al 2008: Natural uranium disturbs mouse folliculogenesis	5, 50 or 400 mg/L	15 weeks	Number and size of ovarian follicles, oocyte maturation and fragmentation	Drinking water	*Arnault, E., Doussau, M., Pesty, A., Gouget, B., Van der Meeren, A., Fouchet, P., and Lefevre, B. (2008). Natural uranium disturbs mouse folliculogenesis in vivo and oocyte meiosis in vitro. Toxicology 247, 80-87.*
Berradi et al 2008: Renal anaemia caused by chronic ingestion	40 mg/L	9 months	Red blood cell count, Renal deterioration	Drinking water	*Berradi, H., Bertho, J. M., Dudoignon, N., Mazur, A., Grandcolas, L., Baudelin, C., Grison, S., Voisin, P., Gourmelon, P., and Dublineau, I. (2008). Renal anemia induced by chronic ingestion of depleted uranium in rats. Toxicol Sci 103, 397-408.*
Briner et al 2005: short and long term effects on open field behaviour	0, 75, or 150 mg/L	2 weeks or 9 months	Behaviour and brain lipid oxidation	Drinking water	*Briner, W., and Murray, J. (2005). Effects of short-term and long-term depleted uranium exposure on open-field behavior and brain lipid oxidation in rats. Neurotoxicol Teratol 27, 135-144*
Bussey et al 2006: Chronic ingestion perturbs acetylcholinesterase activity	40 mg mg/L	1.5, 6 and 9 months	Uranium brain distribution, Holinergic acetylcholinesterase (AChE) activity and onndopaminergic and serotoninergic metabolism	Drinking water	*Bussy, C., Lestaevel, P., Dhieux, B., Amourette, C., Paquet, F., Gourmelon, P., and Houpert, P. (2006). Chronic ingestion of uranyl nitrate perturbs acetylcholinesterase activity and monoamine metabolism in male rat brain. Neurotoxicology 27, 245-252.*
Coryell et al 2006: Molecular analysis of hrpt mutations			Mutations at the hypoxanthine (guanine)phosphoribosyltransferase (hprt) locus in XRCC1-deficient CHO EM9 cells	In vitro study	*Coryell, V. H., and Stearns, D. M. (2006). Molecular analysis of hprt mutations generated in Chinese hamster ovary EM9 cells by uranyl acetate, by hydrogen peroxide, and spontaneously. Mol Carcinog 45, 60-72*
Dublineau et al 2007: Modifications of inflammatory pathways in rat intestine	40 mg/l chronic	3, 6, or 9 months	Effects on the intestine. Several parameters referring to prostaglandin, histamine, cytokine, and nitric oxide (NO) pathways were assessed in ileum	Drinking water	*Dublineau, I., Grandcolas, L., Grison, S., Baudelin, C., Paquet, F., Voisin, P., Aigueperse, J., and Gourmelon, P. (2007). Modifications of inflammatory pathways in rat intestine following chronic ingestion of depleted uranium. Toxicol Sci 98, 458-468*
Feugier et al 2008: Alteration of mouse oocyte quality	10, 20 and 40 mg/L	49 days	Oocyte quality	Drinking water	*Feugier, A., Frelon, S., Gourmelon, P., and Claraz, M. (2008). Alteration of mouse oocyte quality after a subchronic exposure to depleted Uranium. Reprod Toxicol 26, 273-277*

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[111] *Ibid.* p32

[112] WHO 2001*op. cit.* p66

Fukuda et al 2006: Clinical diagnostic indicators of 21 renal and bone damage	0.2, 1.0 or 2.0 mg/kg single dose	28 days	Biomarkers for kidney and bone damage	Injection	*Fukuda, S., Ikeda, M., Chiba, M., and Kaneko, K. (2006). Clinical diagnostic indicators of 21 renal and bone damage in rats intramuscularly injected with depleted uranium. Radiat Prot Dosimetry 118, 307-314*
Goldman et al 2006: Nephrotoxicity of uranyl acetate			Damage to rat kidney tissue	In vitro study	*Goldman, M., Yaari, A., Doshnitzki, Z., Cohen-Luria, R., and Moran, A. (2006). Nephrotoxicity of uranyl acetate: effect on rat kidney brush border membrane vesicles. Arch Toxicol 80, 387-393*
Grignard et al 2008: Contamination affects steroidogenesis metabolism in rat	40 mg/L	9 months	Testicular steroidogenesis	Drinking water	*Grignard, E., Gueguen, Y., Grison, S., Lobaccaro, J. M., Gourmelon, P., and Souidi, M. (2008). Contamination with depleted or enriched uranium differently affects steroidogenesis metabolism in rat. Int J Toxicol 27, 323-328*
Guegen et al 2007: Effects of acetaminophen administration to rats	40 mg/L	9 months	Liver and kidney damage	Drinking water	*Gueguen, Y., Grandcolas, L., Baudelin, C., Grison, S., Tissandie, E., Jourdain, J. R., Paquet, F., Voisin, P., Aigueperse, J., Gourmelon, P., and Souidi, M. (2007). Effect of acetaminophen administration to rats chronically exposed to depleted uranium. Toxicology 229, 62-72.*
Guegen et al 2008: Short-term hepatic effects of depleted uranium	11.5 mg/kg single dose	1, 3 days	Effects on liver enzymes	Injection	*Gueguen, Y., Souidi, M., Baudelin, C., Dudoignon, N., Grison, S., Dublineau, I., Marquette, C., Voisin, P., Gourmelon, P., and Aigueperse, J. (2006). Short-term hepatic effects of depleted uranium on xenobiotic and bile acid metabolizing cytochrome P450 enzymes in the rat. Arch Toxicol 80, 187-195*
Hahn et al 2002: Implanted DU fragments cause soft tissue fragments		Life-span (some euthanised)	Soft tissue sarcomas	Implanted	*Hahn, F. F., Guilmette, R. A., and Hoover, M. D. (2002). Implanted depleted uranium fragments cause soft tissue sarcomas in the muscles of rats. Environ Health Perspect 110, 51-59*
Hartsock et al 2007: Uranyl acetate as a direct inhibitor of DNA-binding proteins			Functioning of DNA-binding proteins	In vitro study	*Hartsock, W. J., Cohen, J. D., and Segal, D. J. (2007). Uranyl acetate as a direct inhibitor of DNA-binding proteins. Chem Res Toxicol 20, 784-789.*
Hu et al 1990: Induction of chromosomal aberrations in male mouse germ cells		1, 13, 36 and 60 days	Chromosome aberrations	Injection	*Hu, Q. Y., and Zhu, S. P. (1990). Induction of chromosomal aberrations in male mouse germ cells by uranyl fluoride containing enriched uranium. Mutat Res 244, 209-214.*
Kalinich et al 2002: DU chloride induces apoptosis in mouse			Cell apoptosis	In vitro study	*Kalinich, J. F., Ramakrishnan, N., Villa, V., and McClain, D. E. (2002). Depleted uraniumuranyl chloride induces apoptosis in mouse J774 macrophages. Toxicology 179, 105-114*
Kundt et al 2009: Uranium in drinking water effects on mouse oocyte quality	2.5, 5, and 10 mg/kg/day	40 days	Number and quality of ovulated oocytes, chromatin organization, and nuclear integrity	Drinking water	*Kundt, M. S., Martinez-Taibo, C., Muhlmann, M. C., and Furnari, J. C. (2009). Uranium in drinking water: effects on mouse oocyte quality. Health Phys 96, 568-574*
Kurttio et al 2005: Bone as a possible target of chemical toxicity		Average of 13 years (human study)	Biochemical indicators of bone formation and marker for bone formation	Drinking water	*Kurttio, P., Komulainen, H., Leino, A., Salonen, L., Auvinen, A., and Saha, H. (2005). Bone as a possible target of chemical toxicity of natural uranium in drinking water. Environ Health Perspect 113, 68-72*
Lestaevel et al 2005: The brain is a target organ after acute exposure	144 ± 10 µg/kg single dose	3 days	Sleep and wake cycles	Injection	*Lestaevel, P., Houpert, P., Bussy, C., Dhieux, B., Gourmelon, P., and Paquet, F. (2005). The brain is a target organ after acute exposure to depleted uranium. Toxicology 212, 219-226*

Lestaevel et al 2009: Different brain activity between depleted and natural U	2 mg/kg/day	9 months	Pro-/anti-oxidant activity in the brain	Drinking water	*Lestaevel, P., Romero, E., Dhieux, B., Ben Soussan, H., Berradi, H., Dublineau, I., Voisin, P., and Gourmelon, P. (2009). Different pattern of brain pro-/anti-oxidant activity between depleted and enriched uranium in chronically exposed rats. Toxicology 258, 1-9.*
Miller et al 2002a: DU uranium catalyzed oxidative DNA damage			DNA damage	In vitro study	*Miller, A. C., Stewart, M., Brooks, K., Shi, L., and Page, N. (2002a). Depleted uraniumcatalyzed oxidative DNA damage: absence of significant alpha particle decay. J Inorg Biochem 91, 246-252*
Monleau et al 2006a: Effect of repeated inhalation on the distribution in rats	Repeated exposures of 190 or 375 mg/m^3	1, 3, 6, 15, 28, 30 or 90	Biokinetics	Inhalation	*Monleau, M., Blanchardon, E., Claraz, M., Paquet, F., and Chazel, V. (2006a). The effect of repeated inhalation on the distribution of uranium in rats. J Toxicol Environ Health A 69, 1629-1649.*
Monleau et al 2006c: Genotoxic and inflammatory effects	116, 190 and 375 mg/m^3	4 h, or 1, 3, 8 and 14 days	DNA strand breaks	Nose-only inhalation	*Monleau, M., De Meo, M., Paquet, F., Chazel, V., Dumenil, G., and Donnadieu-Claraz, M. (2006c). Genotoxic and inflammatory effects of depleted uranium particles inhaled by rats. Toxicol Sci 89, 287-295*
Monleau et al 2006b: Distribution and genotoxic effects after successive exposure	Single or repeated dose, depending on group. Between 116 and 375 mg/ m^3	4 h, 1, 3, 8, 16 days	DNA damage, biokinetics	Nose-only inhalation	*Monleau, M., De Meo, M., Frelon, S., Paquet, F., Donnadieu-Claraz, M., Dumenil, G., and Chazel, V. (2006b). Distribution and genotoxic effects after successive exposure to different uranium oxide particles inhaled by rats. Inhal Toxicol 18, 885-894*
Periyakaruppan et al 2007: Uranium induces oxidative stress in lung cells			Oxidative stress in rat lung epithelial cells	In vitro study	*Periyakaruppan, A., Kumar, F., Sarkar, S., Sharma, C. S., and Ramesh, G. T. (2007). Uranium induces oxidative stress in lung epithelial cells. Arch Toxicol 81, 389-395*
Periyakaruppan et al 2009: Uranium induces apoptosis in lung cells			Induction of apoptosis in lung epithelial cells	In vitro study	*Periyakaruppan, A., Sarkar, S., Ravichandran, P., Sadanandan, B., Sharma, C. S., Ramesh, V., Hall, J. C., Thomas, R., Wilson, B. L., and Ramesh, G. T. (2009). Uranium induces apoptosis in lung epithelial cells. Arch Toxicol 83, 595-600*
Pourahmad et al 2006: Search for cellular and molecular mechanisms in DU			Cytotoxicity	In vitro study	*Pourahmad, J., Ghashang, M., Ettehadi, H. A., and Ghalandari, R. (2006). A search for cellular and molecular mechanisms involved in depleted uranium (DU) toxicity. Environ Toxicol 21, 349-354*
Racine et al 2009: Modification in expression of genes involved in cerebral	40 mg/L	9 months	Gene expression in enzymes involved in cholesterol metabolism in the brain	Drinking water	*Racine, R., Gueguen, Y., Gourmelon, P., Veyssiere, G., and Souidi, M. (2009). Modifications of the expression of genes involved in cerebral cholesterol metabolism in the rat following chronic ingestion of depleted uranium. J Mol Neurosci 38, 159-165*
Souidi et al 2005: In vivo effects of chronic contamination	40mg/L	9 months	Cellular functions	Drinking water	*Souidi, M., Gueguen, Y., Linard, C., Dudoignon, N., Grison, S., Baudelin, C., Marquette, C., Gourmelon, P., Aigueperse, J., and Dublineau, I. (2005). In vivo effects of chronic contamination with depleted uranium on CYP3A and associated nuclear receptors PXR and CAR in the rat. Toxicology 214, 113-122*
Stearns et al 2005: Uranyl acetate induces hprt mutations in ovary cells			Cytotoxicity	In vitro study	*Stearns, D. M., Yazzie, M., Bradley, A. S., Coryell, V. H., Shelley, J. T., Ashby, A., Asplund, C. S., and Lantz, R. C. (2005). Uranyl acetate induces hprt mutations and uranium-DNA adducts in Chinese hamster ovary EM9 cells. Mutagenesis 20, 417-423*

Theibault et al 2007: Uranium induces apoptosis and is genotoxic to rat kidney			Apoptosis and genotoxicity	In vitro study	*Thiebault, C., Carriere, M., Milgram, S., Simon, A., Avoscan, L., and Gouget, B. (2007). Uranium induces apoptosis and is genotoxic to normal rat kidney (NRK-52E) proximal cells. Toxicol Sci 98, 479-487*
Tissandie et al 2006: Effects of DU on vitamin D metabolism in rats	204 mg/kg	1 or 3 days	Enzymes involved in vitamin D metabolism	Intragastric (i.e. into stomach)	*Tissandie, E., Gueguen, Y., Lobaccaro, J. M., Paquet, F., Aigueperse, J., and Souidi, M. (2006). Effects of depleted uranium after short-term exposure on vitamin D metabolism in rat. Arch Toxicol 80, 473-480*
Tissandie et al 2007: In vivo effects chronic exposure on vitamin D metabolism	40 mg/L	9 months	Enzymes involved in vitamin D metabolism	Drinking water	*Tissandie, E., Gueguen, Y., Lobaccaro, J. M., Grandcolas, L., Voisin, P., Aigueperse, J., Gourmelon, P., and Souidi, M. (2007). In vivo effects of chronic contamination with depleted uranium on vitamin D3 metabolism in rat. Biochim Biophys Acta 1770, 266-272*
Wan et al 2006: In vivo immune toxicity of DU on murine macrophages			Effects on immune cells	In vitro study	*Wan, B., Fleming, J. T., Schultz, T. W., and Sayler, G. S. (2006). In vitro immune toxicity of depleted uranium: effects on murine macrophages, CD4+ T cells, and gene expression profiles. Environ Health Perspect 114, 85-91*
Wise et al 2007: Particulate DU is cytotoxic and clastogenic to human lung cells			Cytotoxicity and clastogenicity	In vitro study	*Wise, S. S., Thompson, W. D., Aboueissa, A. M., Mason, M. D., and Wise, J. P., Sr. (2007). Particulate depleted uranium is cytotoxic and clastogenic to human lung cells. Chem Res Toxicol 20, 815-820.*
Xie et al 2010: DU induces neoplastic transformation in human lung cells			Neoplastic transformation of cells	In vitro study	*Xie, H., LaCerte, C., Thompson, W. D., and Wise, J. P., Sr. (2010). Depleted uranium induces neoplastic transformation in human lung epithelial cells. Chem Res Toxicol 23, 373-378*
Zhu 2009: Renal dysfunction induced by long-term exposure to DU in rats		1 or 7 days, 3, 6 or 12 months	Kidney damage	Implanted	*Zhu, G., Xiang, X., Chen, X., Wang, L., Hu, H., and Weng, S. (2009). Renal dysfunction induced by long-term exposure to depleted uranium in rats. Arch Toxicol 83, 37-46*

Descriptions of studies are included here only for the purposes of brief comparison, and may have been simplified and summarised for brevity. This information should not be taken as a full summary & the reader is directed to the studies themselves for a proper overview. In most cases doses are listed only for studies involving inhalation or ingestion.