

The Hazard Posed by Depleted Uranium Munitions

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This paper assesses the radiological and chemical hazards resulting from the use of depleted uranium (DU) munitions. Due to the low radioactivity of DU, radiological hazards to individuals would become significant in comparison to natural background radiation doses only in cases of prolonged contact—for example, when shards of a DU penetrator remain embedded in a soldier's body. Although the radiation doses to virtually all civilians would be very low, the cumulative "population dose" resulting from the dispersal of hundreds of tons of DU, as occurred during the Gulf War, could result in up to ten cancer deaths. It is highly unlikely that exposures of persons downwind from the use of DU munitions or consuming food or water contaminated by DU dust would reach the estimated threshold for chemical heavy-metal effects. The exposures of soldiers in vehicles struck by DU munitions could be much higher, however, and persons who subsequently enter such vehicles without adequate respiratory protection could potentially be at risk. Soldiers should be trained to avoid unnecessary exposure to DU, and vehicles struck by DU munitions should be made inaccessible to curious civilians.

INTRODUCTION

A number of people have suggested that the use of depleted uranium (DU) munitions by the United States during the 1991 Persian Gulf War might be a cause of health problems reported by returning U.S. soldiers and among the population of southern Iraq. More recently, attention has been focused on this issue as a result of the use of DU munitions by NATO aircraft in Yugoslavia. A series of reports and articles have been published which claim that the use of DU munitions has had serious negative effects on the health of soldiers, surrounding populations, and the environment.¹

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Based on such assessments, former U.S. Attorney General Ramsey Clark and former Soviet president Mikhail Gorbachev have advocated a ban on the use of DU in weapons.² In contrast, the U.S. Department of Defense and various government contractors have published studies that conclude that the health impacts of DU are not serious.³ We have used these and other references and our own calculations to try to put the DU controversy into perspective.⁴ In order to make our results more transparent, we also include an Appendix where the most important radiation doses are estimated using a “back-of-the-envelope” approach.

Our tentative conclusion is that concerns about the public health and environmental effects of DU are overblown. The risks appear to be very low to surrounding populations and to persons who were not in direct, unprotected contact with vehicles struck with DU munitions or areas heavily contaminated by burning DU munitions. DU contamination is unlikely to have any measurable effect on public health in Iraq or Yugoslavia.

It is more difficult to assess the risks to soldiers in vehicles struck by munitions, personnel involved in rescue, repair, and cleanup operations, and to people hunting for souvenirs in struck vehicles. Based on anecdotal information, it appears that the training and equipment provided to cleanup crews was inadequate and some of them may have inhaled significant amounts of DU. Unfortunately, despite U.S. Army regulations, no timely measurements of actual body burdens appear to have been carried out so we may never know how many people were heavily exposed.

DEPLETED URANIUM

Natural uranium, which constitutes about 3 parts per million of average crustal rock by weight, consists of 99.3 percent U-238, 0.7 percent U-235 and 0.0054 percent U-234 (a radioactive decay product of U-238). Depleted uranium differs from natural uranium by virtue of having most of its U-235 and U-234 removed to make enriched uranium for nuclear fuel or weapons, but its chemical and biological behavior is virtually identical to that of natural uranium in the same chemical form. Table 1 gives the isotopic composition of the DU used in U.S. munitions.

Table 1: Isotopes in natural and depleted uranium.

Isotope	Half-life ^a <i>yr</i>	Specific Activity ^b <i>Ci/g</i>	Concentration ^c <i>weight %</i>	
			Natural U -----	Depleted U
U-234	2.46 10 ⁵	6.22 10 ⁻³	0.0054	0.0007
U-235	7.04 10 ⁸	2.16 10 ⁻⁶	0.711	0.2
U-236	2.34 10 ⁷	6.47 10 ⁻⁵		0.003
U-238	4.47 10 ⁹	3.36 10 ⁻⁷	99.28	99.8
Natural U		6.85 10 ⁻⁷		
Depleted U		3.85 10 ⁻⁷		

a. Edgardo Browne, and Richard B. Firestone. Table of Radioactive Isotopes. (New York: John Wiley, 1986).

b. Specific activity (Ci/g) = $3.57 \cdot 10^5 / A t_{1/2}$, where A is the atomic mass and $t_{1/2}$ is the half-life in years.

c. The U-235 concentration can vary between 0.2 and 0.3 percent; the DU used by DoD contains about 0.2 percent U-235 and a trace amount of U-236 (from reprocessed uranium). Health and Environmental Consequences of Depleted Uranium Use in the U.S. Army: Technical Report . Atlanta: U.S. Army Environmental Policy Institute (June, 1995). Available at: <http://aepi.gatech.edu/DU/chapter2.html>. For DU containing 0.2 percent U-235, the U-234 concentration is 6.4 to 7.2 ppmw, depending on the concentration of U-235 in the enriched product. Steve Fetter, "Nuclear Archaeology: Verifying Declarations of Fissile-material Production," *Science & Global Security* 3: 3-4 (1993), 257.

The principal U.S. munitions containing DU are the 105- and 120-mm tank-fired rounds, which contain about 4 and 5 kilograms of DU respectively, and the 30-mm rounds fired by the Gatling gun mounted on the A-10 aircraft, which contain about 0.3 kilograms of DU each. The U.S. Army used about 4,000 large-caliber DU rounds in combat during Desert Storm, mostly "in the desert, many miles from the nearest village, on battlefields several hundred square miles in size."⁵ Figure 1 shows a schematic diagram of the 120-mm round. In addition to the rounds used in combat, about 7,000 were used in

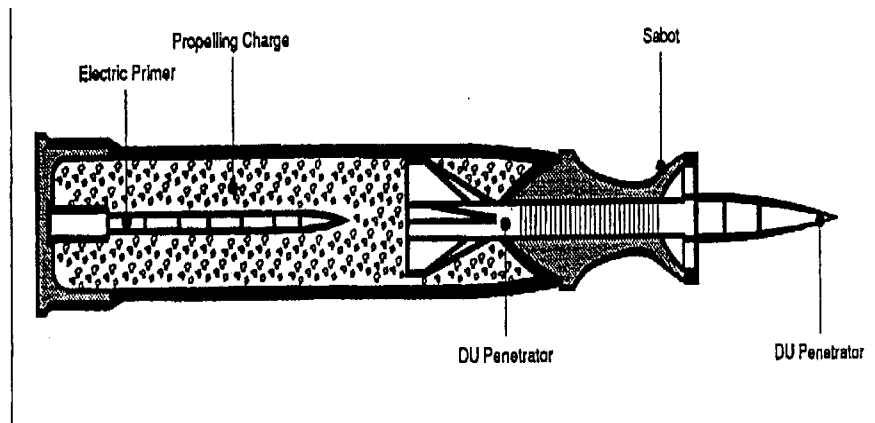


Figure 1: Schematic diagram of 120-mm DU penetrator. Source: *Operation Desert Storm: Army Not Adequately Prepared To Deal With Depleted Uranium Contamination*, U.S. General Accounting Office Report, GAO-NSIAD-93-90 (1993), p. 13.

practice and 3,000 were destroyed—many in a fire at Camp Dohoa, Kuwait.⁶ The U.S. Air Force reportedly fired about 800,000 of the 30-mm DU rounds during Desert Storm. Use of DU munitions by the U.S. Marines, Navy and British forces was relatively minor. In all, about 300 tons of DU was fired in Desert Storm. This is comparable to the amount of natural uranium that was released into the atmosphere from the U.S. Government's Feed Materials Production Center (FMPC) near Fernald, Ohio, between 1953 and 1977.⁷

DU is weakly radioactive. It decays very slowly, with a half-life of 4.5 billion years. Because of the resulting emission of ionizing radiation, internal or

external exposure will result in a radiation dose. It is highly unlikely that exposure to DU would result in doses high enough to produce any short-term radiation effects, but lower doses may result in an increased chance of cancer.⁸ In addition, internal exposure to uranium at sufficiently high levels can result in toxic chemical effects, similar to the effects of nonradioactive heavy metals such as lead.

The main source of external exposure would be the handling of DU munitions or fragments and inhabiting or traversing areas contaminated with DU. Inhalation of fine DU aerosols generated by fires or the impact of munitions would be the source of most internal exposure. Below we estimate and evaluate the risks to individuals and populations from external and internal exposure to DU.

EXTERNAL EXPOSURE

Uranium isotopes and their radioactive decay products emit alpha, beta, and gamma rays. Alpha particles (helium nuclei) cannot penetrate a piece of paper or the inert outer layer of the skin; they are hazardous only if uranium is inhaled or ingested. Beta particles (electrons) have somewhat longer ranges, but they are hazardous only if bare uranium is in direct contact with the skin. Gamma rays (photons) are far more penetrating. The most prominent gamma-ray emission from DU, the 1-MeV photon emitted during the decay of Pa-234m (a radioactive daughter of U-238), has a mean attenuation length of 16 centimeters in water or 1.5 centimeters in lead.

Individual Exposure

The theoretical maximum whole-body gamma-ray dose-rate from external exposure to DU is 2.5 millirem per hour.⁹ Dose rates in this range might be experienced by a person surrounded by DU munitions. The dose rate is considerably smaller in more common situations. According to the U.S. Army, the whole-body dose rate in or near a tank fully loaded with DU munitions is less than 0.2 millirem per hour.¹⁰ To put this in perspective, the average equivalent whole-body dose rate from natural background radiation in the United States is about 300 millirem per year.¹¹ Thus, driving a fully loaded tank for 2000 hours would result in a dose roughly equal to the annual dose from natural background radiation.

As might be expected, the dose rate from DU deposited on the ground is considerably smaller. The dose rate to a person standing on flat ground uni-

formly contaminated with 1 ton of DU per square kilometer (a reasonable upper limit for battlefield areas) would be about 1 millirem per *year*.¹² For comparison, the dose rate from natural uranium in soil is about 10 millirem per *year*.¹³ The U.S. regulatory limit for public exposure to anthropogenic sources of ionizing radiation is 100 millirem per year.¹⁴ Even in the area immediately surrounding a vehicle destroyed by DU munitions, the dose rate from external radiation is unlikely to exceed 30 millirem per year — one tenth the natural background dose rate.¹⁵

Contact of bare DU with bare skin, as might occur when handling pieces of DU penetrators after an impact or fire, would result in a much higher dose rate—about 230 millirem per hour to the skin, mostly from beta particles.¹⁶ Skin is relatively insensitive to radiation; even continuous direct contact is unlikely to produce radiation burns or any other short-term health effects.¹⁷ If, however, a person kept a piece of DU in his or her pocket or wore a necklace or bracelet made of DU, there would be an increased risk of skin cancer.¹⁸ DU fragments should therefore be cleaned up and disposed of safely to avoid radiation exposures from “souvenirs.”

Population Exposure

Although external doses are likely to be well below established exposure limits, low doses might still be worrisome if large numbers of people are exposed. For purposes of protecting public health, there is a presumption that any radiation dose, no matter how small, is associated with a proportionate increase in the probability of cancer death—a “linear, no-threshold” dose-response relationship. It is important to note, however, that the carcinogenic effect of radiation, which has been observed at high doses (above 10 rem), usually is statistically undetectable at low doses due to the incidence of cancer from other causes. For example, there is no epidemiological evidence that exposure to natural or depleted uranium, which are weakly radioactive, is associated with an increased incidence of cancer.¹⁹ Although we believe that the linear model is a conservative estimate of the risk from low doses of ionizing radiation in a genetically heterogeneous population, others believe that it may overestimate the risk.

An important prediction of a linear, no-threshold dose-response relationship is that the number of cancer deaths does not depend on how the dose is distributed among the population.²⁰ A dose of 10 rem to each of 1,000 people would result in the same number of cancer deaths as a dose of 1 rem to each of 10,000 people or 0.1 rem to each of 100,000 people. The number of cancer deaths is proportional to the “population dose,” or the sum of the doses for all

exposed persons. In the above examples the population dose is 10,000 person-rem. The risk coefficient, estimated from data at high doses, is about one cancer death per 2,000 person-rem for doses delivered over an extended period of time.²¹ (The dose in this case is whole-body dose or the “effective dose equivalent” (EDE). The EDE is the weighted average of the dose to various organs, with the weights determined by the relative probability that a fatal cancer will occur in that organ after a uniform whole-body dose.) Using this coefficient, a population dose of 10,000 person-rem would result, on average, in five additional cancer deaths.

The population dose rate from external exposure to contaminated soil, D_e (person-rem/yr) is given by

$$D_e = C_e M \quad (1)$$

where C_e is the EDE dose-rate conversion factor (1.3 mrem/yr per ton of DU per square kilometer),²² M is the total amount of DU dispersed (metric tons), and ρ is the population density (per square kilometer). Average population densities are about 10/km² in Saudi Arabia, 50/km² in Iraq, and 100/km² in Kuwait and Yugoslavia. We will use 50/km² as a conservative (i.e., high) value for the area contaminated by DU during the Persian Gulf War.

Equation (1) ignores shielding by terrain or structures and the effects of weathering, tilling, or decontamination. Actual population dose-rates will be considerably smaller, particularly after several years.²³ Equation (1) also assumes that contamination is uncorrelated with population density—in other words, that the level of DU contamination is the same in urban and rural areas. If DU contamination is heaviest in remote areas, away from cities and villages, the population dose and the expected number of cancers would be reduced. Taking these factors into account, the total population dose over the 50-year period following the initial contamination might be roughly 10 times the first-year dose given by equation (1), give or take a factor of two.

If 300 tons of DU was dispersed over an area with an average population density of 50 per square kilometer, the population dose rate would be about 20 person-rem per year. Over a 50-year period, the total population dose would be roughly 200 person-rem. According to the linear hypothesis, this dose would result in 0.1 cancer deaths—in other words, a ten percent chance that one person would die of cancer.

Thus, we conclude that the health risks to individuals or populations from external exposure to DU are small, with the possible exception of scavengers

who have direct, prolonged contact with bare DU fragments.

INTERNAL EXPOSURE

Internal exposure to DU is more hazardous than external exposure. Internal exposure can occur from inhalation of fine aerosols, ingestion of dust, or from fragments embedded in the body. Fragments, dust, and aerosols are generated when munitions strike hard targets, such as armored vehicles. In the case of some U.S. tanks, which contain DU armor, the armor can contribute to the generation of fragments and aerosols. Aerosols and dust also are generated during fires involving DU munitions or DU-armored vehicles.

Tank-fired DU rounds have a muzzle velocity of 1,500 meters per second; at this velocity, the kinetic energy of a 5-kilogram DU penetrator is equivalent to 3 pounds (1.4 kg) of TNT.²⁴ The kinetic energy of a 30-mm Gatling-gun penetrator is equivalent to about 0.1 pounds of TNT.²⁵ When these penetrators strike a hard target, such as a tank, a large fraction of their kinetic energy is converted into heat in less than a millisecond.²⁶ This rapid release of energy can convert much of the DU into small, hot fragments and particles. The smaller fragments can burn, generating DU-oxide aerosol. When a penetrator strikes a soft target, such as a personnel carrier, truck, or soil, much less aerosol is generated and much of the metal DU penetrator may remain intact.

The U.S. government has sponsored a number of tests to determine the amount, size, and solubility of DU aerosols generated by impacts and fires. In five impact tests involving 25-, 105-, and 120-mm DU rounds with complete, partial, and no penetration of hard armored targets, the fraction of the DU converted to aerosol ranged from 3 to 70 percent; 1 to 96 percent of the aerosol mass was respirable (i.e., particle diameters less than 10 microns); and 17 to 43 percent of the respirable aerosol was in soluble chemical forms.²⁷ Two of these tests apparently suffered from flaws that caused the fraction aerosolized to be significantly under- or over-estimated. All things considered, a reasonably conservative estimate of the fraction of the DU mass converted into respirable aerosol in a hard-target impact is 20 percent. Assuming that no more than half of the rounds strike hard targets, roughly 10 percent of the DU in rounds fired during a military engagement would be converted to respirable aerosol.

In general, fires convert a much smaller fraction of the DU into aerosol than do hard-target impacts, and the fraction of soluble aerosol is lower. In three fire tests involving multiple munitions, 10 to 35 percent of the DU mass was converted into oxide; 0.07 to 0.6 percent of the oxide mass was in the form

of aerosols; 0.007 to 0.07 percent of the oxide mass was in respirable aerosol (particles with diameters less than 10 microns); and 3 to 7 percent of the aerosol was soluble.²⁸ Thus, in a fire the estimated fraction of the total DU mass released as respirable aerosol is less than 0.05 percent.

Health Effects of Internal Exposure

The health risks posed by DU aerosols depend on their size and their solubility in body fluids. Aerosol size determines the fraction of inhaled DU that is deposited in nasal passages, bronchial tubes, and the lung. Solubility determines the rate at which inhaled or ingested DU is absorbed into the bloodstream. Fine, insoluble aerosols result in higher radiation doses, because they are deposited primarily in the lung, where they remain for several years. Soluble aerosols pose greater risks of chemical toxicity because they are absorbed into the bloodstream quickly. Uranium in the blood concentrates in the kidneys and bone. We have used the metabolic model of the International Council on Radiological Protection (ICRP) to calculate the amounts of uranium in various organs as a function of time after inhalation or ingestion, and to estimate the resulting radiation doses.²⁹

Radiation Effects

As discussed above, the primary risk from low doses of radiation is an increased probability of cancer. In the United States, workers are limited to a whole-body dose or EDE of 5 rem per year. In addition, the maximum dose to any organ is limited to 50 rem per year. According to the linear model, a whole-body dose of 5 rem brings with it a 0.25 percent chance of developing a fatal cancer.³⁰

Table 2 gives the 50-year EDE and the 50-year dose commitment to the most exposed organ per gram of inhaled or ingested DU aerosol, as a function of aerosol size and solubility. A person could inhale 3 grams of soluble DU aerosol or 0.05 to 0.3 grams of insoluble aerosol (depending on aerosol size) without exceeding the occupational exposure limits. Similarly, a person could ingest 30 grams of soluble aerosol or 600 grams (more than one pound!) of insoluble aerosol without exceeding the occupational limit. It is extremely unlikely a person could inhale or ingest enough DU aerosol to receive a sufficiently high radiation dose to cause any near-term health effects.³¹

Table 2: 50-year dose commitment, rem per gram of DU inhaled or ingested.^a

	Aerosol Size ^b (μm)	Soluble ^c		Insoluble ^d	
		Skeleton	EDE	Lung	EDE
Inhaled	0.2	18	1.2	770	92
	1	14	0.96	380	46
	5	16	1.1	130	16
Ingested		Skeleton	EDE	Skeleton	EDE
		1.5	0.098	0.058	0.0087

a. Calculated using the model for uranium described in International Commission on Radiological Protection, "Limits for Intakes of Radionuclides by Workers," *ICRP Publication 30, Part 1* (Oxford: Pergamon Press, 1978).

b. Activity median aerodynamic diameter (AMAD).

c. Lung clearance class of "days."

d. Lung clearance class of "years."

Chemical Toxicity

Like many heavy metals, uranium is toxic. The kidney is generally considered the most sensitive organ because the acid environment in the tubules where urine is collected frees the uranium to attack the cells on the tubule surfaces. Based on animal studies, significant cell death is believed to occur above a concentration of 3 parts per million uranium by weight (ppm) in kidney tissue.³² For an average adult male with a kidney mass of 310 grams, such a concentration corresponds to a total of about 1 milligram of uranium in the kidneys.

The U.S. Occupational Health and Safety Administration (OSHA) has set the following "permissible exposure levels" for uranium aerosols: 0.05 milligrams per cubic meter (mg/m^3) for soluble compounds (the same as for lead aerosols) and $0.25 \text{ mg}/\text{m}^3$ for insoluble compounds. These limits are based on

continuous occupational exposure (40 hours per week, 50 weeks per year). Continuous occupational exposure at the OSHA limits would result in a steady-state uranium concentration of about 1 ppm in the kidney. A one-time exposure that results in a uranium concentration of 1 ppm or less in the kidney is therefore unlikely to cause permanent damage.³³

Table 3 gives the maximum amount of uranium in the kidney following the inhalation or ingestion of one gram of uranium aerosol. A uranium concentration of 1 ppm in the kidney would result after inhaling 5 to 6 milligrams of soluble uranium aerosol or 300 to 1400 milligrams of insoluble aerosol. For comparison, the Health Physics Society estimates that the thresholds for transient and permanent renal damage are 8 and 40 milligrams of inhaled soluble uranium aerosol.³⁴ Also note that a uranium concentration of 1 ppm in the kidney would result after ingestion of 60 milligrams of soluble aerosol or 1400 milligrams of insoluble aerosol.

Table 3: Maximum amount of uranium in the kidney (milligrams) following inhalation or ingestion of one gram of uranium aerosol.^a

	AMAD ^b (μm)	Soluble ^c	Insoluble ^d
Inhaled	0.2	58	0.22
	1	48	0.49
	5	61	1.1
Ingested		5.4	0.22

- a. Calculated using the model for uranium described in International Commission on Radiological Protection, "Limits for Intakes of Radionuclides by Workers," ICRP Publication 30, Part 1 (Oxford: Pergamon Press, 1978).
- b. Activity median aerodynamic diameter (AMAD).
- c. Lung clearance class of "days."
- d. Lung clearance class of "years."

Toxic effects at levels of exposure to uranium lower than those required to cause kidney damage have not been reported. However, there has been much less study of low-dose effects of uranium than for lead. In the case of lead, sig-

nificant biochemical and neurological effects have been found at blood levels five to ten times smaller than those which cause kidney damage. Thus, we cannot rule out the possibility that significantly lower uranium doses might have adverse, but as yet unrecognized, health effects.

Table 4 gives the maximum amount of DU aerosol that could be inhaled or ingested in a single event as a function of aerosol size and the percentage of the uranium that is in soluble forms, based on occupational standards for both radiation and toxicity. Exposures below these levels would not result in radiation doses or uranium concentrations in the kidney greater than those permitted by current U.S. occupational standards. For the aerosols produced by fires, for which 3 to 7 percent of the uranium is in soluble form, the limit is 56 to 110 milligrams, depending on aerosol size; for aerosols produced by impacts (17 to 43 percent soluble), the limit is 12 to 36 milligrams.

Table 4: Maximum amount of DU that could be inhaled or ingested (milligrams) without exceeding U.S. occupational toxicity or radiation standards.

Percent in Soluble Forms ^a	Inhalation			Ingestion
	0.2 μm	1 μm	5 μm	
0	54 ^b	110 ^b	290	1400
5	57 ^b	110	76	650
10	51	60	44	420
15	35	41	31	310
25	21	25	19	200
50	11	13	10	110

a. Lung clearance class of "days."

b. Limit set by radiation exposure (5 rem/yr EDE, 50 rem/yr all other organs). Other limits set by chemical toxicity (0.1 $\mu g/g$ in kidney).

Individual Exposure

Estimating exposures to individuals from airborne DU aerosols is difficult because the concentration is highly sensitive to details about the nature of the

release, weather conditions, and other factors. Here we use a combination of rough estimates and test data to estimate the dose that individuals might receive under various circumstances.

Outside Struck Vehicles

First consider the case in which a single DU penetrator strikes a hard target. As noted above, 20 percent of the penetrator mass could be converted into a respirable aerosol during a hard impact. Initially, this aerosol would be distributed throughout the cloud generated by the impact. Since the kinetic energy of the penetrator is converted into heat very rapidly (in less than a millisecond), it is reasonable to assume that the cloud would have roughly the same characteristics as one formed by the detonation of an equivalent amount of high explosive.

Church gives the following empirical relationships for the height, H (meters), and radius, R (meters), of clouds formed by high-explosive detonations.³⁵

$$H = 76W^{0.25} \quad R = 35W^{0.375} \quad (2)$$

where W is the explosive yield in pounds of TNT equivalent. Experiments have shown that about 5 percent of aerosol is initially found between the ground and a height of $H/4$.³⁶ Thus, the average concentration of aerosol near the ground, (milligrams per cubic meter), is roughly

$$\frac{0.05 f_i M}{R^2 [H/4]} = \frac{68 f_i M}{W} \quad (3)$$

where M is the mass of the penetrator (kilograms) and f_i is the fraction converted to respirable aerosol.

Now consider a person standing in the open, directly downwind from the center of the cloud. This person would be immersed in the cloud for a time $t = 2R/u$ where u is the wind speed (meters per second). The total amount of aerosol inhaled, I (milligrams), during this time would be

$$I = tb = \frac{2R}{u} b = \frac{480 f_i M b}{u W^{0.625}} \quad (4)$$

where b is the breathing rate (m^3/s). Thus, if $f_i M = 1$ kilogram, $u = 1$ m/s, and $b = 3.3 \cdot 10^{-4}$ m/s, then $I = 0.16 W^{0.625}$. Higher values of W result in lower

inhaled doses because the energy release disperses and dilutes the aerosol.

As noted above, the kinetic energy of a 120-mm DU penetrator is equivalent to 3 pounds of TNT. If all the kinetic energy is lost on impact, the maximum amount inhaled would be 0.08 milligrams ($u = 1$ m/s). If only 10 percent of the kinetic energy is lost on impact (but the fraction of the penetrator mass aerosolized remains 20 percent), the maximum amount inhaled would be 0.3 milligrams. A similar calculation for a 30-mm DU round, assuming that 25 percent of the kinetic energy is lost on impact ($W = 0.03$ pound TNT), yields a maximum inhaled dose of 0.08 milligrams. As noted above, a person could inhale 12 to 36 milligrams of DU aerosol (of which 17 to 43 percent is in soluble forms) without exceeding 1 ppm of uranium in the kidney. An individual in the open air would have to be exposed at very close range to the aerosol clouds generated by the impact of 40 to 500 such penetrators in order to inhale this much DU aerosol—an unlikely scenario.

More plausible is a situation in which individuals are exposed at greater distances to aerosol clouds generated by the impacts of hundreds of tank-fired rounds or thousands of Gatling-gun rounds. Standard Gaussian plume models can be used to estimate the amount of aerosol that would be inhaled by a person downwind from an aerosol release under a variety of conditions. Table 5 gives the results of one such model for a person standing in the open directly downwind from a release of 1 kilogram of respirable aerosol.³⁷ Such a release might result from the impact of one tank-fired round or the impacts of roughly 20 Gatling-gun rounds on a hard target. At a distance of one kilometer, the inhaled dose is only 2 to 26 *micrograms*—500 to 20,000 times less than the 12 to 36 milligram limit derived above. At a distance of ten kilometers doses are ten times smaller.

In Desert Storm, a total of 4,000 tank-fired rounds and 800,000 Gatling-gun rounds were used over a wide area. An individual that somehow managed to be 10 kilometers *directly downwind* from *every one* of these impacts under *worst-case* weather conditions would have inhaled less than 15 milligrams of DU. Thus, it is virtually impossible that any U.S. soldier outside of a struck vehicle could have inhaled a dangerous amount of DU aerosol from penetrator impacts. It seems unlikely that even Iraqi soldiers on the “highway of death” between Kuwait City and Basra, other than those in vehicles struck by DU munitions, could have received doses in excess of U.S. occupational radiation or toxicity standards.

Table 5: DU inhaled (micrograms) by individual directly downwind from release of 1 kilogram of respirable DU aerosol generated by impact of one 120-mm penetrator ($W = 0.3$ to 3 lb) or 17 30-mm penetrators ($W = 0.03$ lb).^a

Distance (km)	Worst-case Weather ^a			Typical Weather ^b		
	$W=0.03$ lb	$W=0.3$ lb	$W=3$ lb	$W=0.03$ lb	$W=0.3$ lb	$W=3$ lb
0.1	200	59	18	54	22	8.4
0.2	120	42	13	32	15	5.9
0.5	54	22	7.4	11	6.9	3.4
1	26	12	4.5	4.3	3.2	1.9
2	8.8	5.4	2.3	1.5	1.3	0.93
5	1.4	1.3	0.74	0.40	0.36	0.30
10	0.32	0.37	0.30	0.15	0.14	0.13

- a. Source: HOTSPOT 98, assuming standard terrain, 0.01 m/s deposition velocity, 10-min sampling time, 3.3×10^{-4} m/s breathing rate.
- b. Class "F" stability, 1 m/s wind speed, 250 meter mixing height.
- c. Class "D" stability, 5 m/s wind speed, 1,000 meter mixing height.

In contrast to impacts, which are widely distributed, fires could expose nearby individuals to aerosols from hundreds of burning munitions. For example, on 11 July 1991, some 660 120-mm rounds were involved in a fire at Camp Doha, 17 kilometers west of Kuwait City. This event probably represents a near-worst-case fire scenario. Nevertheless, many of the rounds survived the fire without exploding or burning, and many of the DU penetrators in rounds that did explode or burn were found intact or nearly intact.

In the test fires reviewed above, less than 0.025 percent of the DU mass was converted into a respirable aerosol, of which only 3 to 7 percent was in soluble forms. Applying this release fraction to the 660 rounds damaged in the Doha fire results in a release of less than 1 kilogram of respirable aerosol—less than that from the hard impact of one penetrator.

Table 6 gives the amount inhaled by an individual standing directly down-

wind from a fire that releases 1 kilogram of respirable aerosol. Although the size and height of the initial cloud and the time over which the release occurs are very different from an explosive release of aerosol, the resulting doses are roughly the same. Even if the amount released were ten times greater, the amount inhaled would be orders of magnitude below the limits for occupational exposure.³⁸

Table 6: DU inhaled (micrograms) by individual directly downwind from release of 1 kilogram of respirable aerosol generated by a fire.^a

Distance (km)	Worst-case Weather ^b		Typical Weather ^c	
	Smoldering ^d	Hot Fire ^e	Smoldering ^d	Hot Fire ^e
0.1	25	54	11	14
0.2	16	44	7.9	10
0.5	6.9	25	3.8	4.9
1	3.1	13	1.8	2.4
2	1.1	4.6	0.79	0.98
5	0.18	0.79	0.23	0.30
10	0.04	0.16	0.09	0.12

- a. Source: HOTSPOT 98, assuming standard terrain, 0.01 m/s deposition velocity, $3.3 \cdot 10^{-4}$ m/s breathing rate, 50-m release radius.
 b. Class "F" stability, 1 m/s wind speed, 250 meter mixing height.
 c. Class "D" stability, 5 m/s wind speed, 1,000 meter mixing height.
 d. Heat release rate of 10^4 cal/s for 5 hours.
 e. Heat release rate of 10^6 cal/s for 1 hour.

Inside Struck Vehicles

Much higher doses are possible inside a struck vehicle because the wind does not quickly dissipate the aerosol. Measurements taken inside an M1A1 tank after it was struck by a single 120-mm DU penetrator corresponded to average and maximum 15-minute intakes of 12 and 26 milligrams.³⁹ Estimates derived from the concentration of uranium in the urine of 14 soldiers

that were in struck vehicles, but who do not have retained shrapnel, are consistent with inhalation of up to roughly 25 milligrams of DU.⁴⁰ Taking into account various uncertainties and the possibility of multiple penetrator strikes, it is possible that individuals inside struck vehicles could inhale 50 or more milligrams of DU aerosol. According to tables 2 and 3, inhalation of 50 milligrams of aerosol, of which 17 to 43 percent is in soluble form, would result in a 50-year EDE of up to 4 rem and a uranium concentration of up to 4 ppm in the kidney. The radiation dose would not be a major cause for worry, but the possibility of kidney damage or other toxic effects would be.

In addition to inhaled aerosols, individuals in struck vehicles may also retain fragments of uranium in their bodies. Such fragments deliver a high radiation dose to a relatively small volume of surrounding tissue. As the fragments dissolve gradually in body fluids, uranium also is transported to other organs and excreted in the urine. Urinary excretion rates of up to 70 micrograms of uranium per day three to six years after exposure have been reported for veterans with retained shrapnel.⁴¹ Excretion rates this high can only be attributed to the slow dissolution of fragments, not to inhaled aerosols.⁴² Excretion of 70 micrograms per day would imply a steady-state concentration in the kidney of about 0.5 ppm, or six times lower than the 3 ppm threshold for kidney damage.⁴³ The effective dose equivalent from uranium entering the blood at a rate of 70 micrograms per day is less than 0.05 rem per year, and the total dose over 50 years would be less 2 rem.⁴⁴

The radiation dose from the fragments themselves is less straightforward to estimate. If the fragments are in the form of numerous fine slivers of uranium metal 0.1 millimeter in diameter, the 50-year dose commitment to the muscle would be on the order of 100 rem per gram of retained shrapnel, resulting in an effective dose equivalent of about 6 rem per gram of shrapnel.⁴⁵ If, on the other hand, the fragments were 1 millimeter in diameter, the doses would be ten times smaller per gram of DU. In either case, the dose from fragments would be delivered to a very small volume of tissue, and associated risk is likely to be less than that of an equal, uniformly-distributed dose.⁴⁶

Aside from the risks of DU exposure, the probability of death or serious injury is very high in vehicles struck by anti-tank weapons. Of 113 soldiers in U.S. vehicles struck by DU penetrators in the Gulf War, 13 were killed and 50 were wounded—a casualty rate of over 50 percent.⁴⁷ The risks associated with DU exposure are very small in comparison.

A final category of exposure are people who enter vehicles after they have been struck, either to rescue comrades, remove munitions or equipment, or to clean or repair damaged vehicles. If contaminated vehicles are entered with protective clothing and masks, there will be no internal dose and the external

dose will be very small. It may be necessary to enter the vehicle immediately, without protective clothing, in rescue operations, but it seems highly unlikely that rescuers would inhale more DU than those in the vehicle when it was struck.

Unfortunately, during the Gulf War it appears that large numbers of soldiers went inside destroyed enemy vehicles to hunt for souvenirs, decontaminate U.S. vehicles hit by DU munitions, or clean up areas contaminated by burning DU munitions without the benefit of effective protective equipment. Analysis of the amount of uranium in urine shortly after exposure would have been the best way to assess the magnitudes of these exposures. No such tests were made for almost two years after exposure; by then, the concentrations had returned to near background levels.

The dose from clean-up operations is very difficult to estimate. Consider, as a hypothetical example, a vehicle interior contaminated with 100 grams of DU aerosol—2 percent of the mass of a 120-mm DU penetrator and 10 percent of the total aerosol. During clean-up operations, perhaps 1 percent of the aerosol could be resuspended at any given time. If the interior volume is 10 cubic meters, the concentration of aerosol would be 100 milligrams per cubic meter. A man doing moderate work, breathing 1.5 cubic meters of air per hour, would inhale 150 milligrams of DU per hour. Assuming that 17 to 43 percent of the aerosol is soluble, the threshold for permanent kidney damage (40 milligrams of soluble uranium aerosol) would be exceeded in 40 to 90 minutes. Although actual concentrations of resuspended aerosol might be ten or more times lower, total exposure times could be ten or more times higher. This emphasizes the importance of proper education, use of protective equipment, and preventing unprotected individuals from entering contaminated vehicles or areas.

Population Exposure

The aerosols generated by impacts and fires could be carried by the wind for distances up to a hundred kilometers or more before depositing on the ground, vegetation, and other surfaces. The surrounding population can become exposed by inhaling aerosols in the passing cloud, inhaling resuspended aerosols, or by ingesting DU in contaminated food or water.

Inhalation

The concentration of DU aerosols at a particular point would depend on many variables, including atmospheric stability, wind velocity, precipitation, terrain, and the size and height of the initial aerosol cloud. Atmospheric dispersion models of varying complexity have been developed to predict the concentra-

tions downwind from such a release. In situations like the present one, however, a much better feel for the estimates can be obtained by using an extremely simple atmospheric dispersion model, the “wedge model.”⁴⁸ The simplicity of the results obtained with this model stem from the fact that, if cancer risk is linearly proportional to dose, the total number cancers will depend only on the total amount of DU inhaled by the population, not on the distribution of the doses within the population. The accuracy of the predictions of the wedge model in such applications is generally comparable to that of more sophisticated models because most of the cancers are due to very small doses at great distances from the release point.

Using the wedge model, the population dose from DU inhaled from passing clouds of aerosol, D_i (person-rem), is⁴⁹

$$D_i = \frac{C_i f_i M b}{v} \quad (5)$$

where C_i is the dose conversion factor for inhaled DU (rem per gram), M is the mass of DU (metric tons), f_i is the fraction released as respirable aerosol, ρ is the average population density (km^{-2}), b is the average breathing rate (cubic meters per second), and v is the average deposition velocity of the DU aerosol (meters per second).⁵⁰ The average breathing rate for an adult performing light activity is $3.3 \times 10^{-4} \text{ m}^3/\text{s}$ ($1.2 \text{ m}^3/\text{hr}$). Observed deposition velocities for aerosols in the absence of precipitation range from 0.001 to 0.1 meters per second, depending on aerosol size and composition, wind speed, terrain and ground cover; a rough average for one-micron aerosols is 0.01 m/s.⁵¹

To return to a previous example, if 10 percent of 300 tons of DU was converted to a one-micron, 25-percent-soluble aerosol ($C_i = 35 \text{ rem/g}$, $v = 0.01 \text{ m/s}$) and dispersed over an area with an average population density of 50 per square kilometer, the population dose from inhalation during plume passage would be roughly 2,000 person-rem—sufficient, according to the linear model, to cause one additional cancer death. We believe that this is a relatively conservative (i.e., high) estimate of the population inhalation dose resulting from the use of DU in Persian Gulf War, but the uncertainties are very large. The fraction released as respirable aerosol might be twice as high or ten times smaller, and the population density in the most contaminated areas might be considerably smaller than the average for the entire region. Much finer and less soluble aerosols ($C_i = 80 \text{ rem/g}$, $v = 0.002 \text{ m/s}$) would result in a population dose up to ten times greater, while coarser and more soluble aerosols ($C_i = 8 \text{ rem/g}$, $v = 0.05 \text{ m/s}$) would reduce the dose by a factor of twenty. All things considered, the actual population dose in this case is likely to be 40 to 20,000

person-rem.

Resuspension

The ground under the plume would be coated with a thin layer of DU dust that would be kicked up by winds or passing traffic to be inhaled again. Over a period of several years, however, the aerosol would settle into the soil or become attached to larger particles. For the resident population, the dose from inhalation of resuspended DU could be of the same order of magnitude as the dose during the plume passage. For travelers passing through, this resuspension dose would be much smaller than the original inhalation dose.

Using the wedge model, the population dose from resuspension, D_r (person-rem), is⁵²

$$D_r = C_i f_i M \int_0^t K(t) dt \quad (6)$$

where the resuspension factor, K (m^{-1}), is the ratio of the concentration of resuspended aerosol in air (g/m^3) to the concentration of aerosol on the ground (g/m^2). The resuspension factor generally decreases with time due to weathering. Observed values for K range over eight orders magnitude, depending on climate (wind, rain, freeze-thaw cycles), mechanical disturbance, ground cover, aerosol size and chemical composition, and other factors.⁵³

The resuspension factor is often expressed as the sum of a short-term factor that decays exponentially and a long-term factor:

$$K(t) = K_0 e^{-t/\tau} + K \quad (7)$$

Reports published during the 1970s gave values for K_0 , K , and τ that, when integrated over 50 years, resulted in K ranging from 60 to 800 s/m.⁵⁴ Based on data from the Chernobyl accident, the National Council on Radiation Protection and Measurements recently recommended the following alternative:

$$K(t) = \frac{10^{-6}}{t} \quad 1 < t < 1000 \quad (8)$$

$$K(t) = 10^{-9} \quad t > 1000$$

where t is measured in days.⁵⁵ Integrated over 50 years, equation (8) gives K

= 2 s/m. If, in the above example involving the release of 30 tons of DU aerosol, we assume that $K = 2$ to 800 s/m, the population dose from resuspension would be 30 to 14,000 person-rem.

Ingestion

In an agricultural area such as Kosovo, the use of DU munitions could deposit a layer of dust on vegetation that could be eaten by humans or by animals in the human food chain. DU also could leach into drinking water. Ingestion is unlikely to be an important contributor to the population dose, however, because weathered DU aerosols are relatively insoluble. (Even the fraction classified as “soluble” in the above discussion would oxidize to insoluble forms in the environment.) Virtually all the population dose from ingestion results from the consumption of produce that is contaminated by direct deposition onto leaf surfaces, either during plume passage or resuspension. The population dose from other ingestion pathways—root uptake by produce, ingestion of milk or meat from animals that ingest contaminated forage, ingestion of contaminated soil, and ingestion of contaminated water—is very small and can be neglected.⁵⁶

If we assume that the produce raised in the contaminated area is sufficient to supply the entire diet of the population living in that area, the population dose from the deposition of DU aerosols onto produce and forage, D_g (person-rem), is given by⁵⁷

$$D_g = C_g f_g M \left[1 + \int K(t) dt \right] \frac{U_p r_p}{Y_p} \tag{9}$$

where C_g is the dose-conversion factor for ingestion of DU (rem/g), f_g is the fraction of the DU converted to an ingestible aerosol, τ is the mean residence time of DU dust on vegetation (years), U_p is the average annual per-capita consumption of produce (kilograms per year), r_p is the fraction of DU dust retained on produce and forage, and Y_p is the agricultural productivity (kilograms of produce per square meter). Typical values are $\tau = 0.06$ yr, $U_p = 200$ kg/yr, $r_p = 0.2$, and $Y_p = 0.7$ kg/m².⁵⁸ Equation (9) assumes that the length of the growing season is greater than τ and that the initial DU contamination is distributed over the growing season.⁵⁹

As shown in table 2, the dose conversion factor depends on the solubility of the DU aerosol. If 25 percent of the aerosol is soluble, $C_g = 0.03$ rem/g. Returning to our Gulf War example, if $f_g = 0.2$, $M = 300$ tons, $\tau = 50/\text{km}^2$, $\tau = 0.01$ m/

s, and $K = 100$ s/m, the population dose from ingestion is roughly 600 person-rem. The uncertainty in this estimate is very large, but it serves to illustrate that, for DU ingestion, is generally less important than inhalation.⁶⁰

ENVIRONMENTAL EFFECTS

Finally, it is sometimes asserted that the use of DU munitions results in serious negative effects on plants and animals. A full investigation of this possibility would require an analysis of the ecology, geology, and hydrology of particular regions where DU might be dispersed, and is beyond the scope of this article.

However, the fact that natural uranium is ubiquitous in soils and variable in concentration makes us doubt that the dispersal of DU will have serious environmental consequences. The average concentration of uranium in U.S. soils is 1.8 parts per million by weight, with a range of 1 to 4 ppm. In some areas of the world, concentrations as high as 10 ppm are found. If we consider only the top ten centimeters—the “mixed layer” of soil—the average concentration in U.S. soils is 0.3 grams per square meter, plus or minus a factor of two.

A total of 300 tons of DU was fired in the Gulf War over an area of several thousand square kilometers—an average concentration on the order of 0.1 grams per square meter. Although concentrations might be considerably higher in some areas, only a fraction of the DU—perhaps 10 percent—is in the form of biologically accessible aerosol or dust. The remainder is in the form of intact, or nearly intact, uranium metal penetrators, most of which are buried in the soil. And although a significant fraction of the aerosols generated by impacts are initially soluble, these are oxidized in the environment to insoluble chemical forms. Moreover, the radiation dose to animals from internal exposure to DU is only about half that of natural uranium (per milligram of uranium inhaled or ingested), and the external radiation dose rate from DU is more than ten times less than natural uranium in the soil (per gram of uranium per square meter of soil).⁶¹ Thus, when averaged over reasonably large areas, the environmental effects of DU are likely to be perturbations similar in magnitude to those resulting from variations in the concentration of natural uranium.

CONCLUSIONS

In summary, individuals who were not in or did not subsequently enter vehicles struck with DU munitions would receive radiation doses low relative to natural background doses, and uranium exposures far below the thresholds at which toxic effects have been observed. The radiation dose to the entire population also is quite small. The total population dose resulting from the use of DU in the Persian Gulf War is on the order of 3,000 person-rem, which would most likely result in one or two cancer deaths. If this dose was distributed among one million people, the average dose would be 3 millirem per person—about one percent of the average annual background dose. Due to numerous uncertainties, the dose could be ten times higher or, more likely, ten times lower. Even at the upper end of this range the risks of DU to the general population are small.

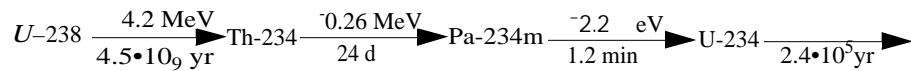
The most exposed soldiers—those in armored vehicles struck by DU munitions, those who entered struck vehicles thereafter, and those involved in decontaminating such vehicles without protective equipment—may have received sufficiently large doses to suffer the known chemically toxic effects associated with uranium. If DU munitions are used again in the future, troops involved in clean-up activities should be appropriately trained and equipped in advance.

Contaminated vehicles and fragments of DU penetrators abandoned on the battlefield represent an “attractive nuisance.” Curious passers-by, both adults and children, will enter the vehicles and thereby be subject to potentially significant levels of uranium exposure from resuspended and ingested aerosols. Fragments of penetrators may be picked up and taken home as souvenirs. The U.S. removed the small number of its armored vehicles that had been struck by “friendly” DU munitions and either decontaminated them or buried them. In the absence of more costly decontamination efforts, we would propose that all DU-contaminated vehicles be filled with concrete and buried and that DU penetrator fragments be picked up and buried as low-level radioactive waste.

APPENDIX

"BACK-OF-THE-ENVELOPE" ESTIMATES OF RADIATION DOSES

Uranium-238 makes up 99.8 percent of DU and the radiation from it and its decay products dominate the radiation doses from DU. The main decay chain of U-238 (with emitted particle type and energy shown above the arrow and half-life below) is



Several months after the production of pure U-238, the decay products Th-234 and Pa-234m, because of their short half-lives, will be in equilibrium (i.e. have the same radioactivity) as U-238. Because of the long half-life of U-234, however, its activity and those of its decay products take more than 100,000 years to build up to the same level. As a result, for thousands of years after its production, the doses from DU will be dominated by the decay of U-238, Th-234, and Pa-234m.

External Gamma-ray Dose

The alpha decay of U-238 does not produce any significant gamma-ray emissions. The beta decay of Th-234 produces low-energy gamma rays; the most important have energies near 0.063 MeV (3.9% of decays), 0.093 MeV (5.5%), and 0.113 MeV (0.28%).⁶² The decay of Pa-234m yields high-energy gamma rays with energies of 0.77 MeV (0.21%) and 1.00 MeV (0.65%).⁶³ Including weaker decays of similar energy, there are approximately 0.1 gamma rays of average energy 0.08 MeV per Th-234 decay and 0.009 gamma rays of average energy 1.0 MeV per Pa-234m decay.

The rate of gamma-ray energy emission from the *i*th isotope in the decay chain of U-238, R_i (J/m²yr), above a flat, infinite plane uniformly contaminated with one metric ton of U-238 per square kilometer (1 gram per square meter), is given by

$$R_i = \frac{(6.0 \cdot 10^{23})(1.6 \cdot 10^{-13})}{A} f_i E_i = 0.063 f_i E_i \quad (\text{A-1})$$

where $6.02 \cdot 10^{23}$ is the number of nuclei per mole, $1.6 \cdot 10^{-13}$ is the number of joules per MeV, A is the atomic weight of U-238 (238 grams per mole) and its mean lifetime $\tau = 4.5 \cdot 10^9 / \ln(2) = 6.5 \cdot 10^9$ years, f_i is the number of

gamma rays emitted per decay of the i^{th} radioisotope in the decay chain, and E_i (MeV) is corresponding average gamma-ray energy.

The rate of energy absorption per kilogram of tissue at a height h above the contaminated plane from gamma rays emitted by the i^{th} radioisotope from a square meter of surface at a distance r , $P_i(r, h)$ ($J/Kg \text{ yr}$), is given by

$$P_i(r, h) = \frac{R_i}{4 \rho_t (r^2 + h^2)} \exp\left[-\frac{\sqrt{r^2 + h^2}}{a_i}\right] \quad (\text{A-2})$$

where ρ_t is mean energy attenuation depth in tissue (about 0.3 meters for 0.08- and 1.0-MeV gamma rays),⁶⁴ ρ_t is the density of tissue (approximately 10^3 kg/m^3) and a_i is the mean energy absorption distance for gamma rays in air. For air at sea level density of 1.2 kg/m^3 , $a_i = 360$ meters for 0.08-MeV gamma rays and 300 meters for 1.0-MeV gamma rays.⁶⁵

Integrating equation (A-2) over the plane gives a dose rate

$$P_i(h) = \int_0^\infty P_i(r, h) dr = \frac{R_i}{2 \rho_t} \ln \frac{a_i}{h} = 1.0 \cdot 10^{-4} f_i E_i \ln \frac{a_i}{h} \quad (\text{A-3})$$

Approximating $h = 1 \text{ m}$ and inserting the average values of f_i , E_i , a_i for Th-234 and Pa-234m gives

$$P = 1.0 \cdot 10^{-4} [(0.1)(0.08) \ln(360) + (0.009)(1.0) \ln(300)] = 1.0 \cdot 10^{-5} \frac{J/kg}{yr} = 1.0 \frac{mrem}{yr}$$

where $1 \text{ mrem} = 10^{-5} \text{ joules/kg}$. For comparison, the more precise value calculated in the main text is 1.3 mrem/yr .

Internal Dose

We have calculated radiation doses from internal exposure using the respiration and internal transport models described in ICRP-30. In the ICRP model, the respiratory system is divided into three regions: nasopharyngeal (NP), tracheobronchial (TB), and pulmonary (P). The fraction of inhaled material initially deposited in these regions is shown in figure A1 as a function of the activity median aerodynamic diameter (AMAD) of the aerosol. For an aerosol with an AMAD of 1 micron, 25 percent of inhaled material is deposited in the pulmonary region.

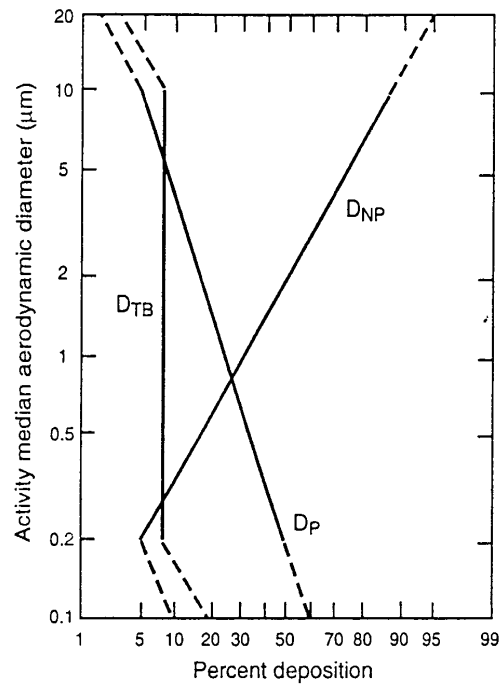


Figure A1: Fraction of inhaled material deposited as a function of the activity median aerodynamic diameter of the aerosol: nasopharyngeal (NP), tracheobronchial (TB), and pulmonary (P).

Some of the material deposited in the pulmonary region is removed from the lung in mucus to the gastro-intestinal tract; the remainder is absorbed into body fluids, either directly or after passing through the pulmonary lymph nodes. Figure A2 summarizes this transport model. Solubility categories are defined according to whether an aerosol will dissolve in bodily fluids in days or less (D), weeks (W), or years (Y). The transfer of material between compartments is specified in terms of the fraction of the aerosol mass in the source compartment and a transfer half-life for removal to another compartment. The uranium oxides, UO_2 and U_3O_8 , are assigned to the most insoluble class

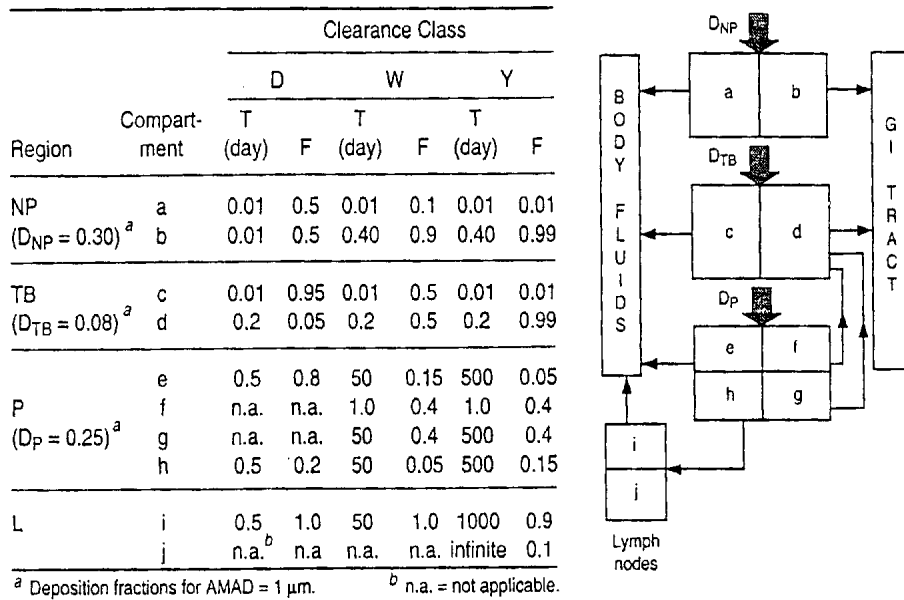


Figure A2: Summary of transport model, showing whether aerosol will dissolve in bodily fluids in days or less (D), weeks (W), or years (Y). The symbols T and F denote the biological half-time (days) and the fractional coefficient, respectively, corresponding to the compartment indicated.

(Y) while UO_3 is assigned to the intermediate class (W).

The most important radiation doses from inhaled insoluble uranium aerosols are to the lung. About 60 percent of insoluble aerosol deposited in the pulmonary region (15 percent of inhaled aerosol mass) is retained there with a mean residence time of $500/\ln(2) = 720$ days = 2.0 years; if we include subsequent transfer and retention in the pulmonary lymph nodes, the total residence time of this material in the lung is 4.2 years.⁶⁶ Most of the energy released by uranium is in the form of very-short-range alpha particles (4.2 MeV per U-238 decay). The carcinogenic effect of the dense ionization along

the alpha-particle track is assumed to be 20 times greater per unit energy deposited than for energy deposited by gamma or beta rays. Taking into account this “quality factor” of 20 Sieverts per J/kg (2000 rem per J/kg), the dose to the lung resulting from the inhalation of one gram of U-238 is given approximately by

$$\frac{0.15 \frac{g}{g_{\text{inhaled}}} (4.2 \text{ yr}) \frac{0.063 \text{ decay}}{g \text{ yr}} \frac{J}{\text{MeV}} \frac{4.2 \text{ MeV}}{\text{decay}} \frac{2000 \text{ rem}}{J/\text{kg}}}{1 \text{ kg}} = 330 \frac{\text{rem}}{g} \quad (\text{A-4})$$

where 0.15 is the fraction of inhaled uranium retained in the lung with a mean residence time of 4.2 years (assuming an insoluble, one-micron aerosol), 0.063 is the number of decays per year per gram of U-238 multiplied by the number of joules per MeV (see equation A-1), and 1 kilogram is the lung mass of an average adult male. For comparison, table 2 gives a lung dose of 380 rem per gram of DU inhaled; the difference is due to the decay of U-234 and U-235 in addition to U-238. The contribution of the lung dose to the effective dose equivalent is derived by multiplying the lung dose by 0.12 (the fraction of additional cancer deaths due to lung cancer if the body were uniformly irradiated).

NOTES AND REFERENCES

1. See, for example, Helen Caldicott, Michio Kaku, Jay Gould, and Ramsey Clark. *Metal of Dishonor: How Depleted Uranium Penetrates Steel, Radiates People and Contaminates the Environment* (New York: International Action Center, 1997); Grace Bukowski, Damacio A. Lopez, and Fielding M. McGehee. “Uranium Battlefields Home & Abroad: Depleted Uranium Use by the U.S. Department of Defense.” (Reno, NV: Citizen Alert, March 1993); Felicity Arbuthnot, et al. “Depleted Uranium: A Post-War Disaster for Environment and Health.” (Amsterdam: Laka Foundation, May 1999); Rob Edwards, “Too Hot to Handle,” *New Scientist* 5 June 1999; and a series of articles by Bill Mesler, in *The Nation*: “The Pentagon’s Radioactive Bullet,” 21 October 1996, “Pentagon Poison: The Great Radioactive Ammo Cover-up,” 26 May 1997, and “The Gulf War’s New Casualties,” 14 July 1997.
2. “Depleted-uranium weapons are an unacceptable threat to life, a violation of international law and an assault on human dignity. To safeguard the future of humanity, we call for an unconditional international ban forbidding research, manufacture, testing, transportation, possession and use of DU for military purposes.” Ramsey Clark, “An International Appeal to Ban the Use of Depleted Uranium Weapons.” Available at: <http://www.iacenter.org/depleted/appeal.htm>. “We should prohibit weapons whose use may have particularly dangerous, long-term environmental and medical consequences. In my view, weapons containing depleted uranium should be among the first to be banned.” Mikhail Gorbachev, “Poison in the air: The environmental costs of the Kosovo

conflict must be exposed." *The Guardian* (London), (June 1999).

3. See, for example, *Environmental Exposure Report: Depleted Uranium in the Gulf*, <http://www.gulfink.osd.mil/du>, July 31, 1998, and the dozens of reports referenced therein. An excellent web site with many relevant government documents not available elsewhere is maintained by Gulf War veteran Chris Kornkven. Available at: <http://www.globaldialog.com/~kornkven>.

4. Reviews of the health effects of uranium also have been published by the U.S. Department of Health and Human Services [*Toxicological Profile for Uranium* (TP-90-29, 1990)] and the National Academy of Sciences [*Health Risks of Radon and Other Internally Deposited Alpha-emitters (BEIR IV)* (Washington: National Academy Press, 1998).]

5. *Health and Environmental Consequences of Depleted Uranium Use in the U.S. Army: Technical Report*. (Atlanta, U.S. Army Environmental Policy Institute, Georgia Institute of Technology, June 1995). Available at: <http://aepi.gatech.edu/DU/chapter4.htm>, p. 79 and A-10. Printed copy available without charge by calling 404-892-3099.

6. *Health and Environmental Consequences of Depleted Uranium Use in the U.S. Army: Technical Report*. (Atlanta, U.S. Army Environmental Policy Institute, Georgia Institute of Technology, June 1995). Available at: <http://aepi.gatech.edu/DU/chapter4.htm>, p. 79 and A-10. Printed copy available without charge by calling 404-892-3099.

7. *Radiation Doses and Risk to Residents from FMPC Operations from 1951-1988* (Radiological Assessments Corporation [RAC] report #1-CDC-Fernald-1998-Final, 417 Till Road, Neeses, SC 29107), Vol. II, Fig. C-6.

8. The genetic damage from ionizing radiation should also cause harmful mutations in eggs and sperm. However, unlike cancers, statistically significant effects have not yet been established in humans. *Health Effects of Exposure to Low Levels of Ionizing Radiation (BEIR V)*, 94-97, Washington, DC: National Academy Press, 1990.

9. The whole-body dose within an infinite slab of radioactive material (mrem/hr) is given by $2.13 \cdot 10^9 \cdot S \cdot \sum_i f_i E_i T_i G_i$, where S is the specific activity ($3.85 \cdot 10^{-7} \text{ Ci/g}$), E_i is the energy of the i^{th} photon emission (MeV), f_i is the intensity of this emission (dis^{-1}), T_i is the ratio of the mass energy-absorption coefficient in tissue to that in uranium, G_i is the fraction of the photon energy absorbed in the whole body, and $2.13 \cdot 10^9$ is the number of disintegrations-gram-millirem per MeV-Curie-hour. Most of the gamma-ray dose is from the 1-MeV emission from Pa-234m, for which $f = 0.0059$, $T = 0.71$, and $G = 0.62$. One rem = 0.01 Sievert.

10. N. Harley, E. Foulkes, L. Hilborne, et al. 1999. *A Review of the Scientific Literature As It Pertains to Gulf War Illnesses*. Vol. 7 of *Depleted Uranium*. MR-1018/7-OSD. (Santa Monica, CA: RAND, 1999). tables G.1, G.2.

11. *Exposure of the Population of the United States and Canada from Natural Back-*

ground Radiation, Report No. 94. (Bethesda, MD: National Council on Radiological Protection, 1987), 148.

12. The effective dose equivalent (EDE) dose-rate conversion factors for U-234, U-235, U-236, and U-238, including the contribution of short-lived decay products in equilibrium, are $1.20 \cdot 10^{-6}$, $7.42 \cdot 10^{-4}$, $9.43 \cdot 10^{-7}$, and $1.12 \cdot 10^{-4}$ rem/s per Ci/m², respectively. [Steve Fetter, "Internal Dose Conversion Factors for 19 Target Organs and 9 Irradiation Times and External Dose-Rate Conversion Factors for 21 Target Organs for 144 Radionuclides" (Idaho Falls: EG&G, Inc., September 1991), p. 135.] Multiplying by the specific activities and concentrations given in table 1 gives 1.3 mrem/yr per gram of DU per square meter. This is for exposure to a uniformly contaminated, infinite flat plane;. Shielding by terrain and structures would reduce the average dose rate to less than 1 mrem/yr.

13. *Exposure of the Population of the United States and Canada from Natural Background Radiation*, 69.

14. Title 10, Code of Federal Regulations, Part 20, Standards for Protection Against Radiation, Subpart D, 20.1301: Dose Limits for Individual Members of the Public.

15. A vehicle struck by two 120-mm penetrators might have 10 kilograms of DU fragments lying within a 100-square-meter area. The dose-rate above an infinite plane contaminated with 100 grams of DU per square meter is 130 mrem/yr; the finite deposition pattern in this case would reduce the dose rate by a factor of four. For comparison, the estimated cumulative deposition of uranium released by the FMCP plant was about 1 g/m² beyond a few kilometers. Near the center of the plant, the deposition level was 100 times higher (*RAC Report*, Fig. O-2).

16. Bureau of Radiological Health, *Radiological Health Handbook* (Rockville, MD: U.S. Department of Health, Education, and Welfare, January 1970), p. 204. The beta-ray surface dose rate for DU is virtually identical to that for natural uranium, because the daughters of U-238 are the primary beta-ray emitters. A 1-MeV beta ray has a range of approximately 0.5 centimeters in tissue.

17. Temporary loss of hair occurs after short-term skin doses of 300 rem; permanent hair loss and first-degree burns after skin doses of 600 rem; and second-degree burns after skin doses of 1000 to 2000 rem. [*Reactor Safety Study* (Washington, DC: U.S. Nuclear Regulatory Commission, 1975), p. F-13, 14.] The estimates are based on doses delivered in a few days or less. The dose rate from contact with DU is so low—less than 40 rem per week—that even continuous contact of bare skin with bare DU is unlikely to produce any of these symptoms, for the same reason that exposure to the sun for two hours in one day will produce a burn but exposure for 2 minutes per day for 60 days will not.

18. The estimated increase in generally non-lethal basal and squamous cell carcinomas for a skin dose of 100 rem is $7.1 \cdot 10^{-6}$ cm² in areas of skin not exposed to sunlight and $3.3 \cdot 10^{-5}$ cm² in areas exposed to sunlight. [*Health Effects of Exposure to Low Lev-*

els of Ionizing Radiation (BEIR V) (Washington, DC: National Academy Press, 1990), p. 327.] Thus, a person who kept a piece of DU in continuous contact with one square inch (6.5 cm²) of bare skin would incur an added skin-cancer risk of roughly 0.1 to 0.5 percent per year of exposure.

19. "Among 18,869 white males employed between 1943 and 1947 at a uranium conversion and enrichment plant in Oak Ridge, Tennessee, no excess cancers were observed through 1974...Several other published epidemiological studies of uranium mill and metal processing plant workers have either found no excess cancer or documented that excess lung cancer was attributable to other known carcinogens (radon and its progeny and cigarette smoke) rather than uranium." Harley, et al., "A Review of the Scientific Literature As It Pertains to Gulf War Illnesses." The excess lung cancers observed in uranium miners has been attributed to exposures to the gaseous uranium decay product, radon-222, and its short-lived decay products, whose concentrations build up in unventilated mines.

20. This is true even if there is a threshold (a dose below which there is no increased risk), so long as the threshold is below the natural background dose.

21. *Recommendations of the International Commission on Radiological Protection* Report No. 60 (Oxford: Pergamon Press, 1991). The National Academy of Sciences has recommended a population-weighted risk coefficient of 1 cancer death per 2,500 person-rem for doses delivered over weeks or months. *Health Effects of Exposure to Low Levels of Ionizing Radiation (BEIR V)*, 6.

22. See footnote 11.

23. Most of the external dose from DU comes from a 1-MeV gamma-ray which is emitted in 0.7 percent of decays of Pa-234m, a decay product of U-238. Shielding by terrain and structures probably would reduce population doses by a factor of two to four initially. Shielding would increase over time as a result of weathering and tilling of the soil. As long as most of the DU mass is contained in fragments with dimensions smaller than 1 cm, which seems likely, self-shielding will not reduce the dose rate greatly.

24. The kinetic energy of the 120-mm round is equal to $1/2 mv^2 = 1/2 (5 \text{ kg})(1500 \text{ m/s})^2 = 5.6 \text{ MJ}$; for comparison, one kilogram of TNT releases about 4 MJ.

25. The 30-mm round has projectile mass of about 0.4 kilograms (of which 0.3 kilograms is DU and the remainder is steel and aluminum) and a muzzle velocity of about 1,000 meters per second; the kinetic energy is equivalent to about 0.12 pounds of TNT.

26. The 120-mm penetrator is about 0.5 m in length, so the kinetic energy could be dissipated in as little as $(0.5 \text{ m})/(1500 \text{ m/s}) = 0.3 \text{ ms}$. The corresponding time for the 30-mm projectile is $(0.1 \text{ m})/(1000 \text{ m/s}) = 0.1 \text{ ms}$.

27. See Reports 6, 10, 22, 24, and 26, in "Research Report Summaries" Tab L, *Health and Environmental Consequences of Depleted Uranium Use in the U.S. Army*.

28. See Reports 12, 16, and 24, in "Research Report Summaries" Tab L, *Health and Environmental Consequences of Depleted Uranium Use in the U.S. Army*.
29. The ICRP assumes that 12 and 0.052 percent of uranium in the blood goes to the kidneys, where it is retained with half-lives of 6 and 1500 days, respectively; that 20 and 2.3 percent goes to the bone and is retained with half-lives of 20 and 5000 days; and that 12 and 0.052 percent are distributed uniformly throughout other tissues and retained with half-lives of 6 and 1500 days. International Commission on Radiological Protection, "Limits for Intakes of Radionuclides by Workers," *ICRP Publication 30, Part 1* (Oxford: Pergamon Press, 1978), p. 102–103. For a more elaborated model with parameters for different age groups, see "Age-dependent Doses to Members of the Public from Intake of Radionuclides," *ICRP Publication 71* (Oxford: Pergamon Press, 1995). We have used the parameters in ICRP-30, because our primary focus is on soldiers, who are overwhelmingly adult males, and because breakdowns by gender and age are unnecessary for our order-of-magnitude estimates of population doses.
30. The RAC report (Table K-2) estimated that the most exposed member of the public, a person living 1.7 km northeast of the center of the FMPC plant for 42 years would have received an EDE of about 5 rem.
31. A person would have to inhale at least 20 grams of DU aerosol to receive a radiation dose sufficient to cause respiratory impairment, and much larger amounts to cause other effects, such as nausea or temporary sterility. Respiratory impairment is possible at lung doses greater than 3,000 rem. Inhalation of 1 gram of insoluble DU aerosol would result in a lung dose of 30 to 160 rem in the first year after exposure, depending on aerosol size. Nausea and vomiting have been observed at whole-body doses of more than 25 rem per day or 75 rem per week; inhalation of 1 gram of DU would result in a whole-body dose of 0.05 rem in the first week after exposure. Temporary sterility has not been observed for single doses of less than 10 rem or dose rates of less than 25 rem per week; inhalation of 1 gram of DU would result in a dose to the gonads of less than 0.01 rem in the first week. Dose-morbidity relationships from *Reactor Safety Study*, pp. 9-11 to 9-20; dose conversion factors calculated by the authors using ICRP model.
32. *Health Risks of Radon and Other Internally Deposited Alpha-emitters (BEIR IV)* (Washington, DC: National Academy Press, 1988), p. 283.
33. The exposure limits are equivalent to an average inhalation rate of 0.34 mg/d of soluble uranium compounds or 1.7 mg/d of insoluble compounds. According to the ICRP model, up to 60 percent of inhaled soluble uranium aerosol and 10 percent of insoluble uranium enters the blood. The rate of transfer to the blood in both cases is therefore about 0.2 mg/d. Of uranium entering the blood, 12 percent and 0.052 percent are retained by the kidney with half-lives of 6 and 1500 days. Thus, for soluble compounds the steady-state concentration of uranium in the kidney would be $(0.2 \text{ mg/d}) [(0.12) (6 \text{ d}) + (0.00052) (1500 \text{ d})] / \ln(2) = 0.4 \text{ mg}$. Dividing the kidney mass (310 g for a standard adult male) gives a steady-state uranium concentration of

1.3 $\mu\text{g/g}$. One-time exposures resulting in a maximum uranium concentration of 1 ppm should be less injurious than long-term exposures resulting in the same concentration. In every case but one (inhalation of 0.2-micron insoluble aerosols), the maximum concentration occurs 1.5 to 2.5 days after inhalation or ingestion, and concentrations near the maximum level persist only for about one day.

34. *Bioassay Programs for Uranium: An American National Standard*, HPS N13.22-1995 (McLean, VA: Health Physics Society, October 1995); quoted in Harley, et al., "A Review of the Scientific Literature As It Pertains to Gulf War Illnesses." Assumes an AMAD of one micron.

35. H.W. Church, *Cloud Rise from High Explosive Detonations*(Albuquerque, NM: Sandia National Laboratory, report TID-4000, UC/41, 1969).

36. *Supplementary Documentation for Environmental Impact Statement Regarding the Pantex Plant: Dispersion Analysis for Postulated Accidents*LA-9445-PNTX-D (Los Alamos, NM: Los Alamos National Laboratory, 1982).

37. Doses were calculated using HOTSPOT 98, version 1.0 (Steven G. Homann, personal communication, 11 June 1999). S.G. Homann and D.V. Wilson, *HOTSPOT Training Manual: Health Physics Codes for the PC* UCRL-MA-118617 (Livermore, CA: Lawrence Livermore National Laboratory, 1995).

38. For example, the maximum radiation doses resulting from a 10-kilogram release under worst-case conditions would be 50, 12, and 0.3 millirem at distances of 0.1, 1, and 10 kilometers, respectively; corresponding maximum uranium concentrations in the kidney would be 0.01, 0.003, and 0.00004 ppm.

39. *Health and Environmental Consequences of Depleted Uranium Use in the U.S. Army*, Tab N.

40. The U.S. Veterans Administration conducted a study in which concentrations of uranium in the urine of 29 veterans who were injured in friendly-fire incidents involving depleted uranium were measured. [Melissa McDiarmid, "Medically Significant health Effects of DU Exposure," transcript of Veterans Administration satellite teleconference for VA doctors on DU exposures, March 1998; Available at: <http://www.global-dialog.com/~kornkven>.] Urine uranium concentrations were measured in 1994 and 1997, roughly 1100 and 2300 days after exposure. Of the 29 veterans, 14 did not have retained shrapnel and therefore were exposed only through inhalation. The highest urine uranium concentration measured in these 14 veterans, both in 1994 and 1997, was 0.14 micrograms of uranium per gram of creatinine, for the same individual; multiplying by the average excretion rate of creatinine (2.2 g/d) gives a urinary uranium excretion rate of 0.3 $\mu\text{g/d}$. (The concentration of uranium in the urine of an unexposed control group ranged from about 0.01 to 0.05 μg per gram creatinine.) Using the ICRP model, and assuming an AMAD of 1 μm , we calculate that the rate of excretion 1100 and 2300 days after a one-time inhalation is about 23 and 13 $\mu\text{g/d}$ per gram of uranium inhaled for insoluble compounds, and 4.4 and 2.6 $\mu\text{g/d}$ for soluble com-

pounds. Thus, if the aerosol was 30 percent soluble, an excretion rate of $0.3 \mu\text{g}/\text{d}$ in 1994 and 1997 would imply the inhalation of 17 to 30 mg of uranium in 1991. The implied amount inhaled would be up to two times smaller for a finer, less-soluble aerosol, and up to two times greater for a coarser, more-soluble aerosol.

41. The highest urine uranium concentration among the 15 veterans with retained shrapnel was 31 micrograms per gram creatinine [Melissa McDiarmid, "Medically Significant health Effects of DU Exposure"]; multiplying by the average excretion rate of creatinine gives a urinary uranium excretion rate of about $70 \mu\text{g}/\text{d}$.

42. If material retained in the lung were the sole source of the uranium excreted in the urine, an excretion rate of $70 \mu\text{g}/\text{d}$ in 1997 would imply inhalation of roughly 7 grams of uranium in 1991 (30 percent soluble, AMAD = $1 \mu\text{m}$). Inhalation of this much uranium aerosol would result in lethal concentrations of uranium in the kidney—up to 300 ppm, or 100 times greater than the damage threshold about 2 days after exposure. The fact that, in the McDiarmid study, the highest uranium concentrations in veterans with shrapnel were more than 100 times greater than the highest concentrations in exposed veterans without shrapnel also implies that shrapnel, rather than inhaled aerosol, is the source of uranium in the urine.

43. In equilibrium (which is a good approximation so long after the initial exposure), the rate at which uranium is mobilized and enters body fluids is equal to the rate of excretion— $70 \mu\text{g}/\text{d}$ in the most exposed individual. Of uranium in body fluids, the ICRP assumes that 12 percent is retained in the kidney with a half-life of 6 days and 0.052 percent is retained with a half-life of 1,500 d. Thus, the equilibrium concentration of uranium in the kidney is $(67 \mu\text{g}/\text{d})[(0.12)(6 \text{ d}) + (0.00052)(1500 \text{ d})]/\ln(2) = 145 \mu\text{g}$; dividing by the mass of the kidneys gives a concentration of $0.47 \mu\text{g}/\text{g}$, or about 0.5 ppm.

44. The 50-year EDE from DU dissolved into the blood is 1.86 rem per gram. Thus, the mobilization of $67 \text{ mg}/\text{d}$ of DU into the blood would produce an annual dose of $(67 \cdot 10^{-6} \text{ g}/\text{d})(365 \text{ d}/\text{yr})(1.86 \text{ rem}/\text{g}) = 0.046 \text{ rem}/\text{yr}$. The dose is unlikely to be constant over a 50-year period, since this would amount to excretion of $(67 \cdot 10^{-6} \text{ g}/\text{d})(365 \text{ d}/\text{yr})(50 \text{ yr}) = 1.2$ grams of DU.

45. Almost 99 percent of the internal dose from uranium is due to alpha particles, which have a range of only about $4 \mu\text{m}$ in uranium metal. If the fragments are much less than $4 \mu\text{m}$ in diameter, which seems highly unlikely, then nearly all of the alpha energy would be deposited in the surrounding tissue and the dose rate $R = 1.87 \cdot 10^{10} SA Q E / M \text{ rem per year}$, where SA is the specific activity of DU $3.85 \cdot 10^7 \text{ Ci}/\text{g}$, E is average alpha energy per decay (4.2 MeV), Q is the quality factor (20 rem/rad), M is the muscle mass (28,000 g), and $1.87 \cdot 10^{10}$ is the number of $\text{Bq g rad s per Ci MeV yr}$. Thus, $R = 22 \text{ rem}/\text{yr}$, or about 1000 rem over 50 years if the fragments do not largely dissolve during this time. Applying a weighting factor of 0.06, the contribution of the muscle dose to the EDE would be about 60 rem. Beta particles, which have a range of $200 \mu\text{m}$ in uranium, contribute nearly all of the

remaining dose; in this case $E = 0.87$ MeV, $Q = 1$, and $R = 0.22$ rem/yr, or roughly 10 rem over 50 years. If the fragments initially have a combined mass of 1 gram, and are dissolved at a constant rate of 20 mg/yr ($55 \mu\text{g}/\text{d}$), the 50-year dose would be reduced by a factor of two. If, as seems very likely, the fragments have diameters much larger than $4 \mu\text{m}$, then only those alpha particles emitted in the top $4\text{-}\mu\text{m}$ layer of the fragment could escape the fragment. The fraction emitted in the top $4 \mu\text{m}$ and directed outward is approximately $(4/r)$; thus, for fragments in the form of long slivers of radius r (μm), the dose is roughly $1000(4/r) + 10$ rem per gram of DU. For a fragment radii much greater than $200 \mu\text{m}$, the dose is roughly $1000(4/r) + 10(200/r) = 6000/r$. For fragment diameters of 0.1, 1, and 10 millimeters, the dose is roughly 100, 10, and 1 rem per gram DU. EDE assumes a weighting factor of 0.06 for muscle; the dose to organs other than muscle would be negligible.

46. This is because the primary effect of the radiation is to kill rather than damage cells. International Commission on Radiological Protection, "Recommendations of the International Commission on Radiological Protection," *ICRP Publication 26*, (Oxford: Pergamon Press, 1977), p. 8.

47. *Health and Environmental Consequences of Depleted Uranium Use in the U.S. Army*, chapter 4; *Health and Environmental Consequences of Depleted Uranium Use in the U.S. Army* Tab G.

48. "Report to the American Physical Society by the Study Group on Light-Water Reactor Safety," *Reviews of Modern Physics* 47 (1975), p. S45.

49. Steve Fetter and Frank von Hippel, "The Hazard from Plutonium Dispersal by Nuclear-warhead Accidents," *Science and Global Security* 2 no. 1 (1990): 24–27.

50. More accurately, the reciprocal of the mass-weighted average of the reciprocal of the deposition velocity, $\langle v^{-1} \rangle^{-1}$ should be used instead of the average deposition velocity.

51. *Reactor Safety Study* table VI B-1. The *RAC Report* (tables L-3, M-1) estimated the average deposition velocity for uranium particles released from the FMPC plant to be about 0.02 m/s for aerodynamic diameters of less than 5 microns.

52. Fetter and von Hippel, "The Hazard from Plutonium Dispersal," 29–31.

53. See, for example, *Reactor Safety Study* table VI E-3.

54. Based on a review of the data then available, a 1974 Atomic Energy Commission study suggested for populated areas the values $K_0 = 10^{-5} \text{ m}^{-1}$, $K = 10^{-9} \text{ m}^{-1}$, and $\tau = 0.2$ yr; when integrated over 50 years, this gives $K = 65 \text{ s/m}$. [*U.S. Atomic Energy Agency Proposed Final Environmental Impact Statement Liquid Metal Fast Breeder Reactor Program* (WASH-1535, 1974), appendix II-G.] The Reactor Safety Study used the same values of K_0 and K , but assumed $\tau = 1.5$ yr, so that $K = 470 \text{ s/m}$. [*Reactor Safety Study*, p. E-13.] Anspaugh proposed that $K(t) = 10^{-4} e^{-0.15\sqrt{t}} + 10^{-9}$, which gives $K = 800 \text{ s/m}$. [L.R. Anspaugh, L. H. Shinn, P. L. Phelps, and N.C. Kennedy, "Resuspension and Redistribution of Plutonium in Soils," *Health Physics* 29:571–582.]

55. *Recommended Screening Limits for Contaminated Surface Soil and Review of Factors Relevant to Site-specific Studies* Report No. 129 (Bethesda, MD: National Council on Radiation Protection and Measurement, 1999); J.A. Garland, N.J. Pattenden, K. and Playford, "Resuspension Following Chernobyl" IAEA-TECDOC-647 (Vienna: International Atomic Energy Agency, 1992).

56. Regarding contaminated leaf surfaces, the ratio of the dose from milk and meat to the dose from produce is given by $(Y_p/Y_f)(r_f/r_p)U_f(F_mU_m + F_tU_t)/U_p$ where U_p , U_m and U_t are the average annual per-capita consumption of produce, milk, and meat (kg/yr), U_f is the ingestion rate of forage by animals (kg/d), r_p and r_f are the fractions of deposited DU dust retained on produce and forage, Y_p and Y_f are the agricultural productivity for produce and forage (kg/m²), and F_m and F_t are the transfer coefficients for the forage-milk and forage-meat pathways [(kg_{DU}/kg)/(kg_{DU}/d)]. Typical values are $U_p = 200$ kg/yr, $U_m = 100$ kg/yr, $U_t = 100$ kg/yr, $U_f = 16$ kg/d, $r_p = 0.2$, $r_f = 0.6$, $Y_p = 0.7$ kg/m², $Y_f = 0.3$ kg/m², $F_m = 3.7 \cdot 10^{-4}$ d/kg, and $F_t = 3.4 \cdot 10^{-4}$ d/kg, which gives a dose ratio of 0.004. The population dose rate from root uptake is equal to $C_g f_g M [U_p B_p + U_f B_f (F_m U_m + F_t U_t)] / z_{rz} \rho_s$, where ρ_s is the density of soil (kg/m³), z_{rz} is the depth of the root zone (m), and B_p and B_f are the transfer coefficients for the soil-produce and soil-forage pathways [(kg_{DU}/kg_{veg})/(kg_{DU}/kg_{soil})]. Typical values are $B_p = 1.1 \cdot 10^{-3}$, $B_f = 3.1 \cdot 10^{-2}$, $\rho_s = 1600$ kg/m³, and $z_{rz} = 0.3$ m. The population dose rate from contaminated water is given very approximately by $C_g M U_w / P W$ where U_w is the average rate of water consumption from stream and river runoff (m³/yr), P is the average precipitation rate (m/yr), and W is the residence time of uranium with respect to dissolution in runoff. Typical values are $U_w = 0.8$ m³/yr, $P = 1$ m/yr and $W = 10^4$ yr. The population dose from ingestion of soil is given by $C_g f_g M U_s / z_{DU} \rho_s$, where U_s is the average annual consumption of soil (kg/yr) and z_{DU} is the soil depth into which the DU is assumed to be uniformly mixed (m). Typical values are $U_s = 0.04$ kg/yr, $z_{DU} = 0.1$ m. In the Gulf War example given in the text, the root uptake, water, and soil ingestion pathways would together contribute a population dose of about 0.1 person-rem per year. Typical values are taken from R.E. Moore, C.F. Baes III, L.M. McDowell-Boyer, A.P. Watson, F.O. Hoffman, J.C. Pleasant, and C.W. Miller, "AIRDOS-EPA: A Computerized Methodology for Estimating Environmental Concentrations and Dose to Man from Airborne Releases of Radionuclides," EPA 520/1-79-009 (Washington: U.S. Environmental Protection Agency, December 1979); "Calculation of Annual Doses to Man from Routine Releases of Reactor Effluents for the Purpose of Evaluating Compliance with 10 CFR Part 50, Appendix I," Regulatory Guide 1.109 Revision 1 (Washington, DC: U.S. Nuclear Regulatory Commission, October 1977); and Y.C. Ng, "A Review of Transfer Factors for Assessing the Dose from Radionuclides in Agricultural Products," *Nuclear Safety* 23(1): (January-February 1982).

57. For a derivation of equation 9, see Steve Fetter, "Radiological Hazards of Fusion Reactors: Models and Comparisons," (Ph.D. Diss., University of California, Berkeley, 1985).

58. See footnote 54.

59. If the contamination occurs just before harvest, the quantity $[1 + K]$ in equation 9 should be replaced by $[^{-1} + K]$; if contamination occurs between harvests, it should be replaced by $[K]$.

60. The RAC estimates (Table K-2) of doses from uranium releases from FMPC concluded that the doses from ingestion of contaminated food have been only a few percent of those from inhalation.

61. The dose rate from environmental uranium per gram is higher than from depleted uranium because of the presence of the 80,000-year half-life uranium-238 decay product, thorium-230, and its decay product, 1600-year half-life radium-226. Their short-lived decay products, Pb-214, Bi-214, and Bi-210, each release a high-energy gamma ray during almost every decay. In contrast, the 1-MeV gamma ray which accounts for most of the external dose from depleted uranium during the first century after its production is released by only about 0.6 percent of Pa-234 decays [C. Michael Lederer and Virginia S. Shirley, *Table of Isotopes seventh edition* (John Wiley & Sons, 1978)]. The resulting dose rate from a given level of uranium in the soil cannot be calculated simply, however, because of the difference in the chemistry of uranium, thorium and radium in the soil and, more importantly, because the 4-day half-life radioactive gas radon-222 separates thorium-230 from lead-214, bismuth-214, and bismuth-210, allowing their escape into the air.

62. *Table of Radioactive Isotopes* Edgardo Browne and Richard Firestone; Virginia S. Shirley, ed. (John Wiley and Sons, 1986).

63. 98.6 % of the Pa-234m decays go directly to the ground state of U-234.

64. L.T. Dillman, "Absorbed Gamma Dose Rate for Immersion in a Semi-Infinite Radioactive Cloud," *Health Physics*, 27 (1974): 571.

65. J.H. Hubbell, "Photon Cross Sections, Attenuation Coefficients, and Energy Absorption Coefficients from 10 keV to 100 GeV," NSRDS-NBS 29 (Washington, DC: National Bureau of Standards, August 1969).

66. One quarter of the material deposited in the pulmonary region with half-life of 500 days is removed to the pulmonary lymph nodes (considered, for purposes of radiological protection, as part of the lung), where 90 percent is retained with a half-life of 1000 days (a mean residence time of 4 years) and 10 percent is retained indefinitely (when calculating the 50-year dose, the residence time in this compartment is 50 years). Thus, the overall residence time of this material in the lung is $[2 + 0.25(0.9 \cdot 4 + 0.1 \cdot 25)] = 4.15$ years.