Radiological Terrorism
Fact Sheet
Radiological Weapons - Nuclear Blast/Reaction

Gamma Emitters: Uranium
- Gamma rays are uncharged radiation similar to x-rays. They are high-energy particles that easily pass through matter. With such high penetrability, gamma radiation can result in whole-body exposure.

- Naturally occurring uranium is a mixture of three isotopes, of which uranium-238 (half-life: $4.5 \times 10^9$ years) is the most abundant (99.2%) and most stable. The other isotopes are uranium-235 (half-life: $7 \times 10^8$ years) and uranium-234 (half-life: $2.5 \times 10^5$ years). These isotopes emit high levels of gamma and beta radiation and their containment canisters weigh thousands of pounds, making them difficult to use and transport by terrorists. Inhaled uranium compounds are metabolized and excreted in the urine. Urinary levels of 100 µg per deciliter following acute exposure may cause renal failure. Absorption is determined by the chemical state of the uranium. Soluble salts are readily absorbed; the metal is not.

Beta Emitters: Radioiodine and Strontium
- Beta particles, found primarily in fallout radiation, are very light, charged particles that can travel a short distance in tissue. In large quantities they can damage the basal stratum of the skin causing a “beta burn” that is similar to a thermal burn. Their main threat comes from being internalized through inhalation or ingestion.

- Radioiodine (Iodine–131, 132, 134, 135), a beta emitter, is created during nuclear fission and is found in reactor fuel rods or after a nuclear explosion. Following a reactor accident, it can be released from a ruptured reactor core and containment vessel. Its primary toxicity is to the thyroid gland, where thyroid uptake concentrates the RAI and allows local irradiation similar to therapeutic thyroid ablation. A high incidence of childhood thyroid carcinoma was documented following the Chernobyl disaster.

- Strontium-90 is a direct fission product of uranium. It and its daughters emit both beta and gamma rays and can be an external irradiation hazard if present in quantity. Strontium will follow calcium and is readily absorbed by both respiratory and GI routes. Up to 50% of a dose will be deposited in bone.

Alpha Emitters: Americium, Plutonium
- Alpha particles are massive, charged particles that cannot travel far and are completely stopped by the dead layers of the skin or by clothing. Alpha particles offer minimal external hazard, but can cause significant regional cellular damage when internalized.

- Americium-241 is a decay daughter of plutonium and an alpha emitter. Its main threat is heavy metal poisoning, but, in large quantities, it can cause whole-body irradiation. Seventy-five percent of the initial lung burden is absorbed, with 10% of the particles retained in the lung. Gastrointestinal absorption of americium is minimal, but it may be absorbed rapidly from skin wounds. It is eliminated by urinary and hepatic excretion.
Alpha Emitters: Americium, Plutonium (continued)

- Plutonium-239/238 is produced from uranium in reactors. It is the primary fissionable material in nuclear plants and weapons. The primary radiation is in the form of alpha particles, and it is always contaminated with americium. Primary toxicity is from inhalation. GI absorption will depend upon the chemical state of the plutonium, but the metal is not absorbed. Stool specimens will be positive after 24 hours and urine specimens after 2 weeks. Wound absorption is variable. Plutonium may be washed from intact skin.

Health Risks

Radiation causes biological damage by direct and indirect means. When radiation interacts with atoms, energy is deposited, resulting in ionization (electron excitation). Such ionization can damage critical molecules or structures in a cell directly by hitting a particularly sensitive atom or molecule in the cell. The damage from this is irreparable and the cell either dies or malfunctions. Changes in cellular function, such as delays in phases of the mitotic cycle, disrupted cell growth, permeability changes, and changes in motility, can occur at lower radiation doses than those causing cell death. Indirect cell damage occurs when the radiation interacts with water molecules in the body, resulting in unstable, toxic hyperoxide molecules that can damage sensitive molecules and afflict subcellular structures. In general, actively dividing cells are the most sensitive to radiation, so the two most significant radiosensitive organ systems are the hematopoietic and gastrointestinal (GI) systems.

Without appropriate medical care, the median lethal dose of radiation, the LD50/60 (that which will kill 50% of the exposed persons within a period of 60 days), is estimated to be 3.5 Gy. However, with modern medical care, nearly all radiation casualties are considered treatable if care is quickly made available.

From a statistical perspective, for radiation, tumor induction is the most important long-term sequelae of exposure. These statistics are derived from known data on exposures greater than 100 rem. There is also reliable data showing that exposure to just 10 rem causes a 0.8% increase in the lifetime risk of death from cancer. Thus, out of 5000 people with such an exposure, 40 may develop a fatal cancer. The latency period for the various radiation-induced cancers seen in humans may be several years.

Additional potential sequelae of radiation exposure include cataract formation, decreased fertility and fetal teratogenesis. Cataracts can develop as early as 6 months to many years after exposure. The threshold for detectable cataract development is 200 rem for acute gamma-radiation doses and 1500 rem for chronic exposures. Although the testes and ovaries are highly radiosensitive, the effects of exposure are short term. Whole-body irradiation above 12 rem can produce a transient azospermia, lasting from months to several years, but natural fertility does recover. There are four main effects of ionizing radiation on the fetus: growth retardation; severe congenital malformations (including errors of metabolism); embryonic, fetal, or neonatal death; and carcinogenesis. Irradiation in the fetal period leads to the most evident permanent growth retardation. The peak incidence of teratogenesis, or gross malformations, occurs when the fetus is irradiated during organogenesis; however, malformations of organs other than the central nervous system are uncommon in humans.

Decontamination

The most common contaminants will primarily emit alpha and beta radiation. It is impossible for a patient to be so contaminated that he is a radiation hazard to health care providers; so medical or surgical treatment should not be delayed because of possible contamination. Most of the time, the simple removal of outer clothing and shoes will reduce contamination by 90%. External contamination of the skin and hair is from particulate matter that can be washed off. If practical, the clothing and effluent from washing should be sequestered and disposed of properly.
Treatment

Acute Radiation Syndrome
During the prodrome of ARS, supportive care and the use of oral antiemetics, such as granisetron (Kytril®) and ondansetron (Zofran®), may be indicated. These medications will decrease the nausea and vomiting in a high percentage of exposed individuals and reduce the likelihood of a compromised individual being injured because he was temporarily debilitated. These antiemetics are not radioprotectants and do not change the degree of radiation injury.

As bone marrow suppression develops, the prevention and management of infection governs therapy. Antibiotic prophylaxis must be considered in afebrile patients who have a profound neutropenia (< 0.1 x 10⁶ cells/l). As the duration of the neutropenia increases, the risk of secondary infections such as invasive mycoses increases and the use of cytokine hematopoietic growth factors, such as filgrastim (Neupogen®), a granulocyte colony-stimulating factor (G-CSF), and sargramostim (Leukine®), a granulocyte-macrophage colony-stimulating factor (GM-CSF), to stimulate hematopoiesis may prove invaluable if started within 72 hours of exposure.

It must be assumed during the care of all patients that even those with a typical gastrointestinal syndrome may be salvageable. Replacement of fluids and prevention of infection by bacterial transmigration is mandatory.

General Management
Inhaled particles less than 5 microns in size will end up in the alveolar area, while the mucociliary apparatus will clear larger particles. Soluble particles are then directly absorbed into the blood stream or moved into the lymphatic system. Insoluble particles, until cleared from the respiratory tract, will continue to irradiate surrounding tissues. In the alveoli, the localized inflammatory response can produce fibrosis and scarring.

Absorption of ingested radioactive material depends on the solubility and chemical makeup of the contaminant. For example, radioiodine is rapidly absorbed; plutonium and strontium are slowly absorbed, if at all. The target organ for ingested radionuclides that pass unchanged in the feces is the lower GI tract. Gastric lavage and emetics can help empty the stomach promptly, while purgatives, laxatives, and enemas can reduce radioactive materials in the colon. Ion exchange resins limit gastrointestinal uptake of ingested or inhaled radionuclides.

The skin is impermeable to most radionuclides, but wounds and burns allow particulate contamination to bypass the epithelium. Also, fluid in the wound may hide weak beta and alpha emissions from detectors. Because of this, all contaminated wounds must be meticulously and aggressively cleaned and debrided.

Once absorbed, a radionuclide crosses capillary membranes through passive and active diffusion and is distributed throughout the body. Organ metabolism, the ease of chemical transport, and the affinity of the radionuclide for chemicals within the organ determine the rate of distribution, with the liver, kidney, adipose tissue, and bone having greater capacities for binding radionuclides because of their high protein and lipid makeup.

Heavy metal poisoning is also a potential threat depending upon the isotope used. Chelation agents should be administered as needed. Calcium edetate (EDTA) is used primarily to treat lead poisoning but must be used with extreme caution in patients with preexisting renal disease. Diethylenetriaminepentaacetic acid (DTPA, an investigational drug) is more effective in removing many of the heavy-metal, multivalent radionuclides. Dimercaprol forms stable chelates with mercury, lead, arsenic, gold, bismuth, chromium, and nickel and may be considered for the treatment of internal contamination with radioisotopes of these elements. Another consideration is penicillamine, which chelates copper, iron, mercury, lead, gold, and possibly other heavy metals.

Signs and Symptoms (continued)

death. Convulsions may or may not occur, and signs of increased intracranial pressure may or may not be evident. Individuals receiving such high doses following a nuclear blast would have to be close enough to be well within the range of 100% lethality due to blast and thermal effects.

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The signs and symptoms of chronic radiation poisoning (CRS) are based upon victims who were exposed to radiation for at least 3 years and who had received at least 100 rem or more to the marrow. Such victims may complain of sleep and/or appetite disturbances, generalized weakness with rapid fatigue, increased excitability, loss of concentration, impaired memory, mood changes, vertigo, ataxia, paresthesias, headaches, epistaxis, chills, syncopal episodes, bone pain, and hot flashes. On exam, clinical findings may include localized bone or muscle tenderness, mild hypotension, tachycardia, intention tremor, ataxia, asthenia, and hyperreflexia (sometimes hyporeflexia). Reproductive effects in exposed children may include delayed menarche and underdeveloped secondary sexual characteristics. Lab findings include mild to marked pancytopenia and bone dysplasia. Gastric hypoaacidity and dystrophic changes may be present. Once the patient is removed from the radiation environment, clinical symptoms and findings slowly resolve, and complete recovery has occurred from the lower doses.
Treatment (continued)
Specific Treatments:

- **Americium-241**: DTPA or EDTA chelation in the first 24 to 48 hours following pulmonary exposure is effective.
- **Plutonium-239/238**: Administer 1 g CaDTPA, by nebulizer or IV, within 24 hours of exposure; followed by 1 g ZnDTPA qd while monitoring urine levels.
- **Radioiodine**: See Potassium Iodide.
- **Strontium-90**: Immediately after ingestion, oral administration of aluminum phosphate can decrease absorption by as much as 85%. Administration of stable strontium can competitively inhibit the metabolism and increase the excretion of strontium-90. Large doses of calcium and acidification of the urine with ammonium chloride will also increase excretion.
- **Uranium-238/235/234**: Sodium bicarbonate makes the uranyl ion less nephrotoxic. Tubular diuretics may be beneficial. Laboratory evaluation should include urinalysis, 24-hour urine for uranium bioassay, serum BUN creatinine, beta-2-microglobulin, creatinine clearance, and liver function studies.

**Potassium Iodide**
Radioactive iodine (RAI) is a product of nuclear fission and a potent cause of thyroid cancer. Potassium iodide (KI), if taken in time and at the appropriate dosage, blocks the thyroid gland’s uptake of RAI and reduces the risk of cancer and other diseases that might be caused by exposure to RAI.

After careful review of the data from Chernobyl, relating estimated thyroid radiation dose and cancer risk in exposed children, the FDA is revising its recommendation for administration of KI based on age, predicted thyroid exposure, and pregnancy and lactation status. For adults over 40 (with an exposure greater than 500 rem), adults 18 through 40 (with exposures over 10 rem), and pregnant or lactating women, the dose is 130 mg of KI. For children and adolescents ages 3 to 18, the dose is 65 mg, unless the adolescent is near adult size (>70 kg). Such teens would get the adult dose. Children ages 1 month to 3 years should receive 32 mg and infants (<1 mo, with an exposure over 5 rem) should get 16 mg.

The protective effect of KI lasts approximately 24 hours, so for optimal prophylaxis, it should be dosed daily until the risk of significant exposure to inhaled or ingested RAI no longer exists. Individuals intolerant of KI, as well as neonates and pregnant and lactating women should be given priority with regard to other protective measures.

Pregnant women should be given KI for their own and their baby’s protection since iodine (stable or radioactive) readily crosses the placenta. With the risk of blocking fetal thyroid function with excess stable iodine, repeat dosing with KI of pregnant women should be avoided. Lactating females should receive KI for their own protection, and potentially to reduce the radioiodine content of the breast milk. The infant should get his/her KI directly. Since stable iodine as a component of breast milk may increase the risk of hypothyroidism in nursing neonates, repeat dosing with KI should be avoided in the lactating mother, except during continuing severe contamination. If repeat dosing of the mother is necessary, the nursing neonate should be monitored.

**Units of Radiation**
Although the rad is still used widely as a unit of radiation, the current trend is toward use of the international unit called a gray (Gy), which may be used to describe all types of radiation. In man, higher energy radiation has greater effects as it is absorbed in tissue. A quality factor (QF) is used to adjust the difference. The dose in rads times the QF yields the rem (roentgen equivalent, man). The international unit for this radiation equivalency is the sievert (Sv) and is appropriately utilized when estimating long-term risks of radiation injury. Since the QF for x-ray or gamma radiation = 1, then for pure gamma radiation: 100 rad = 100 cGy = 1000 mGy = 1 Gy = 1 Sv = 100 rem.

**Disclaimer**
Information contained in this fact sheet was current as of September 2002, and was designed for educational purposes only. Medication information should always be researched and verified before initiation of patient treatment.

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