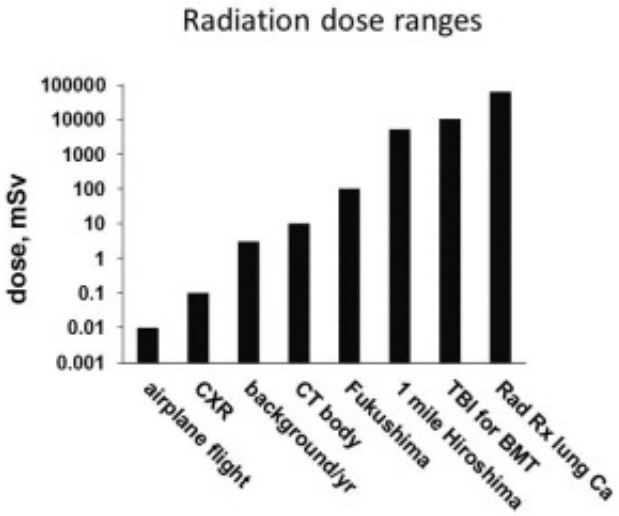
# Kidney disease after radiation exposure

Radiation exposure on earth is usually at a very low dose. Diagnostic x-rays are essential for modern medical care and pose little or no radiation risk. High dose radiation is much less common than diagnostic x-ray, but it is also very useful, because it can cure cancer. But those high doses pose a risk of injury to the surrounding normal tissues, with both early and late side effects. Depending on dose and site of irradiation, these normal tissue side effects can include injury to the blood-forming bone marrow, gastrointestinal tract, lungs, brain, heart, and kidneys. To these is added the delayed risk of radiation-induced cancer, but that is five years or more after exposure.




The typical doses for some common radiation exposures.

The range of doses in question is shown on the adjoining figure. Note that background radiation is more than that of a diagnostic chest x-ray. The added cancer risk becomes significant at single doses of 30 mSv, i.e. ten times those of yearly background exposure. Early gastrointestinal effects such as nausea or vomiting may occur after single exposures of 1000 mSv or more (100 rads or cGy), and hematopoietic marrow suppression will occur after single doses of 3000 mSv or more.

The early and late side effects of radiation are well-known in clinical Radiation Oncology, in which single doses of 200 cGy and cumulative doses of 60 Gy are common. Modern radiotherapy protocols use image-guided “sculpting” of the radiation fields and dose fractionation to reduce the injury to normal tissue. But sequelae such as radiation pneumonitis and chronic kidney disease still occur. Radiation pneumonitis complicates radiotherapy for lung cancer in about 20% of cases, and chronic kidney disease occurs in 10 to 20% subjects who have undergone hematopoietic stem cell transplant. This justifies research on the mechanisms of radiation-induced lung or kidney injury and development of mitigating pharmaceuticals.

Accidental or war-time radiation exposures also result in injury to normal tissue. Thus, marrow suppression and gastrointestinal toxicity occurred as the acute radiation syndrome after the atomic bombs of Hiroshima and Nagasaki. Similar effects occurred at the Chernobyl accident. Increased cardiovascular disease is now also recognized in long-term Hiroshima/Nagasaki survivors, many years after the bombs. The increased long-term risk of cardiovascular disease may depend on renal injury, even for estimated single doses of only 100 rads.

There is a persistent threat of accidental, belligerent, or war-time radiation exposures. Timely measurement of the amounts of radiation is critical, because exposures below 30 mSv (~3 rads) may cause great anxiety but will not cause human harm. Higher dose exposures pose a risk of immediate and long-term effects, as stated above. Those who survive the blast within a one mile radius of a 10 kiloton atomic bomb will have partial or total body exposures of 100 rad or more, and fallout beyond that radius poses similar risk for up to 48 hours. Such dose exposures are in a range where normal tissue radiation injury will occur, including to kidneys.

Current research on renal radiation injury is focused on identifying mitigators of that injury. Mitigation is the use of an agent after radiation exposure but before expression of organ injury. Angiotensin-converting enzyme inhibitors are effective mitigators of renal radiation injury, and there are promising new data for epoxyeicosatrienoic acid analogues. Successful mitigation of normal tissue radiation injury will have a major impact, to limit the adverse effects of accidental, terrorist, or war-time radiation exposure.

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## Publication

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