A crash course in radiation, biology, and health physics

D. M. Wood, November 2017
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Contents

As of 2023 Fig. 4 has been modified and our first sidebar note added. The purpose of this document is to introduce every aspect of the usual approach for estimating radiation doses from 'ionizing radiation', such as the α particles and γ rays produced by plutonium.

(How to translate these doses into cancer risks is described in a separate document.) The 'health physics' of radiation exposure is interesting but can be confusing because of the need to conceptually 'partition' incoming radiation into doses acting on different organs, which differ considerably in their sensitivity to radiation.

Documents on the website as of 2019 or later demonstrate that almost no gamma rays are emitted by Pu. The short range of alpha particles (see below) means that only plutonium in dirt which is inhaled or swallowed can provide a radiation dose. This is discussed in detail in other website documents.

Radiation

"Ionizing radiation" consists of particles, emitted by unstable ("radioactive") nuclei, with enough energy to break bonds[1] (or 'ionize' electrons from atoms) in biological molecules. Less unfamiliar are (uncharged) particles of light ("photons") in the range from the ultraviolet [sunburn] through X rays (which penetrate bone and tissue) to 'gamma rays' (γ rays, with energies from hundreds of

[1] Such bonds have a 'breaking energy' in the range of eV ("electron volts"), a unit of energy at the atomic scale.

keV to several MeV, characteristic of the transitions between nuclear energy states), which can penetrate several centimeters of metal or hundreds of meters in air.

In addition, some nuclei emit 'alpha particles' (α), which are doubly charged and not very penetrating precisely because they interact so strongly with atoms and molecules. This means that they dump their energy rapidly and are thus quite dangerous to biological tissue. Some nuclear decays emit high-energy electrons; these are termed 'beta particles' (β). These also obviously have a charge but are much lighter and are much more easily deviated from straight-line paths. They may penetrate up to 10 meters of air, but only a few millimeters of tissue. [There are a few 'cosmic rays' too, and neutrons if (ugh) we are near where they are being produced.]

The process of radioactive decay (and thus the particles per second we measure) is itself statistical, as you can see from Fig. 2. Given a specified amount[2] of radioactive material (and some information about its shape and size and the geometry and efficiency of measurement for the particles we are measuring for the detector we use) we can predict the average number of particles per second we should measure.

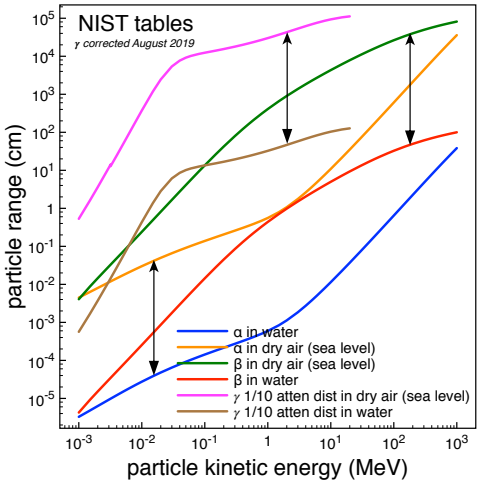

Figure 1: NIST data for ranges of α and β particles and γ rays in air (sea level, room temperature) and liquid water as a function of their energies. [1]

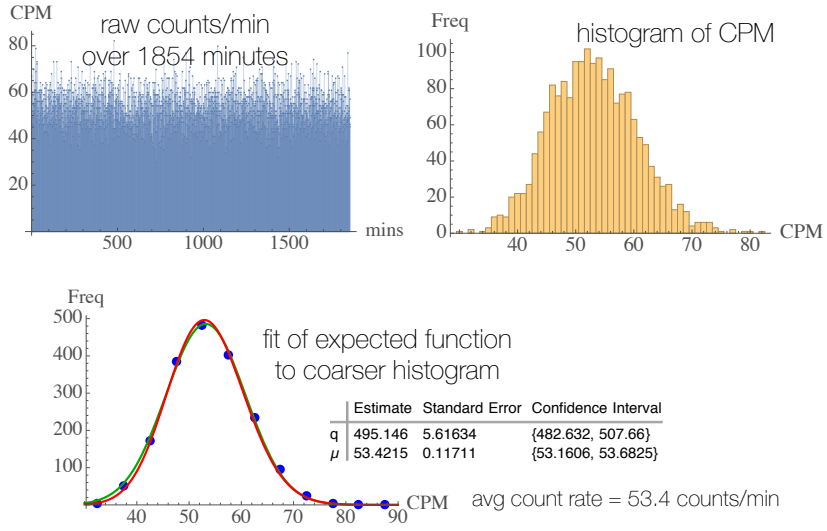

Figure 2: Extracting average count rate from a long count using a sensitive Geiger-Müller counter on the desk in my office. There is one free parameter in the fit (which determines the average count rate and the entire shape of the curve), so that this process, although statistical, is extremely well understood.

[2] Because the sources are radioactive (unstable) nuclei, their number changes (decays) in time t, according to the formula

$$N(t) = N(t=0)\ 2^{-t/t_{1/2}}$$

where $t_{1/2}$ is the 'half life' of the excited state of the nucleus. If you plug this in in place of t above, you would find that after this time has passed only half of the excited nuclei remain.

Health effects of radiation exposure

The health consequences of radiation have been recognized since the early use of X rays; by 1925 radiologists and those experimenting with recently-discovered radioactive elements such as radium had set up health guidelines. After World War II the survivors of Hiroshima and Nagasaki were carefully studied to assess the health impact of "large" doses or radiation.

Tumors subject to radiation therapy receive thousands of times more radiation.

Leukemia was among the first cancers to be associated directly with radiation exposure because its 'incubation time' (latency) is among the shortest of all cancers. We will take it as a given that *cancer is the principal health risk for people exposed to low doses of nuclear radiation*.

Non-ionizing radiation like light or radio, by contrast, is deliberately broadcast; sunlight on the ground (mostly non-ionizing radiation) comes from a source so large that the statistical aspects of the photons [light particles] it emits are not evident under most conditions.

How does radiation affect living tissue?

As we have already seen, every aspect of nuclear radiation is statistical in nature, because we have only statistical information about whether a given nucleus will emit a particle or not; for a large number of decaying nuclei, in contrast, outcomes are very well understood statistically.

The interaction of ionizing radiation with living tissue is extremely complicated. We can naively think of the process for a single subatomic particle as shown in panel (a) of Fig. 3: a series of encounters with atoms punctuated by more-or-less straight-line motion until all of its energy is used up as it rips electrons from ('ionizes') atoms. However, a radioactive source produces such particles with a range of energies, a range of trajectories, and of course the location of atoms in biological molecules is extremely complex. This means that the real behavior of a 'beam' of radioactive particles is a statistical average over huge numbers of possibilities. As complicated as the microscopic path may be there is nonetheless, as shown by panel (c) of the Figure, a well-defined quantum mechanical description of the energy loss by a single charged particle along its macroscopic path. The *range* of, for example, an α particle in tissue is about 30×10^{-4}

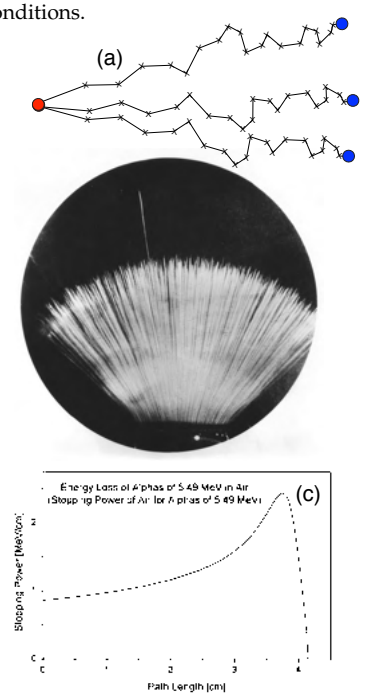

Figure 3: Microscopic and macroscopic behavior of α particles as an example. (a): crude atomic-scale trajectory of incoming particle (at left, in red) as it interacts with atoms, sometimes kicking electrons loose, until it is at rest (blue, right); (b): a photograph taken by Lise Meitner in the 1930s (from the British Science Museum, collection of high energy α particles in a 'cloud chamber'; (c) the energy loss of an α particle in air (Wikipedia). Note the typical range of a few cm in air (much less in tissue) and the large energy loss just before stopping.

cm (30 μ) [2] (see also Fig. 1). Although on average most biological tissue has a density close to that of water, distinct tissue types can be very different in their sensitivity ("radiosensitivity") to various types of radiation. Rapidly-dividing cells are in general most sensitive (a fact which is exploited in cancer radiation therapy, since cancer cells exhibit uncontrolled rapid growth). Cells with short replacement times, such as blood cells or the lining of the small intestine) also are sensitive, which is why, for instance, leukemia is among the earliest cancers to emerge after serious radiation exposure. 'Radiation weighting factors' which take into account the biological impact of nuclear radiation (and how much energy they deposit per unit length along their paths) have long been used.

In our case the relevant radiation types are γ and α (and, primarily because of ^{241}Am, even β), whose weights (often called 'relative biological effectiveness' or RBE) are given in Table 1 in the margin. The sensitivity of various *tissue types* to various forms of radiation has been more and more quantitatively understood since the 1950s. See ICRP Publication 119 for details. We must add all the forms of radiation present, and all of the types of tissue present, to quantitatively describe the total *effective* dose to an organism (*e.g.*, a human):

$$H_{tt} = \sum_i w_r D_{t,r} \quad (1)$$

$$E_{eff} = \sum_{tt} w_{tt} H_{tt} \quad (2)$$

The tissue type weights are defined so that $\sum_{tt} w_{tt} = 1$. These tissue type weights are updated by international radiation regulatory bodies such as the International Commission on Radiological Protection (the ICRP). By convention the quantity H_{tt} is called the *tissue equivalent dose* to a particular tissue type; similarly, H_{equiv} (sometimes labeled E_{eff}, as we do above) is called the *effective dose*. As we will see below, both are measured in Sieverts.

It is worth noting that if, for example, we can neglect α particles (because of their very short range, even in air)

It should not be surprising that extremely sophisticated computer codes exist (for example, GEANT) to accurately simulate the passage of particles through matter, with applications from design of CERN particle detectors through detailed medical applications. A downloadable Windows package for computing similar range curves may be found at the SRIM (SRIM = Stopping and] Range of Ions in Matter). It is relevant to technological applications.

radiation type	weighting factor w_r
$\simeq$ 5 MeV α	20
β (electrons)	1
X rays and γ	1
>2 MeV protons	5

Table 1: Weighting factors w_r for radiation types. For more details, consult Wikipedia's excellent document.

'Weapons-grade' plutonium must contain less than about 7% of the isotope ^{240}Pu (which spontaneously fissions and initiates a chain reaction prematurely). This isotope becomes ^{241}Pu upon two neutron captures (I'd guess this occurs only within 'bulk' Pu), however, and with a half-life of about 14 years, ^{241}Pu decays by emitting a β particle to ^{241}Am, which is why the latter is also present around the former Rocky Flats plant.

tissue type	w_{tt}
breast, lung, bone marrow, stomach, colon	0.12
gonads	0.08
thyroid, liver, bladder, esophagus	0.04
skin, brain, salivary glands, bone surfaces	0.01

Table 2: Weighting factors for tissue types, taken from the ICRP 2007 standards

and β particles (because they are absent), then we are left only with γ contributions to doses. Because the 'radiation weighting factor' of γs is 1 *and* the quantity H_{tt} can then be pulled out of the sum in Eq. (2), we discover that the effective dose is then the absorbed dose (provided it is delivered to the entire body). The logical sequence is shown graphically in Fig. 4.

It is important to note that the 'effective dose' was introduced by the International Commission on Radiological Protection to estimate cancer risks to a population, not to an individual. There have been strong criticisms [3, 4] because, in effect, it was designed by a committee with somewhat conflicting, subjective goals. It may eventually be replaced by something more accurately named (based, say, on 'lifetime risk of cancer death per 100,000 people', or even based on age-range specific values. In many ways this is closer to how epidemiological cancer risk data is presented, as you will see in a document elsewhere.

Nonetheless, the discussion above reflects common practice *right now*, and is how, for example, the software tool RESRAD (described elsewhere) would estimate lifetime cancer risks based on radiation exposure.

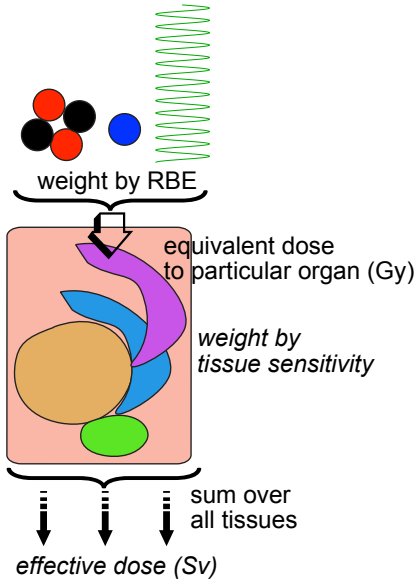

Figure 4: Schematic decomposition into absorbed dose into contributions from radiation types and then contributions to doses to tissue types, culminating in 'effective dose'.

Response of an **organism** *to radiation*

In radiation biology it is common to distinguish between two classes of response to radiation exposure:

1. 'Deterministic' effects, which will certainly occur beyond a particular *threshold* dose level. These include (for example, for human beings) hair loss or radiation sickness, for extremely large radiation doses. Cells must have been *killed* in order for deterministic effects to occur. Cancer radiation therapy produces a deterministic effect: the death of tumor cells.

2. 'Stochastic' effects, in which probabilities alone can predict outcomes, since cells are affected in some way but are not killed outright. Usually the probability of

occurrence of a stochastic effect increases with dose but its severity does not. There is generally no threshold.

The induction of *cancers* of all types is believed to be stochastic. For the very low levels of radiation around the former Rocky Flats plant, all health effects are expected to be stochastic.

For deterministic effects, a variation in the sensitivity of individual humans (due, for example, to a genetic predisposition) will give rise, in a large population, to a curve like the upper in the panel of Fig. 6. Some (very high dose) thresholds (in gray, Gy) are given in Table 3 in the margin.

More about stochastic effects

The overt effects of some radiation exposure disappear eventually: the body 'heals'. It has been known for many decades that the same is true at the level of organs and tissue. Since the 1950s *cellular* repair systems have been studied and it has been possible in the last 20 years to examine individual breaks in DNA molecules due to radiation. For example [5], the average repair time for a single strand break in a DNA molecule in a cell is about 10 minutes, while double strand breaks take several hours.

It is thus extremely likely (more in a separate document) that for doses low enough not to have defined (deterministic) effects there are dose *thresholds* (which may depend on tissue type and radiation type) below which *no* radiation effects occur, because 'free radical' scavengers within a cell can protect even the DNA from damage and mutations.

Distinction: units

If a radiation particle (say, a 250 MeV proton being used as a 'proton scalpel' used for radiation therapy) enters your body, the energy it carried is conserved—it has to go somewhere (ideally, into the tumor). This 'absorbed dose' is conventionally measured in the international unit called the Gray (Gy), which you have encountered above,

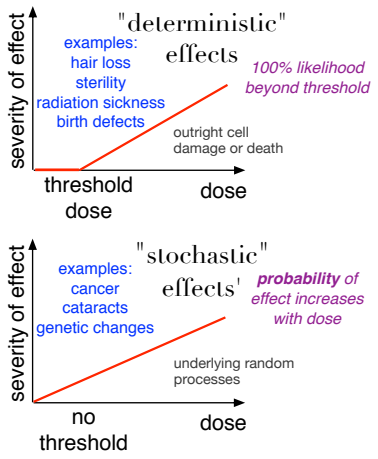

Figure 5: Schematic behavior of 'deterministic' vs. 'stochastic' radiation effects.

Effect	Dose threshold (Gy)
fetal abnormality	0.1-0.5
sterility	2-3
skin reddening	2-5
hair loss	2-5
death	3-5
cataracts	5

Table 3: Deterministic effects of radiation and their threshold dose.

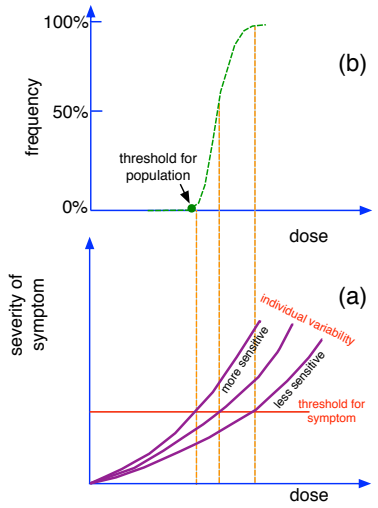

Figure 6: How individual variability in sensitivity to radiation [panel (a)] gives rise to the threshold for a large population, panel (b). Redrawn from Fig. 15 of [6]

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which has units of energy per unit mass (joules per kilogram). Until we know the biological impact of this energy we cannot assess biological harm (such as the risk of induced cancer). Precisely because we have to model how this absorbed dose energy is partitioned among internal organs (as we did above when describing weighting factors for tissue types), it is conventional (and conceptually very important) to use a different *name* for how we measure the health impact of the very same energy. The absorbed dose, when weighted by the biological impact on various tissues (remember that the sum of the tissue-type weights is 1), is measured in the international unit called the Sievert (Sv). They each measure energy in J/kg but the Sievert is meant to reflect biological damage while the Gray measures simply radiation energy absorbed (both, per unit mass).

If we know the energy of each proton and how many protons per second are being beamed into a tumor and the length of time the beam is on, we can compute the dose in Gy, even though this dose occurs inside the body. By contrast, the dose in Sv is our best guess about how effective the beam will be in killing the tumor. Useful Wikipedia entries by Doug Sim, a chart and help distinguish between these two quantities and their units: see also the helpful graphic, again by Doug Sim.

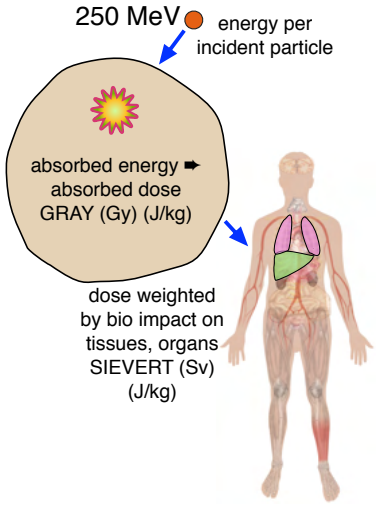


Figure 7: How incident particles of radiation become an absorbed dose (Gy) and an tissue-weighted effective dose (Sv).

Distinction: Committed vs. external dose

As those inundated with dire warnings about the dangers of 'hot particles' already know, it is possible for some radioactive material or gas (such as radon) to be inhaled or swallowed. This becomes an *internal* source of radiation which may not be eliminated from the body. Except under catastrophic circumstances (cleaning up Chernobyl, for instance) there are no deterministic (*i.e.*, guaranteed to occur) effects; this holds for Pu as well as other radionuclides. Thus such internal doses give rise to a 'committed' dose—one we know will be present even in the absence of external sources. If such exposure is important (as

It is estimated [7] that most humans receive a liver dose of 10-20 microsieverts (μSv) per year. About 1 atom per 5000 cells in the body is ^{239,240}Pu.

in the case of nuclear processing plant workers) the radiation source can be permanently fixed in the body. It gives rise to a committed dose which depends on what happens to the radiation source in your body.

Thus the total radiation dose a person has received at a given time is the *sum* of the 'committed dose' up to that time and the sum of all the external doses he or she has received up to that time. Under many situations the lifetime risk of cancer is estimated using 50 years for the committed dose period.

To be precise the committed dose D after a time τ is defined by

$$D(\tau) = \int_0^\tau dt\, r(t),$$

where $r(t)$ is the internal dose rate (say, in Gy per year). If the radionuclide has a long half life, $D(\tau) = r\tau$. Because the Chernobyl accident was monitored in almost real time, it was possible to generate accurate committed dose predictions based on real-time monitoring of radiation levels [8].

In the context of Rocky Flats: We assume we are able to neglect doses due to α particles, which will be true provided no Pu is inhaled—discussed in the context of 'hot particles' later—since α particles cannot reach living tissue except through skin wounds. It is worth noting that *if* all of the radiation is γ rays, there is no 'committed dose' (that is, all radiation is due to *external* gamma rays, and we are interested in whole-body doses (which no longer considers individual doses to internal organs because they have been summed over), then

$$H_{tt} = D_t \quad (3)$$

$$H_{equiv} = D_t. \quad (4)$$

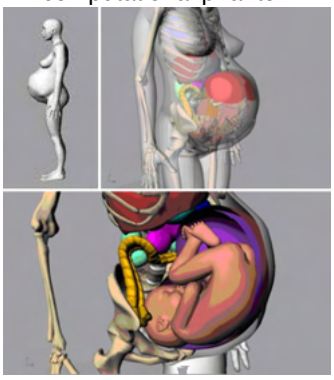
This means that *under these conditions* the dose in grays (Gy) is the same as the dose in sieverts (Sv). We will use this fact when discussing the Rocky Flats situation in a later document.

However, it is not relevant to unshielded nuclear plant workers directly in contact with Pu or who may have inhaled or ingested Pu.

Refinements of dose technology

More recent applications of radiation for therapeutic and diagnostic purposes requires much more precise and systematic data and modeling. As we have seen above, the *effective dose* description lends itself to a more careful accounting of the spatial variations of tissue radiation sensitivity inside a human body. Such approaches have used a 'computational human phantom'—a mathematical and geometrical description of a human body—to estimate how an incident specified radiation beam delivers a dose.

Rendering of RPI 9 month pregnant female computational phantom



RPI Radiation and Dosimetry Group/Wikimedia

Figure 8: Computational phantom used for modeling radiation dose.

Physical phantoms are generally calibrated using small 'dosimeters' (radiation measuring units or film badges) tuned to reproduce the sensitivity of internal organs and placed in a full-sized physical model of a human being (or parts thereof). For example, astronaut radiation exposure in the International Space Station has been treated in this way.

In the 1980s a voxel (volume element) computationally-intensive second-generation approach began. Third generation descriptions include the effects of a breathing human body in addition to organ-specific features, and are available for male and females ranging in age from birth to adulthood. These have been incorporated into ICRP references so are part of international radiation protection and monitoring. See the web page for the CP-2017 workshop or download the program for the ICRP-ERPW 2017 conference for examples of how extremely sophisticated such phantoms have become.

Takeaway messages

- Different forms of radiation penetrate tissue to very different extents.
- Every aspect of prediction about health effects of radiation is statistical.
- The principal risk to human health of exposure to radiation is cancer.
- It is important to distinguish between radiation *exposure* (being in the presence of radioactive substances) and *dose* (the amount of energy from radiation actually absorbed by the body) from the sources.
- The 'effective dose' concept is extremely widely used in assessing the risk of cancer due to radiation exposure. It acknowledges both the different biological impacts of α, β, and γ radiation and the different sensitivities of distinct tissue types. It is used for

  - *Planning* of occupational or public exposure to external radiation sources
  - After-the-fact for *regulatory* purposes to show compliance with established dose limits,

  but is also often used on an individual basis.

- Only with specific assumptions about the radiation type can we readily relate the absorbed radiation energy (measured in Gray) to the whole-body radiation dose (measured in Sieverts). Around Rocky Flats we can assume that almost all radiation exposure is due to γ rays (like high-energy X-rays, but more penetrating), since there are very few β emitters and the range of α particles in air is a few centimeters.

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