22.01 Fall 2015, Quiz #3 Solutions

December 24, 2015

Complete all the assigned problems, and show all intermediate work. Partial credit *will* be given generously for showing the correct approach, even if you can't solve the problem. Make sure to write whatever you can, to show what you really know. Define any variables which you don't know and keep cranking. HINT: Read all questions before starting, to help you budget your time.

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1 Conceptual Questions (10 Points Each)

1.1 Mechanistically explain five ways in which irradiation prevents the early spoilage of food, and/or the spread of harmful agricultural pests.

1. Irradiation can kill insects in high enough doses, though these are very high. This would be the equivalent of inducing neurovascular syndrome in the insects, or the most severe and immediate form of acute radiation poisoning.
2. Irradiation can sterilize insects so they can't reproduce and decimate crops. Only enough radiation has to be applied to destroy all the gametes of the bugs involved.
3. Irradiation can kill cells in the food, stopping their oxidating metabolism and slowing overripening or spoilage, by delaying ripening.
4. Irradiation can kill foodborne pathogens, which may infect people, plauts, or animals without directly spoiling the food (essentially creepy crawly stowaways on food).
5. Irradiation can kill the embryos in many seed- or sprout-based foods, stopping the sprouting process and retaining the quality of the food by preventing germination-related chemical changes.
6. Irradiation can kill the bacteria, molds, and yeasts responsible for foodborne illnesses, but *not* the toxins (like botulism toxin) that they produce.
7. Irradiation can *inactivate*, or neutralize, some of the bacteria present, preventing them from multiplying further.

Radiation *cannot* destroy enzymes, prions, viruses, or bacterial toxins, as the doses would be prohibitively high.

1.2 Suggest three reasonable ways (lead underpants are *not* an option) in which airlines could help flight attendants and pilots incur less radiation dose per unit of flight time. Mention why you choose each one, and state which sources/forms of radiation you are mitigating.

1. Fly at a lower altitude, giving more atmospheric shielding between the crew/passengers and the gammas produced by neutral pion decay in the atmosphere, as well as any other cosmic rays which make it through the atmosphere.
2. Wrap the plane in a thin foil of lead, to block as many of the gamma rays (same source as (1) as possible.
3. Either decrease the cabin pressure or fill the plane with radon-free air, to remove as much of the radon present in the air as possible. This would be a rather negligible reduction.

1.3 Studies still disagree on whether second-hand smoke is actually dangerous. Explain from a radiological point of view why you think it is/isn't (choose a side), and set up an equation to estimate the increase in equivalent dose from living with a smoker.

Second-hand smoke still contains most of the radon precursor products (mostly radium) that deposit on/in tobacco leaves, which are dried and concentrated. Some of these particles enter the lungs of the smoker, while some will others will either be expelled as second-hand smoke, or directly emitted from the burning cigarette. Many of these particles hang out in the air, or deposit on free surfaces, releasing radon when the radium decays. This greatly increases the radon dose indoors, particularly those with poor ventilation (well insulated buildings, first floors, basements).

An equation to estimate the increase in equivalent dose from living with a smoker would take into account the following factors:

1. The existing radon background rate
2. The number of cigarettes smoked per unit time
3. The total radium content per cigarette (as regulars, slims, cigars, whatever other kinds of smokeables), as that will vary based on its size, source, etc.
4. The fractional refresh rate of the air in any unit (let's say L/min) divided by the volume of the room.
5. The radiation quality factors of the various radon decay products (Q=20 for α, Q=1 for β^-)
6. The tissue quality factor for the lungs ($w_T = 0.12$)
7. The energy per decay product

An example equation to get the radon concentration in the air would be built something like this:

$$\frac{d\left(N_{Rn-222}\right)}{dt} = \left[\frac{\left[\lambda_{Ra-226} \underbrace{\frac{\left(\frac{Cigarettes}{second}\right)\left(\frac{Atoms\ Ra-226}{Cigarette}\right)(Fraction\ Ra\ expelled)}{V_{Room}}}_{N_{Ra-226}} \right] - \lambda_{Rn-222} N_{Rn-222}}{1 + \frac{Refresh\ Rate}{V_{Room}}} \right] \tag{1}$$

When solved for the steady-state radon concentration *(Rn)* in the air, the rest of the equation proceeds exactly like the homework question concerning the internal dose exposure rate from radon inhalation:

$$\dot{Sv} = (Rn\,Conc.)\,(Decay\,rate\,in\,lungs)\left[\sum_{i=1}^{n} E_i Q_i\right] w_{T,lungs} \tag{2}$$

where i represents each of the radon decay products up to the long-lived one (Pb-210). You don't have to have any isotopes memorized to get full credit, just know that radon comes from some solid parent isotope, and has a bunch of α and β^- decaying isotopes.

1.4 Set up, but do not solve, an expression for the equivalent dose rate in $\frac{Sieverts}{second}$ for a person standing at a distance D from a point source of gamma activity A_γ. Define any variables you need to complete the expression.

A person of surface area S_{human} standing at a distance D from a point source of gamma activity A_γ first sees the following fraction of gamma rays emitted from the point source:

$$f_{\gamma-solid-angle} = \frac{\Omega}{4\pi} = \frac{S_{human}}{4\pi D^2} \tag{3}$$

Then we must account for the fraction of gamma rays attenuated by the air in between the point source and the person:

$$f_{air-attenuation} = e^{-\left(\frac{\mu}{\rho}\right)_{air}\rho_{air}D} \tag{4}$$

Then we account for how many gamma rays attenuate inside the person, causing them to accrue dose:

$$f_{human-attenuation} = 1 - e^{-\left(\frac{\mu}{\rho}\right)_{human}\rho_{human}x_{human}} \tag{5}$$

where x_{human} is the thickness of the person. Finally, we multiply by the energy of the gamma ray E_γ, assume that all interactions occur via the photoelectric effect (to avoid dealing with fractions of Compton scattering or pair production, a conservative assumption), multiply by the quality factor for gamma rays (1) and the tissue equivalency factor (1), and divide by the mass of the person:

$$\dot{Sv} = (A_\gamma)\left(\frac{S_{human}}{4\pi D^2}\right)\left(\frac{\left(e^{-\left(\frac{\mu}{\rho}\right)_{air}\rho_{air}D}\right)\left(1 - e^{-\left(\frac{\mu}{\rho}\right)_{human}\rho_{human}x_{human}}\right)E_\gamma Q_\gamma w_{T,body}}{m_{human}}\right) \tag{6}$$

1.5 Rank the following types of cells in order of increasing radiation sensitivity, based on your knowledge of acute radiation effects: Bone marrow cells, cerebral neurons, endometrial (blood vessel liner) cells, hair follicle cells, intestinal cells in villi.

Looking at the tables of acute radiation symptoms, the most sensitive cells will feel the largest effect in the shortest amount of time given the same dose. Therefore, the ranking proceeds as follows, from least to most sensitive:

$$neurons < villi < hair < endometrial < marrow \tag{7}$$

Neurons are only affected by cerebrovascular syndrome, which is caused by the highest dose. Next, the onset of diarrhea only starts in the severe phase, which would indicate killing or stopping reproduction of villi cells. Next, depilation (hair loss) starts in the moderate dose range. Finally, blood cell counts go down even with a mild dose, making them the most sensitive.

1.6 Qualitatively and mechanistically explain the observed trend (below) in G-values as a function of chemical species and particle energy, using your knowledge of charged particle creation, diffusion, and stopping power:

Table 13.3 *G* Values (Number per 100 eV) for Various Species in Water at 0.28 μs for Electrons at Several Energies

Species	Electron Energy (eV)							
	100	200	500	750	1000	5000	10,000	20,000
OH	1.17	0.72	0.46	0.39	0.39	0.74	1.05	1.10
H_3O^+	4.97	5.01	4.88	4.97	4.86	5.03	5.19	5.13
e^-_{aq}	1.87	1.44	0.82	0.71	0.62	0.89	1.18	1.13
H	2.52	2.12	1.96	1.91	1.96	1.93	1.90	1.99
H_2	0.74	0.86	0.99	0.95	0.93	0.84	0.81	0.80
H_2O_2	1.84	2.04	2.04	2.00	1.97	1.86	1.81	1.80
Fe^{3+}	17.9	15.5	12.7	12.3	12.6	12.9	13.9	14.1

Table 13.4 *G* Values (Number per 100 eV) for Various Species at 10^{-7} s for Protons of Several Energies and for Alpha Particles of the Same Velocities

Species	Protons (MeV)				Alpha Particles (MeV)			
Type	1	2	5	10	4	8	20	40
OH	1.05	1.44	2.00	2.49	0.35	0.66	1.15	1.54
H_3O^+	3.53	3.70	3.90	4.11	3.29	3.41	3.55	3.70
e^-_{aq}	0.19	0.40	0.83	1.19	0.02	0.08	0.25	0.46
H	1.37	1.53	1.66	1.81	0.79	1.03	1.33	1.57
H_2	1.22	1.13	1.02	0.93	1.41	1.32	1.19	1.10
H_2O_2	1.48	1.37	1.27	1.18	1.64	1.54	1.41	1.33
Fe^{3+}	8.69	9.97	12.01	13.86	6.07	7.06	8.72	10.31

First, group all the primary species (those directly made by the splitting of water by radiation) into one group, and notice how they all follow the same patterns with increasing electron energy. First the G-values are high, because electron stopping powers are quite low at very low energies. This results in a less dense charged particle track, even though very few charged particles are made on an absolute sense. Next, at higher energy (˜1000eV), stopping powers increase, causing more ionizations per unit length, and more chemical reactions due to denser reactions before diffusion can spread the species out. Finally, at much higher energies, the stopping power decreases again, so even though lots of charged particles are made per unit energy, the density of those particles is low.

Now look at H_2 and H_2O_2, which are secondary products made by the reaction of primary species. These follow the exact opposite trend, showing how when charged particle tracks are denser, more reactions take place to create secondary species before dffusion can spread them out.

2 Analytical Questions (20 Points Each)

2.1 Set up, but do not solve, a complete set of equations to calculate the added risk of long-term radiation effects (cancer, mutations) for a person standing and permanently living within 1 km of an atomic bomb detonation delivering a 1 MeV neutron flux of Φ_n and a 1 MeV gamma flux of Φ_γ. Consider all possible sources of elevated radiation exposure that would result from being in close proximity to an atomic bomb blast, and how they would become incorporated in the human body. Define any symbols you need to represent quantities which you do not know: Numbers aren't important here, concepts are!

For this problem, consider all the potential sources of increased radiation exposure, including the prompt gamma flux, the prompt neutron flux, radioactive fallout, and continued modes of intake/ingestion of fission and decay products. Living within 1 km would certainly incur added background doses and fallout. First, the acute gamma dose, which are applied as whole-body doses:

$$Sv_{acute,\gamma} = \Phi_\gamma \left(\frac{S_{human}}{4\pi D^2} \right) \left(\frac{\left(e^{-\left(\frac{\mu}{\rho}\right)_{air} \rho_{air} D} \right) \left(1 - e^{-\left(\frac{\mu}{\rho}\right)_{human} \rho_{human} x_{human}} \right) E_\gamma Q_\gamma w_{T,body}}{m_{human}} \right) \tag{8}$$

where D is 1 km in the unit of choice. This part is strikingly similar to problem 1.4! The energy of the gammas is 1 MeV as stated in the problem, while the quality and tissue factors are unity. Next, the acute neutron dose:

$$Sv_{acute,n} = \Phi_n \left(\frac{S_{human}}{4\pi D^2} \right) \left(\frac{\left(e^{-\Sigma_{air} D} \right) \left(1 - e^{-\Sigma_{human} x_{human}} \right) E_n Q_n w_{T,body}}{m_{human}} \right) \tag{9}$$

where instead of gamma attenuation factors, macroscopic cross sections (Σ) are used. The units all work out here! In this part the tissue quality factor is still unity, while the radiation quality factor is 20 according to the tables attached. Now onto the long-term effects: Consider both the elevated, externally applied (full-body) background dose, and any internal routes of exposure. For example, these could include iodine from milk or seaweed to the thyroid, airborne α/β^- emitting fallout in the lungs, heavy metals to the brain, etc. It isn't important which organs/routes you choose, as long as they take the following general form:Ex_

$$Sv_{background} = \sum_{i=1}^{n} \left[\int_0^{t_{lifetime,human}} \left(C_i(t) - C_{i,background} \right) \frac{\lambda_i E_{decay} Q_\gamma w_{T,full-body}}{m_{human}} dt \right] \tag{10}$$

where *C(t)* is the concentration of the isotope i in question, with the normal background level of that isotope, λ is its decay constant, and all other quantities are as described above. Note how the first two terms take the form of an increased activity over background (λN), while the rest takes that activity and converts it into a dose in Sieverts. The increased dose to fallout takes a rather similar form, while noting a couple of key exceptions:

1. Some of the fallout isotopes have finite ingestion and excretion rates in addition to decay, let's call them In_i and Ex_i
2. Each isotope acts on the organ which concentrates it, and only blasts the mass of the organ involved for modes other than gamma decay

Therefore, it would take the following form:

$$Sv_{fallout} = \sum_{i=1}^{n} \left[\int_0^{t_{lifetime,human}} \left(In_i(t) - Ex_i \right) \frac{\lambda_i E_i Q_i w_{T,organ\,i}}{m_{organ\,i}} dt \right] \tag{11}$$

Then, the total increased risk of cancer as a function of time is the sum of these time-dependent increases in radiation dose, multiplied by the fractional increase in risk per unit dose:

$$\%\, Risk\, Increase = \sum_{i=1}^{n} Sv_i RiskRate_i \tag{12}$$

This keeps in mind that dose rate effects are real, meaning that the rate of radiation dose accrual can change the level of increased cancer risk somewhat.

2.2 Set up, but do not solve, a complete set of equations to determine the acute, equivalent dose to which a person was exposed by taking time-dependent measurements of their white blood cell count. Graph what this function would look like for any general case starting at the time of acute radiation exposure, and point out the major features, slopes, or other parameters of this graph. (HINT: Also graph the concentration of cells which produce white blood cells, and think back to our discussion of isotope production & decay)

First, we will need to set up an equation for the number of white blood cell-producing marrow cells. After an acute radiation exposure, some fraction of these will be instantly killed, while some fraction will be sterilized (unable to reproduce), but still keep producing white blood cells until they die naturally. Assuming the marrow cell fraction never hits zero, we can write an equation for the number of marrow cells $[M]$:

$$\frac{dM}{dt} = \underbrace{-f_s \lambda_M M}_{Decay\ due\ to\ natural\ death} + (1-f_s)\underbrace{aM\left[1-\frac{M}{M_{max}}\right]}_{Logistic\ growth}; \quad M(0) = M_0 \tag{13}$$

This accounts for the fact that some fraction of marrow cells M_0 immediately survive the radiation onslaught, while some further fraction f_s become unable to divide, but still able to produce white blood cells. Meanwhile, marrow cells which weren't sterilized $(1-f_s)$ grow with their characteristic doubling time a, and yet they cannot exceed their maximum concentration M_{max}, which was the old, pre-irradiation steady state concentration.

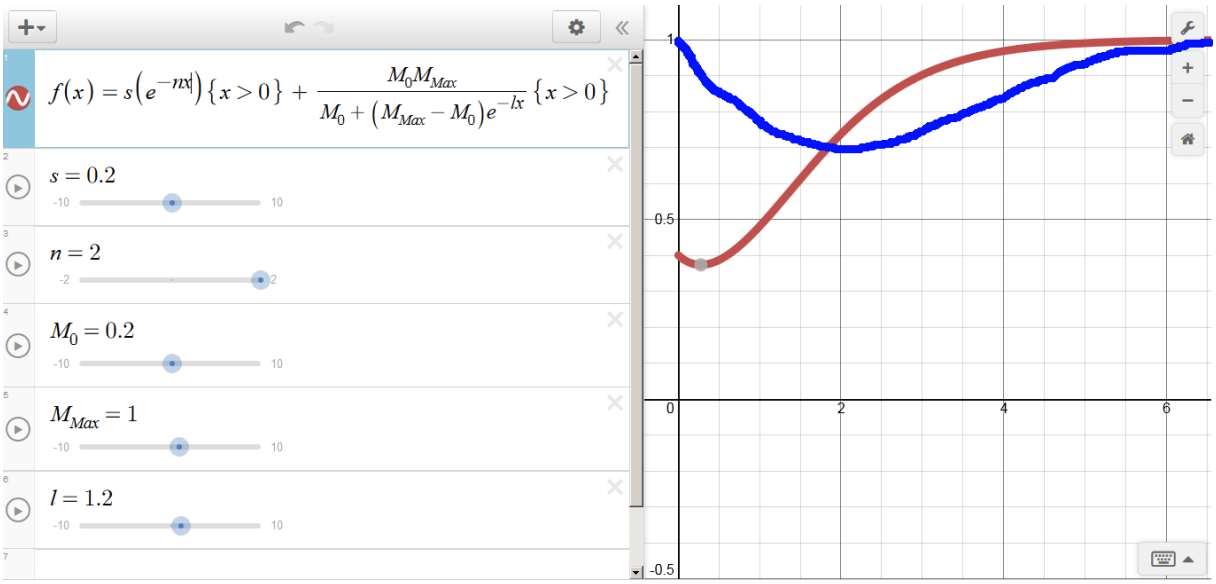
Then, we can assume that the production of white blood cells $[W]$ is proportional to the existing population of marrow cells $[M]$, while the removal term for white blood cells dies off with some average lifetime, or decay constant, λ_W. We then get a differential equation as follows:

$$\frac{dW}{dt} = bM - \lambda_W W \tag{14}$$

Finally, we recognize that both the fraction of marrow cells killed immediately $(1-M_0)$ and the fraction of marrow cells sterilized (f_s) should be directly proportional to the dose of radiation applied, according to some *linear threshold* model at its simplest:

$$(1-M_0) = cD; \qquad f_s = dD \tag{15}$$

where D is the dose incurred by the person. The graph of the marrow cell count $[M]$ is graphed in red as follows:



Don't worry if you didn't solve the equation, you don't have to! The important part is to note that some fraction of cells die immediately, then a few more decay away, while the rest bounce back to settle at a normalized concentration of unity via a logistic equation. Just like the Bateman equations (the series decay equations), the slope of the white blood cell count should mirror the value of the marrow cell count at that time. This function is graphed in blue.

Note how as soon as there are fewer marrow cells to make up for the steady-state white blood cell death rate, they start to die off at a rate proportional to $(1 - M(t))$. As $M(t)$ recovers, then white blood cells also start to regenerate. That's why the steepest part of the blue curve is at the zero-derivative point of the red curve (that's not a Microsoft Paint goof, it's intentional!)

22.01 Introduction to Nuclear Engineering and Ionizing Radiation
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