Compensating Radiation Harm

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Underwriter Certification, our proposed system for regulating nuclear power, requires a firm, automatic procedure for compensating people who have been harmed by a release of radioactive material. If humanity is to enjoy the benefits of nuclear power, it is essential that this system be reasonably realistic. It must be at least qualitatively correct, reflecting the essentials of what happens when radioactive particles interact with a human. The system must be well enough defined that underwriters can reliably estimate their loss associated with a given release. Before we can come up with such a system, we need a little background.

1 Radiation Damage and Repair

The overriding safety concern about nuclear power is the health hazard associated with a release of radioactive material. We have been told over and over that any such release is a catastrophe. But we live in a sea of radiation. Depending on where you are reading this, in the last minute your body has absorbed between 1 and 10 million particles with enough energy to produce cell damage. Life evolved in an environment where the natural level of radiation was 5 times higher than it is now.[7] If radiation is so damaging, why are we here?

The answer is life evolved a system, an extraordinarily clever system, for handling this onslaught. The system is so automatic that we are unaware of it. For many hazards, evolution developed sensors and responses, so we can react to a danger. Too much heat will destroy tissue. So we developed nerves that sense temperature and send a signal called pain to the central nervous system that tells us "stop touching, get away". But there's no getting away from radiation. So evolution went with a system that repairs radiation damage without our needing to do anything. This system can be overwhelmed if the dose rate is high enough.

When it comes to cancer, the main concern is DNA damage. Living tissue is made up of cells. Cells are mostly water. If a radioactive particle enters a cell, it transfers a portion of its energy to the cell mainly by breaking the chemical bonds that hold the water molecule together. This creates highly reactive, *free radicals* which can disrupt the cell's chemistry including damaging the cell's DNA.

Most of the DNA damage is single strand breaks, in which only one side of the double helix is disrupted. Single stand breaks are astonishingly frequent, tens thousand per cell per day. Almost all these breaks are caused by free radicals produced by the cell's own metabolism. These are repaired almost automatically by the clever structure of the DNA molecule itself, with the undamaged side serving as a template.

But occasionally we get a double strand break (DSB). It's the DSB's that can start the process that results in cancer. Cell metabolism generates a DSB about once every ten days per cell. Average natural background radiation creates a DSB about every 10,000 days per cell.[4] However the break was caused, the DNA molecule is split in two.

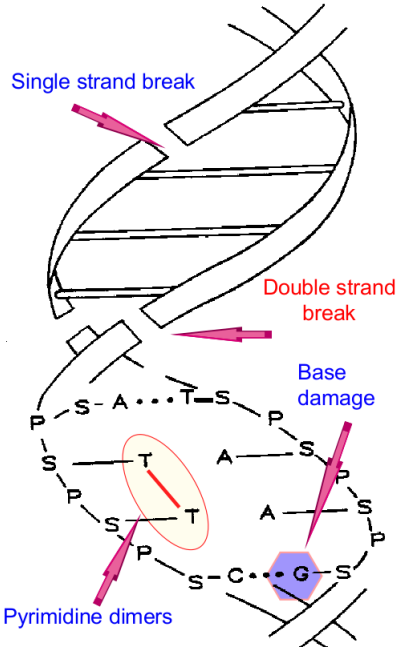

Figure 1: DNA damage scenarios

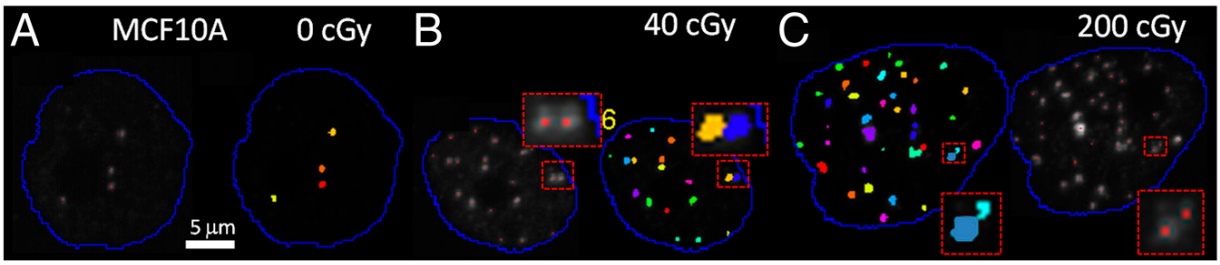

Figure 2: UCB pictures of cell repair. The bright spots in the three screenshots are clusters of damage sensing and repair proteins, dubbed Radiation Induced Foci (RIF). Berkeley found that the number of RIF's increases less than linearly with dose, which they measured in units called grays (Gy). At 0.1 Gy, they observed 73 RIF's/Gy. At 1.0 Gy, they saw 28 RIF's/Gy. If an RIF is faced with a single DSB, the repair is almost always correct. If an RIF is faced with more than one DSB, the error rate skyrockets. We expect 25 to 40 DSB's per gray. Do the math. 4 DSB's and 7.3 RIF's, no problem. 40 DSB's and 28 RIF's, trouble.

Experiments show that double strand breaks result in RIF's, clusters of damage sensing and repair proteins. But the number of RIF's does not increase linearly with the dose rate. As long as the number of RIF's is as large as the number of DSB's, there is very little unrepaired damage. But if the number of DSB's is larger than the number of RIF's, the amount of unrepaired cells goes up drastically. By tagging the ends of the break, Berkeley actually has pictures of the DSB repair process, Figure 2, which is largely complete in about 2 hours for doses below 100 mSv and 10 hours for doses around 1000 mSv.[1]

A few of these unrepaired cells will survive and a few of those will result in a viable mutation that will eventually result in cancer. This process is both highly non-linear and critically dose rate dependent. Society needs a reasonable model of radiation harm to properly set the compensation for the dose received in a radioactive release. Such a model must capture both the non-linearity and the importance of the repair period. ***What counts is the dose received within the repair period.***

[1] Radiation dose is the amount of energy deposited in tissue in joules per kilogram of tissue. The shorthand for joules per kg (J/kg) tissue is called a gray (Gy). A modified unit called a sievert (Sv) adjusts the gray by a factor that attempts to account for how damaging that particular form of radiation is. The adjustment factor is 1.0 for the types of radiation that are important to the public in a nuclear power plant release. For present purposes, grays and sieverts are numerically equal. I will use grays when talking about ambient dose rates measured at a location, and sieverts when talking about the dose that is actually absorbed by a person.

We will also need to distinguish between *acute* doses, doses received over a very short period, and *chronic* doses, doses which are spread out over multiple repair periods.

2 How good is our radiation repair systems?

Effective radiation dose is measured in units called millisieverts (mSv). Table 1 summarizes most of the cases where people have received far above normal background doses.[2]

Table 1: High Dose Populations.
Single acute dose above top horizontal line; repeated doses below. Belarus/Ukraine kids: thyroid dose

Group	Size	Period	Cumulative dose mSv	Dose rate mSv/day	Result
Bomb survivors	33,459	seconds	5 to 150	5 to 150	Insignificant decrease in leukemia
Bomb survivors	5,463	seconds	150 to 300	150 - 300	Insignificant increase in leukemia.
Bomb survivors	6,793	seconds	300-5000+	300-5000+	Significant increase in leukemia.
5-20	14,555	seconds	5 to 20	5 to 20	Insignificant decrease in solid cancers.
20-40	6,411	seconds	20 to 40	20 to 40	Solid cancers same as control
40-125	10,970	seconds	40 to 125	40 to 125	Insignificant increase in solid cancers.
125+	16,166	seconds	125+	125+	Significant increase in solid cancers.
Louis Slotin	1	seconds	21000	21000	Died in 9 days
H. Daghlian	1	seconds	5900	5900	Died in 25 days
Norway tech	1	< hour	38500	38500	Died in 13 days
Tokaimura	3	seconds	3000-17000	3000-17000	>10,000 mSv died
Goiania	≈46	hrs or less	1000-6000	1000-6000	50% mortality abv 4000 mSv
Thai scrap	≈10	hrs or less	1000-6000	1000-6000	100% mortality abv 6000 mSv
Chern 1st responders	134	<2 hrs	1000-16000	1000-16000	Sigmoid mortality, 50% mortality at 6000 mSv.
Chernobyl liquidators	220,000	2 min to 90 days	1-1500	nil to 1500 most < 2	Low/high dose rate mushed together. 6% increase in cancer. Decrease in mortality.
Litvenko	1	3 weeks	96,000	4,000	Died in 23 days
Belarus kids	13,127	2-3 weeks	ave 780 max 48k	39-2400	45 thyroid cancer, eventual 50? deaths
Ukraine kids	11,611	2-3 weeks	ave 560 max 33k	28-1600	87 thyroid cancer, eventual 50? deaths
Eben Byers	1	2 years	366,000	300	Horrible bone cancer. Died in 3 years.
Evans radium hi	127	10 years	>80000	80+	Cancers. Hi mortality >200 mSv/d
Dial painters hi	273	up to 15 yrs	190000-440000	35 to 80+	96 bone cancers
Evans radium mid	17	10 years	20000-80000	20 to 80	Abnormalities. Nil clinical symptoms.
Dial painters lo	2,110	up to 15 yrs	200 - 160000	up to 30	Zero bone cancers.
Evans radium lo	59	10 years	up to 20000	max 20	Nil abnormalities.
Albert Stevens	1	20 years	61,000	8	Died at age 79 of heart failure.
UPPU Club	26	≈10y	up to 7200	0.03-2	Lower mortality than coworkers.
Taipei Apt hi	1,100	18 years	up to 4000	up to 3	Decrease in cancer, maybe non-rad.
Taipei Apt mid	900	18 years	ave 420	up to .160	Decrease in cancer, maybe non-rad.
Taipei Apt low	8,000	18 years	ave 120	up to .050	Decrease in cancer, maybe non-rad.
Keralans	69,956	10-15 yrs	50-650	.016 to .160	Insignificant decrease in cancer
NRX Clean Up	≈1000	90s jumps	up to 200	up to 150	Insignificant decrease in cancer

Table 1 indicates that daily doses below roughly 20 mSv show no effect, even when those daily doses are repeated over a decade or more. Below 20 mSv/day, it almost does not matter

[2] The book *Why Nuclear Power has been a Flop* describes most of these examples in some detail.

what the cumulative dose is. Our repair system is remarkably effective, as long as it is not overwhelmed. We must reflect this in our release harm model. However, once the daily dose gets much above 30 mSv, we start to see an increase in cancer. Our harm model must capture this as well.

3 S-shaped Mortality Curves

On the night of April 25, 1986, Unit 4 of the Chernobyl nuclear power station exploded. The reactor was a water cooled, graphite moderated design originally designed for weapons plutonium production. There was no radiation containment structure.

Much worse, the design was unstable. It is not difficult to build inherently stable reactors, reactors in which any increase in temperature automatically decreases power output. This decrease does not depend on operator or control system action. It is part of the reactor physics. All commercial reactors built in the west and all commercial reactors currently being built anywhere have this property. But with the Chernobyl design, it was possible to put the reactor in a state where an increase in temperature, increased power, further increasing temperature, creating a run away power excursion. The Chernobyl explosion was a nuclear power disaster in the same way the Hindenburg was an air transportation disaster. It showed us how not to do it.

Figure 3: Chernobyl Unit 4.

In the explosion at Chernobyl over a hundred plant workers and first responders received doses of 1000 mSv or more in a few hours or less. 134 were treated for Acute Radiation Sickness (ARS). 28 of these men died. ARS kills by messing with the immune system. The blood forming cells in bone marrow stop or cut production depending on the dose. The immune system can't function, and deadly infections follow. If the dose is less than about 5000 mSv, the bone marrow will normally recover. It typically takes about 3 or 4 weeks for the marrow cells to resume production. If the victim survives for more than about 30 days, then a full recovery can be expected.

Figure 4 plots the death rates of the 134 Chernobyl ARS victims against dose.[11][page 58]

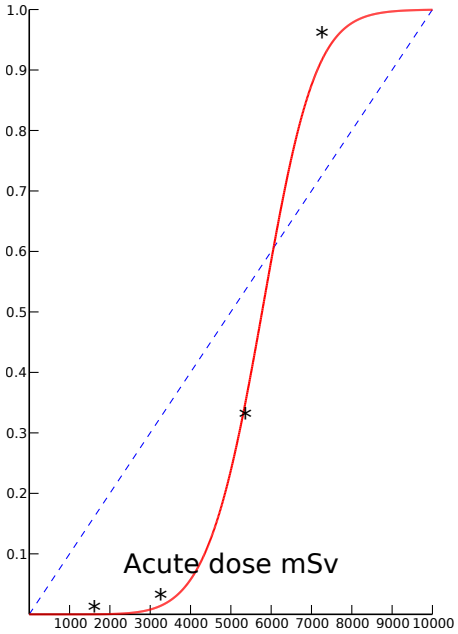

Figure 4: Chernobyl ARS deaths as a function of acute dose

The mortality curve, Figure 4, is S-shaped. Below about 4000 millisieverts and above about 6000, the curve is quite flat. This reflects the fact that a probability cannot be smaller than 0.00 nor larger than 1.00. To put it another way, a smooth dose-response curve must have a slope of zero at 0.00 mortality and a slope of zero at 1.00 mortality. In between, the curve can be fairly steep. In the Chernobyl data, the curve rises by 0.3 in the 2000 to 4000 mSv interval and another 0.6 in the 4000 to 6000 mSv interval. ***Using S-shaped curves to model the response to any toxic material is standard medical practice.***

If a student in Biology 1.01, attempted to fit a straight line, such as the dashed blue curve, to this data, he would be rewarded with an F.

If the dose is much below 1000 mSv, even if it is received in a short period, we do not see Acute Radiation Sickness. However, as Table 1 indicates, daily doses much above 30 mSv result in increased cancer incidence. We need to put numbers on this cancer harm.

The atom bombs that were dropped on Hiroshima and Nagasaki produced a large population of people, who suffered high to extremely high doses in less than the cellular repair time. 86,611 atom bomb survivors have been studied and their cancer incidence and mortality estimated as a function of dose.

Figure 5 fits an S-shaped curve to the atom bomb survivor data. The technical details of this fit are outlined in Appendix A. In all the nuclear power plant releases to date including Chernobyl, daily doses to the public almost never exceeded 5 mSv and almost always were less than 1 mSv. So we need to focus on the low end, which is also where most of the data is. Our S is very lopsided, with the low end hook much smaller than the high end; but the low end hook is definitely there. The people who received between 20 and 40 mSv have the same cancer incidence as the control group. The 5 to 20 mSv group showed a slight reduction in cancer relative to the control group.

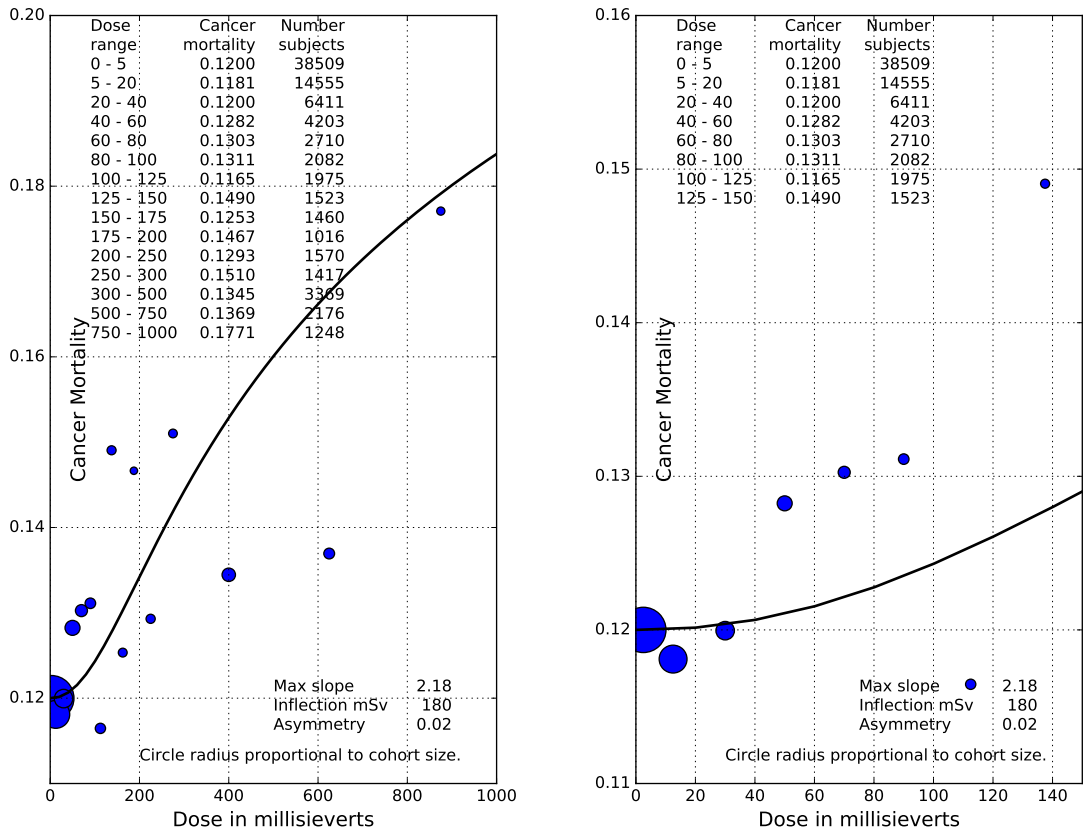

Figure 5: S-shaped Curve Fit to Bomb Survivor Cancer Data. Left: 0 to 1000 mSv. Right: 0 to 150 mSv. At the low end, reducing the dose by a factor of two, reduces the harm by a factor of 4.5. See Appendix A.

4 Handling Chronic Dose Profiles

An increase in cancer mortality is life shortening. We can use mortality tables to estimate the average amount of life shortening for a given individual associated with a fatal cancer. This is called his Lost Life Expectancy or LLE. For Americans as a whole, the LLE associated with a fatal cancer is about 12 years. This is on the high side worldwide. More sophisticated estimates based on age, sex, etc. are possible; but for now to keep things overly-simple, let's assume the LLE associated with a fatal cancer is 12 years.

Once you have the cancer LLE number, it is an easy matter to estimate the LLE associated with any given increase in cancer mortality. For example, the LLE associated with an increase in cancer mortality of 0.01 is 0.01 * 12 = 0.12 years or about 44 days.

The atom bomb doses were a one-shot affair, essentially all the dose was received within a single repair period. In the jargon, these are called *acute* doses. But in a nuclear power plant release, the dose is spread over weeks and months and years, many repair periods, at least for the public. These are called *chronic* dose profiles. How can we use our acute dose harm data to estimate the harm from chronic doses?

Here's one possibility:[3]

1. Assume an overly long legal repair period. We know most of the intra-cellular mechanisms operate on time scales of ten hours or less. Radiotherapy effectively assumes a repair period of a day or two in fractionating very high dose rates, far higher than the public will see in a release. If we assume a regulatory repair period of a day, we are being conservative.
2. Apply our S-shaped harm fit to each such legal repair period separately, assuming incorrectly that all the radiation received in that period is received as an acute dose at the start of the period. This conservative fabrication allows us to use our acute dose harm curve to (over-)estimate chronic dose harm.
3. Calculate the Lost Life Expectancy associated with the dose in each repair period.
4. Add the individual repair period LLE's up.

This procedure is called the Sigmoid No Threshold (SNT) model of radiation harm. SNT captures both the non-linearity in the response, and the fact that essentially all the damage will be repaired, as long as the dose rate is not too large.

[3] Allison has suggested a similar procedure.[1]

Table 2 shows the SNT Lost Life Expectancy associated with a single repair period dose, assuming a fatal cancer shortens life by 12 years.

The Sigmoid No Threshold model has several important implications:

Fatal cancer LLE=12 years	
Repair period dose mSv	SNT Loss of Life Expectancy Days
0.12	0.0000092
0.25	0.0000455
0.50	0.0002062
1.00	0.0009343
5.00	0.0312002
7.00	0.0649561
10.00	0.1412820
15.00	0.3414995
20.00	0.6381213
25.00	1.0351992
30.00	1.5353158
40.00	2.8494991
50.00	4.5815432
80.00	12.1303065
100.00	18.8424029
200.00	62.2848524
300.00	106.2430770

Table 2: SNT LLE's.

There is a cumulative effect. The model treats each day as a independent event. Thus the LLE's add. However, we are adding LLE's, not doses. If the dose in day 1 is 25 mSv, and in day 2 is 10 mSv, and in day 3 is 5 mSv, then we can add the LLE's of each of those days to end up with $1.035 + 0.141 + 0.031 = 1.208$ days.[4] This is quite different from the LLE associated with an acute dose of $25 + 10 + 5 = 40$ mSv or 2.849 days.

Suppose a person lives in a area which has a high background dose rate of 7.3 mSy/y. Then his weekly dose is 0.02 mSv, which according to Table 2 has an LLE of 0.00000018 days. If he lives in this area for 80 years (29,200 days) the model claims his LLE will be $29,200 \cdot 0.00000018 = 0.005$ days. The Sigmoid No Threshold model is consistent both with the fact that we can't see any increase in cancer incidence in high background dose areas, and the fact that an ***acute*** dose of much more than 50 mSv will generate observable increases in cancer.

Under SNT, dilution is an effective countermeasure This is true, even if in doing so, we increase the exposed population proportionally. If we are able to dilute from a single person dose of 50 mSv's down to a dose of 1 mSv, at the cost of increasing the exposed population by a factor of 50, the collective LLE goes from 2.48 days to $50 \cdot 0.0005 = 0.025$ days. If a necessary task requires significant radiation exposure, spread it over a large number of people.

The importance of buffer zones. At repair period doses of much less than 100 mSv, the SNT repair period harm drops off at little more than the square of the repair period dose. See Appendix A. To first order the dose rate drops off as the square of the distance from the source, at least for the first few kilometers. Combining these two factors, near the plant doubling the distance will on average reduce the harm by close to factor of 20.

[4] Actually, SNT combines each day's harm probabilisticly assuming independence. This means each day must be weighted by the probability of no mortal cancer up to that time. In these examples, that probability is very nearly 1.00.

At Chernobyl, the closest permanent residence to the plant was about 3 kilometers from Unit 4. At Fukushima, the closest permanent residence to the units that had containment breaches was more than 1 kilometer away. The non-evacuation LLE numbers would have skyrocketed, if a sizable number of people had been living closer to the damaged reactors.

At Chernobyl at the time of the explosion, two off duty workers were fishing in the plant's discharge channel, taking advantage of the fact that fish are attracted to the warm water. It is unlikely they were much less than 500 meters from Unit 4. They exhibited ARS symptoms, vomiting, tanning, and had to be treated. Probably received an acute dose of 1000 mSv or more. Their SNT cancer Lost Life Expectancy was at least 279 days each. Under the plan we are proposing, their compensation would be at least $100,000 each.

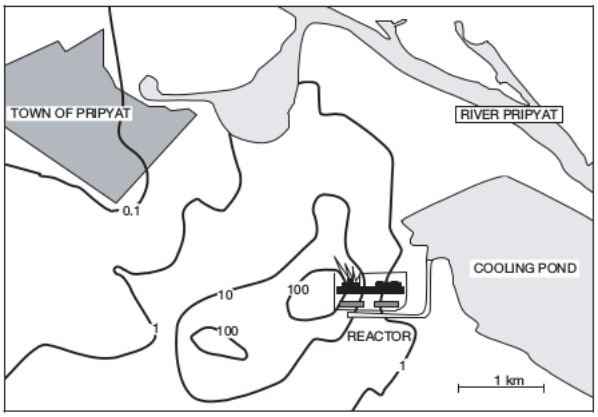

FIG. 6.3. Measured exposure rates in air on 26 April 1986 in the local area of the Chernobyl reactor. Units of isolines are R/h (1 R/h is approximately 0.2 Gy/d) [6.12].

Figure 6: Air dose rates next to reactor.[10] Multiply R/h by 8300 to get μGy/h.

There must be a buffer zone around any nuclear reactor. For a large reactor, there should be at least two kilometers to the nearest non-plant residence or high density workplace.[5]

At Fukushima, the dose rates fell off by more than a factor of ten in the first kilometer. At Chernobyl, the 1 kilometer fall off was at least a factor of 10 in all but the downwind direction, Figure 6. Directly downwind the fall off was a factor of 10 in two kilometers.[6]

The requirement for a buffer zone can be turned into a plus.

1. Most nuclear plants are on the water. In the 1960's, the California State Resources Agency strongly supported nuclear. They realized that the buffer zones could be turned into state parks and beaches, assuring public access to the ocean.[13][p 123] That shore front was protected from private development. Nuclear power plants need not use up shoreline; they can preserve it.
2. Buffer zones can replace Emergency Planning Zones (EPZ). EPZ's are areas within which evacuation drills must take place. Holding up those drills was an effective delaying tactic of the anti-nukes in the late 1970's.
3. Buffer zones will make it much easier for weak politicians to not call for an evacuation.

[5] Some industrial processes can be in the buffer zone. Obvious candidates include electricity intensive, labor extensive processes such as CO2-free metals manufacture, hydrogen production, and desalination.

[6] In a totally uncontained release combined with an explosion such as Chernobyl, highly radioactive chunks of non-volatile isotopes will be ejected from the reactor. However, these will fall out almost immediately. We need almost all that fallout to be in the buffer zone.

They can instead tell people to leave the buffer zone, go home, and shelter in place. They've done something. According to the EPA, shelter-in-place can put your dose rate by a factor of ten or more, Figure 7.

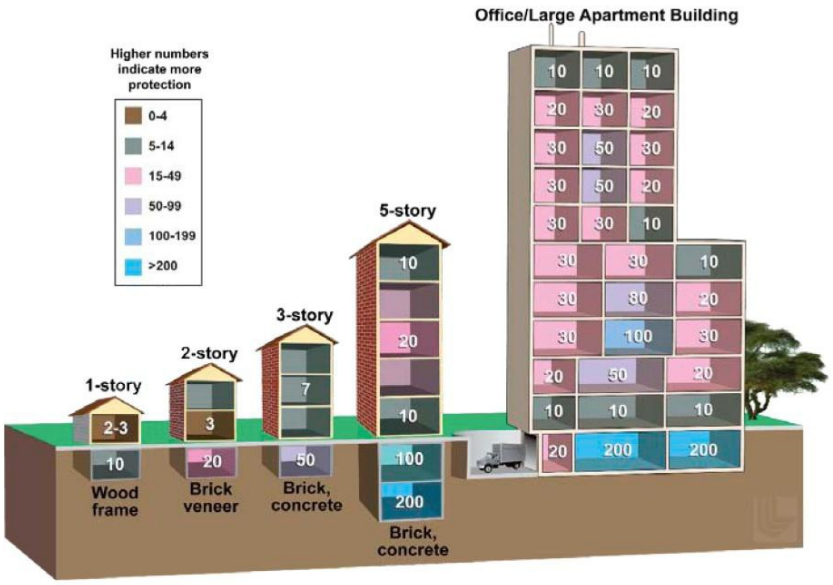

Figure 7: EPA estimate of dose reduction from staying in doors. 10 means the indoor dose is one-tenth the outdoor dose.

4. Buffer zones make it easier to allow controlled venting. At Fukushima, at 12:20 AM on March 12, the site manager, Maseo Yoshida wanted to vent Unit 1 and asked Tepco-Tokyo for permission. Tepco forwarded the request to Prime Minister Naoto Kan. Kan said not until a 3 km radius around the plant has been fully evacuated. Evacuation of this area was not confirmed complete until around 9:00 AM, and venting did not start until 10:00 AM. By then enough hydrogen had seeped into the outer building to cause the first explosion, which not only released a large amount of radioactive material, but also knocked out the mobile emergency diesel, which six minutes earlier had started sending power into Units 1 and 2, allowing high pressure water injection and core cooling. The debris also obstructed the attempt to get another mobile diesel generator to Unit 3. Without the delay in venting, Fukushima might have looked a lot like Three Mile Island.
   With proper buffer zones, the plant management should be given discretion to vent, if they decide it is warranted.

4.1 An LLE Based compensation System

To complete our compensation system, the body politic needs to put a value on a year of life. At this point, we are making the ethically defensible assumption that all life-years are equal, Estimates of this value vary by orders of magnitude; but one of the more generous numbers is the American dialysis standard, which is $127,000 or about $350 per day. Let's assume we decide to use this number.

We now have a compensation system. In a release,

1. Estimate each affected person's daily dose profile. Measuring ambient radiation is cheap and accurate. The area around any nuclear plant should be fitted with an array of dosimeters.
2. Compute each individual's Lost Life Expectancy by combining SNT with her dose profile, adding the LLE's associated with each weekly dose she received.
3. Her compensation is her total expected lost life years times the legal value of a life year.

Under Underwriter Certification, the federal government also must put a cap on the liability insurance required for each plant. This can be done by postulating a reasonable worst case release for the plant's design. We can then model this release plume for the site where the plant is located, and estimate the resulting dose profiles for the site's population distribution, and the total compensation that would be required if the postulated release occured. That number becomes the cap for that plant.

4.2 Handling Evacuation

As we shall see, under SNT and assuming decent buffer zones, evacuation will almost never be indicated. The straightforward way to handle evacuation would be to ignore it. By this I mean evacuation would be voluntary. If a person chooses to evacuate, he would receive the same compensation as if he had not evacuated; but that's it.

5 SNT at Fukushima

Suppose SNT was enforced at Fukushima. We'd have enough radiation monitors around to be able to estimate reasonably accurately the ambient dose rate anywhere in the surrounding area through time.[7] We'd know the population distribution quite precisely. We'd know a great deal about the behavior of people: how much time they spend indoors, how much time they spend in paved areas, how much time they spend in open fields. We'd calibrate our estimates by distributing personal dosimeters to a sample of the population. We'd end up doing a pretty good job of estimating the daily dose for each person in the area for every day since the release.

I can do none of the above. Despite that, I have made a stab at estimating the total SNT Lost Life Expectancy associated with the radiation release at Fukushima, assuming no evacuation.

[7] And the sensors would have battery back up. At Fukushima, when the grid went down, so did the sensors.

The details of this estimate are outlined in reference [2]Chapter 5. The estimate required a chain of heroic assumptions and approximations, so the results are at best illustrative.

But at every step of the way, I tried to err on the high side. With all these caveats, Table 3 shows the overall results. Using SNT I came up with a total public LLE of about 400 days. This does not include plant workers or responders. At $350 per day, the total compensation would be less than $500,000. Most of that money would go to the 2% of the population with the highest dose rates.

Table 3: Fukushima SNT Compensation at $127,000 per LLE year, 40 year cutoff.

fuku				Internal factor = 1
Cancer LLE yrs: 12.00				$/life-day = 350
Area	Initial μSv/h	Population	LLE(days)	Compensation
1-5 km	200	6000	309.91	382,604
5-10 km	50	18000	45.28	55,896
10-20 km	20	76000	25.94	32,019
20-40 km	10	200000	15.06	18,594
Total		300000	396.18	489,115

In a real world analysis, we would use much more accurate numbers, based on the actual doses received. But I don't think my overall numbers are out of the ballpark. They are consistent with the UNSCEAR conclusion that, if there is any radiation impact on the public from the release, it will not be detectable.[12] And in fact ten years on, UNSCEAR could detect no radiation related increase in cancer.

Fukushima was a massive industrial casualty. The cost to TEPCO ratepayers, shareholders, and Japanese taxpayers from the loss of three large reactors and their electricity is in the many billions of dollars. And the Fukushima locals suffered not only horrible losses from the tsunami; but also the loss of jobs and local revenue that the plants would have created. But, sensibly handled, with no mandatory evacuation, there would have been no significant off-site impact from the release of radioactive material at Fukushima.

6 SNT at Chernobyl

Chernobyl was a much larger release than Fukushima. And according to SNT, the harm goes at better than the dose squared. So when I applied SNT to Chernobyl — see reference [2][Chapter 5] for details — I came up with quite different results, Table 4. This table does not include plant workers, first responders, nor liquidators; just the residents in a pretty sparsely populated area. It assumes no behavior changes, and no evacuation. However, it does assume contaminated food is kept away from kids, as was done at Fukushima. Under these assumptions, the total residential public LLE was about 290 life-years. At $350 per day, the total compensation would be about 130 million dollars.[8]

[8] I made an attempt to come up with the liquidator LLE. The result was an LLE of 5800 years, At $350 per day, the liquidator compensation would be about 750 million dollars.

Table 4: Chernobyl SNT Compensation at $127,000 per LLE year, 40 year cutoff.

chern			Internal factor = 2	
Cancer LLE yrs: 12.00				$/life-day = 350
Area	Initial μSv/h	Population	LLE(days)	Compensation
Pripyat	1000	50000	82267	101,469,432
3-10 km	600	10000	12815	15,813,411
10-30 km	300	40000	8979	11,084,677
30+ km	150	16000	999	1,233,271
Total		116000	105061	129,600,793

Once again these calculations are illustrative only. The Chernobyl dose rate data by location are far more uncertain than at Fukushima. But I don't think my overall numbers are misleading, at least not on the low side. In 2019, a team from Harvard Medical School queried the Ukrainian National Cancer Registry.[8] They could find no statistical differences in the solid cancer incidence rates in the districts close to Chernobyl compared to the country as a whole, Figure 8, no pattern that pointed to increased incidence in the high dose districts.

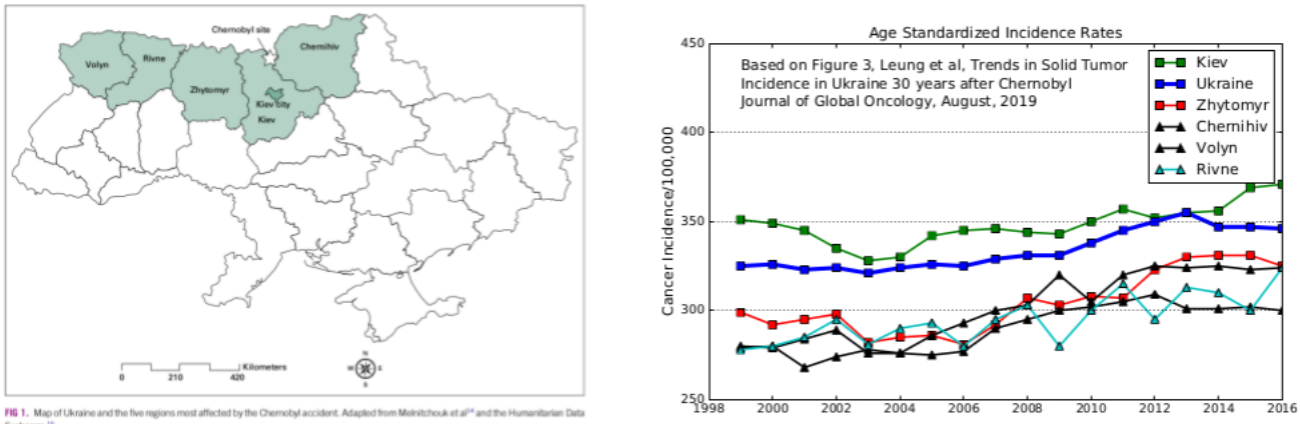

Figure 8: Ukraine cancer incidence, blue line is all of Ukraine

Breast cancer is one of the more radiosensitive diseases. Zupunski et al studied breast cancer incidence in the rayons (roughly counties) closed to the plant.[14] The rayon averaged dose rates varied by more than a factor of ten, Figure 9.

There was no statistically significant difference in the breast cancer rate. It any thing, the higher dose rayons tracked below the lower, Figure 10.

Table 4 is based on conservative assumption after conservative assumption. From the point of the people living in the region, Chernobyl, properly handled, was at worst a bad airplane crash spread over some 100,000 people. But this depends on a dose-response model that recognizes our ability to repair radiation damage, and the acceptance of this model by all concerned.

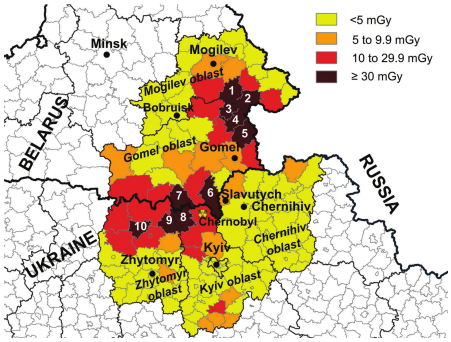

Figure 9: Breast Cancer Doses in the Rayons most affected by the release

Otherwise, the response will turn a bad casualty for the people living in the vicinity of the plant into something far, far worse, which is precisely what happened.

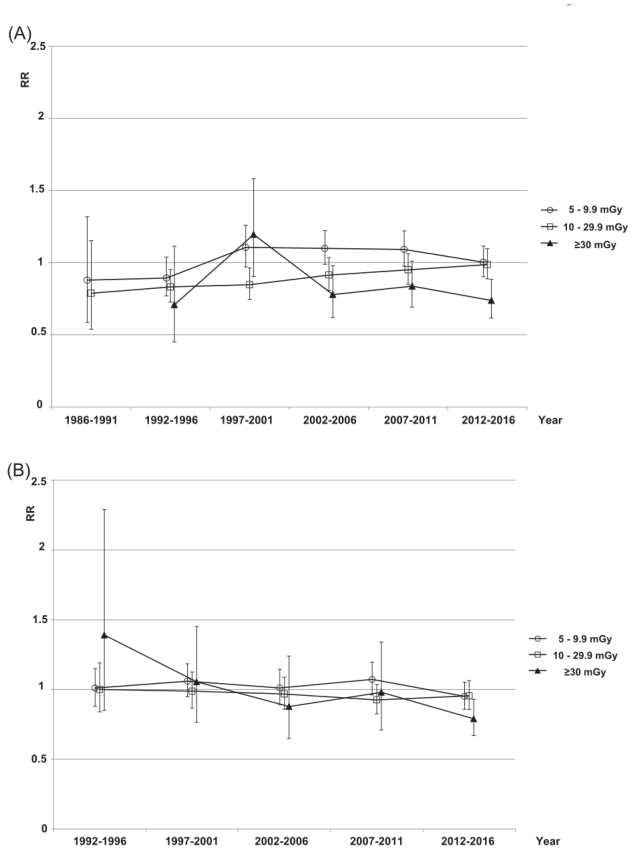

FIGURE 2 Breast cancer relative risk (RR) estimates by 5-year-lagged cumulative absorbed breast dose categories compared to the reference category with dose of <5.0 mGy (RR = 1.00) adjusted for attained age, urban/rural status and stratified by 5-year intervals in (A) Belarus and (B) Ukraine

Figure 10: Breast Cancer Relative Risk

7 Food and Land Contamination

In addition to the human health impact of a large release, there will be other impacts. The most important of these will be food and land contamination. Food that exceeds the legal contamination limits must be purchased at market prices and disposed of. If land exceeds the legal contamination limits for grazing or agriculture, then the farmer must be compensated for the income lost. All the legal contamination limits must be fixed before hand.[9] They cannot be changed during a release, or for that matter during a plant's life, unless the plant is compensated for any additional costs.

The key food contaminant is ^{131}I in milk. Iodine concentrates in the thyroid gland; but ^{131}I has a half-life of 8 days. It is effectively gone inside of three months.[10] Assuming reasonable contamination limits, in any real world release, food/land payments will be a small proportion of the LLE harm payments. Put another way, control of contaminated food, properly implemented, will reduce the LLE harm and the LLE payments, more than the cost of the contamination payments.

8 Loss of income

Finally, we must deal with loss of income. This must be done carefully, lest we set up an open ended invitation to the ambulance chasers.

Here's a possibility. The government sets the dose rate above which a business can shut down and be eligible for compensation of loss of income. Let's call this the Shutdown Level. A reasonable value would be 1 mSv/d absorbed dose. No one has ever been identifiably harmed by a 1 mSv/d dose rate. Table 1 indicates 1 mSv/d is roughly a factor of 20 below the dose rates at which we start to see harm. Appendix A argues that a 4 mGy/d air dose equates to about 1 mSv/d absorbed.

As long as the air dose is above 4 mGy/d at the business's location, the owner can elect to shut down, and be compensated for his loss of income. His employees would also be compensated for their loss of income. Even if the business does not shut down, employees who don't show up for work will still be compensated for the loss of wages.

If an owner elects to keep operating at air dose rates above the Shutdown Level, he still gets the loss of income compensation, as if he had shutdown. Similarly, an employee who chooses to work also gets the loss of wages compensation, even though he is still being paid by the business.

The reasoning here is:

[9] When you apply SNT to ingested dose, you find that the marginal harm associated with consuming say a kilogram of contaminated food depends critically on the dose profile that would have been received if the individual had not consumed this kilogram.[3] The harm is far higher in high dose rate areas than low. At least in theory, the contamination limits should depend on the local dose rate.

[10] Contaminated milk could be turned into cheese, aged for a few months, and be safely consumed.

1. Without this double dip, the owner and employees have no incentive to stay and keep the local economy moving.
2. It does not cost the plant's Underwriter anything. Without the double dip, the owner would almost certainly shut down and the employees would almost certainly not show up.

As soon as the air dose rate drops below 4 mGy/d at the business's location, the loss of income compensation stops.

At Chernobyl, the air dose rate in Pripyat was above 4 mGy/d for roughly four weeks. Pripyat was a 50,000 person company town whose business was supporting the power plant; but, if we conservatively assume 25,000 non-plant wage earners at $1000 per month, the loss of income compensation would be roughly 25 million dollars, a small proportion of the LLE compensation. At Fukushima, only 1 or 2 measuring points outside the plant were briefly above 4 mGy/d.

Loss of income compensation must be limited to direct, physical impacts of a release. In the American tort system, all sorts of indirect, psychological effects are claimable. After the Deepwater Horizon blowout in the Gulf of Mexico, one bar in Key West was awarded $600,000 for lost business. The oil spill never came within 700 miles of Key West. If the bar actually lost business, it was not the fault of the spill; but rather the lurid, grossly exaggerated media coverage of the casualty. A system in which such tenuous impacts are compensatable is uninsurable and will stifle even the most beneficial project.

9 Implementing SNT based Compensation

These calculations suggest that an SNT based compensation scheme combined with reasonable buffer zones would be feasible. A cap of 500 million dollars would almost never be exceeded. The inusrance market is already happily and very profitably writing nuclear liability coverage for 500 million. According to the underwriters there's plenty of appetite for more. Each plant can and should be responsible for its own insurance. That way if the underwriting market is unhappy with the way the plant is performing, the insurance will be yanked, and the plant shut down.

We suggest a cap of one billion 2023 dollars. But this only works with a fixed compensation scheme based on the dose rate profile actually incurred by the person or business, combined with a radiation harm model that recognizes our ability to repair low dose rate radiation damage.

Provided we intelligently site our nuclear power plants, an SNT based compensation scheme is implementable. Once such a scheme is implemented, the federal government can leave it up to the underwriters and the locals to regulate nuclear power.

A Technical Exposition of SNT Model

A.1 The basic assumptions underlying SNT

1. Assume there is no threshold below which there is absolutely zero harm, even though there are many cases where the harm if any is so low that it cannot be detected.
2. Accept harm is monotonic in dose, ignoring compelling evidence that in some situations some radiation can be beneficial.
3. Assume we have repair systems. Harm results if damage is unrepaired. $H(d) = D(d)P_{rf}(d)$. $D(d)$: damage from dose d. $P_{rf}(d)$: probability repair fails if dose is d.[11] It is important that we distinguish between damage and harm. In this context, they are not synonyms. Harm is unrepaired damage.
4. Assume the probability that repair fails goes to zero at zero dose. This implies slope of harm curve goes to zero at zero dose, which can be seen by taking derivative of the product.
5. Assume slope goes to zero at always fatal high end. As we approach the always fatal end, there's nobody left to kill.
6. (1) through (5) is a long winded way of saying, we need an S-shaped response curve. *Duh!*
7. Five parameter logistic works. 5PLogisitic is a generalization of a normal logistic, which allows the low end hook to be smaller than the high end via the g parameter. This is essential in modelling radiation harm.

$$H(d) = H_{inf} + \frac{H_0 - H_{inf}}{\left[1 + \left(\frac{d}{d_{mid}}\right)^s\right]^g} \qquad s > 1.0, \quad d_{mid}, g > 0.0$$

If we assume zero harm at zero dose ($H_0 = 0.0$), and 100% harm at a very large (infinite) dose ($H_{inf} = 1.0$), we have three free parameters.[12]

d_{mid} The location of the inflection point of the S.

s The slope parameter. For the standard logistic, it is the slope at the inflection point. For all g, it is the slope of the low end curve in log-log space, as we shall see.

g The asymmetry. $g = 1$, standard symmetric logistic. $g < 1$, low end hook smaller than high end.

There is nothing radical or original here. The logistic is the standard dose-response model everywhere except in radiation.

[11] If we assume damage is linear in dose, and the probability of repair decreases linearly with dose, then we end up with a quadratic dose response curve. This is qualitatively different from a linear-quadratic model at the all important low end. The slope of a quadratic model goes to zero at zero dose. The slope of a linear- quadratic model goes to the linear coefficient at zero dose.

[12] In fitting the RERF data, c_0 was set to the control group mortality (0.12). The excess risk for a given dose was computed by substracting 0.12 from the resulting curve.

A.2 Fitting the 5 Parameter Logistic to the Bomb Survivor Data.

The Radiation Effects Research Foundation (RERF) has studied the cancer mortality of 86,611 survivors of the Hiroshima and Nagasaki atom bombs. Figure 11 shows a fit of the 5PLogistic to the RERF cancer mortality data. The parameters for this fit are $g = 0.02$, $d_{mid} = 180$ mSv, $s = 2.18$. A $g = 0.02$ means the low end hook is far smaller than the high end.

This is an eyeball fit. The RERF data is a total mess. It bounces up and down like a slinky. This is not normal scatter. Each one of the circles below 1000 mSv is the mean of thousand or more data points. How can the average response for 5000 people between 300 and 500 mSv be far below the average response of 6000 people between 125 and 300 mSv? The eyeball fit purposely gives less weight to the numbers that make the least sense.

But no one can claim that it is too optimistic. There are 34,642 data points below the curve, and 13,440 above. This fit is purposely biased to the high side.

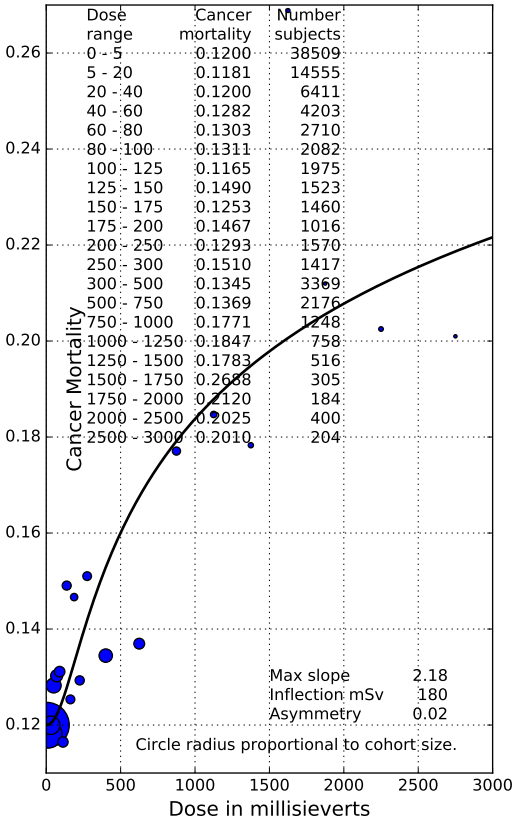

Figure 11: Bomb survivor cancer mortality, 0 to 3000 mSv

A.3 Low end asymptotic behavior

From the broad perspective of Figure 11, the low end hook is barely visible. But for nuclear power plant releases, where the daily doses to the public are rarely above 1 millisievert, all we are interested in is the very low end, Figure 12. Our fit is slightly above the data below 40 mSv but not outrageously so.

The 5PLogisitic exhibits a surprising asymptotic behavior at the low dose end. It turns into a power law in which the exponent is the slope parameter, s.

Letting $\delta = (\frac{d}{d_{mid}})^s$, our harm model can be written

$$H = 1 - (1 + \delta)^{-g}$$

If $\delta \ll 1$, then by Taylor's Theorem,

$$(1 + \delta)^{-g} \cong (1 - g\delta)$$

Substituting this approximation into the equation for harm,

$$H = g\delta = g(\frac{d}{d_{mid}})^s$$

At the low end, moving the inflection point down, increases the harm. A smaller hook goes the other way.[13]

For our fit, the low end harm goes as the 2.18 power of the repair period dose.

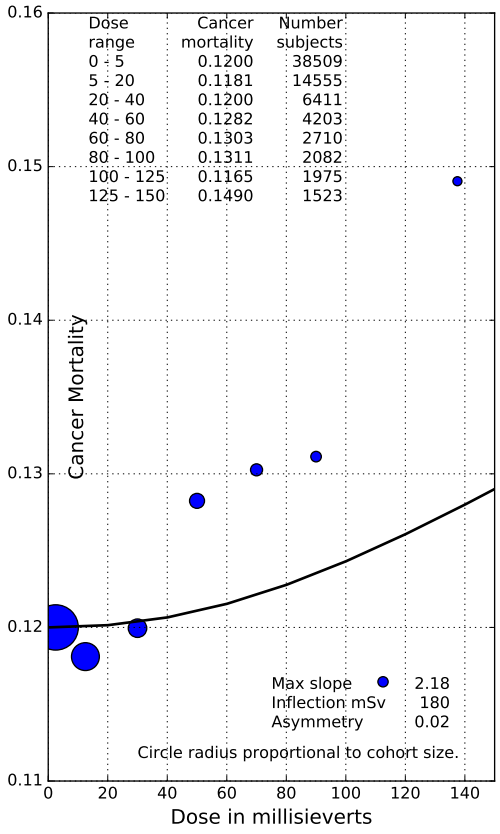

Figure 12: Bomb survivor cancer mortality, 0 to 150 mSv

[13] Corollary: for the slope to go to zero as d goes to zero, s must be greater than 1.0.

Figure 13 is a log-log view of our SNT curve. This power law approximation sets in pretty quickly. The curve is pretty much a straight line in log-log space from 100 mSv (about half the inflection point dose) on down.

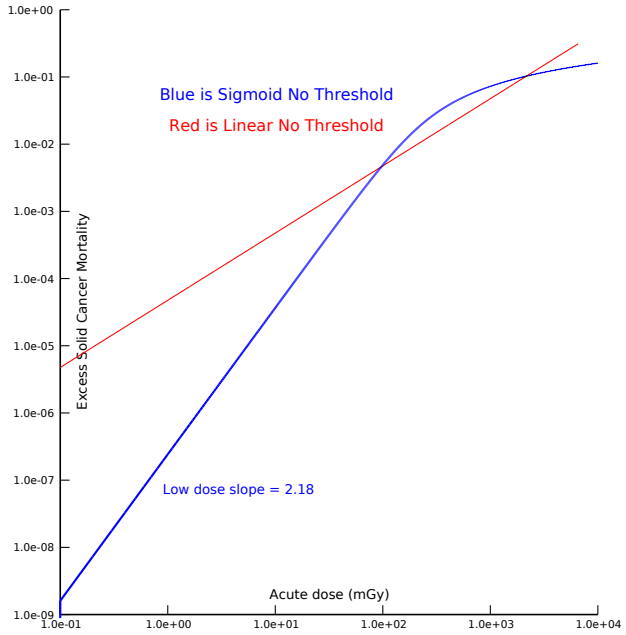

Figure 13: Log-log view SNT 5Pl curve

This has an important implication. According to SNT in any real world release, the harm goes at about the inverse 2.2 power of the dose within the repair period. But to first order the dose rate is inversely quadratic with the distance from the source of the release. This means the harm tends to drop off at better than the fourth power of the distance from the source. A rough rule of thumb is doubling the distance from the source will reduce the harm by about a factor of 18.[14]

For comparison, I have plotted LNT on Figure 13. LNT is also a power law with an exponent of 1. The figure makes a couple of points. LNT is often defended because it is conservative. But that's only true at the low and high end. In this case, the lower cross over point is about 110 mSv.

However as we move down in dose, the curves diverge rapidly, Table 5. At 1 mSv, the LNT mortality is 200 times higher than the SNT; at 0.1 mSv, the LNT mortality if 3000 times

[14] Really large NPP releases such as Fukushima and Chernobyl tend to be spread over a week or two. During that period, the wind will move around. At any time, locations at the middle of the plume will see a roughly cubic drop off in momentary harm with plume distance from the source.

higher than SNT. At the daily doses experienced by the public in a power plant release, LNT over-estimates the harm by multiple orders of magnitude.

Table 5: SNT versus LNT Lost Life Expectancy

Cancer LLE years=12.00					2023-10-14T00:10:09Z
Acute Dose mSv	SNT Fit	LNT Fit	LNT LLE days	SNT LLE days	LNT excess risk over SNT excess risk
0.01	0.120000000	0.120000	0.0021	0.00000004	51011.91
0.02	0.120000000	0.120001	0.0042	0.00000018	22514.38
0.10	0.120000001	0.120005	0.0208	0.00000617	3370.35
0.50	0.120000047	0.120024	0.1040	0.00020618	504.53
1.00	0.120000213	0.120048	0.2081	0.00093431	222.68
2.50	0.120001572	0.120119	0.5201	0.00688624	75.53
5.00	0.120007123	0.120237	1.0403	0.03120022	33.34
10.00	0.120032256	0.120475	2.0805	0.14128196	14.73
15.00	0.120077968	0.120713	3.1208	0.34149949	9.14
20.00	0.120145690	0.120950	4.1610	0.63812132	6.52
25.00	0.120236347	0.121187	5.2012	1.03519920	5.02
30.00	0.120350529	0.121425	6.2415	1.53531576	4.07
40.00	0.120650571	0.121900	8.3220	2.84949908	2.92
50.00	0.121046014	0.122375	10.4025	4.58154318	2.27
80.00	0.122769476	0.123800	16.6440	12.13030655	1.37
100.00	0.124301918	0.124750	20.8050	18.84240291	1.10
200.00	0.134220286	0.129500	41.6100	62.28485239	0.67
300.00	0.144256410	0.134250	62.4150	106.24307699	0.59
500.00	0.160060512	0.143750	104.0250	175.46504373	0.59
1000.00	0.183778013	0.167500	208.0500	279.34769700	0.74

A.4 Repair Period Choice

Since SNT harm is a bit more than quadratic in repair period dose, if we cut the repair period in half, the repair period dose is cut by a bit more than a factor of four. But we have twice as many repair periods, so the overall LLE is cut by a bit more than a factor of 2.

Conversely, if harm is linear in dose, it does not make any difference what the repair period is. However, you chop up the dose profile, when you add the repair periods up, you will end up with the same LLE. LNT's assumption of linearity implies that harm is cumulative in dose. The reverse is also true. If harm is cumulative in dose, the dose response curve must be linear. Linearity and cumulative are not two separate assumptions. Each implies the other.

B Converting Ambient Air Dose Rates to Absorbed Dose

Radiation release analyis starts out with an estimate of the dispersion and contamination by major isotope through time. This is usually measured by the air dose rate 1 meter above ground in μGy/h. A vexing problem for any radiation harm compensation scheme is how to convert measurements of ambient air dose into individual absorbed dose.

One possible solution is to give everybody a personal dosimeter and use the weekly reading on this device to determine that person's dose profile. This is an invitation to fraud. It would be an easy matter for an individual to find a radiation source and pump his readings up by orders of magnitude. Criminal syndicates offering this service would quickly develop.

The Fukushima/Chernobyl computations in Sections 5 and 6 used the Golikov Model to do the conversion.[5] This model:

1. Accounts for weathering by locale. Weathering refers to the migration of an isotope deeper into the soil, or its being washed away, or any other process other than radioactive decay, which reduces the air dose rate at a location. Weathering tends to occurs faster in built up areas, slower in undisturbed area such as forests. Table 6 shows Golikov's locale breakdown.

Table 6: Golikov Locales

Rural	Wood Home	Brick Home	High Rise Apartment	Work Shop	Office	Outside	Work Yard	Ploughed Field	Virgin land
Urban	Wood Home	Brick Home	High Rise Apartment	Office	Dirt	Paved	Park	Garden	Virgin land

2. Accounts for the time each segment of the population spends in each locale, most importantly the time spent indoors. The dose rate indoors can be one-tenth to one-hundreth the dose rate outside. To do this, Golikov divided the population into 10 groups, and measured the time each groups spent in each locale. Table 7 shows Golikov's population breakdown.

Table 7: Golikov Population Groups

Rural:	Indoor Workers	Outdoor Workers	Pensioners	School Kids	Pre-school kids
Urban:	Indoor Workers	Outdoor Workers	Pensioners	School Kids	Pre-school kids

3. Account for the fact that there will be variation within each group. A log-normal distribution is applied to each group, and the LLE of each of 20 sub-groups in that group computed separately, and then added up. This is important for a non-linear harm model such as SNT.

Such a model is necessary if we are to have a reasonably accurate estimate of the LLE's. However, for the purposes of compensation, such a model would be extremely difficult to implement. Instead I suggest we use a Golikov-like model to estimate the total LLE associated with a release, and then apply a single overall factor to the air-dose rates which results in the same total LLE. The population as a whole will be properly compensated; but some individuals will

be over-compensated, some under. This is hardly ideal; but we have sent the right signal to the plant owner/operator and his insurer.

It turns out that if we assume an overall factor of 0.25 in converting air dose in grays to absorbed dose in sieverts, the total compensation at Fukushima would be 1.3 million and that at Chernobyl 453 million. This is definitely on the generous side. When the Japanese government issued dosimeters to the citizens of the town of Date-shi, just outside the Fukushima evacuation zone, they found the absorbed doses were about 0.15 times the measured air dose.[6]

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