

A Guide to Radiation Hazard Evaluation, Applied to Fukushima Recovery Workers

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ABSTRACT

The hazard function model employed internationally in radiological risk assessment for deterministic (threshold-type) biological effects of moderate and high doses of ionizing radiation is used to distinguish three levels of risk for Fukushima recovery workers: life-threatening damage unlikely (RADHAZ low), possible (RADHAZ moderate), and likely (RADHAZ high). Based on hypothetical exposures considered, life-threatening damage could reach the possible or likely range if the worker is exposed to a very large gamma spike during a work shift, say owing to a hydrogen explosion. Projected long-term increase in cancer risk from low-dose exposures of down-wind populations is purely hypothetical, since it is based on the discredited linear no-threshold (LNT) hypothesis. Such projections can lead to radiation phobia, with avoidable casualties.

The Fukushima Disaster

A radiological emergency situation currently exists at the Fukushima Dai-ichi (No. 1) nuclear power station (NPS) in Japan as a result of the Mar 11, 2011, magnitude 9.0 earthquake, which triggered a massive tsunami that killed thousands of Japanese citizens. Thousands are also still unaccounted for. The Fukushima Dai-ichi NPS is operated by the Tokyo Electric Power Company (TEPCO) and is located on the eastern coast of the main Japanese island of Honshu about 225 km north of Tokyo. The facility consists of six boiling water reactors, three (units 1 to 3) of which were operating when the earthquake occurred.¹

The large tsunami caused by the earthquake overwhelmed the site's sea defenses and disabled the heat exchangers and also the diesel generators, so that the site fuel-rod cooling services were then dependent on back-up batteries, which could only supply limited power for a short period. The radiological emergency arose because of the overheating of reactor fuel rods, due to a loss of electrical power for the cooling system.¹ The ¹³⁷Cs released from the damaged rods poses a risk to the emergency workers since its gamma emission can penetrate the body and seriously damage the radiosensitive bone marrow. Serious damage to the bone marrow can lead to radiation deterministic (threshold-type) effects that include loss of life. The workers wear protective garments and respirators to prevent inhalation intake of airborne radioactive material; thus, the focus here is on exposure to the external gamma rays that penetrate the body and protective garments.

This paper presents tools for evaluating potential life-threatening harm from hematopoietic system damage. The tools are used for assigning radiation hazard (RADHAZ) categories for Fukushima recovery workers. The tools are based on estimates of radiation doses to bone marrow delivered at work-shift-specific, average rates and on the hazard function (HF) model previously developed by this author for radiological risk assessment.²⁻⁴ The HF model is used internationally for radiological risk assessment, including the health consequences of nuclear accidents and other radiological incidents. For completeness, deterministic health effects of large radiation doses are reviewed in the next section. These include some effects that do not directly relate to hematopoietic system damage, but may occur among Fukushima radiological emergency workers if unexpectedly exposed to a very high gamma dose (e.g., due to a hydrogen explosion damaging the reactor containment vessel).

Radiation Deterministic Effects

If a person is exposed to a large gamma dose delivered to the entire body, cells in irradiated tissues can be destroyed in large numbers. This can lead to deterministic radiobiological effects.

Deterministic effects are those radiobiological effects that increase in severity as the radiation dose increases and for which a dose threshold exists. Risks for these effects are characterized using statistical models that reflect the distribution of individual thresholds (i.e., individual tolerances) for harm.⁴⁻⁶ The statistical models usually generate sigmoid-type (i.e., slanted-S-shaped) risk vs. dose curves.

Examples of deterministic effects are hypothyroidism arising from large radiation doses to the thyroid gland, as can occur after ingesting large amounts of radioiodine (not a present concern in Japan); skin burns arising from radioactive media (e.g., hot particles, highly radioactive water) deposition on the skin as may have occurred for Fukushima workers who stood in what was apparently highly radioactive water while carrying out emergency work; permanent suppression of ovulation in females; temporary suppression of sperm production in males; growth and mental retardation caused by exposure of a fetus during pregnancy; and death from severe damage to critical organs such as the bone marrow, lung, or small intestine or to the central nervous system (CNS). Of the critical targets indicated, bone marrow is the most radiosensitive, and the median lethal dose (LD₅₀) for whole-body exposure to gamma rays is based on dose-rate-dependent damage to this target.

Dose thresholds are required for deterministic effects because large numbers of cells must be destroyed simultaneously to produce such effects. This is not likely after low radiation doses and is less likely when high doses are delivered at a low rate than when delivered at a high rate.²⁻⁸ For example, if the cell lethality probability per cell at risk (for a specific cell type) is 0.1 (i.e., 1 in 10 cells) at a given gamma dose, then the corresponding probability for simultaneously killing 1 million of these cells with the same dose is $0.1^{1,000,000}$, which is essentially zero. The threshold dose for a specific deterministic effect depends on the type of radiation, on the dose-rate pattern over time (i.e., dose-rate history), and possibly other factors.³ Exceeding the threshold for a specific deterministic effect can lead to a related radiation syndrome.⁹⁻¹⁸ The different syndromes are discussed below.

Prodromal Syndrome and Related Symptoms

The early prodromal syndrome is a group of symptoms and signs of acute gastrointestinal (GI) and neuromuscular effects beginning within hours after brief high-rate exposure to a very large radiation dose to the whole body. The GI symptoms include anorexia, nausea, vomiting, diarrhea, intestinal cramps, salivation, and dehydration.^{9,15-17} Anorexia, nausea, and vomiting occur as part of the earliest signs of radiation sickness. The neurovascular symptoms include fatigue, listlessness, apathy, sweating, and headache. After a massive radiation dose, hypotensive shock may occur due to vascular damage. At LD_{50} , the principal symptoms of the prodromal reaction are anorexia, nausea, vomiting, and fatigue.^{9,15-17} Early diarrhea, fever, and hypotension occur primarily in victims who have received a massive radiation dose.¹¹⁻¹²

Central Nervous System Syndrome

Massive gamma radiation doses (e.g., >50 Gy) to the total body of humans when delivered in a short time (minutes to several hours) can lead to death within about 2 days because of severe cerebrovascular and neurological damage (the neurological or CNS syndrome).¹² For chronic exposure at moderate dose rates, the deaths associated with the CNS syndrome can be delayed, and larger doses are generally required than when the dose is delivered in a short time at a high rate. Whole-body gamma doses that cause the CNS syndrome likely also cause lethal damage to other vital systems (e.g., GI, hematopoietic, and respiratory).

Gastrointestinal Syndrome

Brief, total-body exposure to external gamma doses between about 10 and 50 Gy can lead to the GI syndrome.¹⁸ As with the CNS syndrome, the GI syndrome is generally associated with lethal damage. The length of survival relates to the renewal time

of the depleted intestinal lining cells and is influenced by factors such as infection, bleeding, fluid loss, and loss of protein and electrolytes. Most deaths associated with the GI syndrome occur during the second week after brief exposure to radiation.¹²

For chronic exposure at moderate dose rates, the deaths associated with the GI syndrome can be delayed, and are associated with higher radiation doses than for brief exposure at a high rate. Whole-body gamma doses that cause the GI syndrome likely also cause lethal damage to other vital systems (e.g., hematopoietic and respiratory).¹³

Hematopoietic Syndrome

Brief, uniform exposure of the total body to external gamma doses between about 1.5 Gy and 10 Gy can cause the hematopoietic (or bone marrow) syndrome.¹³ The loss of hematopoietic stem cells in the bone marrow due to irradiation leads to a reduced number of white cells in the blood, the lymphocytes being the most sensitive indicators of injury. Neutrophils may show an initial increase over the first few days following brief exposure, and afterward a dose-related decrease.¹² About 10 days after a brief whole-body gamma dose of 2–5 Gy, a second abortive (increasing and then decreasing) rise in the blood cell counts begins. However, if the damaged bone marrow fails to recover adequately, a final decline occurs.¹² Fever is associated with the loss of neutrophils, and the magnitude of the loss has been used to evaluate the chance of surviving a high dose of radiation.¹² Platelet losses also occur, and the time course is similar to that for granulocytes. Platelet levels in the blood below 30,000–50,000 per μL are associated with bleeding.¹² Persons undergoing the hematopoietic syndrome are susceptible to infection due to injury to the hematopoietic and immune systems.

Modeling Methods

Hazard Function Equation for Life-threatening Damage

The cumulative normalized dose is indicated by X , and for a given absorbed radiation dose differs for different deterministic effects. For life-threatening damage to the hematopoietic system, the value $X=1$ corresponds to the LD_{50} absorbed dose to bone marrow, which for gamma exposure depends on the dose-rate pattern (i.e., dose rate history) to bone marrow.^{2,3,6} This is because the bone marrow is the critical target for determining the risk of life-threatening damage from whole-body exposure to external gamma rays. Lethal damage to other organs (e.g., GI tract, CNS) is accompanied by lethal damage to bone marrow with essentially a probability of 1.

The dose-rate-dependent value for the LD_{50} can be evaluated based on the following equation, with $D_{50}(y)$ representing LD_{50} .^{2,3}

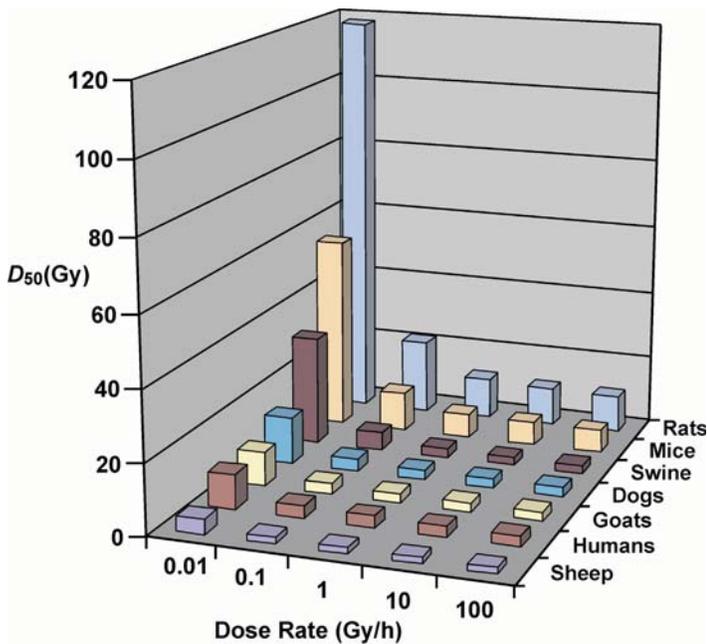


Figure 1. Central estimates of the median lethal gamma dose D_{50} in Gy for different mammalian species as a function of the average dose rate in Gy h^{-1} to bone marrow, based on research reported elsewhere.^{2,14}

$$D_{50}(y) = \theta(y) = \theta_1/y + \theta_\infty \quad (1)$$

The variable y as used here is the average (spatial) dose rate to bone marrow in gray per hour (Gy h^{-1}); θ_1 has the value $0.07 \text{ Gy}^2 \text{ h}^{-1}$ (central estimate) for humans of all ages, and when divided by y accounts for the steep rise in the D_{50} as dose rate decreases. θ_∞ has the value 3.0 Gy (central estimate) for humans.^{2,3} The more general notation $D_{50}(y)$ is used in equation (1) instead of $\text{LD}_{50}(y)$ to be consistent with previous publications. The notation $D_{50}(y)$ can be applied to either lethality (i.e., LD_{50}) or morbidity (i.e., ED_{50}); however, different values for θ_1 and θ_∞ apply to lethality and morbidity.⁶ For a given endpoint, the parameter values differ for different mammalian species including different rodent strains.² This is the basis for the different dose-rate dependences for different mammalian species (Fig. 1). The illustrated feature of the D_{50} approaching an asymptotic value significantly >0 as the dose rate becomes very high is called the Ainsworth phenomenon in honor of the late John Ainsworth, who first reported the observation.¹⁴

When the dose rate changes over time (e.g., during different work shifts at the Fukushima plant), the normalized dose can be evaluated in increments X_1, X_2, \dots, X_n , for n consecutive work shifts (based on the average dose rate to bone marrow for each work shift). The indicated average dose rates are given by y_1, y_2, \dots, y_n , respectively, and can be estimated based on personal dosimeter measurements that are recorded for each worker for each work day. The monitoring is to ensure that a worker does not exceed the effective dose limit (which was raised after the

disaster to 250 mSv per year). For uniform exposure of the total body to gamma rays, the effective dose to the total body in mSv , as measured with a personal dosimeter, estimates the equivalent dose to bone marrow.

If $Y_1^*, Y_2^*, \dots, Y_n^*$ are respective work-shift-specific estimates of equivalent doses to bone marrow in sieverts (Sv), based on personal dosimeter readings, then corresponding bone marrow absorbed radiation dose estimates Y_1, Y_2, \dots, Y_n in Gy can be divided by respective work-shift-specific, exposure times t_1, t_2, \dots, t_n , in hours to obtain corresponding estimates of average bone marrow dose rates y_1, y_2, \dots, y_n in Gy h^{-1} . Here Y_j^* is assumed to be a reasonable estimate of Y_j averaged over the entire bone marrow. The estimates of y_j in Gy h^{-1} can be used to obtain a normalization factor $D_{50}(y_j)$, in Gy, which divides each corresponding dose Y_j (in Gy) to yield estimates of each X_j , for $j = 1, 2, \dots, n$.

The normalized doses obtained for each shift are then added to obtain the cumulative normalized dose X , which then is used along with the HF equation below to arrive at a RADHAZ category. The HF, $H_{hem}(X)$, relates to the risk, $R_{hem}(X)$, of life-threatening damage (same as the risk for lethal damage) to the hematopoietic system through equation (2):¹³

$$H_{hem}(X) = -\ln[1 - R_{hem}(X)] = [\ln(2)]X^V \quad (2)$$

Equation 2 is only applied when X exceeds a threshold value, otherwise $H_{hem}(X) = 0$. Currently the lower bound on the threshold is $X = 0.4$.¹⁹ In equation 2, V is the shape parameter, which determines the steepness of the dose-response relationship. A value of $V = 6$ (central estimate) is used for life-threatening damage to the hematopoietic system.¹³

Results

Categories of RADHAZ

Three categories of RADHAZ indication are considered:¹⁹ Category 1, life-threatening damage unlikely (RADHAZ low):

$$0 \leq H_{hem}(X) < 0.003.$$

Category 2, life-threatening damage possible (RADHAZ moderate): $0.003 \leq H_{hem}(X) < 0.1$.

Category 3, life-threatening damage likely (RADHAZ high): $H_{hem}(X) \geq 0.1$.

RADHAZ categories 2 and 3 would be avoided if the current effective dose limit of 250 mSv is not exceeded. However, the limit could be exceeded in unexpected circumstances such as shortly after a hydrogen explosion where a spike in the gamma levels occurred. This is addressed in hypothetical examples that are discussed below.

Hypothetical Gamma Exposure Scenario

For this hypothetical exposure scenario, an adult male emergency worker works nine consecutive 10-h work shifts with

successively estimated (based on personal dosimeter readings), bone marrow equivalent doses of 20, 18, 25, 16, 28, 18, 22, 24, and 20 mSv (total = 191 mSv). On the tenth shift, there is a hydrogen explosion leading to a spike (1,500 mSv) in the gamma dose (equivalent dose to bone marrow) to the worker. Assuming the absorbed gamma dose to be uniformly distributed over the body, the total effective dose would equal the equivalent dose to bone marrow, i.e., 1,691 mSv, and therefore greatly exceeding the limit.

The shift-specific, equivalent doses and their cumulative values over the different consecutive shifts are shown in Fig. 2. The corresponding results for the shift-specific, normalized doses to bone marrow and their cumulative value over the different shifts are shown in Fig. 3. The lower-bound threshold estimate ($X=0.4$) is not exceeded until the 10th shift.

The buildup of the HF, $H_{hem}(X)$, over the 10 work shifts reflects the cumulative hematopoietic damage hazard (Fig. 4). Zero values for $H_{hem}(X)$ are plotted at the value 0.0001 because of the logarithmic scale. Two horizontal dashed lines are presented for RADHAZ references. The region below the lower dashed line corresponds to a category-1 RADHAZ, i.e., life-threatening damage unlikely. The region between the two dashed lines corresponds to a category-2 RADHAZ, i.e., life-threatening damage possible. The region above the upper horizontal dashed line corresponds to a category-3 RADHAZ, i.e., life-threatening damage likely. Thus, for the exposure scenario simulated here, the worker would not be expected to have life-threatening damage up to and including the ninth work shift. However, due to the large spike in the gamma dose on the tenth shift (greatly exceeding the annual effective dose limit), the worker would be considered to possibly have life-threatening damage to the hematopoietic system and would qualify for close medical monitoring with possible application of radiation countermeasures (e.g., supportive care) by medical professionals.

Had the radiation exposure spike on the tenth work shift been twice as large (bone marrow equivalent dose increment of 3000 mSv), then $H_{hem}(X)$ would equal 0.46, resulting in a category-3 RADHAZ (life-threatening damage likely) (Fig. 5). In this case radiation countermeasures would likely be provided.^{19,20}

The countermeasures are generally treated as effectively increasing the value for the D_{50} by a fixed proportion called a protection factor (PF).¹⁴ For a $PF = 2$, the D_{50} would be treated as increasing by a factor of two (reducing X by a factor of two), which for the results in Fig. 5 would lead to changing from a category-3 RADHAZ to a category-2 RADHAZ.

Low-dose Exposures

Following the Chernobyl accident, irresponsible news media coverage frightened millions worldwide, which caused massive radiation phobia in Eastern Europe, leading to tragic abortion of more than 100,000 wanted births, apparently on the advice of

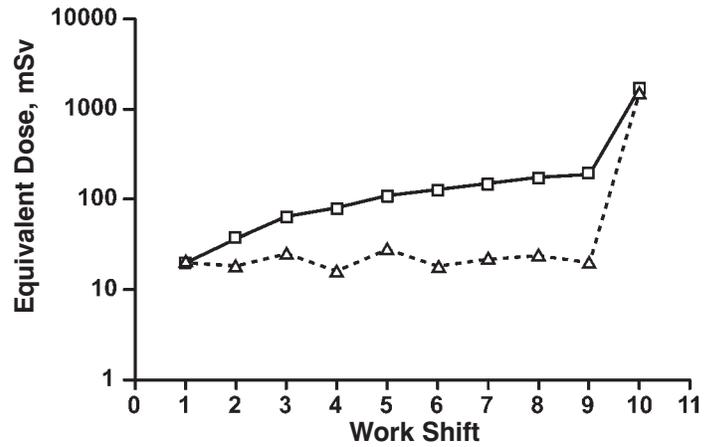


Figure 2. Work-shift-specific (triangles) and cumulative (squares) bone marrow equivalent doses for a hypothetical 10-shift exposure scenario for a Fukushima radiological emergency worker.

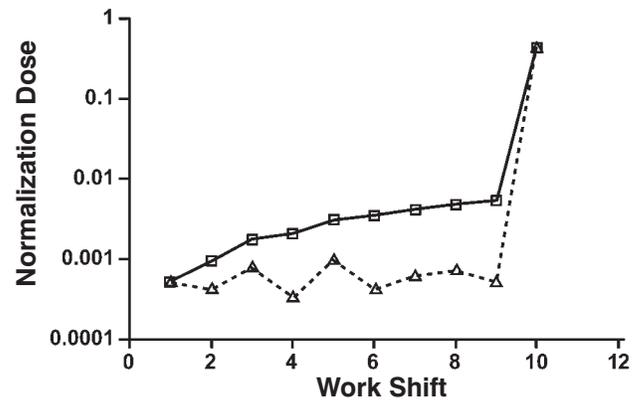


Figure 3. Work-shift-specific (triangles) and cumulative (squares), normalized doses to bone marrow for a hypothetical 10-shift exposure scenario for a Fukushima radiological emergency worker.

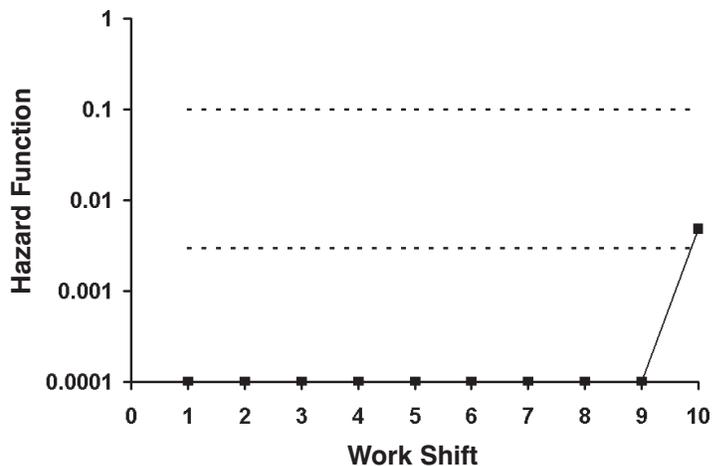


Figure 4. Calculated hazard function, $H_{hem}(X)$, buildup (i.e., hematopoietic system damage cumulative hazard) as a function of the number of work shifts for the gamma dose patterns in Fig. 2 (equivalent dose) and 3 (normalized dose). The upper horizontal dashed line is located at $H_{hem}(X) = 0.1$ and the lower horizontal line is located at $H_{hem}(X) = 0.003$. Zero values for $H_{hem}(X)$ for shifts 1 through 9 are plotted at the value 0.0001 because of the logarithmic scale.

misinformed physicians.²¹ Based on the linear-no-threshold (LNT) hypothesis, a single radiation hit of a given size to each member of a very large population will cause at least one cancer case. Two radiation hits to each person will double the number of cancers, according to the hypothesis. This is why some scientists in the United States have stated in news media interviews that no radiation dose is safe, no matter how small. Unlike the hypothetical harm (LNT-hypothesis based) to Chernobyl victims from low-level radiation exposure that were projected from back-of-the-envelope calculations (up to hundreds of thousands of cancer deaths for example), the abortions were real and unnecessary casualties.²²

The Japanese citizens have shown remarkable patience and courage in spite of the thousands of lives that were lost as a result of the earthquake and tsunami. In addition, unlike the LNT-advocating experts who have been interviewed on television in the U.S. regarding the potential health consequences of the Japanese radiological emergency to workers and the general public, Japan has its own experts (none of whom have appeared in U.S. television interviews) on the biological effects of low-dose and low-dose-rate exposure to ionizing radiation. These include Sohei Kondo, Yoshiya Shimada, Taisei Nomura, Hiroshi Tanooka, Teruisa Tsuzki, Kazou Sakai, and many others. While many so-called experts in the U.S. rely on the LNT hypothesis for assessing risks from low-level exposure to radiation, Japanese scientists have their own published research (most of which is unknown to U.S. experts) to rely on, and they are aware of the abundant evidence against the validity of the LNT hypothesis.

Recent publications in *Dose-Response*²³⁻²⁹ and *Health Physics*³⁰ discuss some of the abundant information against the validity of the "one-radiation hit to each person in a very large population causes at least one cancer" hypothesis. A related video

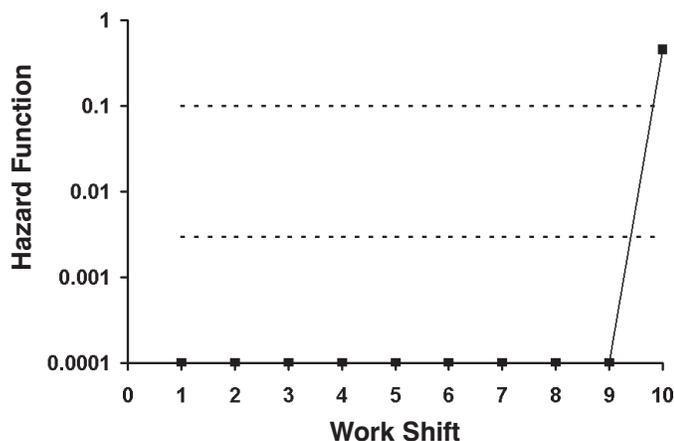


Figure 5. Calculated hazard function, $H_{hem}(X)$, build up (i.e., hematopoietic system damage cumulative hazard) as a function of the number of work shifts for the gamma dose patterns in Fig. 2 (equivalent dose) and Fig. 3 (normalized dose) up to and including work shift number nine. For this scenario, the radiation exposure is twice as large on the tenth shift as in Fig. 2. Zero values for $H_{hem}(X)$ for shifts 1 through 9 are plotted at the value 0.0001 because of the logarithmic scale.

presentation from the International Dose-Response Society and Lovelace Respiratory Research Institute is available on the internet at <http://dSPACE.Irri.org:8080/xmlui/handle/123456789/891>.

Lessons from the Chernobyl accident related to managing radiological emergencies have apparently not been learned by many members of the news media in the U.S.,²² who have largely ignored the thousands of lives lost and thousands still unaccounted for in Japan related to the earthquake and tsunami, and have instead devoted an inappropriately large amount of time to a supposed threat to the U.S. west coast from minuscule levels of radioiodine coming from Japan. Many residents on the west coast as well as in other parts of the U.S. rushed to purchase potassium iodide tablets to supposedly protect them from a harmless amount of radioiodine. Interestingly, some of the prodromal symptoms (nausea, vomiting, blackened stool, fatigue) associated with high-level radiation exposure can also be caused by potassium iodide pills. The pills can also cause irregular heartbeat. In addition, the radioactive potassium contained in potassium iodide tablets likely deliver more radiation to the thyroid than has been received from stray iodine-131 atoms coming across the Pacific Ocean from Japan to the U.S.

Much better education about ionizing radiation is needed, especially concerning the natural radiation environment in which we all live. Too few people in the U.S. and elsewhere are aware that everyone is radioactive and that more than a million harmless natural radiation hits (deposition events) take place in our bodies each second of our lives.³¹ Multiplying by the number of seconds in a day [(1 x 10⁶ hits s⁻¹) x (60 s min⁻¹) x (60 min h⁻¹) x (24 h d⁻¹)] shows that each day of our lives we humans experience more than 86 billion harmless natural radiation hits to our bodies. Worrying about a few additional hits from a stray radioiodine atom coming across the Pacific Ocean from Japan, when the next day we U.S. citizens are each going to receive more than 86 billion additional harmless natural radiation hits, makes absolutely no sense. In addition, it now appears that the natural radiation hits may be helping to prevent cancer and other adverse effects by occasionally stimulating the body's natural defenses (DNA repair, selective apoptosis that removes aberrant cells, antioxidant defenses, and anticancer immunity).^{23,28}

The recent paper by Hiroshi Tanooka supports the existence of a dose-rate dependent threshold for cancer induction, which could be quite large (greater than 1,000 mGy) for gamma exposure at a low rate.³² Low-rate exposure to gamma rays has also been demonstrated to prevent chemical-carcinogen-induced cancer, likely by up-regulating anticancer immunity and other adaptive response genes.^{23,28} In addition, new research shows that residential radon (a source of natural radiation) levels near the U.S. Environmental Protection Agency's action level of 4 pCi L⁻¹ (approximately 150 Bq m⁻³) of air may be significantly suppressing lung cancer; thus, eliminating radon from the home

may actually increase the risk of lung cancer,³³ a conclusion that agrees with earlier reported research.³⁴

This study did not focus on cancer risks for Japanese radiological emergency workers or general public; however, any projected cancer cases for the Japanese population based on the LNT hypothesis should be rejected because the hypothesis is not valid.²³⁻³⁷ In addition, use of the LNT hypothesis for projecting future cancer cases in Japan or elsewhere (e.g., west coast of the U.S.) can lead to thousands of radiation-phobia-related casualties as occurred after the Chernobyl accident.²²

Conclusion

From personal dosimetry and a standard hazard-function model, risk of deterministic effects of ionizing radiation on exposed recovery workers can be calculated. Barring an exceptional event that caused the 250 mSv/y limit to be greatly exceeded, such effects in Fukushima workers are unlikely. The discredited LNT risk model should not be used to project cancer deaths in down-wind populations with low-dose and low-dose-rate exposures.

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