Commentary on Fukushima and Beneficial Effects of Low Radiation

Jerry M. Cuttler
Cuttler & Associates, Mississauga, Ontario, Canada
(jerrycuttler@rogers.com)

Abstract

Approximately 160,000 people evacuated the area around the Fukushima Dai-ichi NPP shortly after it was damage by the earthquake and tsunami. The evacuation order applied to 70,000 of them, while the other 90,000 left voluntarily and returned soon afterward. After more than two years, most of the 70,000 are still not allowed to return to their homes. The 1100 disaster-related deaths caused by the evacuation order show that this precautionary action, taken to minimize cancer risks, was not "conservative." In this paper, recent studies are reviewed on the consequences of the radioactive releases and on the benefits of many medical treatments with low doses of radiation that were carried out until the 1950s, before the radiation scare was created. Recent research has shed light on the high rate of spontaneous double-strand breaks in DNA and the adaptive protections in cells, tissues and humans that are up-regulated by low radiation. These defences prevent, repair, remove and replace damage, from all causes including external agents. Cancer mortality is reduced. The ICRP's concept of radiation risk is wrong. It should revert to its 1934 concept, which was a tolerance dose of 0.2 roentgen (r) per day based on more than 35 years of medical experience.

1.   Fukushima

Approximately 160,000 people evacuated the area around the Fukushima Dai-ichi NPP shortly after it was damaged by the March 11, 2011 earthquake and tsunami. The evacuation order applied to 70,000 of them, while the other 90,000 left voluntarily and returned soon afterward. After more than two years, most of the 70,000 are still not allowed to return to their homes. Recently, a number of reports were issued on health effects, mechanisms of low radiation effects, lessons learned, and a health risk assessment based on preliminary dose estimation, as discussed below.

UNSCEAR indicates that no health effects attributable to radiation were observed among the workers, among children or any other member of the population (UNSCEAR 2012a, Chapter IIB, Section 9(a)). Chapter III, Section 1 discusses the difficulties in attributing health effects to radiation exposure and inferring *risks*, meaning radiation-induced cancer and hereditary effects (so-called "stochastic" effects). *"In general, increases in the incidence of health effects in populations cannot be attributed reliably to chronic radiation exposure to radiation at levels that are typical of the global average background levels of radiation. This is because of the uncertainties associated with the assessment of risks at low doses, the current absence of radiation-specific biomarkers for health effects and the insufficient statistical power of epidemiological studies."* Section 2 points out that failure to properly address uncertainties can cause anxiety and undermine confidence among the public, decision-makers and professionals.

The UNSCEAR report on mechanisms (UNSCEAR 2012b) is a short document reviewing the biological mechanisms of action of radiation at low doses. It highlights major advances in the field for guidance on future work programs. Understanding of the mechanisms is improving, but there is a lack of consistency and coherence. UNSCEAR states that as yet there is no indication of a causal relationship with radiation-related disease and no consensus on the impact of radiation exposure.

ICRP Task Group 84 compiled a considerable amount of detailed information and developed recommendations on efforts to protect people against radiation exposure during and after the accident (ICRP 2012). Eighteen issues were identified as requiring action and relevant ICRP Recommendations were scrutinized. The Task Group prepared suggestions and recommended eleven actions. The ICRP should ensure:
- proper interpretation of radiation risk coefficients
- understanding of the limitations of epidemiological studies on radiation effects
- resolution of confusion on protection quantities and units
- proper interpretation of the hazard from intake of radioactivity
- an ad hoc system to protect rescuers and volunteers
- clear recommendations for crisis management and medical care and for recovery and rehabilitation
- consistent and understandable recommendations about public protection levels (infants, children, pregnant women, fetus) and related issues (categorizing accident exposures, transit from an emergency, and rehabilitation)
- updated public monitoring policy
- definition of tolerable contamination levels for consumer products, rubble and residues
- strategies to mitigate the serious psychological consequences from radiological accidents
- information-sharing regarding radiological protection policy after an accident, with recommendations to minimize communication lapses.

Using the ICRP methodology and atomic bomb survivor risk estimates (Ozasa 2012), the World Health Organization issued a health risk assessment (WHO 2013) that estimates the lifetime risks of cancer and calculated the cumulative risks for the 15 years following the radioactivity release from the power plant. The findings in the executive summary indicate that in the two most affected locations of the Fukushima Prefecture, the preliminary estimated radiation "effective" doses for the first year ranged from 12 to 25 mSv. In the highest dose location, the estimated additional lifetime risks for the development of leukemia, breast cancer, thyroid cancer and all solid cancers over the baseline rates are likely to represent an upper bound of the risk as methodological options were consciously chosen to avoid underestimation of risks.

The WHO report predicted that the lifetime risks for leukemia increased by up to 7% over the baseline cancer rates in males exposed as infants. For breast cancer, the estimated lifetime risks increased by up to 6% over the baseline rates in females exposed as infants. For all solid

cancers, the estimated lifetime risks increased by up to 4% over the baseline rates in females exposed as infants. For thyroid cancer, the estimated lifetime risk increased by up to 70% over the baseline rates in females exposed as infants. Less than 1% of the Fukushima NPP workers received an effective dose in the range 100 – 200 mSv, while several workers received up to 700 mSv. Their lifetime cancer risks are estimated in Section 5.3 of the WHO report and are much higher.   Section 7.4 discusses the psychological consequences. Attributing a cancer risk to a low radiation exposure produces a radiophobia (Jaworowski 1999) that far outweighs all other health consequences.

The author has great difficulty understanding the methodology used by the WHO to estimate risk. It is very complex and uses many assumptions. The bomb survivor information (Ozasa 2012) relates to a short-term exposure and is subject to many confounding factors. The linear extrapolation of high-dose (> 1 Gy) risk to calculate health effects of low radiation is very controversial; the biology is absent. In 1980, a founder of the ICRP, Lauriston Taylor stated (Taylor 1980):

"Today we know about all we need to know for adequate protection against ionizing radiation. Therefore, I find myself charged to ask: why is there a radiation problem and where does it lie?" "No one has been identifiably injured by radiation while working within the first numerical standards (0.2 r/day[†]) set by the NCRP and then the ICRP in 1934." "An equally mischievous use of the numbers game is that of calculating the number of people who will die as a result of having been subjected to diagnostic X-ray procedures. An example of such calculations are those based on a literal application of the linear, non-threshold, dose-effect relationship, treating the concept as a fact rather than a theory. ... These are deeply immoral uses of our scientific knowledge."

The tsunami-only refugees number 250,000. The 160,000 Fukushima refugees include about 90,000 who voluntarily evacuated and have returned home. However, 70,000 were forced to leave the mandated zones by the government's overly-restrictive and arbitrary emergency evacuation to comply with the ICRP's ALARA principle.   They receive compensation payments each month from TEPCO. This was not a "conservative" precautionary measure (Cuttler 2012). Prolonged evacuation was enforced because of widespread radiation phobia (Brumfiel 2013), and the "Reconstruction Headquarters" has reported approximately 1100 disaster-related (pre-mature) deaths among the evacuees, due to psychosomatic effects (67%) and disruption of medical and social welfare facilities (18%) (Saji 2013, Table A5).

2.   Beneficial Effects

Beneficial radiation health effects were identified by medical scientists and practitioners very shortly after the discoveries of x-rays and radioactivity in 1895/6. This ionizing radiation was employed initially for diagnosing bone fractures and other medical conditions. They soon

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[†] The SI radiation level that corresponds to 0.2 r/day is ~ 1.86 mGy/day or 680 mGy/year (68 rad/year).

learned that large exposures were harmful; however, low exposures produced remarkably beneficial effects, such as rapid healing of wounds and cures of infections. They discovered that a low radiation dose to the entire body increased the action of protective processes in living organisms, including the overproduction of lymphocytes that significantly prevented or impaired tumor growth (Murphy and Morton 1915).

Many very important beneficial applications of low radiation, other than curing cancer, were identified in the early 1900s. Many thousands of patients were treated with low radiation with no apparent increases in the incidence of "stochastic effects" (cancer or other genetic effects), long after these radiation treatments. The applications include healing of wounds (Calabrese 2013a) and the cure of a wide variety of infections, such as gas gangrene (Calabrese and Dhawan 2012), carbuncles and boils (Calabrese 2013b), sinus infections (Calabrese and Dhawan 2013a), and inner ear infections (Calabrese and Dhawan 2013b). Other applications include the treatment of arthritis and other inflammatory conditions (Calabrese and Calabrese 2013a, 2013b; Rodel et al. 2012) and swollen lymph glands (Schenck 1935; Hurwitz and Zuckerman 1937).

Most people and many scientists are very puzzled when they are informed about the extensive evidence of radiation-induced beneficial effects that apply to so many different characteristics in living things (Luckey 1980, 1991). They try to disregard this information because it contradicts what they have been carefully taught all of their lives, namely that exposure to ionizing radiation, in any amount, carries a "risk of health effects." The implied meaning of the term health effects is <u>adverse</u> health effects, i.e., cancer and harmful genetic effects. Scientists request a detailed explanation of the mechanism of this action before they will believe the evidence of positive health effects. However, the detailed mechanism of action of most natural phenomena, such as gravitational attraction, is not well understood, yet we accept and employ them as needed. The biological effects of radiation have been carefully and extensively studied for more than a century. We likely know more about these effects than those of any other stressor (Taylor 1980).

An excellent explanation of the complex processes whereby ionizing radiation induces beneficial effects in biological organisms has been provided by Feinendegen et al (2012). The occurrence of spontaneous DNA damage was discovered more than 25 years ago. Its rate is at least six orders of magnitude greater than the damage rate caused by the average background level, ~2.4 mGy/year (Cuttler 2012). While single-strand breaks are readily repaired, double-strand breaks (DSBs) are more serious and relevant to induction of cancer and other genetic changes. Measurements have determined that nonirradiated cells, depending on the type and age, contain on average from about 0.1 to numerous DSBs at steady state. This value corresponds well to the calculated probability of 0.1 for a DSB to occur per average cell in the human body per day from endogenous, nonradiogenic sources (Pollycove and Feinendegen 2003). In contrast, at background level, the probability of a radiogenic DSB to occur per day was calculated to be on average only about 1 in 10,000 cells. So the ratio of nonradiogenetic

to radiogenetic DSBs produced per day is about 1,000, i.e., the natural damage rate is a thousand times greater than the rate due to background radiation (Feinendegen et al 2012).

The key determinant is the effect of radiation on the biological defences and protective systems, which involve the actions of more than 150 genes. They act on all the damage occurring (and its consequences), from internal causes and the effects of external agents, to restore good health. In contrast to high-dose irradiations, low-dose irradiations can up-regulate adaptive protections in cells, tissues, animals and humans. The detailed behaviours of the mechanisms are very complex, but the evidence of beneficial health effects is very clear, from cancer prevention and cures to the very important medical treatment applications mentioned earlier.

The evidence of beneficial effects from low radiation requires the definition of the range for harmful effects. This was known when the first radiation protection standard was set in the early 1930s. There have been many studies on mammals, especially since the 1940s. The recent review by Fliedner et al. (2012) on the response of the hematopoietic system[‡] to low dose-rates of ionizing radiation is very important because it focuses on the damage accumulating in the cell renewal of bone marrow tissue (which has a rapid turnover) and is generally more radiosensitive than the gastrointestinal cell system or skin.

The article assesses many human exposures and animal studies. A study of dogs exposed to cobalt-60 gamma radiation during their entire lives allows the range for harmful effects to be determined. Figure 1 shows the mortality curve for each dose-rate group. At dose rates higher than 18.8 mGy/day (1.88 cGy/d), death was nearly always due hemopoietic insufficiency.   In the dose-rate group 18.8 mGy/day, still some dogs died from myeloproliferative disorders (MPD), but below this dose-rate the relative number of deaths from fatal tumors increases to the level seen in the control dogs. Figure 2 shows the lifespan, at the 50% mortality level, for each dose-rate group, normalized to the lifespan of the control dogs (4300 days). Lifespan decreases below that of the controls when the radiation level exceeds about 700 mGy/year.   Some dogs succumbed earlier than others, indicating individually varying radiosensitivities for tolerance or failure of the blood-forming system.

There was no group of dogs in the dose-rate range between 1100 mGy/year and background radiation level. Extending the fitted line from 1100 mGy/year to background (2.4 mGy/year) suggests the likelihood of a lifespan longer than the controls in this range, a beneficial effect of low radiation.

In the group of 92 dogs exposed to 3 mGy/day (1,100 mGy/year), there were no significant changes in the concentrations of the blood cells in a clinically relevant way; however, radiation effects were apparent beyond 1000 days. In this group, some dogs survived up to 5000 days within the radiation field—a full life span. The cause of death in these dogs was similar to the control dogs, dominated by fatal tumors (Fliedner et al. 2012).

[‡] stem cells in the bone marrow that produce the blood cell components

3.   Non-scientific Influences on Radiation Protection

This data brings into question the dose limits in radiation protection. Current limits are fixed numbers without much attention to dose rate. The dose rate should be built into the exposure limits. The great discrepancy between the recommended dose rate limit, 1 mGy/year for the general public, and the observed dose rate of 1,100 mGy/year, at which the hemopoietic system keeps providing stability and full function in service of the entire body without apparent radiation-induced increase in tumor incidence, questions the justification of the radiation protection recommendations (Fliedner et al. 2012).

As pointed out in an earlier article (Cuttler 2012), the 1934 radiation protection standard that was based on the "tolerance dose" concept of 0.2 r/day (680 mGy/year) was changed in the 1950s because of strong political pressure by scientists and other influential people to create a social fear of low radiation from a-bomb testing during the arms race and abhorrence of nuclear war. The concept adopted was a radiation-induced probabilistic (stochastic) risk of cancer death and genetic harm that is to be kept small compared to other hazards in life. The risk is calculated using the linear-no-threshold (LNT) hypothesis of radiation carcinogenesis being promoted by Hermann Muller and other geneticists in the early 1900s. The incredible irony is the continued use of this concept, six decades later, in spite of more than a century of contradictory radiobiological evidence. The flood of assessments based on the LNT theory of cancer and genetic risks continues and many research studies based on this model are funded.

Calabrese has described "the road to linearity" in great detail (Calabrese 2009). The eugenics movement was an important factor in the widespread acceptance of the LNT dose-response model. *"Eugenics is the applied science or the biosocial movement, which advocates the use of practices aimed at improving the genetic composition of a population, usually a human population"* (National Library of Medicine 2013). The word was coined in 1883 by Francis Galton, a cousin of Charles Darwin, founder of the science of evolutionary biology. Galton wanted eugenics to develop from a science to a policy to a religion (Cavanaugh-O'Keefe 1995). Natural evolution occurs slowly and progressively; significant improvements occur over a period of centuries. However, the eugenicists wanted to expedite improvements in the human race (its gene pool) by social and political interventions. This movement became very popular throughout the world, beginning in the early 1900s and continuing through to the present. In 1970, the American Eugenics Society (I. Gottesman) defined it in this way: *"The essence of evolution is natural selection; the essence of eugenics is the replacement of 'natural' selection by conscious, premeditated, or artificial selection in the hope of speeding up the evolution of 'desirable' characteristics and the elimination of undesirable ones."*

In the 1920s, Hermann Muller, a biologist and proponent of eugenics, became interested in the genetics of fruit flies (*Drosophila Melanogaster*), focusing on the gene mutation rate and lethal mutations. He found a strong temperature dependence leading him to believe that spontaneous mutation was the dominant mode. In his Science article on his discovery of radiation-induced mutations (Muller 1927a), he states that the study of gene mutations is very seriously hampered by their extreme infrequency and by the generally unsuccessful attempts

to modify organisms for utilitarian purposes. Following reports of germinal changes induced by radium or x-rays, he performed a series of experiments using relatively heavy doses of x-rays. Mutations were induced in a high proportion of the treated germ cells causing a rise of about 15,000 percent in the rate over that in the untreated cells. The experimental data appears in his paper at the 5th International Congress of Genetics in Berlin (Muller 1927b). Four irradiation times were used: 12, 24, 36 and 48 minutes. The x-ray tube target was at a distance of 16 cm from the flies; the voltage was 50 kV, and the current was 5 milliamperes. This suggests that a dose-rate of about 100 roentgen per minute was used, based on information in a related article. Therefore, these radiation doses were in the range from about 1200 to 4800 roentgen. There should be an exact proportionality between point mutations and dosage if the former directly result from chance hits by the rays, but his data suggested a square-root relation. Subsequently, Muller became an activist promoting the fear of genetic damage from any exposure to x-rays or nuclear radiation stating that the risk was linearly proportional to dose without any threshold.

Many other scientists carried out similar research on fruit flies. For example, a paper in 1930 showed that the mutation frequency was linear with dose between 285 and 4560 roentgen (Oliver 1930). However, a critical study using special flies supplied by Hermann Muller revealed in 1946 that there was no evidence of a significant difference between the controls and those that were irradiated for 21 days with radium gamma rays to a dose of ~ 50 roentgens (Caspari and Stern 1948). Muller knew about this result, weeks before he delivered his Nobel Prize lecture in which he declared that there is no safe level of radiation exposure—*"no escape from the conclusion that there is no threshold"* (Calabrese 2012).

Later research demonstrated that mutation frequency depends not only on the total dose but also the dose rate. Because repair capacity is limited, a higher dose rate results in a greater number of mutations for the same dose. A study by Koana et al. (2004) demonstrated there is a threshold at 1 Gy for fruit flies, and another study (Koana et al. 2007) demonstrated a reduction in the mutation frequency in sperm irradiated with a low dose rate of 0.05 Gy/minute (300 rad/hour). The mutation frequency was 0.79% for a dose of 10 Gy and 0.07% for 0.2 Gy. The latter was significantly lower than 0.33% for the controls, which indicates that a threshold exists between 0.2 and 10 Gy. Ogura et al. (2009) irradiated flies at the much lower dose rate of 22.4 mGy/hour (2.2 rad/hour). As shown in Figure 3, the mutation frequency at 0.5 mGy (0.09%) is much lower than in the control group (0.32%), whereas the mutation frequency in the 10 Gy group (0.77%) is significantly higher. It is very clear that the LNT model, which predicts harm at low dose, is wrong. The biological evidence shows a beneficial effect below a dose of about 1 Gy.

4.   Conclusion

In light of the on-going crisis of suffering and economic hardship in Japan, the appropriate action for the radiation protection establishment is to abandon the concept of stochastic cancer risk, based on the LNT dose-response model, and adopt the previous tolerance dose concept.

It is supported by extensive biological evidence and credible models. This change in concept would dispel the psychosis of fear surrounding the use of radiation in medical diagnostics and the treatment of serious diseases and illnesses. It would also improve social acceptance of using nuclear energy for many very important peaceful applications. In view of the 1100 disaster-related deaths caused by the evacuation order, it is clear the long-term precaution to avoid a low radiation exposure was not a "conservative" emergency measure.

Dedication: This commentary is dedicated to the memory of Theodore Rockwell, a pioneer in the development of nuclear energy and a tireless campaigner against the spread of false information about radiation and nuclear energy, as described at:
http://ansnuclearcafe.org/2013/04/02/ted-rockwell-atomic-pioneer-and-tireless-campaigner-for-facts/?utm_source=ANS+Nuclear+Cafe&utm_campaign=0f5e7280c9-RSS_EMAIL_CAMPAIGN&utm_medium=email

5. References

Brumfiel G. 2013. "Fallout of Fear." *Nature* 493: 290-293. January 17. Available at: http://www.nature.com/news/fukushima-fallout-of-fear-1.12194

Calabrese EJ. 2009. "The road to linearity: why linearity at low doses became the basis for carcinogen risk assessment." *Arch Toxicol* 83: 203-225. Available at: http://db.tt/DrdIz9xT

Calabrese EJ. 2012. "Review: Muller's Nobel Prize lecture: when ideology prevailed over science." *Tox Sci* 126 (1): 1-4.

Calabrese EJ. 2013a. "Historical foundations of wound healing and its potential for acceleration: dose-response considerations." *Wound Rep Regen* 21(2): 180-193.

Calabrese EJ. 2013b. "X-ray treatment of carbuncles and furuncles (boils): a historical assessment." *Hum Exp Toxicol* (in press)

Calabrese EJ and Calabrese V. 2013a. "Low dose radiation therapy (LD-RT) is effective in the treatment of arthritis: animal model findings." *Int J Rad Biol* 89(4): 287-294.

Calabrese EJ and Calabrese V. 2013b. "Reduction of arthritic symptoms by low dose radiation therapy (LD-RT) is associated with an anti-inflammatory phenotype." *Int J Rad Biol* 89(4): 278-286.

Calabrese EJ and Dhawan G. 2012. "The role of x-rays in the treatment of gas gangrene: a historical assessment." *Dose-Response* 10(4): 626-643. Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3526332/

Calabrese EJ and Dhawan G. 2013a. "The historical use of radiotherapy in the treatment of sinus infections." *Dose-Response* (in press)

Calabrese EJ and Dhawan G. 2013b. "Historical use of x-rays: treatment of inner ear infections and prevention of deafness." *Hum Exp Toxicol* (in press)

Caspari E and Stern C. 1948. "The influence of chronic irradiation with gamma rays at low doses on the mutation rate in *Drosophila Melanogaster*." *Genetics* 33: 75-95. Available at: http://www.genetics.org/content/33/1/75.full.pdf+html?sid=cb861a39-fb63-48c4-bcbe-2433bb5c8d6a

Cavanaugh-O'Keefe J. 1995. "Introduction to eugenics." Available at: http://www.emmerich1.com/EUGENICS.htm

Cuttler JM. 2012. "Commentary on the appropriate radiation level for evacuations." *Dose-Response* 10: 473-479. Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3526322/

Feinendegen LE, Pollycove M and Neumann RD. 2012. "Hormesis by low dose radiation effects: low-dose cancer risk modeling must recognize up-regulation of protection." Therapeutic Nuclear Medicine. Springer. ISBN 973-3-540-36718-5. Available at: http://db.tt/UyrhlBpW

Fliedner TM, Graessle DH, Meineke V and Feinendegen LE. 2012. "Hemopoietic response to low dose-rates of ionizing radiation shows stem cell tolerance and adaptation." *Dose-Response* 10: 644-663. Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3526333/

Hurwitz S and Zuckerman SN. 1937. "Roentgen rays in the treatment of acute cervical adenitis." *Journal of Pediatrics* 10: 772-780.

International Commission on Radiological Protection (ICRP). 2012. "Report of ICRP Task Group 84 on initial lessons learned from the nuclear power plant accident in Japan vis-à-vis the ICRP system of radiological protection." Available at: http://www.icrp.org/docs/ICRP%20TG84%20Summary%20Report.pdf

Jaworowski Z. 1999. "Radiation Risk and Ethics." *Physics Today* 59(9): 24-29. Am Institute of Physics. Available at: http://dl.dropbox.com/u/71478013/Jaworowski-1999_Radiation-Risk-Ethics_PhysToday_copyright.pdf

Koana T, Takashima Y, Okada MO, Ikehata M, Miyakoshi J and Sakai K. 2004. "A threshold exists in the dose-response relationship for somatic mutation frequency indicated by x irradiation of *Drosophila*." *Rad Res* 161: 391-396.

Koana T, Okada MO, Ogura K, Tsujimura H and Sakai K. 2007. "Reduction of background mutations by low-dose x irradiation of *Drosophila* spermatocytes at a low dose rate." *Rad Res* 167: 217-221.

Luckey TD. 1980. "Hormesis with Ionizing Radiation." CRC Press.

Luckey TD. 1991. "Radiation Hormesis." CRC Press.

Murphy JB and Morton JJ. 1915. "The effect of roentgen rays on the rate of growth of spontaneous tumors in mice." *J Exper Med* 22(6): 800-803. Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2125377/pdf/800.pdf

Muller HJ. 1927a. "Artificial transmutation of the gene." *Science* 66(1699): 84-87.

Muller HJ. 1927b. "The problem of genetic modification." Proceedings of the 5th International Congress of Genetics. Berlin. pp234-260.

Ogura K, Magae J, Kawakami Y and Koana T. 2009. "Reduction in mutation frequency by very low-dose gamma irradiation of *Drosophila Melanogaster* germ cells." *Rad Res* 171: 1-8.

Oliver CP. 1930. "The effect of varying the duration of x-ray treatment upon mutation frequency." *Science* 71: 44-46.

Ozasa K, Shimizu Y, Suyama A, Kasagi F, Soda M, Grant EJ, Ritsu S, Sugiyama H and Kodama K. 2012. "Studies of the mortality of atomic bomb survivors, Report 14, 1950-2003: an overview of cancer and noncancer diseases." *Rad Res* 177: 229-243.

Pollycove M and Feinendegen LE. 2003. "Radiation-induced versus endogenous DNA damage: possible effect of inducible protective responses in mitigating endogenous damage." *Hum Exp Toxicol* 22: 290-306. Available at: http://www.belleonline.com/newsletters/volume11/vol11-2.pdf

Rodel F, Frey B, Gaipl U, Keilholz L, Fournier C, Manda K, Schollnberger H, Hildebrandt G and Rodel C. 2012. "Modulation of inflammatory immune reactions by low-dose ionizing radiation: molecular mechanisms and clinical applications." *Current Medical Chemistry* 19(12): 1741-1750.

Saji G. 2013. "A post accident safety analysis report of the Fukushima Accident – future direction of evacuation: lessons learned." Proceedings of the 21st International Conference on Nuclear Engineering. ICONE21. July 29 – August 2. Chengdu. China. ASME.

Schenck SG. 1935. "Roentgen therapy for acute cervical adenitis." *American Journal of Disease of Children* 49: 1472-1486.

Taylor LS. 1980. "Some nonscientific influences on radiation protection standards and practice, the 1980 Sievert Lecture." *Health Physics* 39: 851-874.

UNSCEAR. 2012a. "Report of the United Nations Scientific Committee on the Effects of Atomic Radiation." Fifty-ninth session (21-25 May 2012). Available at: http://daccess-dds-ny.un.org/doc/UNDOC/GEN/V12/553/85/PDF/V1255385.pdf?OpenElement

UNSCEAR. 2012b. "Biological mechanisms of radiation actions at low doses, a white paper to guide the Scientific Committee's future program of work." Available at: http://www.unscear.org/docs/reports/Biological_mechanisms_WP_12-57831.pdf

US National Library of Medicine. 2013. "Glossary. Eugenics." Available at: http://ghr.nlm.nih.gov/glossary=eugenics

WHO. 2013. "Health risk assessment from the nuclear accident after the 2011 Great East Japan Earthquake and Tsunami, based on a preliminary dose estimation." World Health Organization. Available at: http://apps.who.int/iris/bitstream/10665/78218/1/9789241505130_eng.pdf
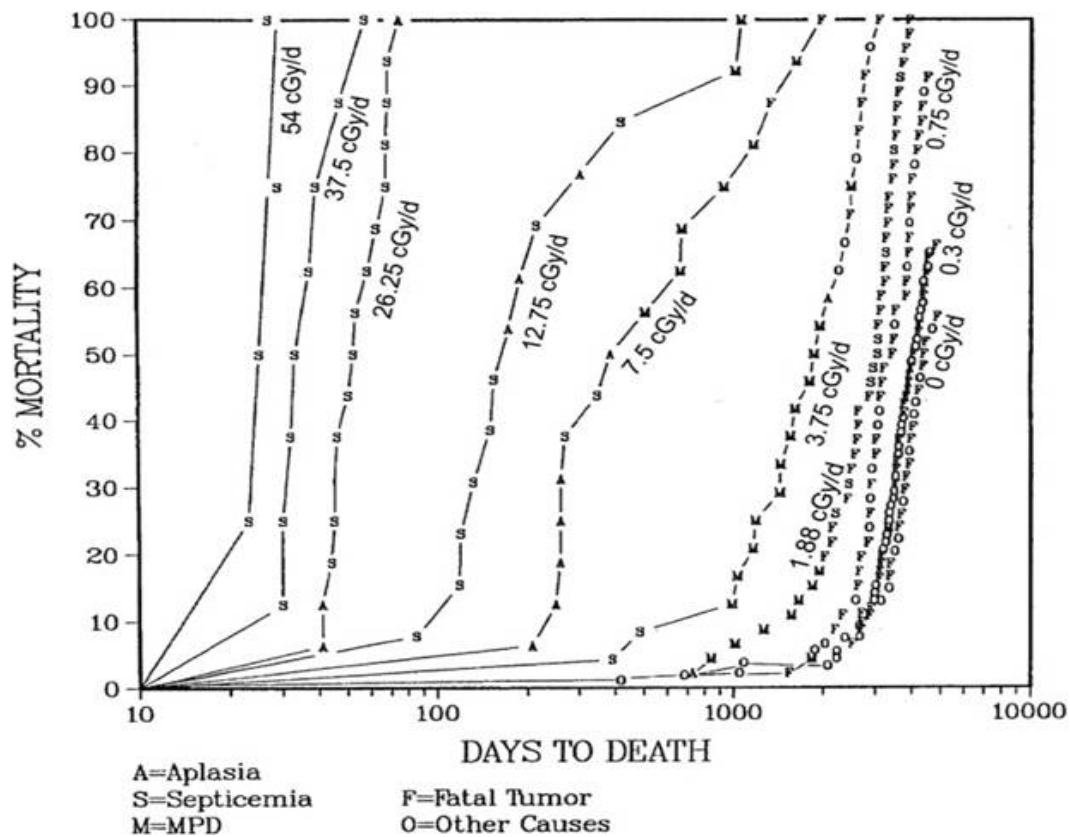
Figure 1   Mortality curves of dogs subjected to whole-body chronic gamma irradiation at different dose rates (Fliedner et al. 2012, Figure 3)
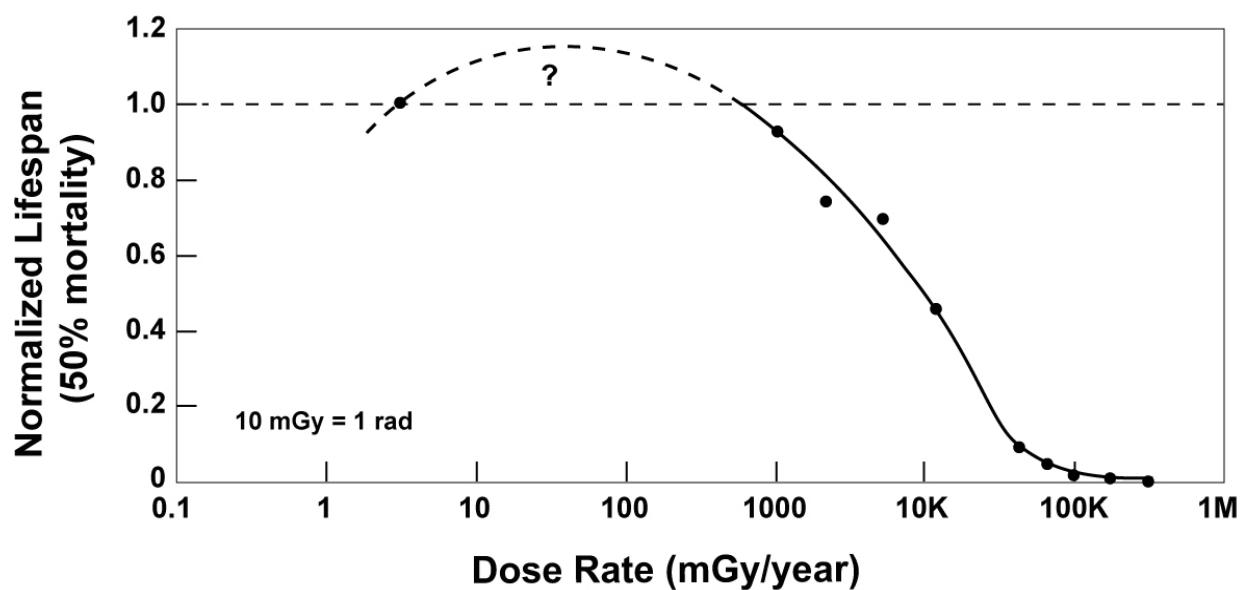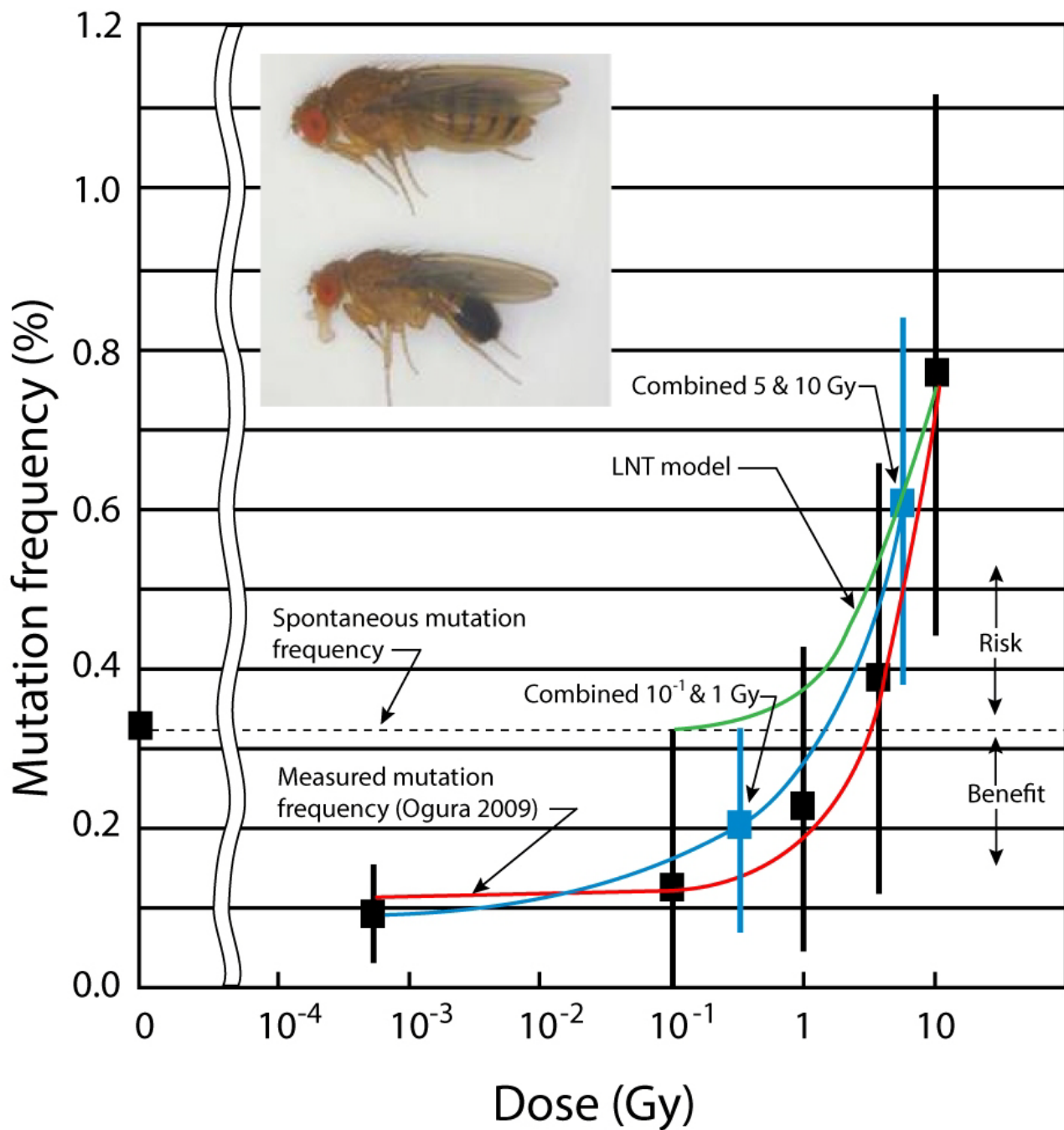

Figure 2.   Lifespan versus radiation level

Figure 3. Fruit fly mutation frequency versus radiation dose. A binomial distribution is assumed for the occurrence of the mutations. Each error bar is two standard deviations from the mean frequency. The (blue) data points at 0.3 Gy (0.19%) and at 7 Gy (0.61%) are obtained by "pooling" the data measured by Ogura et al (2009) at 10^{-1} and 1 Gy, and at 5 and 10 Gy, respectively. Note that the mean mutation frequency is below the spontaneous level (0.32%) when the dose is below 1 Gy.