Radiation Risk

The purpose of considering amendments to Title 10 of the *Code of Federal Regulations* (10 CFR) Part 20 is to modify the U. S. Nuclear Regulatory Commission's (NRC's) radiation protection standards to reflect developments in the scientific knowledge underlying radiation protection that have occurred over the last 30 years. These developments not only include scientific information on radionuclide uptake and metabolism, but include an increased understanding of the inherent health risks associated with radiation exposure.

The current 10 CFR Part 20 is based upon the radiation risk estimates from the 1970's, as reflected in the International Commission on Radiological Protection (ICRP) recommendations in 1977. The radiation risk was estimated to be 1.25×10^{-4} per rem (1.25×10^{-2} per Sv), and considered cancer mortality and risks of heritable disease.

Evolution of Risk Estimates:

Following its 1987 meeting in Como, Italy, the ICRP issued a statement that reviewed the existing estimates of the biological risks of ionizing radiation and the preliminary data from the reanalysis of the Hiroshima-Nagasaki atomic bomb follow-up studies. Reanalysis of these data indicated that the risks from gamma radiation are approximately a factor of 2 higher than previous estimates for the general population and are also higher, but by a smaller factor, for workers. The ICRP concluded in 1987 that this information was not considered sufficient at the time to warrant a change in the dose limits for occupational exposure. The ICRP also noted that the potential higher risks indicated by the reanalysis of the atomic bomb data should not be a major consideration as the dose limits should not be of primary importance in controlling doses if the principle of keeping radiation exposures "As Low As is Reasonably Achievable" (ALARA) is being practiced. This position has since been modified by the ICRP 1990 recommendations.

The 1988 report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) contains information on the health risks of ionizing radiation determined from a reevaluation of the data on the survivors of the Hiroshima-Nagasaki atomic bombings. Based on these data, the radiation risk at high doses and high dose rates was estimated to be 7.1×10^{-4} fatal health effects per rad (7.1×10^{-2} per Sv). For estimating the risk from radiation doses below 100 rads (1 Gy), UNSCEAR recommended that a dose rate reduction factor be applied to account for the reduced effectiveness of lower doses and lower dose rates. This would lead to an estimated risk between (0.7 and 3.5) $\times 10^{-4}$ health effects per rad ((0.7 - 3.5) $\times 10^{-2}$ per Sv) for low doses such as those encountered in routine occupational exposure. The fatal cancer risk as estimated by the 1988 UNSCEAR report for low doses are between 1.7 times lower to 2.8 times greater than the 1977 ICRP estimate.

The 1990 report of the National Academy of Sciences' Committee on the Biological Effects of Ionizing Radiation (BEIR-V) was a comprehensive reevaluation of the health risks of radiation exposure based upon the revised dose estimates for the survivors of the Hiroshima-Nagasaki atomic bombings. The BEIR-V report, like the 1988 UNSCEAR report, indicates that a

reduction factor should be applied to the risk estimates derived from high doses and dose rates in order to apply them to low dose and low dose situations. Assuming a factor of 2 reduction, the BEIR-V would give a lifetime risk of a radiation-induced cancer fatality of about 4×10^{-4} fatal cancers per rem (4×10^{-2} per Sv) for workers and 5×10^{-4} fatal cancers per rem (5×10^{-2} per Sv) for the general population, the higher value for the public being associated with the higher sensitivity and the longer period of elevated risk associated with the younger ages present in the general population. This value is four times as large as the estimate in the 1977 ICRP recommendations.

The 1990 ICRP Recommendations contain another comprehensive reevaluation of the health risks of radiation exposure. The ICRP concluded, after reviewing the available experimental information on dose-response relationships and the influence of dose and dose rate, that the most probable response is linear quadratic in form for low Linear Energy Transfer (LET) radiation. The linear coefficient at low doses or low dose rates is obtained from the high dose, high dose rate estimates of risk. Averaging estimates of probability for total fatal cancer from the 1988 UNSCEAR report and the BEIR V report with estimates derived by ICRP, the average nominal risk acute high dose exposure is 10×10^{-4} fatal cancers per rem (10×10^{-2} per Sv). The ICRP applied a dose and dose rate effectiveness factor of 2 to yield a nominal value of 5×10^{-4} per rem (5×10^{-2} per Sv) for the probability of induced fatal cancer in a population of all ages. This value is four times as large as the risk estimate contained in the 1977 ICRP recommendations. A smaller value would be obtained for a working population of age 18 to 64 years, at about 4×10^{-4} per rem (4×10^{-2} per Sv). The ICRP made its own estimate of how this fatal cancer risk is distributed among organs and the length of life lost for cancer in each of these organs by further analysis of the data on the atomic bomb survivors.

Based in part on the four fold increase in the average estimates of cancer mortality for low dose and low dose rate radiation exposure, the ICRP recommended a limit on effective dose of 2 rem (20 mSv) per year, averaged over 5 years with the further provision that the effective dose should not exceed 5 rem (50 mSv) in any single year. The ICRP noted that dose limits are needed as part of the control of occupational exposure, both to impose a limit on the choice of dose constraint and to provide a protection against errors of judgment in the application of optimization. The dose limit forms only a part of the system of protection aimed at achieving levels that are ALARA. It represents the point at which regular, extended, deliberate, occupational exposure can reasonably be regarded as only just tolerable.

In 2000, UNSCEAR issued another comprehensive review of the broad field of experimental studies of the radiation effects in cellular systems and in plants and animals. Many of the responses to ionizing radiation observed in plants and animals form a basis for the knowledge of human radiation effects and can often be evaluated in more detail than studies of humans. Furthermore, fundamental radiobiology includes the field of molecular radiobiology which is contributing to an understanding of the mechanisms of radiation response. The UNSCEAR concluded that even at low doses radiation may act as a mutational initiator of tumorigenesis and that anti-tumorigenic defenses are unlikely to show low-dose dependency. Hence, it is unlikely there might be a threshold level of exposure below which biological response does not occur. Such a threshold could only occur if DNA repair processes were totally effective in that dose range or if a single radiation track were unable to produce an effect. The cellular

processes such as apoptosis and cellular differentiation that can protect against later phases of tumorigenesis are judged to be efficient but can be bypassed; there is no reason to believe that those defenses act differently on spontaneous and radiation-induced tumors or have specific dose dependencies.

In their 2000 report, UNSCEAR derived risk estimates for radiation-induced cancer. For a global population of all ages and both genders, it is suggested that the lifetime risk estimate for solid cancer mortality might be taken as 9×10^{-4} per rem (9×10^{-2} per Sv) for men and 13×10^{-4} per rem (13×10^{-2} per Sv) for women. The uncertainties in the estimates may be a factor of 2, higher or lower. The estimates could be reduced by 50 percent for chronic exposures and solid cancer incident risks can be taken as being roughly twice those for cancer mortality. Lifetime cancer risk estimates for those exposed as children might be twice the estimates for a population exposed at all ages. Finally, UNSCEAR reiterated that the experience of the Japanese atomic bomb survivors provides compelling evidence for linearity in estimating excess risks of solid cancers; therefore, as a first approximation, linear extrapolation of the estimates of cancer risk at 100 rem (1 Sv) could be used for estimating solid cancer risks at lower doses.

In contrast to the recommendations of the ICRP and the technical reviews by UNSCEAR and the BEIR committee, the French National Academy of Medicine and the French Academy of Sciences published a report in March 2005 that cautioned against using the linear, no threshold (LNT) hypothesis to estimate radiation risk at very low radiation exposures. The French report suggested that there is a lower effectiveness of inducing biological damage from low doses (compared to acute exposures of high dose radiation), or the existence of a practical threshold. The report suggested that the lower effectiveness could be related to either the failure of a very low dose to sufficiently activate cellular signaling for DNA repair mechanisms, or to an association between apoptosis, error-free DNA damage repair, and increased immune surveillance. However, the French did acknowledge that they did not find it possible to define the threshold level (between 0.5 and 5 rem (5 and 50 mSv)) or to provide the evidence for it.

This French report raises doubts on the validity of using the LNT hypothesis for evaluating the carcinogenic risk of low dose (< 10 rem; 100 mSv) radiation exposure and even more for very low dose (< 1 rem; 10 mSv) exposures. While acknowledging that the LNT concept can be a useful, pragmatic tool for instituting and managing a system of radiological protection, the report asserts that LNT is not based on current biological concepts, so it should not be used for assessing the risks associated with low and or very low doses.

The 2006 report of the National Academy of Sciences' Committee on the Biological Effects of Ionizing Radiation (BEIR-VII) is their most recent reevaluation of the health risks of radiation exposure based on a review of all relevant biological information important to the understanding of modeling of those health effects. The BEIR committee considered radiation risk information from studies of persons exposed for medical, occupational, and environmental reasons. Along with the review of these bodies of information, the committee reviewed data on cancer mortality and incidence from the survivors of the Hiroshima-Nagasaki atomic bombings based on

improved dose estimates that were published in 2002. Other phenomena reviewed by the committee that suggests enhancement or reduction in radiation effects include adaptive response, low-dose hypersensitivity, bystander effect, hormesis, and genetic instability; biological effects based on phenomenological data with little mechanistic information. The committee concluded that the current scientific evidence is consistent with a hypothesis that there is a linear dose-relationship between exposure to ionizing radiation and the development of radiation-induced solid cancers in humans. The committee further judged that it is unlikely that a threshold exists for the induction of cancers but notes that the occurrence of radiation induced cancers at low doses will be small. Results based on linear models and reduced by a dose and dose rate effectiveness factor of 1.5 (versus 2) for the U.S. population varies from 4.1 to 6.1 x 10^{-4} per rem (4.1 to 6.1 x 10^{-2} per Sv) for fatal solid cancer. Results for leukemia are based on a linear-quadratic model.

Other health effects such as heart disease and stroke occur at high radiation doses, but the report concluded that additional data must be gathered before an assessment can be made of any possible connection between low doses of radiation and noncancer health effects.

The biological risks of radiation exposure that were documented by UNSCEAR in their 2006 report note that risk estimates vary for different populations (e.g., U.S. vs Japanese) and with different risk models. The 2006 UNSCEAR risk estimates are somewhat lower, although not much lower, than those previously estimated by UNSCEAR (2000) or the U.S. National Academy of Science (BEIR – V, BEIR – VII). A reduction of about 10 percent in the solid cancer risk estimate may be due to the new atomic bombings dosimetry that was revised in 2002 and another small 3 – 7 percent reduction may be due to increased follow-up of the atomic bomb survivors. The statistical uncertainties in the above estimates may be on the order of a factor of 2 higher and the lower bound includes zero. The UNSCEAR also noted that lifetime solid cancer risk estimates for those exposed as children might be factors of 2-3 times higher than the estimates for members of the public; studies of *in utero* radiation exposures show that the fetus is particularly sensitive with elevated risk being detected at doses of 1 rad (10 mGy) and above. Finally, UNSCEAR concluded that the experience of studies of the survivors of the atomic bombings is consistent with a linear dose response for the risk of all solid cancers combined. Therefore, as a first approximation, linear extrapolation of the estimates of risk following an acute dose of 100 rem (1 Sv) can be used for estimating solid cancer risks at lower doses.

The ICRP issued new recommendations on radiological protection in 2007, which formally replaced the 1990 recommendations. The revised recommendations included consideration of the detriment arising from cancer, non-cancer, and heritable effects of radiation on health. The accumulation of cellular and animal data relevant to radiation protection since 1990 has strengthened the view that DNA damage response processes in single cells are critically important to the development of cancer after radiation exposure. For the purposes of radiation protection the ICRP continues to judge that the weight of evidence on fundamental cellular processes coupled with dose-response data supports the view that in the low dose range, below 10 rem (100 mSv), it is scientifically plausible to assume that the incidence of cancer or heritable effects will rise in direct proportion to an increase in the equivalent dose in the relevant organ or tissue.

The ICRP also continues to emphasize that while the LNT model remains a scientifically plausible element in its system of radiological protection, biological/epidemiological information that would unambiguously verify the hypothesis that underpins the model is unlikely to be forthcoming. In arriving at this judgment, the ICRP considered potential challenges associated with information on cellular adaptive responses, the relative abundance of spontaneously arising and low-dose-induced DNA damage and the existence of post-irradiation cellular phenomena of induced genetic instability and bystander signaling. Since the estimation of cancer risk incidence and mortality is based upon direct human epidemiological data, any contribution from these biological mechanisms would be included in that estimate.

Since 1990, further epidemiological information from medical, occupational, and environmental exposures has accumulated. Much of the new information reviewed by ICRP came from additional follow-up of survivors from the Hiroshima-Nagasaki atomic bombings. Overall, the 2007 ICRP recommendations reported that the current cancer risk estimates derived from the survivors of the atomic bombings are not greatly changed but the inclusion of cancer incidence data provides a firmer foundation for cancer risk modeling.

A follow-up assessment conducted by UNSCEAR in 2010 reconfirmed many of UNSCEAR's 2006 risk estimates derived from the atomic bomb survivors. By contrast, many other groups were exposed over long periods to low doses, and sometimes the exposure was from internally incorporated radionuclides. Valuable information has been provided by epidemiological studies of the health of workers at the Mayak nuclear complex in the southern Urals of the Russian Federation, and of the population near the Techa River whose exposure was due to radioactive discharges from that facility. Follow-up of those exposed as a consequence of the Chernobyl accident has provided useful information of the effects of low-dose external radiation exposure, and on the effects of thyroid exposure to radioiodine. Overall, the cancer risk estimates from these studies do not differ significantly from those obtained from the studies of the atomic bombing survivors in Japan. By contrast, studies on human populations living in areas with elevated natural background radiation in China and India do not indicate that radiation at such levels increases the risk of cancer.

In 2011, the ICRP issued a statement concerning recent epidemiological evidence which suggests that there are some tissue reaction effects (e.g., heart disease, stroke, and cataracts); where threshold doses might be significantly lower than previously considered. The threshold in absorbed dose for these tissue reaction effects is now considered to be 50 rad (0.5 Gy). As a result, the ICRP recommended that the occupational equivalent dose limit to the lens of the eye be reduced from 15 rem (150 mSv) in a year to 2 rem (20 mSv) in a year, averaged over defined periods of 5 years, with no single year exceeding 5 rem (50 mSv).

In April 2011, the U.S. Environmental Protection Agency (EPA) published a revision of their methodology and estimates for radiogenic cancer risk for the U.S. population (EPA 402-R-11-001). This revision was based upon the estimates and methodology from the BEIR VII report, with additional extensions and modifications for the risk from low LET radiation, estimates for additional types of cancer, and modifications for estimating breast cancer mortality risk and thyroid cancer risk. Summary risk coefficients were calculated for a stationary population defined by the 2000 U.S. vital statistics. For uniform whole-body exposure of low-dose gamma radiation to the entire population, the cancer incidence risk coefficient is 1.16×10^{-3} per rad (1.16×10^{-1} per Gy) with a 90% confidence interval of 5.6×10^{-4} to 2.1×10^{-3} per rad (5.6×10^{-2} to 2.1×10^{-1} per gy). The corresponding coefficient for cancer mortality is about one-half that for

incidence: 5.8×10^{-4} per rad (5.8×10^{-2} per Gy). The EPA plans to use this information to develop a revision of Federal Guidance Report 13, "Cancer Risk Coefficients for Environmental Exposure to Radionuclides."

Conclusions:

The NRC staff has concluded that there have been significant changes in radiation risk estimates, and the methodologies for recommending dose limits. These changes have been developed by both national and international organizations. Given these changes, the staff has concluded that there is a sufficient risk informed scientific basis to move the NRC's regulatory framework to a greater degree of alignment with the ICRP recommendations.