

The Science Behind The ICRP 2005 Recommendations:
Biological and Epidemiological Information

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The ICRP 2005 Recommendations are stated to be “based on a simple, but widely applicable, general system of protection that will clarify its objectives and will provide a basis for the more formal systems needed by operating managements and regulators”. Underlying each of the broad areas of recommendation is a series of Foundation Documents that provide details of the most recent and/or appropriate scientific research in support of the recommendations. This presentation will focus on the approaches for quantitating health effects attributable to radiation exposure and the biological mechanisms that underlie these effects. The adverse health effects are grouped as (1) tissue reactions (2) cancer development in exposed individuals and heritable disease in their offspring. For considerations of tissue injury, ICRP has conducted a comprehensive review of experimental and epidemiological studies to revisit current estimates of thresholds for tissue reactions (deterministic effects) and consider revising these. For hereditary risks, ICRP has significantly modified its approach by using spontaneous human mutation rates in conjunction with radiation-induced mutation rates from mouse studies. In addition, hereditary risks are considered up to the second generation only. The overall result is a reduction in the genetic risk that will tend to reduce the value of tissue weighting factor for gonads. New epidemiological data on cancer mortality and incidence have been considered by ICRP and the broad conclusion is that the linear no threshold model remains as the best overall fit to the data. An important component of such a conclusion is that extensive data on cancer incidence are available from the A-bomb Life Span Study (LSS); these provide more accurate diagnosis and take account of relatively high prevalence but low mortality tumors. In addition, models were developed to facilitate transport of risk across populations, particularly based on data from the LSS population. The risk models also incorporated the concept of quality of life detriment, which is a departure from the detriment based on mortality alone. The recent data that impacted the value of DDREF were reviewed, but it was concluded that the current value of 2 was compatible with both human and laboratory animal data. A review of data for the production of non-cancer diseases after radiation was conducted, particularly for the LSS. While it is clear that there is an increase in several such diseases, the data available are not sufficient for their inclusion in the estimation of detriment at doses in the range of a few tens of mSv. Some of these judgments will be considered in greater detail during the presentation.

Educational Objectives:

1. To understand the biological data that for the basis for radiation risk assessments.
2. To understand how new data have necessitated a reconsideration of the basis for radiation protection standards.

This abstract has been reviewed in accordance with EPA guidelines but it does not necessarily reflect EPA policy.

