8 Radiation Safety Guides

Radiation safety standards and regulations undergo continuous review and changes. These changes occur mainly as a response to a public policy based on attitudes of the public and on the philosophy of preventive conservatism, and also because of the increasing sensitivity of radiation-measuring instruments. The continual restriction of acceptable dose limits implies that earlier limits were unsafe. However, there has been no verifiable increase in radiogenic diseases among radiation workers whose radiation doses were within the limits recommended by scientific advisory committees (the International Commission on Radiological Protection (ICRP) and the National Council on Radiation Protection and Measurements (NCRP)) in 1934 and the limits established by governmental regulatory agencies after 1945.

ORGANIZATIONS THAT SET STANDARDS

The hazards of ionizing radiation became apparent almost immediately. Wilhelm Roentgen discovered X-rays in November 1895, and he announced his discovery on January 1, 1896, in a paper that he had prepared for presentation to the Physical-Medical Society of Würzburg. At about the same time, Emile Grubbe, an American physicist who was experimenting with a Crooke's tube (a cathode-ray tube) similar to the one used by Roentgen, suffered severe burns on his hands as a result of holding the energized tube in his hands. Then, in May of that same year, a man who had a diagnostic radiograph made of his head suffered skin burns and loss of hair on the side of his face that had been exposed to the X-rays. Henri Becquerel, who discovered radioactivity in 1896, developed an ulcer on the skin of his chest as a result of having kept a test tube containing a radium salt in his vest pocket. After surgical treatment, the wound healed, but it left a painful scar. The year 1899 marked the first successful use of X-rays to cure a cancer, a basal-cell carcinoma on the face of a woman. Thus, from the very beginning of the use of radiation sources for beneficial purposes, harmful effects were observed.

As the usefulness of radiation in medicine and science was being discovered, reports of harmful radiation effects continued, causing various practitioners to suggest a variety of radiation safety rules. The first organized action in radiation safety was taken in 1915 by

the British Roentgen Society. The X-ray and Radium Protection Committee of the British Roentgen Society published further recommendations in 1921 and in 1927.

International Commission on Radiological Protection

In 1925, the radiological societies of several countries met in London at the First International Congress of Radiology. Among the main topics discussed at the meeting were radiation protection and the need for a committee to deal with questions of radiation safety. Then, in 1928, at the Second International Congress of Radiology, a committee called the International X-ray and Radium Protection Committee was established to provide guidance in these matters. At that time and for many years afterward, its main concern was regarding the safety aspects of medical radiology. Its interests in radiation protection expanded with the widespread use of radiation outside the sphere of medicine, and, in 1950, its name was changed to the International Commission on Radiological Protection (ICRP) in order to describe its area of concern more accurately. Since its inception, the ICRP has been recognized as the leading agency for providing guidance in all matters of radiation safety. In describing its operating philosophy, the ICRP states: "The policy adopted by the Commission in preparing recommendations is to deal with the basic principles of radiation protection, and to leave to the various national protection committees the responsibility of introducing detailed technical regulations, recommendations, or codes of practice best suited to the needs of their individual countries" (ICRP Publication 6, p. 1, Pergamon Press, Oxford, U.K., 1964). In discussing the development of its recommendations, the ICRP says: "Since there is little direct evidence of harm at levels of annual dose at or below the limits recommended by the Commission, a good deal of scientific judgment is required in predicting the probability of harm resulting from low doses. Most of the observed data have been obtained at higher doses and usually at high dose rates." The ICRP goes on to say: "The estimation of these consequences and their implications necessarily involves social and economic judgments as well as scientific judgments in a wide range of disciplines" (ICRP Publication 60, pp. 1 and 2, Pergamon Press, Oxford, U.K., 1991). The ICRP expended its philosophy to add ". . . an approach for developing a framework to demonstrate radiological protection of the environment" (ICRP Publication 103, p. 1, Pergamon Press, 2007). An approach for environmental protection was next added: ". . . the Commission's approach to environmental protection needs to be applied sensibly and in a manner that is commensurate with the (potential) risk of harmful effects under different exposure situations." (ICRP Publication 114, Elsevier, 2009) The ICRP's published reports and recommendations are listed in the Suggested Readings, at the end of this chapter.

Initially, the recommendations of the ICRP were based on the *tolerance dose*. The tolerance dose was believed to be a dose that the body can tolerate, and thus adherence to this dose limit would prevent observable harmful radiation effects. To this end, the dose to tissue deeper than 1 cm (the *deep dose*) and the skin dose to skin at a depth of 0.007 cm (the *shallow dose*) of 300 and 600 mrems per week, respectively, were recommended. When genetic damage was assumed to be the effect to be prevented, a deep-dose equivalent of 5 rems/yr was recommended in ICRP Publication 2 in 1959. By 1977, continued observation of radiation effects on the survivors of the atomic bombings in Japan, including the absence of any observable genetic effects, led the ICRP to update its radiation safety recommendations. Its new recommendations, which were published in ICRP Publication 26, are based on an *acceptable-risk* concept. This new basis for radiation safety standards recognized cancer as the main biological effect of concern. The biomathematical model for radiation carcinogenesis postulates that a single radiation-induced change in a DNA molecule can initiate

an oncogenic process. According to this model, there is no dose below which cancer cannot occur. This means that every increment of radiation dose carries a proportional increase in risk of radiogenic cancer. Accordingly, radiation safety standards were recommended on the basis of a risk that would be accepted by society in exchange for the benefits resulting from radiation use at the recommended limit.

ICRP 26 also recognized that different organs and tissues have different likelihoods of developing radiogenic cancer. This fact led to the introduction of the concept of effective dose, which considers the risk of stochastic effects from nonuniform irradiation relative to the risk from uniform whole-body radiation. As a consequence, ICRP Publication 26 recommended a maximum effective dose equivalent (EDE) of 50 mSv (5000 mrems) in 1 year and also said that this limit should include the sum of external radiation dose and the dose from internally deposited radionuclides. By 1990, the continuing studies of the Japanese survivors of the atomic bombings suggested that the probability of fatal radiogenic cancer might have been underestimated by a factor perhaps as great as 4 in the earlier recommendations. Accordingly, in ICRP Publication 60, which was issued in 1990, the commission recommended a limit on EDE for occupational exposure of 20 mSv (2000 mrems) averaged over a 5-year period (100 mSv, or 10,000 mrems in 5 years), with a limit of 50 mSv (5000 mrems) in any single year. ICRP Publication 103 was issued in 2007, reaffirming and updating Publication 60 recommendations, adding and modifying radiation and tissue weighting factors, and incorporating the information and models published over the previous years. ICRP Publication 130 (2015) was issued to replace ICRP 30, 68, 54, and 78. The use of voxel phantoms (ICRP 110), the updated alimentary tract model (ICRP 100), revised decay schemes (ICRP 107), revisions to the Human Respiratory Tract Model (HRTM), as well as male, female, and pediatric phantoms were incorporated into ICRP 130. These changes have (admittedly) made the models more complex than that required for radiological protection, and the derivation and use of some have been relegated almost exclusively to computer programs. The majority of the computations for internal, and many external, doses are now performed using Monte Carlo programs. Finally, in 2011, the ICRP recommended that the dose limit to the lens of the eye be changed to 20 mSv per year averaged over 5 consecutive years, and 50 mSv in any single year, with a lifetime limit of 0.5 Gy to the eye.

International Atomic Energy Agency

The International Atomic Energy Agency (IAEA), a specialized agency of the United Nations that was organized in 1956 in order to promote the peaceful uses of nuclear energy, recommends basic safety standards that are based, to the extent practically possible, on the ICRP recommendations.

> Under its Statute the International Atomic Energy Agency is empowered to provide for the application of standards of safety for protection against radiation to its own operations and to operations making use of assistance provided by it or with which it is otherwise directly associated. To this end authorities receiving such assistance are required to observe relevant health and safety measures prescribed by the Agency.
>
> (From *Safe Handling of Radioisotopes.* Safety Series No. 1. IAEA, Vienna, 1962.)

The health and safety measures prescribed by IAEA are published according to subject in its *Safety Series*. The first set of recommendations was published in 1962, and a revised set

of basic safety standards, which was based on ICRP Publication 26, was published in 1982. The appearance of ICRP Publication 60 in 1990 led the IAEA, in 1995, to publish a third major revision of its basic safety standards for protection against ionizing radiation and for the safety of radiation sources. These safety standards serve as the basis for the regulation of both *practices* (any human activity that may increase the likelihood of additional dose to anyone) and *interventions* (an action to mitigate the consequences of an accidental exposure or of a practice that has gone out of control).

> The IAEA's safety standards are not legally binding on Member States but may be adopted by them, at their own discretion, for use in national regulations in respect of their own activities. The standards are binding on the IAEA in relation to its own operations and on States in relation to operations assisted by the IAEA. Any State wishing to enter into an agreement with the IAEA for its assistance in connection with siting, design, construction, commissioning, operation, or decommissioning of a nuclear facility or any other activities will be required to follow those parts of the safety standards that pertain to the activities to be covered by the agreement. However, it should be recalled that the final decisions and legal responsibilities in any licensing procedures rest with the States.
>
> (From IAEA Safety Standards Series. *Application of the Concepts of Exclusion, Exemption and Clearance*. Safety Guide No. RS-G-1.7, 2004.)

International Labor Organization

The International Labor Organization (ILO), which was founded in 1919 and then became part of the League of Nations, survived the demise of the League to become the first of the specialized agencies of the United Nations. Its concern generally is with the social problems of labor. Included in its work is the specification of international labor standards dealing with the health and safety of workers. These specifications are set forth in the *Model Code of Safety Regulations for Industrial Establishments for the Guidance of Governments and Industries*, in the recommendations of expert committees, and in technical manuals. In regard to radiation, the model code has been amended to incorporate those recommendations of the ICRP that are pertinent to control of occupational radiation hazards, and several manuals dealing with radiation safety in the workplace have been published.

International Commission on Radiological Units and Measurements

The International Commission on Radiological Units and Measurements (ICRU), which works closely with the ICRP, has had, since its inception in 1925, as its principal objective, the development of internationally acceptable recommendations regarding the following:

1. quantities and units of radiation and radioactivity,
2. procedures suitable for the measurement and application of these quantities in clinical radiology and radiobiology, and
3. physical data needed in the application of these procedures, the use of which tends to assure uniformity in reporting.

In terms of its operating policy: "The ICRU feels it is the responsibility of national organizations to introduce their own detailed technical procedures for the development and maintenance of standards. However, it urges that all countries adhere as closely as possible

to the internationally recommended basic concepts of radiation quantities and units" (ICRU Report 32, 1979).

Nuclear Energy Agency

The Nuclear Energy Agency (NEA) is a division of the Organization for Economic Cooperation and Development (OECD), which is an international organization of 27 industrialized states that cooperate to further economic development among its members. The function of the NEA is to promote the development of scientific, engineering, and legal principles for the safe and beneficial use of nuclear energy for peaceful purposes.

International Organization for Standardization

The International Organization for Standardization (ISO) is a nongovernmental organization that is a response of industries to the globalization of commerce. Its objective is to standardize business and manufacturing practices so that customers or clients in various parts of the world will operate on a level-playing field. That is, they will all adhere to the same standards. Although not explicitly radiation-related, two series of ISO standards are relevant to health physicists. The ISO 9000 series, which was adopted in 1987 in expectation of trade among the members of the European Common Market, deals with quality standards for manufactured items. ISO 9000 series includes five parts: Parts 9000 and 9004 deal with general guidelines, while parts 9001, 9002, and 9003 are well-defined quality standards that deal with all the commercial aspects of engineering, manufacturing, installing, and servicing a product. ISO 9000 certification means that the product was manufactured according to rigid standards and actually performs according to the maker's claims. The application of ISO 9000 standards to obtain uniformity among radiometric instruments made in various countries will facilitate the international interchange of radiation measurements and will add confidence in the accuracy of the measurements.

Another series, ISO 14000, was developed in order to minimize the adverse environmental impact of an organization's activities and products. General objectives of ISO 14000 are the reduction of waste and of the cost of waste management, the conservation of energy and materials, and the optimization of distribution. In addition to requiring demonstration of compliance with all applicable regulatory requirements, certification to ISO 14001 requires identifying each aspect of an operation that might have an environmental impact, prioritizing these impacts, and establishing operating procedures to eliminate or to mitigate the detrimental environmental impacts. It also requires that all employees be trained in sensitivity to the environment, the prevention of pollution, and safe practices. Of particular interest to health physicists in the context of the "NIMBY" (Not In My Back Yard) complex is ISO 14031. This part requires that the organization gives due consideration to the viewpoints of the affected public regarding any possible pollution resulting from the operations of the organization.

National Agencies

Although international scientific agencies recommend radiation safety standards and practices, legal authority for radiation safety is exercised by regulatory agencies established by national states. In almost all cases, the national agencies base their regulations on the recommendations of the international scientific agencies. In the United States of America, for example, the Environmental Protection Agency (EPA) sets radiation safety standards, while several different regulatory agencies, including the Nuclear Regulatory Commission (NRC),

the Occupational Health and Safety Administration (OSHA), and the Department of Energy (DOE), promulgate radiation safety regulations, according to the EPA standards, within their areas of responsibility. In Canada, the radiation safety regulatory body is the Canadian Atomic Energy Authority; in the United Kingdom, it is the Health Protection Agency's Radiation Protection Division; and in France, the Commissiarat d'Energie Nucleaire is the national regulatory agency.

PHILOSOPHY OF RADIATION SAFETY

Public Health and Radiation Safety Practice

Public health is that responsibility which rests on the organized community for the prevention of disease and the promotion of health. Prevention of disease through community efforts is necessary as—because of the population explosion and communal living—we are no longer able to structure our own individual environments. Our environment is determined mainly by the activities of others. The objectives of public health differ significantly from those of "private" health (clinical medicine). The aim of clinical medicine is to cure sick people, while the aim of public health is to keep healthy people healthy. A comparison of public health characteristics to "private" health characteristics is shown in Table 8-1.

Radiation safety standards and public policy regarding radiation are public health concerns because (1) we cannot structure our own individual environments, and (2) the effects of low-level radiation are not unique and, if they occur, are detectable only by epidemiologic means. No verifiable, detrimental radiation health effects have ever been observed among populations exposed within the range of variability of background radiation. Until recently, no detrimental radiation effects were found among the population of radiation workers whose doses were within the limits recommended in ICRP Publication 2. A study in 2005 of 407,391 nuclear workers in 15 countries found a small (1–2%) excess risk of cancer, although a re-analysis of the study (2014) has found there may be some difficulty with this interpretation of the data.

Cancer and genetic defects are the principal radiation effects of public health concern. Both these stochastic effects are attributed to the same biological phenomenon, namely, the loss of information in a base pair by the breaking of the base-pair bond in a DNA molecule. The zero-threshold model is believed to be conservative because of base-pair repair and because the information is replicated several times within the DNA molecule. According to this model, breaking 100 base pairs in a single individual or 1 base pair each in 100 individuals leads to the same probability of initiating an oncogenic lesion or a point mutation. This leads to the concept

TABLE 8-1 Comparison of "Private" Health (Clinical Medicine) to Public Health

PRIVATE HEALTH	PUBLIC HEALTH
Patient is an individual	Patient is the community
Particular disease is either present or absent	All diseases present all the time
Health status evaluation: blood pressure, temperature, blood count, etc.	Statistical and epidemiologic data
Causes: microbial, biochemical, trauma, psychological	Ecological causes, social ills
Therapy: physical, chemical, psychological	Engineering, medical, sociopolitical
Individual pays	Society, or the community, pays

of the *collective dose*, which was introduced by the ICRP in 1977. The collective dose, which is a measure of the total amount of DNA damage in a population, is simply the sum of all the dose equivalents received by the individual members of a population and is expressed in person-rems in the traditional system of health physics units and in person-sieverts in SI units:

$$S = \sum_i n_i H_i, \tag{8.1}$$

where n_i is the number of individuals who receive a dose equivalent to H_i. For example, if, during a given year, 800 workers in a certain nuclear facility received an average dose equivalent of 0.2 rem (0.002 Sv), 199 workers averaged 0.6 rem (0.006 Sv), and 1 worker received 2.6 rems (0.026 Sv), then the collective dose equivalent would be

$$S = (800 \text{ persons} \cdot 0.2 \text{ rem}) + (199 \text{ persons} \cdot 0.6 \text{ rem}) + (1 \text{ person} \cdot 2.6 \text{ rems}),$$

$$S = 282 \text{ person-rems } (2.82 \text{ person-Sv}).$$

The collective dose is the basis for calculating the stochastic impact of radiation exposure to a large group or to a population and, thus, for public health control of radiation. It should be emphasized that the collective-dose concept applies only to the postulated stochastic effects in a large population. The collective-dose concept is applied by postulating that a given collective dose will result in the same total number of detrimental effects regardless of the size of the population and the distribution of the dose. The ICRP postulates 500 excess cancer deaths among a population whose collective dose is 10^4 person-Sv (10^6 person-rems) regardless of how the collective dose is distributed among the population (in this case we are assuming a population in the United States). Thus, 10 mSv (1 rem) among 1 million people, 1 mSv (100 mrems) among 10 million people, or 0.01 mSv (1 mrem) among 1 billion people are postulated as equivalent in their carcinogenic potential. That is, in every one of these populations, the ICRP model postulates 500 excess deaths from cancer. It should be pointed out that the model used to postulate these excess deaths is inherently unverifiable since the statistical variability in the annual number of cancer deaths is far greater than the postulated number of excess radiogenic cancer deaths. In the United States, for example, the proportion of all deaths due to cancer has remained relatively constant during the years 1999–2004, at about 23%. The number of U.S. cancer deaths during these years ranged from 549,838 to 553,888 (out of a U.S. population of 279 million in 1999 to 292.8 million in 2004). This variability in number of cancer deaths of more than 4000 swamps any excess cancer deaths that the linear zero-threshold model may postulate in the United States based on collective-dose considerations. The linear zero-threshold model, therefore, is inherently unverifiable.

In a societal or public health context, an acceptable collective dose for a large population is determined by policymakers on the basis of societal benefits that will accrue to the population versus the postulated detrimental effects as a result of the radiation exposure. Under these conditions, the probability of a detrimental radiogenic effect in any individual is vanishingly small. The recommended risk coefficients for lifetime stochastic effects, such as ICRP's 7.3×10^{-5} per mSv (which includes fatal and nonfatal cancers and detrimental heritable effects), are intended for use in ranking radiation risk among all other public health risks for the purpose of public health decision making. It is not intended to be used as a metric for counting dead bodies or other detrimental effects that are postulated at the low radiation levels that are associated with those practices that are limited by the recommended safety standards.

Dose-Limitation System

Deterministic (Nonstochastic) Effects

Engineering control of the environment by industrial hygienists and public health personnel is usually based, in the case of nonstochastic effects, on the concept of a tolerance dose, that is, a threshold dose. If the threshold dose of a toxic substance is not exceeded, then it is assumed that the normally operating physiological mechanisms can cope with the biological insult from that substance. This threshold is usually determined from a combination of experimental animal data and clinical human data; it is then reduced by an appropriate factor of safety, which leads to the maximum allowable concentration (MAC) for the substance. The MAC is then used as the criterion of safety in environmental control. The MAC was defined by the International Association on Occupational Health in 1959 as follows: "The term maximum allowable concentration for any substance shall mean that average concentration in air which causes no signs or symptoms of illness or physical impairment in all but hypersensitive workers during their working day on a continuing basis, as judged by the most sensitive internationally accepted tests."

Stochastic Effects

A different philosophy underlies the control of environmentally based agents, such as ionizing radiation and radionuclides, that lead to increased probability of cancer and genetic effects. Although molecular biologists have found the existence of intracellular mechanisms for the repair of damaged DNA in bacteria, geneticists have observed a dose–rate dependence of radiogenic mutagenesis, and both these observations imply the existence of a threshold for stochastic effects. Although the postulated stochastic effects have not been seen in populations that had been exposed to low-dose radiation ($\leq$0.1 Gy, or 10 rads), public health policy nevertheless is based on the conservative belief that absence of proof of an effect is not proof of the absence of the effect. Accordingly, *we assume, for the purpose of setting safety standards for radiation as well as for chemical carcinogens and mutagens, that the threshold dose for stochastic effects is zero dose*. The dose–response curves for carcinogenesis and mutagenesis are assumed to be linear down to zero dose. The slopes of the dose–response curves for the various stochastic effects are postulated to be the same at low doses, all the way until zero dose, as at the high doses. Since this means that every increment of dose, no matter how small, increases the probability of an adverse effect by a proportional increment, the basis for control of man-made radiation is the limitation of the radiation dose to a level that is compatible with the benefits that accrue to society and to individuals from the use of radiation.

Based on the preventive conservatism principle, it can be argued that the distinction between those agents that cause deterministic effects and those that increase the probability of stochastic effects, which is based on the existence or absence of a threshold dose, is not as clear-cut as may first appear. For those substances where a threshold has indeed been established, the threshold is for an *individual*. Different individuals have different thresholds. Thus, although the average threshold value for blood changes because of gamma radiation is taken as 0.25 Gy (25 rads), changes have been observed in persons whose doses were as low as 0.10 Gy (10 rads), while others whose doses reached as high as 0.4 Gy (40 rads) showed no blood changes. If a much larger population of exposed people were to be examined for blood changes, it is likely that changes would be seen among some whose dose was even less than 0.10 Gy (10 rads). It is not unreasonable to expect a distribution of sensitivity to most

noxious agents somewhat like that shown in Figure 8-1, in which the sensitivity distribution curve is skewed to the right. The curve should actually intersect the abscissa on the high-dose end of the distribution, since we are reasonably certain that there exists some dose that will affect everyone. On the other hand, it is known that there are "hypersensitive" individuals who respond to extremely low doses, which would not affect most people. On this basis, it is reasonable to assume that the distribution curve to the left of the mode extends to the origin of the coordinate axes. In effect, the distribution of susceptibility among the individuals of a population means that the concept of a threshold dose cannot be applied to a very large population. In setting a maximum acceptable dose (MAD) for a large population group, therefore, a value judgment must be made.

Someone must decide what is an acceptable fraction of the population that may be adversely affected by the agent for which the MAD is being set in return for the benefits to be derived by that population from the use of that agent. The MAD is usually set so conservatively that an extremely large number of people would have to be exposed at that level before the hypersensitive person was found. This same type of reasoning prevails among those who are concerned with recommending radiation dose limits. For occupational exposure, the question of recommending dose limits as a guide to radiation protection is relatively simple. A vast amount of human experience was gained from the promiscuous exposures to radium and X-rays and the consequent harmful radiation effects during the first quarter of the twentieth century, from survivors of the nuclear bombings in Japan, from exposure for medical reasons, and from large population groups living in areas of high- and low-radiation background. Additionally, much more data were obtained from laboratory studies with animals. On the basis of this information, and on the assumption that every additional increment of radiation dose has a corresponding increment of risk, dose limits can be set, which, when applied to occupationally exposed radiation workers, will result in a level of risk no greater than that in other occupations that are recognized as having high safety standards and are considered to be "safe." If any uncertainty arises about where to set an acceptable limit, the uncertainty is resolved by preventive conservatism rather than by scientific realism. Dose limits for nonoccupationally exposed individual members of the general public are set at a level where the resulting postulated radiation risk is very much smaller than the risks that society already accepts in return for other technological benefits. From these societally acceptable doses, we derive annual limits on intake (ALI) and environmental concentrations of the various radionuclides that would result in radiation doses within the prescribed dose limits.

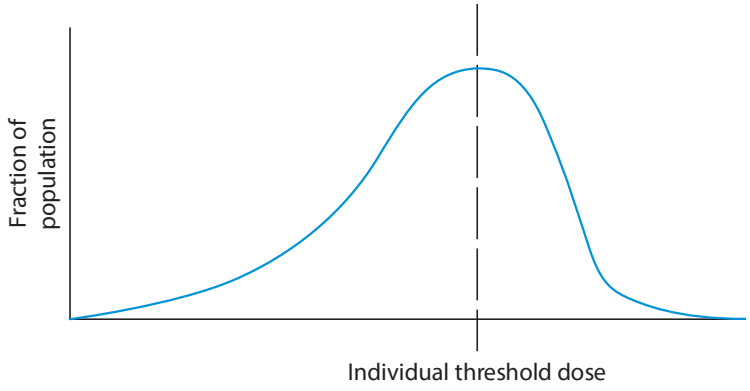

Figure 8-1. Distribution of individual thresholds among a population.

The system of dose limitation recommended by the ICRP is founded on three basic tenets stated in its Publication 26 and reiterated in its Publication 60 and 103:

1. Justification—No practice shall be adopted unless its introduction produces a net positive benefit. It should be pointed out that justification is a societal decision, not a radiation decision.
2. Optimization—All exposures shall be kept as low as reasonably achievable (ALARA), economic and social factors being taken into account.
3. Dose limitation—The dose equivalent to individuals shall not exceed the limits recommended for the appropriate circumstances by the Commission.

It should be emphasized that the second point above urges that actual operational dose limits for any radiological activity be more restrictive than the maximum recommended dose limit. This means that processes, equipment (such as shielding, ventilation, etc.), and other operational factors should be designed so that workers do not exceed the operational dose limit, which is usually much smaller than the maximum recommended dose limit. This operating philosophy is known as the ALARA concept. To apply the ALARA concept, the ICRP recommends that cost–benefit analyses of alternative lower operational dose limits be made, and then that level of radiation protection be selected that optimizes the cost of the detrimental effects of the radiation versus the benefits to be derived from the radiation practice. Since economic and social factors must be considered in implementing ALARA, it is clear that widely differing interpretations can be made by equally competent authorities on what is "as low as reasonably achievable." In the United States, the official interpretation is made by the U.S. NRC and is published in the *Regulatory Guide* series.

Societal benefits and detriments from radiological activities usually are not uniformly distributed among all members of society. Furthermore, different members and segments of society may be exposed to radiation from several different sources. The ICRP, therefore, recommends restrictions, or *constraints*, on radiation sources to try to ensure that no member of the general public will exceed the maximum dose. For example, the U.S. EPA's annual dose limit for public drinking water is 4 mrems (40 μSv), and the U.S. NRC requirement is that the annual dose to a member of the public from the entire nuclear fuel cycle may not exceed 25 mrems (250 μSv). Water treatment and operations in the nuclear fuel cycle must be designed accordingly.

The validity of the radiation safety standards was emphasized by Lauriston Taylor, the founder of the NCRP, who said, in 1980: "No one has been identifiably injured by radiation while working within the first numerical standard set by the NCRP and the ICRP in 1934." Since then, the radiation safety standards have been made about 10 times more restrictive. It is, therefore, reasonable to expect that the current radiation safety standards are sufficiently restrictive to preclude identifiable radiation injury.

ICRP BASIC RADIATION SAFETY CRITERIA

For purposes of radiation safety standards, the ICRP recognizes three categories of exposure:

1. Occupational exposure to adults who are exposed to ionizing radiation in the course of their work. Persons in this category may be called radiation workers. This category contains two subgroups:

   (a) Pregnant women
   (b) All other radiation workers

2. Exposure of members of the general public.
3. Medical exposure. This category deals with the intentional exposure of patients for diagnostic and therapeutic purposes by technically qualified medical and paramedical personnel. It does not include exposure to the personnel involved in the administration of radiation to patients.

Occupational Exposure

For occupational exposure, the ICRP 26, in 1977, recommended the following annual dose-equivalent limits:

1. To prevent nonstochastic effects, the limit is

   (a) 0.5 Sv (50 rems) to all tissues except the lens of the eye.
   (b) 0.15 Sv (15 rems) to the lens of the eye.

   These limits applied whether the tissues were exposed singly or together with other organs.
2. To limit stochastic effects, the dose-equivalent limit from uniform whole-body irradiation is 50 mSv (5 rems) in 1 year.

Limits on intake of radioisotopes in order to meet the ICRP 26 dose limits from internal exposure are listed in ICRP 30 and its supplements.

The ICRP 26 recommendations were superseded in 1990 by ICRP 60 2007 by ICRP 103, and in 2015 by ICRP 130 recommendations for radiation safety limits. The ICRP 60, 103, and 130 recommendations are based on a combined concept of stochastic and nonstochastic (deterministic) effects. These two categories were considered together in a single index of harm called the *detriment*, which includes consideration of both stochastic and deterministic effects. The dose limits in ICRP 60, 103, and 130 are based on a dose, which, if exceeded, may lead to unacceptable consequences, be they either stochastic or deterministic, for an individual. These dose limits are shown in Table 8-2.

TABLE 8-2 ICRP 26/60/103/130 Recommended Dose Limits

APPLICATION	ICRP 26 OCCUPATIONAL	ICRP 26 PUBLIC	ICRP 60 OCCUPATIONAL	ICRP 60/103 PUBLIC	ICRP 103/130 OCCUPATIONAL
Whole body	50 mSv	1 mSv	2 mSv/yr averaged over 5 years, maximum dose in any year 50 mSv	1 mSv	2 mSv/yr averaged over 5 years, maximum dose in any year 50 mSv
Annual dose to lens of eye	150 mSv	15 mSv	150 mSv	15 mSv	2 mSv/yr averaged over 5 years, maximum dose in any year 50 mSv[a]
Skin	500 mSv	50 mSv	500 mSv	50 mSv	500 mSv
Hands/Feet	50 mSv	50 mSv	50 mSv	50 mSv	50 mSv
Fetus/Embryo	5 mSv	—	2 mSv	—	1 mSv

[a]ICRP statement issued April 21, 2011.

Effective Dose

On the principle that the risk of a stochastic effect should be equal whether the whole body is uniformly irradiated or whether the radiation dose is nonuniformly distributed, the ICRP introduced the concept of *effective dose* in the 1977 review of its radiation safety recommendations (ICRP 26).

For the purpose of setting radiation safety standards, we assume that the probability of a detrimental effect in any tissue is proportional to the dose equivalent to that tissue.

TABLE 8-3 Tissue Weighting Factors, w_T

TISSUE, w_T	ICRP 26	ICRP 60	ICRP 103
Gonads	0.25	0.20	0.08
Breast	0.15	0.05	0.12
Red bone marrow	0.12	0.12	0.12
Lung	0.12	0.12	0.12
Thyroid	0.03	0.05	0.04
Bone surface	0.03	0.01	0.01
Colon	—	0.12	0.12
Stomach	—	0.12	0.12
Bladder	—	0.05	0.04
Liver	—	0.05	0.04
Esophagus	—	0.05	0.04
Skin	—	0.01	0.01
Salivary glands	—	—	0.01
Brain	—	—	0.01
Remainder*	0.30	0.05	0.12
Total	1.00	1.00	1.00

*The handling of remainder tissues varies based upon ICRP report.

w_R and w_T are the same for ICRP 60 and NCRP 116.

ICRP 26—remainders: Weighting Factor Remainder: The five other organs or tissues, excluding the skin and lens of the eye, with the highest dose (e.g., liver, kidney, spleen, thymus, adrenal, pancreas, stomach, small intestine, and upper large intestine). The weighting factor for each remaining organ or tissue is 0.06.

ICRP 60—Tissue Weighting Factor Remainder: The following additional tissues and organs and their masses, in grams, following parenthetically: adrenals (14), brain (1400), extrathoracic airways (15), small intestine (640), kidneys (310), muscle (28,000), pancreas (100), spleen (180), thymus (20), and uterus (80). The equivalent dose to the remainder tissues (H_{rem}) is normally calculated as the mass-weighted mean dose to the preceding 10 organs and tissues. In those cases in which the most highly irradiated remainder tissue or organ receives the highest equivalent dose of all the organs, a weighting factor of 0.025 (half of remainder) is applied to that tissue or organ and 0.025 (half of remainder) to the mass-weighted equivalent dose in the rest of the remainder tissues and organs to give the remainder equivalent dose.

However, because of the differences in sensitivity among the various tissues, the value for the proportionality factors differs among the tissues. The relative sensitivity to detrimental effects, expressed as tissue weighting factors w_T of the several organs and tissues that contribute to the overall risk, is shown in Table 8-3. If radiation dose is uniform throughout the body, then the total risk factor has a relative weight of 1. For nonuniform radiation, such as partial-body exposure to an external radiation field, or from internal exposure where the radionuclide concentrates to different degrees in the various organs, the weighting factors listed in Table 8-3 are used to calculate an EDE (ICRP 26 terminology). The EDE, H_E, is given by

$$H_E = \sum_T w_T H_T, \tag{8.2}$$

where w_T is the weighting factor for tissue T and H_T is the dose equivalent (equivalent dose for ICRP 60, 103 and 130) to tissue T. When using ICRP 60, 103, or 130, the corresponding term for EDE is the effective dose. Table 8-3 shows the weighting factors recommended in ICRP 26, 60, and 103. The U.S. NRC used the ICRP 26 values for the tissue-weighting factors in Title 10 of the *Code of Federal Regulations*, Part 20, which usually is cited as 10 CFR 20, that were approved in 1991 and became effective in 1994.

Conceptus (Embryo/Fetus)

Limits on occupational exposure for women are the same as for men. However, the ICRP has set restrictive limits on the conceptus. After pregnancy has been declared, a maximum of 2 mSv, which is the sum of external radiation and dose from internally deposited radionuclides, is recommended for the balance of the pregnancy based on ICRP 60. ICRP 103 has recommended 1 mSv to the embryo/fetus. For purposes of ICRP 60, the external radiation dose assigned to the conceptus is the deep dose registered by the pregnant woman's dosimeter. The internal dose per unit intake of a radionuclide depends on the age of the conceptus.

Dose coefficients (DC) from acute and chronic intakes by inhalation and by ingestion may be found in ICRP 88 (2001). For example, the DC due to an acute inhalation of elemental ^{131}I vapor during the 25th week of pregnancy is listed as 3.1×10^{-8} Sv/Bq. Thus, in the case of a nuclear technologist who accidentally inhaled 1000 Bq during her 25th week of pregnancy, her fetus would be assigned a dose of

$$3.1\times10^{-8}\,\frac{\text{Sv}}{\text{Bq}}\cdot 1\times10^{3}\,\text{Bq} = 3.1\times10^{-5}\,\text{Sv, or } 31\ \mu\text{Sv}\ (3.1\ \text{mrems}).$$

If her dosimeters showed a total external dose of 100 μSv (10 mrems), then the total dose assigned to her fetus would be 31 μSv + 100 μSv = 131 μSv (13.1 mrems).

Medical Exposure

No specific dose limit was recommended by the ICRP for medical exposure. The commission, however, recommended that only necessary exposure should be made, that these exposures should be justifiable on the basis of benefits that would not otherwise have been received, and that the administered doses should be limited to the minimum dose consistent with the medical benefit to the patient. Recommendations for doses to medical research volunteers are based on the benefit to society:

BENEFIT[a]	DOSE (mSv)
Minor	<0.1
Intermediate	0.1 to 1
Moderate	1 to 10
Substantial	>10
Comforters and carers	5 per episode

[a]ICRP 62, 94, 98, and 103.

Exposure of Individuals in the General Public

For members of the general public, the ICRP recommends an effective dose limit of 1 mSv (100 mrems) in a year (Table 8-2). It is believed that the average dose to members of an exposed group will be less than the dose limit. The ICRP points out that the average dose to members of the public would increase if the number of sources increase, even though the

dose to no single individual exceeds the 1-mSv effective dose limit. For this reason, the commission recommends that regional or national authorities should maintain surveillance over all the separate sources of exposure in order to control the collective total effective dose.

Exposure of Populations

The ICRP made no specific recommendations for the dose limit to a population. Instead, it emphasized that each man-made contribution to the population dose must be justified by its benefits, and that limits for individual members of the population refer to the total effective dose from all sources. The dose limit to a population is thus considered to be the sum of several minimum necessary contributory doses rather than a single permissible total dose limit that is available for apportionment among several sources.

Dose Coefficient

In its current (2015) recommendations, the ICRP does not list maximum acceptable concentrations of radionuclides in air or water, nor does it list the ALI as it did in ICRP 30. (The ALI is defined in the paragraph below.) Because the primary safety standard is either the dose limit to an organ (nonstochastic effect) or the effective whole-body dose (stochastic effect), the ICRP as well as the IAEA list the DC rather than the ALI. The DC is defined as the committed equivalent dose to an organ or tissue per unit intake, or the committed effective dose per unit intake. ICRP 119 lists the DC for six different age categories, from 3 months of age to adulthood (ages <1 year, 1 year, 5 years, 10 years, 15 years, and adult), and the IAEA lists them for adult workers as Sv per Bq intake. In traditional units, the DCs are expressed as rem per μCi. The ALI for any radionuclide may be calculated, for use as a secondary safety criterion, from the dose limit and the DC for that nuclide.

Annual Limit on Intake

The annual limit on intake (ALI) is defined in ICRP 30 as that quantity of activity of a radionuclide that would lead to the annual dose limit if inhaled or ingested by a "reference person."

According to ICRP 30 Criteria

ICRP 30 criteria are important in the United States because the U.S. NRC's radiation safety standards are based on the ICRP 30 recommendations. In the ICRP 30, the ALI was restricted by the basic requirements for stochastic and nonstochastic effects and was defined as the annual intake that would lead to an effective committed dose equivalent (a 50-year dose commitment) not exceeding 50 mSv (5 rems) and an annual dose equivalent to any single organ or tissue not exceeding 500 mSv (50 rems). Expressed symbolically, these requirements are

$$\sum_T w_T H_{50,T} \leq 0.05 \text{ Sv} \tag{8.3}$$

$$H_{50,T} \leq 0.5 \text{ Sv} \qquad \text{for every } T, \tag{8.4}$$

where w_T is the weighting factor shown in Table 8-3 and $H_{50,T}$ is the 50-year total committed dose equivalent in tissue T resulting from intakes of radioactive materials from all sources during the year in question. Equation (8.3) assures that the annual limit on effective

whole-body dose is not exceeded in order to control stochastic effects, while Eq. (8.4) assures that the annual limit on the dose to a single tissue or organ is not exceeded in order to remain below the damage threshold for nonstochastic effects. Thus, an effective dose of 0.05 Sv (5 rems) is assigned to an intake of one ALI that is based on stochastic effects (SALI), while an organ dose of 0.5 Sv (50 rems) is assigned to an intake of one ALI based on nonstochastic effects (NALI) to the organ.

It should be noted that the intake limit is placed on the total intake of radioisotopes in any single year, and that no restrictions were placed by the ICRP on the instantaneous rate of intake. That is, the limit may be met by a single large intake or by continuing intake of small quantities. The principles involved in the application of Eqs. (8.3) and (8.4) may be illustrated by the calculation of the ALI for ingested ^{137}Cs according to ICRP 26 and 30 criteria (Example 8.2).

Lung Models

Radiation dosimetry and calculation of inhalation ALIs and acceptable atmospheric concentrations of radioactivity are based on dosimetric models of the respiratory tract. The first such dosimetric model was introduced by the ICRP in 1959. It was a relatively simple model that considered only aerosols and modeled the lung as a two-compartment system: the upper respiratory tract and the deep respiratory tract, and only two classes of particle solubility, "soluble" and "insoluble." The ICRP 2 recommendations for maximum permissible concentrations (MPCs) of airborne particulate radioactivity in the workplace were based on this model. These recommendations were incorporated into the IAEA's first edition of its basic safety standards (1962) and into the radiation safety regulations of the various countries, including the United States, in the Atomic Energy Commission's (AEC) Part 20 of 10 CFR.

ICRP 30 Lung Model

In 1978, in its Publication 30, the ICRP introduced a more sophisticated dosimetric model of the respiratory tract in order to account for the oversimplification and deficiencies of the

earlier two-compartment model. The newer model accounted for the fact that deposition of particles in the respiratory tract is governed by airflow patterns in the respiratory tract and by the size distribution of the inhaled aerosol, and that the clearance rate of the deposited particles is governed by the deposition site as well as by chemical and physical properties of the particles. The ICRP 30 dosimetric lung model was designed to calculate the mean dose to blood-filled lungs from inhaled particles in the size range of 0.2–10 μm in size. This model was used as the basis for safety standards for inhaled radioactive aerosols that were published in ICRP 30 and again in 1990 in the revised standards published in ICRP 61. The U.S. NRC based its 1991 revision of 10 CFR 20 atmospheric concentrations on the ICRP 30 dosimetric model.

Figure 8-7 is a graphic representation of the ICRP 30 dosimetric lung model used to calculate the inhalation ALIs in ICRP 30, and in the U.S. NRC's 1991 revision of its 10 CFR 20 regulations. This lung model consists of three regions where inhaled aerosols may be deposited: the nasopharyngeal region (NP), the tracheobronchial region (TB), and the pulmonary region (P), representing the deep respiratory tract where gas exchange occurs. The NP region is divided into two compartments, *a* and *b*. Compartment *a* represents that part in which the dust deposited in the NP region dissolves and is absorbed directly into the blood. Compartment *b* represents the region from which dust is cleared into the GI tract by swallowing. The TB region is also represented by two compartments, *c* and *d*, from which deposited particles are cleared by the same two mechanisms as above. Compartment *c* represents the region in which dissolution and absorption into the blood takes place, whereas

REGION	COMPARTMENT	CLASS D T	CLASS D F	CLASS W T	CLASS W F	CLASS Y T	CLASS Y F
NP (D_{NP} = 0.30)	a	0.01	0.5	0.01	0.1	0.01	0.01
	b	0.01	0.5	0.40	0.9	0.40	0.99
TB (D_{TB} = 0.08)	c	0.01	0.95	0.01	0.5	0.01	0.01
	d	0.2	0.05	0.2	0.5	0.2	0.99
P (D_P = 0.25)	e	0.5	0.8	50	0.15	500	0.05
	f	n.a.	n.a.	1.0	0.4	1.0	0.4
	g	n.a.	n.a.	50	0.4	500	0.4
	h	0.5	0.2	50	0.05	500	0.15
L	i	0.5	1.0	50	1.0	1000	0.9
	j	n.a.	n.a.	n.a.	n.a.	∞	0.1

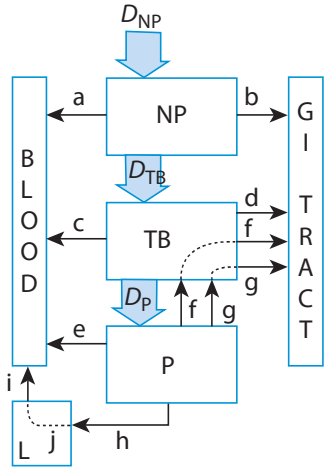

Figure 8-7. ICRP 30 respiratory tract model used to calculate inhalation limits for airborne radioactive particles. The values for removal half-times, T_{a-j}, and compartmental fractions, F_{a-j}, are given in the tabular portion of the figure for each of the three classes of retained materials. The values given for D_{NP}, D_{TB}, and D_P (left column) are the regional depositions based on an aerosol with an activity median aerodynamic diameter (AMAD) of 1 μm. The schematic drawing identifies the various clearance pathways, *a*–*j*, in relation to the deposition D_{NP}, D_{TB}, D_P and the three respiratory regions: nasopharyngeal (NP), trachobronchial (TB), and pulmonary (P). The entry "n.a." indicates "not applicable." (From Watson SB, and Ford MR. *A User's Manual to the ICRP Code: A Series of Computer Programs to Perform Dosimetric Calculations for the ICRP Committee 2 Report*. Oak Ridge, TN: Oak Ridge National Laboratory; February 1980. TM-6980.)

the mechanical transfer by way of the ciliary escalator to the throat and into the GI tract by swallowing is represented by compartment *d*. The pulmonary region, P, is modeled by four compartments. One of these compartments, *e*, represents dissolution and absorption into the blood. Compartments *f* and *g* represent transfer of undissolved particles into the GI tract via the upper respiratory tract (the TB region). Compartment *f* is cleared by mechanical transport, presumably by unbalanced forces during respiratory excursions, and compartment *g* is cleared by alveolar macrophages that migrate into the TB region. Compartment *h* empties into the pulmonary lymph nodes. The pulmonary lymph nodes are represented by two compartments, *i* and *j*. Compartment *i* empties into the bloodstream after the particles have dissolved, while compartment *j* permanently retains some highly insoluble particles.

The exact fraction of the deposited aerosol that is cleared by each route and the respective clearance rates are governed by the chemical composition of the aerosol and particle size. However, since it is not practical to determine each of these parameters for every compound of every element, the various compounds of all the elements have been assigned, to one of three classes: D, W, and Y. Class D aerosols are rapidly cleared from the deep respiratory tract with a clearance half-time on the order of a day or a fraction of a day. Class W aerosols are cleared on the order of weeks, while class Y materials are retained in the lungs on the order of years. Of the various forms of dust that may be transported to the lymph nodes, only class Y materials are permanently retained in the lymph nodes. For health physics purposes, the lung and the pulmonary lymph nodes are considered as a single organ. That is, the activity in the lung and in the lymph nodes is added together, and the total weight of the lungs and pulmonary lymph nodes is used to calculate dose from inhaled aerosols. The ICRP's recommendations for inhaled aerosols are based on inhalation and deposition of an aerosol whose AMAD is 1 μm and whose geometric standard deviation is 4. This assumed distribution leads to deposition of 30% of the inhaled dust in the NP region, 8% in the TB region, and 25% in the P region. The balance 37% is exhaled. Deposition for other size distributions is shown in Figure 8-8.

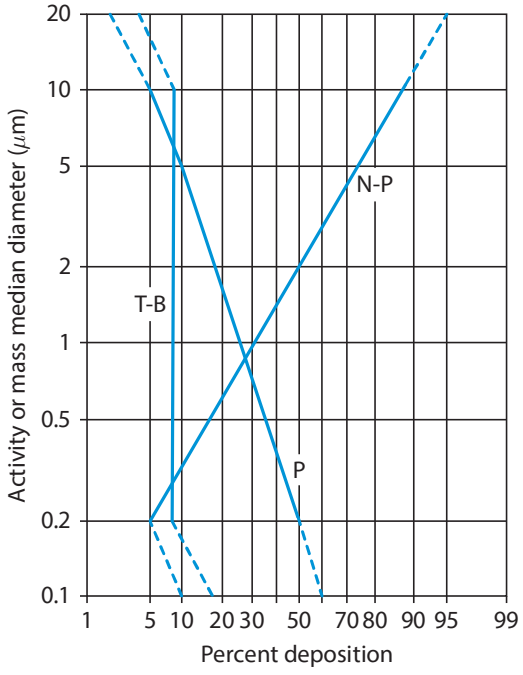

Figure 8-8. ICRP 30 particle deposition model. The radioactive or mass fraction of an aerosol, which is deposited in the nasopharyngeal (NP), trachobronchial (TB), and pulmonary (P) regions, is given in relation to the activity or mass median aerodynamic diameter (AMAD or MMAD) of the aerosol distribution. This model is intended for use with aerosol distributions having an AMAD or MMAD between 0.2 and 10 μm and whose geometric standard deviations are less than 4.5. Provisional deposition estimates further extending the size range are given by the dashed lines. For the unusual distribution having an AMAD or MMAD greater than 20 μm, complete NP deposition is assumed. The model does not apply to aerosols with AMAD or MMAD below 0.1 μm.

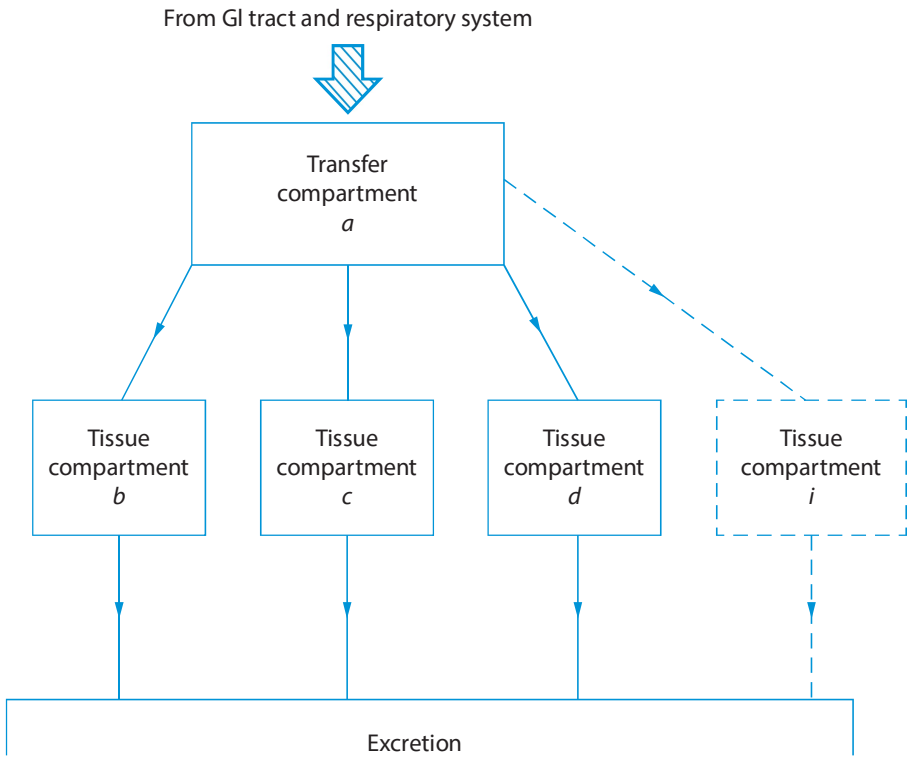

Figure 8-9. Model used to describe the kinetics of radionuclides in the body. *Abbreviation*: GI, gastrointestinal. (Reproduced with permission from ICRP Publication 30, Part 1: Limits for Intakes of Radionuclides by Workers. *Ann ICRP*. 1979; 2(3/4):17. Copyright © 1979 International Commission on Radiological Protection.)

Material brought up from the lung and swallowed enters the GI tract, from which it may subsequently be eliminated in the feces, irradiating the various parts of the GI tract and other organs during its passage. It may also undergo dissolution in the GI tract, and the dissolved portion may be absorbed into the blood and transferred to organs where it may be deposited (Fig. 8-9). Thus, for example, inorganic mercury is deposited mainly in the kidneys, iodine in the thyroid, strontium and radium in the skeleton, and plutonium in the liver and skeleton. The fraction of the activity deposited in the lung that is subsequently transferred to the blood may be calculated with the information given in the lung model. In Figure 8-8, we see that inhaled 1-μm AMAD particles are deposited in the respiratory tract with the regional distribution shown in Table 8-7.

TABLE 8-7 Pulmonary Deposition of 1-μm AMAD Particles

REGION	PERCENT DEPOSITED
NP	30
TB	8
P	25
Total	63

Abbreviations: AMAD, activity mean aerodynamic diameter; NP, nasopharyngeal; TB, tracheobronchial; P, pulmonary.

TABLE 8-8 Fractions of Activity in the Lung from Inhaled 1-μm Particles That Are Transferred to the Blood

CLASS	FRACTION
D	$0.48 + 0.15\,f_1$
W	$0.12 + 0.51\,f_1$
Y	$0.05 + 0.58\,f_1$

Figure 8-7 tells us the fractions of the deposited particles that are cleared from the lung by each of the two clearance mechanisms: (1) dissolution and direct absorption into the blood and (2) physical transport to the throat followed by swallowing into the GI tract. In the case of highly soluble class D particles, for example, one-half of the 30% deposited in the NP region, 95% of the inhaled aerosol deposited in the TB region, and all of the inhaled particles (25%) deposited in the P region are transferred directly to the blood by dissolution:

$$(0.5 \cdot 0.3) + (0.95 \cdot 0.08) + (1 \cdot 0.25) = 0.48.$$

From the particles deposited in the respiratory tract and transported to the GI tract, the activity transferred to the blood is

$$(0.5 \cdot 0.3 \cdot f_1) + (0.05 \cdot 0.08 \cdot f_1) = 0.15\,f_1,$$

where f_1 represents the fraction of the radionuclide in the GI tract that is absorbed into the blood.[2] Similar calculations can be made for class W and Y particles. The results of these calculations are given in Table 8-8.

The GI tract is modeled by four distinct regions, each with its own kinetic parameters, so that organ doses during passage of radionuclides can be calculated. Similarly, the organs where the radionuclides are deposited are also modeled by appropriate equations, usually one or more first-order linear differential equations that allow the organ doses to be calculated.

Inhalation ALI According to ICRP 60 Criteria

Publication 60 of the ICRP recommends a primary dose limit of 100 mSv over a 5-year period, with a maximum in any 1 year of 50 mSv, or an average of 20 mSv/yr. In addition, it does away with the nonstochastic classification, and bases all its recommendations for derived limits on a committed effective dose of 20 mSv/yr. It also changed the nomenclature from "dose equivalent" to "equivalent dose." The ALIs for ingestion and for inhalation are thus calculated by

$$\text{ALI} = \frac{0.02\ \text{Sv}}{\sum_T w_T H_{50,T}\,\dfrac{\text{Sv}}{\text{Bq}}}, \tag{8.25}$$

where the denominator is the committed effective dose either by ingestion or by inhalation. For example, in the case of ^{137}Cs, we found the CEDE from ingested activity to be 1.3×10^{-8} Sv/Bq. By substituting this value into Eq. (8.25), we find the ingestion ALI to be

$$\text{ALI (ingestion)} = \frac{0.02\ \text{Sv}}{1.3\times10^{-8}\,\dfrac{\text{Sv}}{\text{Bq}}} = 1.54\times10^{6}\,\text{Bq} = 2\ \text{MBq},$$

and for inhalation, we have

$$\text{ALI (inhalation)} = \frac{0.02\ \text{Sv}}{8.3\times10^{-9}\,\dfrac{\text{Sv}}{\text{Bq}}} = 2.14\times10^{6}\,\text{Bq} = 2\ \text{MBq}.$$

ICRP 66 Human Respiratory Tract Model

A still more sophisticated dosimetric model for the HRT, called the human respiratory tract model (HRTM), was recommended by the ICRP in 1994, in Publication 66. This dosimetric model is the result of increased knowledge of the biokinetics of the respiratory processes involved in the inhalation of aerosols and gases, the radiosensitivity of the several different tissues within the respiratory tract, and the biological effects of inhaled radioactivity. This increased knowledge makes the new model applicable to all population groups, old and young, male and female, and at different levels of physical activity (heavy exercise, light exercise, resting, and sleeping), rather than only to occupationally exposed, unisexual adults at work. It accounts for the effects of other air pollutants and for smoking, and considers respiratory disease and the health status of the individual. While with the ICRP 30 model only the average dose to the lung was calculated, the ICRP 66 dosimetric model allows the calculation of the doses to the various tissues within the respiratory tract and then the weighting of the mean doses to the various tissues within the respiratory tract according to the radiosensitivity of the tissue. The ICRP 66 HRTM is used by the IAEA and by most regulatory agencies outside the United States as the basis for the safety standards and dose conversion factors (DCFs) for airborne radioactivity. At this time (2016), the United States has not yet adopted the new HRTM, and the NRC safety standards are based on the ICRP 30 lung model.

When dealing with the safety aspects of exposure to airborne radioactivity, which includes aerosols and gases or vapors, we are interested in the answers to several questions:

1. What is in the air, and what is being inhaled?
2. Which of the inhaled aerosols are exhaled and which are deposited in the respiratory tract?
3. Where in the respiratory tract are inhaled particles deposited?
4. What is the fate of the deposited particles?
5. What is the radiation dose from this inhalation exposure?
6. How much of the airborne radioactivity may be safely inhaled?

The new model of the HRT gives a more realistic response to these questions than does the previous model by dealing quantitatively with the inhalability of aerosols, the deposition of inhaled aerosols based on particle size and on airflow velocity in the various airways in the respiratory tract, and on the time-dependent decreased clearance rates from the lungs. Calculation of the lung dose with the new model is fundamentally different from the calculation with the earlier lung models. While the mean dose to uniform blood-filled lungs was calculated with the previous model, the new model considers the several different cell types in the respiratory tract, their masses, and their relative sensitivities to radiation. The dose to each of these different tissues is ca lculated, and then the pulmonary tissue doses are combined, through the use of appropriate weighting factors, to obtain the effective lung dose.

While the previous models were designed for the purpose of calculating secondary safety standards for occupational exposure to aerosols on the size range of 0.2–10 μm, the new model was made to be universally useful by extending its range of applicability to include

- particle sizes from 0.0006 μm to 100 μm,
- males and females,
- 3-month-old infants to adults,
- nose and mouth breathers,

- breathing rates for four different levels of exertion: sleeping, sitting, light exercise, and heavy exercise,
- the effects of smoking, air pollutants, and pulmonary diseases,
- classification of particles on the basis of the rate of absorption of the inhaled radioactivity into the blood (instead of classifying particles on the basis of their solubility as classes D, W, and Y):
- type F (fast)—100% absorbed into blood in $\leq$10 minutes,
- type M (moderate)—10% absorbed into blood in $\leq$10 minutes, 90% absorbed in $\leq$140 days,
- type S (slow)—0.1% absorbed in $\leq$10 minute, 99.9% absorbed in >140 days, and
- gases as well as aerosols.

The ICRP 66 HRTM is designed to calculate the DC, which is defined as the committed dose per unit intake, rem/μCi or Sv/Bq, of an airborne radionuclide. The HRTM supplies only the first part of this calculation—the dose to the lung and the rate of transfer of the inhaled radioactivity to the body fluids and to the GI tract. The biokinetic model for the particular radionuclide must then be used to complete the calculation of the DC.

The ICRP 66 model consists of five interrelated submodels: anatomical (morphometric), physiological, deposition, clearance, and dosimetry models.

Anatomical Model. The anatomical model describes the overall structure, including airway dimensions, of the HRT. ICRP 66 models the respiratory tract by four sequential anatomical regions (Fig. 8-10):

1. Extrathoracic (ET) region—the portion of the respiratory tract outside of the chest, which contains two subparts:
   - ET_1, consisting of the anterior nasal airways.
   - ET_2, consisting of the posterior nasal airways, pharynx, and larynx.
2. Bronchial (BB) region, which includes the trachea and the bronchi.
3. Bronchiolar (bb) region, consisting of bronchioles and terminal bronchioles.
4. Alveolar-interstitial (AI) region, which consists of the respiratory bronchioles, the alveoli, and interstitial connective tissue.

Each region is drained by lymphatic fluid, which flows into lymph nodes. The lymph nodes that drain the ET region are symbolized by LN_{ET}, and those that drain the three thoracic regions are labeled LN_{TH}. For dosimetry purposes, only LN_{TH} nodes contribute to the lung dose. The ET lymph nodes, LN_{ET}, are considered as "other tissues" when calculating the effective dose.

The physical dimensions and branching angles of the air pathways in the tracheobronchial tree are listed in ICRP 66 for the adult male. For example, the trachea is given as 1.65 cm (diameter) × 9.1 cm (length)—each of the five primary bronchi is 1.2 cm (diameter) × 3.8 cm (length)—and is at an angle of 36° (which represents the change in direction of the bulk flow of air from the trachea into the primary bronchi). Continuous bifurcation of the bronchi leads to increasing numbers of smaller airways, until the dead-ended alveoli are reached. The alveoli are the functional part of the respiratory tract, where inhaled oxygen diffuses into the blood and carbon dioxide diffuses out of the blood into the alveoli to

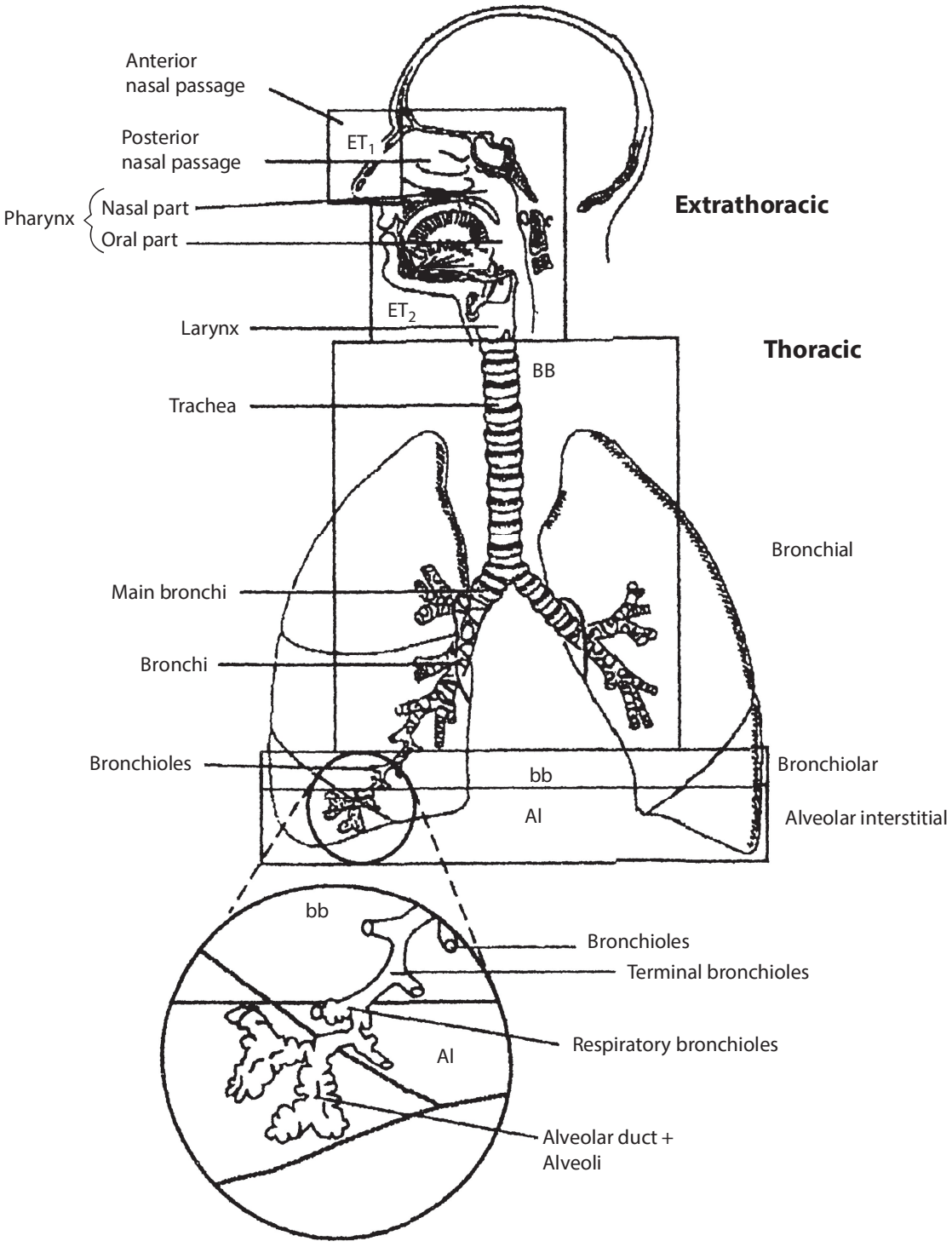

Figure 8-10. Anatomical divisions of the human respiratory tract. (Reproduced with permission from ICRP Publication 66: Human Respiratory Tract Model for Radiological Protection. *Ann ICRP*. 1994; 24(1–3). Copyright © 1994 International Commission on Radiological Protection.) *Abbreviations:* Al, alveolar-interstitial; bb, bronchiolar; ET, extrathoracic; BB, bronchial.

be exhaled. The total surface area available for gas exchange in the alveoli is 140 m^2. These dimensions are scaled down for females and for younger persons.

Physiological Model. The physiological model describes the functional aspects of the HRT. The kinetics of respiration, including volumes of inhaled air and inhalation rates, are given for males and females of various ages and for the four different levels of physical exertion that the model considers, and for nose and for mouth breathers. Correction factors are also given for conditions that modify or impair the normal functioning of the respiratory tract, such as old age, various illnesses, and smoking.

The importance of these physiological considerations may be illustrated by comparing the velocity of inhaled air in the trachea of a male worker when he is seated and when he is engaged in heavy exercise. While sitting, he inhales air at a maximum rate of 300 mL/s. Using the default tracheal diameter of 1.65 cm, this leads to a maximum airstream velocity in the trachea of 140 cm/s. While engaged in heavy physical exertion, the worker's maximum inhalation rate is 1670 mL/s, which leads to a maximum airstream velocity of 781 cm/s. These two very different velocities lead to significant differences in deposition patterns of inhaled particles.

Deposition Model. Deposition of particles in the respiratory tract is calculated on the basis of particle size, velocity of the air, and the geometrical contours of the air path. Deposition, therefore, depends on the person's age, sex, and ventilation rate.

When the mean particle size of an aerosol distribution exceeds about 0.5 μm, deposition is determined mainly by the aerodynamic properties of the particle, and the AMAD or the MMAD is used in the description of the aerosol size. (For a solid radioactive particle, the activity is directly proportional to the particle's mass.) When the mean size is less than about 0.5 μm, diffusion is the main deposition mechanism, and the mean size is expressed as the activity median thermodynamic diameter (AMTD).

To simulate particle deposition, the respiratory tract is modeled as a prefilter followed by a successive series of filters (Fig. 8-11). The prefilter represents the nares and the anterior nasal airways. Each of the successive filters represents the successive anatomical regions in the respiratory tract. Therefore, smaller fractions of the inhaled particles pass through each successive filter. In this model, filtration occurs during both inhalation and exhalation. Using this model, and considering the simultaneous deposition mechanisms of inertial impaction, gravitational settling, and diffusion of particles in the respiratory tract, deposition fractions for each region were calculated for equivalent sizes of 0.0006–100 μm. The deposition of 0.001–100 μm particles in the respiratory tract of a male worker is plotted in Figure 8-12. Table 8-11 lists the regional depositions of a 5-μm AMAD aerosol inhaled by an adult male reference worker and the regional depositions of a 1-μm AMAD aerosol in an adult male member of the public.

Clearance Model. Radioactive particles are cleared from the HRTM by three independent processes: mechanical transfer, dissolution of particles, and radioactive decay. Actual clearance is the sum of these three processes acting simultaneously.

The clearance model deals with the transfer, to the throat, of the deposited radioactive particles up the respiratory tract from the deposition sites and then into the GI tract by swallowing. Concurrently with this mechanical transfer via the ciliary action, the model deals with dissolution of deposited particles and the absorption of the dissolved radioactivity into the blood. It also accounts for the time-dependent changing clearance rates from each of the intrathoracic regions.

Mechanical Transfer. Mechanical transfer, which accounts for the transport of particles to the GI tract and to the lymph nodes, is affected by ciliary action and phagocytosis within the lungs and by sneezing and coughing in the ET airways. The modeled mechanical clearance rates are independent of particle type, sex, and age. However, in vivo laboratory studies on animals and bioassay studies on humans show a time dependence of pulmonary clearance rate. That is, initially most particles are rapidly cleared, and the remaining particles are cleared more slowly. The time dependence is modeled by dividing each region into several

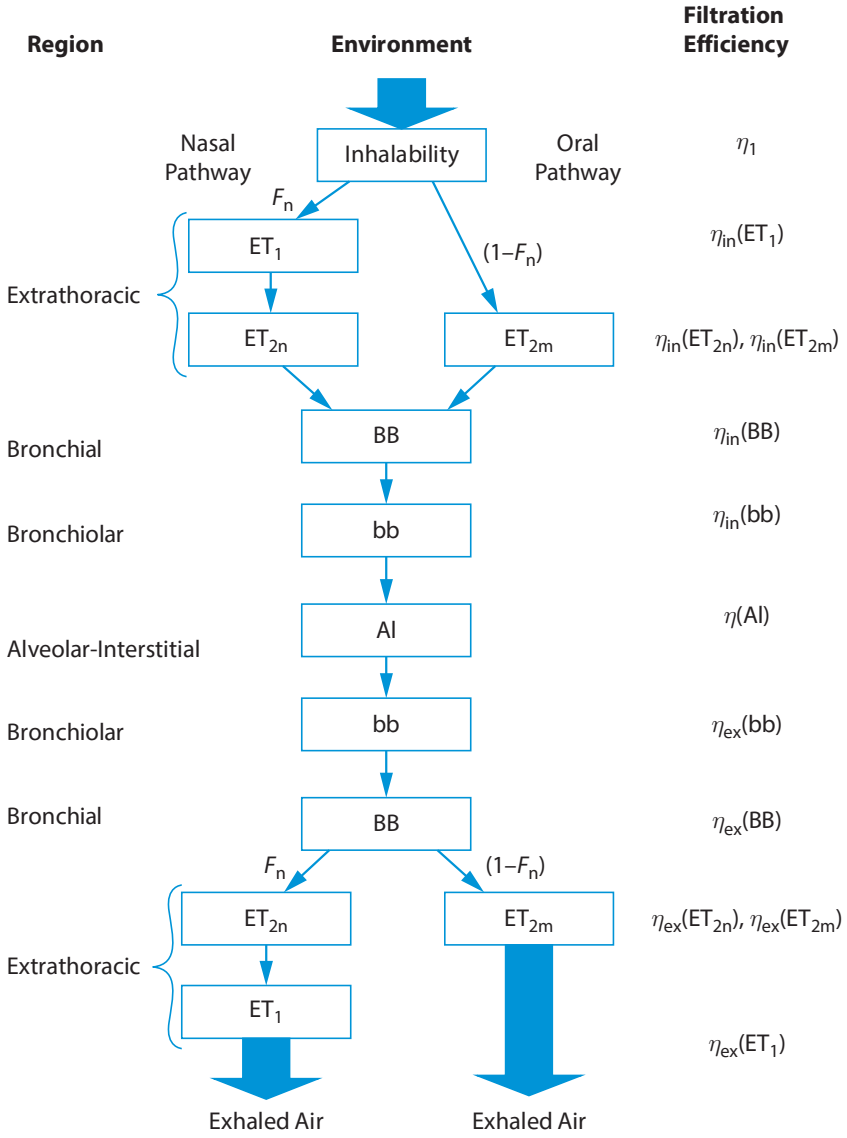

Figure 8-11. Filter model for deposition of inhaled particles in the respiratory tract of a reference worker. Two intake pathways are considered: the nasal pathway for which the fractional airflow is F_n; and the oral pathway, for which the fractional airflow is $1 - F_n$. The subscripts "in" and "ex" of the filtration efficiency, η, represent the inhalation and exhalation phases of the breathing cycle. (Reproduced with permission from ICRP Publication 66: Human Respiratory Tract Model for Radiological Protection. *Ann ICRP*. 1994; 24(1–3). Copyright © 1994 International Commission on Radiological Protection.)

compartments that empty at different rates, as shown in Figure 8-13. Each region contains a compartment that is very slowly cleared. For ET_2, BB, and bb regions, the very slowly cleared compartment is subscripted "seq" (for sequestered). The fraction of each regional deposit that is assigned to the several compartments is specified by the model, and is listed in Table 8-12.

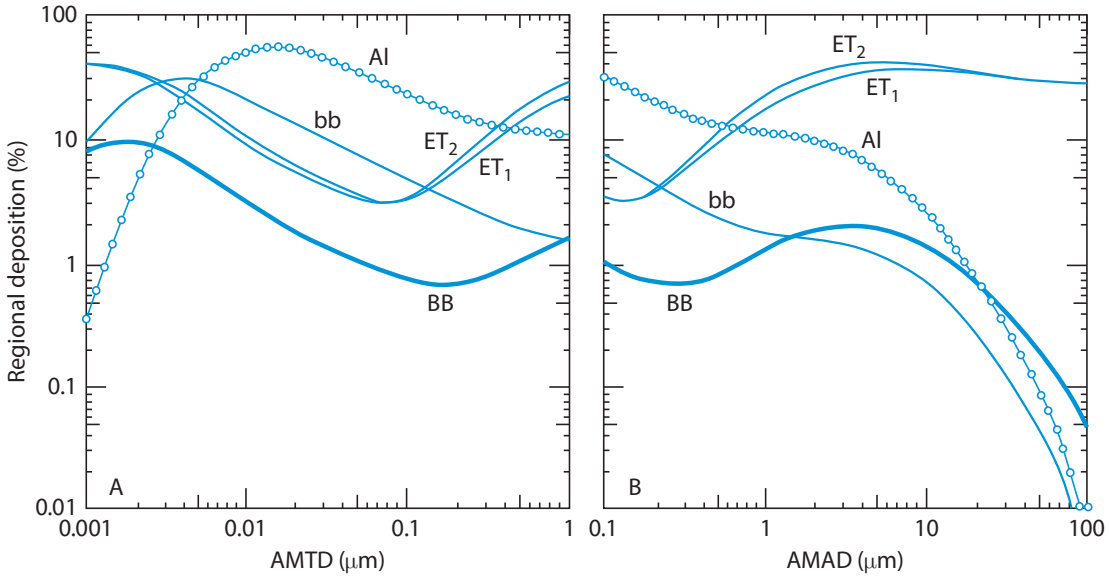

Figure 8-12. Fractional deposition in each region of the respiratory tract of a reference nose-breathing worker as functions of (**A**) activity median thermodynamic diameter, AMTD, and (**B**) the activity median aerodynamic diameter, AMAD. Deposition is expressed as a fraction of the activity present in the volume of inspired air, and the radioactive particle sizes are log-normally distributed. The particles' specific gravity is 3 and the shape factor is 1.5. *Abbreviations*: AI, alveolar-interstitial; bb, bronchiolar; ET, extrathoracic; BB, bronchial. (Reproduced with permission from ICRP Publication 66: Human Respiratory Tract Model for Radiological Protection. *Ann ICRP*. 1994; 24(1–3). Copyright © 1994 International Commission on Radiological Protection.)

TABLE 8-11 Regional Deposition of 5-μm and 1-μm AMAD Aerosols in Two Persons

REGION	WORKER, 5 μm (%)[a]	WORKER, 5 μm (%)	ADULT MALE, 1 μm (%)
ET_1	47.94	33.9	16.5
ET_2	25.82	39.9	21.1
BB	1.78	1.8 (33% in BB_2)	1.2 (47% in BB_2)
bb	1.10	1.1 (40% in bb_2)	1.7 (49% in bb_2)
AI	5.32	5.3	11.7
Total	81.96	82.0	51.2

Abbreviations: AI, alveolar-interstitial; BB, bronchial; bb, bronchiolar; ET, extrathoracic.

[a]ICRP 130 modified the HRT model. Note that for ICRP 130, "The particles are assumed to have density 3.00 g/cm^3 and shape factor 1.5. The particle aerodynamic diameters are assumed to be log-normally distributed with geometric standard deviation sg of approximately 2.50."

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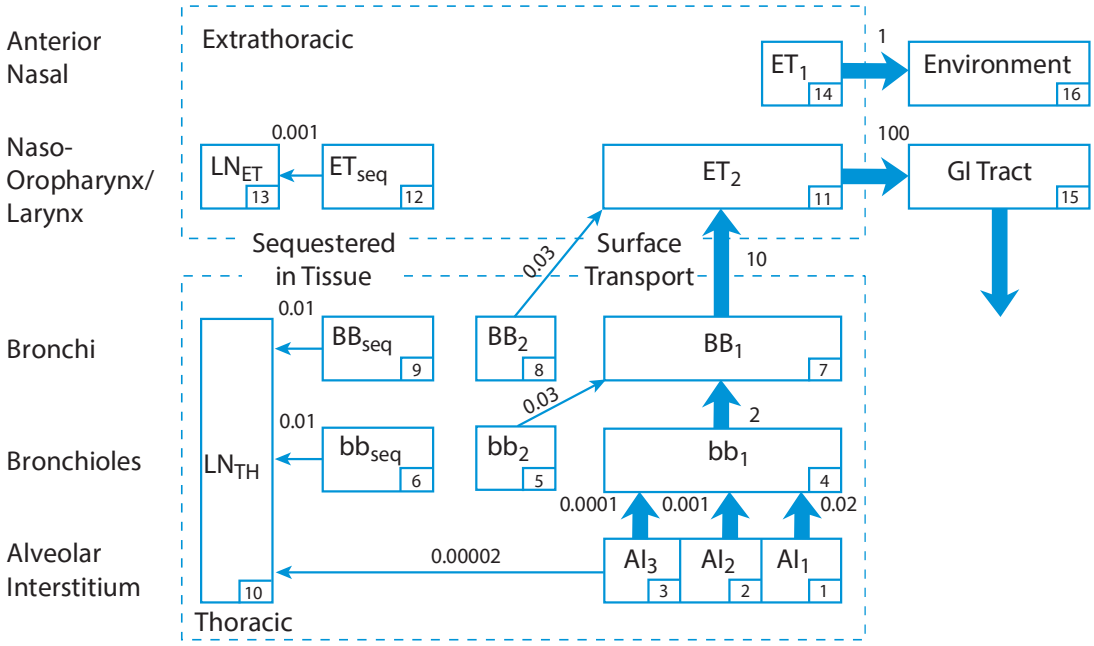

Figure 8-13. Compartmental model to represent time-dependent particle transport in the respiratory tract. The arrows show the transport pathway, and the numbers represent the compartmental clearance rates, per day. *Abbreviations:* ET, extrathoracic; LN_{ET}, lymph nodes (extrathoracic); LN_{TH}, lymph nodes (thoracic); BB, bronchial; bb, bronchiolar; AI, alveolar-interstitial; GI, gastrointestinal. (Reproduced with permission from ICRP Publication 66: Human Respiratory Tract Model for Radiological Protection. *Ann ICRP*. 1994; 24(1–3). Copyright © 1994 International Commission on Radiological Protection.)

TABLE 8-12 Factors for Partitioning Regional Deposits Among the Regional Compartments

REGION	COMPARTMENT	FRACTION TO COMPARTMENT
ET_2	ET_2	0.9995
	ET_{seq}	0.0005
BB	BB_1	$0.993 - f_s$
	BB_2	f_s
	BB_{seq}	0.007
bb	bb_1	$0.993 - f_s$
	bb_2	f_s
	bb_{seq}	0.007
AI	AI_1	0.3
	AI_2	0.6
	AI_3	0.1

Abbreviations: AI, alveolar-interstitial; BB, bronchial; bb, bronchiolar; ET, extrathoracic.

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For modeling purposes, the numerical values for the size-dependent parameter f_s in Table 8-12 is given for two categories of aerodynamic diameter, d_{ae}:

- if $d_{ae} \leq 2.5\left(\frac{\rho}{x}\right)^{0.5}$ μm,
  then $f_s = 0.5$.
- if $d_{ae} > 2.5\left(\frac{\rho}{x}\right)^{0.5}$ μm,
  then $f_s = 0.5 \exp\left[-0.63\left(d_{ae}\sqrt{\frac{x}{\rho}} - 2.5\right)\right]$,

where

ρ = particle density
x = shape factor, with 1.5 as the default.

The partitioning among the compartments in the bb and BB regions is dependent on the particle size; the partition factors for the other regions are independent of the particle size. For example, for ICRP 66, for the particles deposited in the AI region, 30% of the deposit is in the AI_1 compartment, which empties to the bb_1 compartment at a rate of 0.02 (2%) per day, 60% of the AI deposit is in the AI_2 compartment, whose clearance rate to bb_1 is 0.001 per day. Ten percent of the deposit is in the AI_3 compartment, which is cleared very slowly at a rate of 0.0001 per day to bb_1 and at a rate of 0.00002 per day to the thoracic lymph nodes, LN_{TH}. Additionally s_t per day (see paragraph below and Table 8-13) dissolves and is absorbed into the blood. The effective clearance rate for compartment AI_3 is the sum of the clearance rates for each of the three pathways: $\lambda_E(AI_3) = 0.0001 + 0.00002 + s_t$ per day. The quantity of activity in the AI region at time t days after deposition of activity Q Bq (or μCi) in region AI can be described mathematically by the three compartment retention curve:

$$Q_{AI}(t) = 0.3e^{-0.02t} + 0.6e^{-0.001t} + 0.1e^{-(0.00012+S_t)t}. \quad (8.26)$$

TABLE 8-13 Default Values of Absorption Parameters for Type F, M, and S Materials

PARAMETER	F	M	S
f_r	1	0.1	0.001
s_r (d^{-1})	100	100	100
s_s (d^{-1})	—	0.005	0.0001
s_p (d^{-1})	100	10	0.1
s_{pt} (d^{-1})	0	90	100
s_t (d^{-1})	—	0.005	0.0001

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In the BB and bb regions, 0.007 of the deposit is sequestered and is cleared to LN_{TH} at a rate of 0.01 per day. The partition fractions of the regional deposits among the several different compartments are listed in Table 8-12.

Particle Dissolution. Transfer of particulate radioactivity to the blood is modeled as a two-stage process: dissolution of the particle followed by its absorption into the body fluids, including the blood. The model assumes that absorption into the body fluids occurs at the same rate from all the parts of the HRTM except ET_1, where no absorption occurs.

The rate of solubilization of a particle is a function of its size, because dissolution is a surface phenomenon. As a particle dissolves, its surface area rapidly decreases. The rate of dissolution, therefore, decreases with time as the particle continues to dissolve. The HRTM deals with this decreasing rate of dissolution in two alternate ways. In the first time-dependent alternative, a fraction of the deposited activity, f_r, dissolves rapidly and is absorbed at a rate of s_r per day. The remaining fraction, $1 - f_r$, dissolves slowly and is absorbed at a rate s_s per day. According to this model, the overall fractional dissolution rate, f_d, of the intrapulmonary deposit dissolving and being absorbed at time t days after deposition is

$$f_d(t) = f_r e^{-s_r t} + (1 - f_r) e^{-s_s t}. \tag{8.27}$$

A situation where the dissolution and absorption rates increased with time could be modeled by the second alternative through a suitable choice of values for the parameters. In the alternative model, shown in Figure 8-14, the regional deposits are said to be in an "initial" state. Some of these particles dissolve at a constant rate S_p per day, and the rest of the particles are simultaneously changed into a "transformed" state at a rate S_{pt} per day. In the transformed state, the particles dissolve and the dissolved activity is absorbed into the body fluids at a rate of S_t per day, which is different from the absorption rate of the untransformed particles. For the usual case where the dissolution and absorption rates decrease with time,

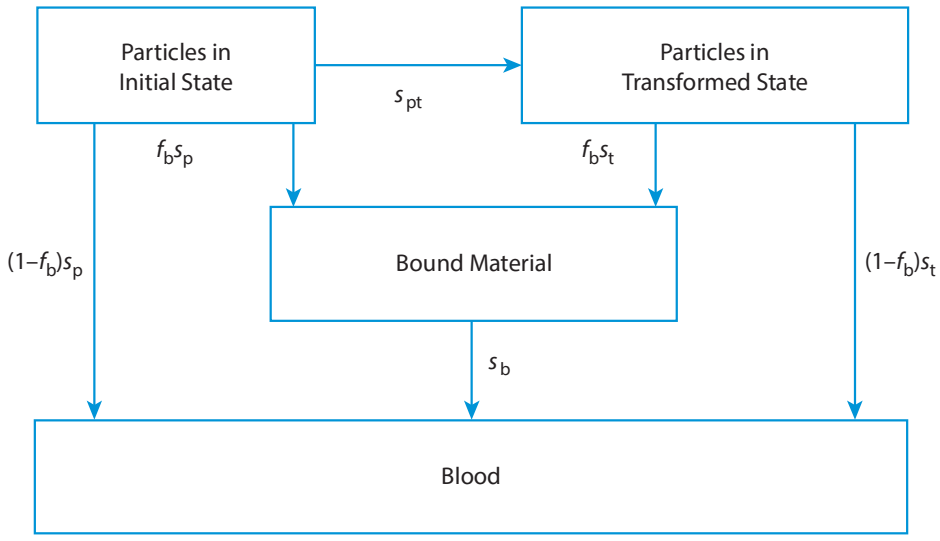

Figure 8-14. Compartmental model for time-dependent absorption into blood. *Source:* Human respiratory tract model for radiological protection. (Reproduced with permission from ICRP Publication 66: Human Respiratory Tract Model for Radiological Protection. *Ann ICRP.* 1994; 24(1–3). Copyright © 1994 International Commission on Radiological Protection.)

both models are equivalent. The parameters of the two alternative absorption models are related by the following equations:

$$S_p = S_s + f_r\left(S_r - S_s\right), \tag{8.28}$$

$$S_{pt} = \left(1 - f_r\right)\left(S_r - S_s\right), \text{and} \tag{8.29}$$

$$S_t = S_s. \tag{8.30}$$

In the absence of material-specific absorption rates, the default values for the solubility and absorption parameters recommended by the ICRP for each of the three solubility–absorption categories are listed in Table 8-13. Both alternatives postulate that a certain fraction, f_b, of the dissolved particles is chemically bound to the tissue, and that the bound material eventually diffuses into the body fluids. This "bound" state is a special case for which specific binding data must be available. Therefore, the "bound" state is not used for setting default values, that is, $f_b = 0$ for all three solubility–absorption categories.

The model representing the overall clearance of particles from the respiratory tract is shown in Figure 8-15.

Dosimetric Model. The HRT is considered as two separate organs for dosimetric purposes. The thoracic region is considered to be the lungs, and the ET region is considered as one of the "remainder" tissues when we calculate the EDE. Each of these organs consists of several different types of cells of differing radiosensitivity, and lie at different depths below the tissue–air interface (Table 8-14). Figure 8-16 shows the modeled tube that contains

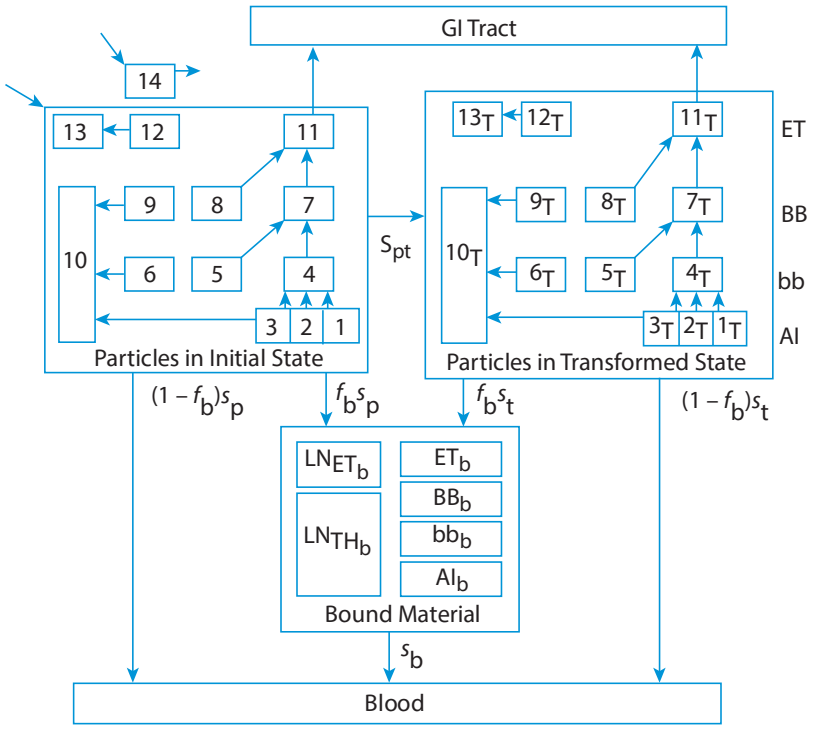

Figure 8-15. Overall compartmental model for respiratory tract clearance, including both time-dependent particle transport and absorption into the blood. *Abbreviations:* GI, gastrointestinal; ET, extrathoracic; BB, bronchial; bb, bronchiolar; AI, alveolar-interstitial; LN_{ET}, lymph nodes (extrathoracic); LN_{TH}, lymph nodes (thoracic).

TABLE 8-14 Target Cells and Assigned Fraction of w_T (Lung)

REGION	COMPARTMENT	TARGET CELL	CRITICAL TISSUE DEPTH (μm)	MASS (kg)	(*A*) of w_T
Extrathoracic airways	ET_1 (anterior nose)	Basal	40–50	2.0N5[a]	0.001
	ET_2 (posterior nose, mouth, pharynx, larynx)	Basal	40–50	4.5N4	0.998
	LN_{ET} (lymphatics)			1.5N2	0.001
Thoracic airways (lungs)	BB (bronchial)				0.333
		Basal	35–50	4.3N4	
		Secretory	10–40	8.6N4	
	bb (bronchiolar)	Secretory	4–12	1.9N3	0.333
	AI (alveolar-interstitial)			1.1	0.333
	LN_{TH} (lymphatics)			1.5N2	0.001

[a] 2.0N5 means 2×10^{-5}, 4.5N4 means 4.5×10^{-4}, etc.

Note: Regional doses, with weighting factors *A* assigned for the partition of the radiation detriment, are summed to give a value of committed dose equivalent for the extrathoracic region and another for the thoracic region, as follows:

$$H_{ET} = H_{ET1} \times A_{ET1} + H_{ET2} \times A_{ET2} + H_{LN(ET)} \times A_{LN(ET)}$$
$$H_{TH} = H_{BB} \times A_{BB} + H_{bb} \times A_{bb} + H_{AI} \times A_{AI} + H_{LN(TH)} \times A_{LN(TH)}$$

H_{TH} is considered the lung, $w_T = 0.12$.

When calculating effective dose equivalent, H_{ET} is considered a "remainder" tissue dose.

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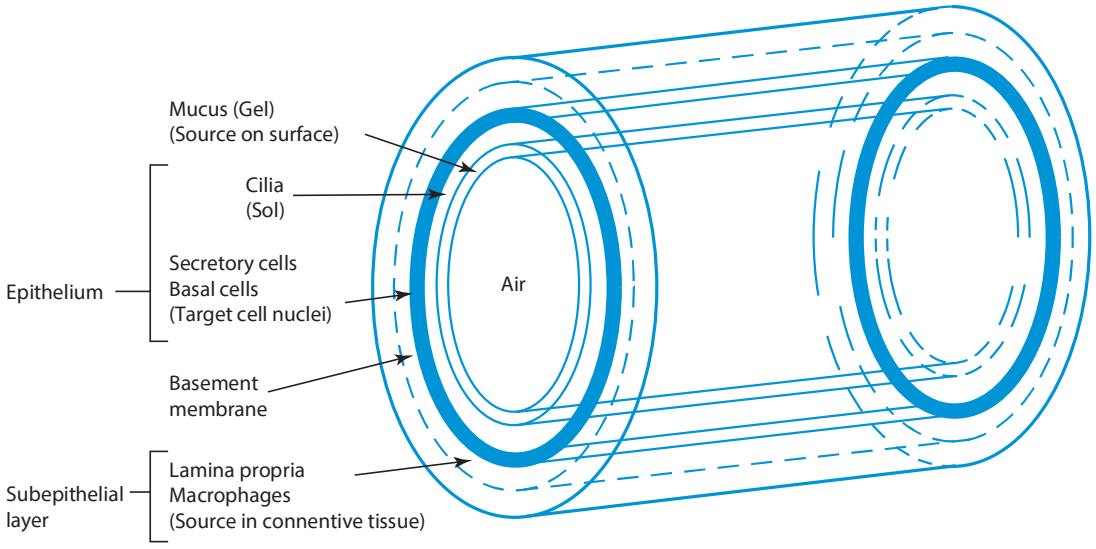


Figure 8-16. Simplified geometrical model of the tissue–air interface and the source and target tissues in dosimetry of the extrathoracic, bronchial, and bronchiolar regions. (Reproduced with permission from ICRP Publication 66: Human Respiratory Tract Model for Radiological Protection. *Ann ICRP*. 1994; 24(1–3). Copyright © 1994 International Commission on Radiological Protection.)

the tissue–air interface and the source and target tissues in airways in the ET, BB, and bb regions. For example, the sensitive target cells in the bb region are the nuclei of the secretory (Clara) cells (Fig. 8-17) that lie within the epithelial layer shown in Figure 8-16. These cells are believed to be the progenitor cells for squamous cell carcinoma, the most frequently occurring lung cancer. These depths are important because alphas, betas, and electrons that are emitted from radioactive particles that are deposited on the interface surface dissipate some of their energy in passing through the less-sensitive tissue. Thus, only a fraction of the energy of the emitted radiation is absorbed by the sensitive target cells. Figure 8-18 shows the absorbed fractions, AF (T←S) of beta particle energy that is absorbed by the target cells in the bb region. ICRP Publication 66 contains values for the AFs of all the target cells from alphas, betas, and electrons that originate in the various parts of the respiratory tract, as well as tables of the specific AFs of photon energy in various tissues and organs with the lungs as the source.

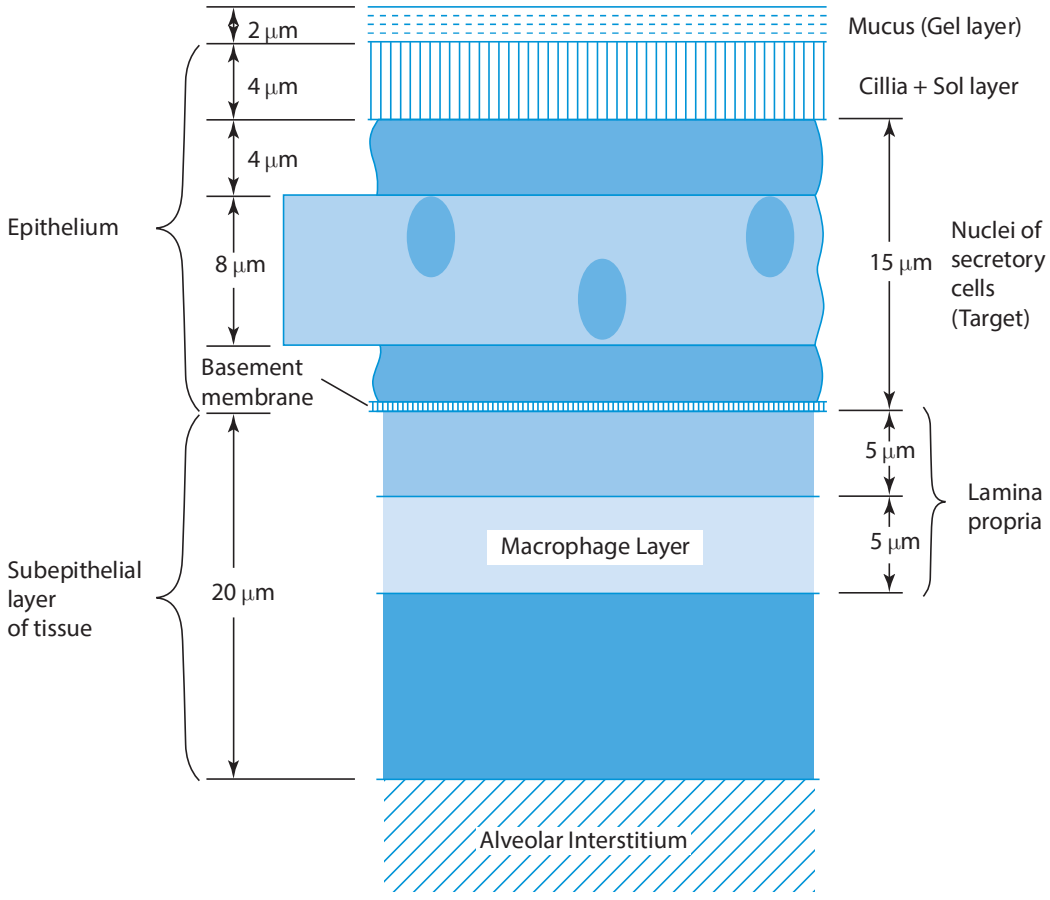


Figure 8-17. Dosimetric model of the target cells (secretory cells) in the bronchiolar wall of the bronchiolar region. (Reproduced with permission from ICRP Publication 66: Human Respiratory Tract Model for Radiological Protection. *Ann ICRP*. 1994; 24(1–3). Copyright © 1994 International Commission on Radiological Protection.)

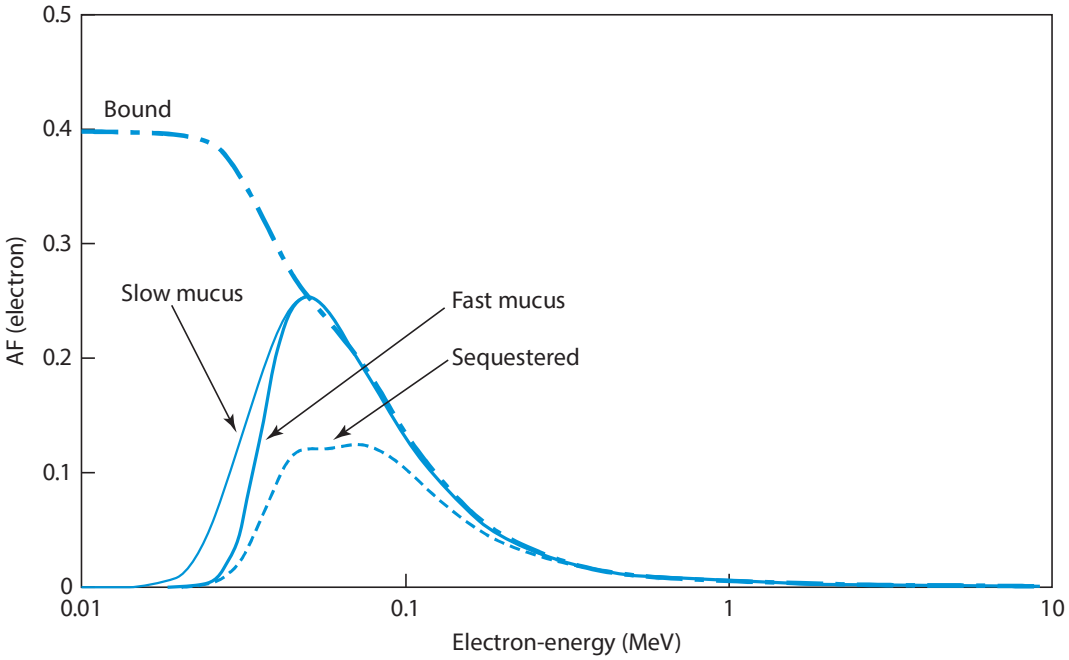


Figure 8-18. Absorbed fractions for betas emitted in the bronchiolar (bb) region. Curves are shown for emissions from the mucous gel layer (fast mucous), sol layer (slow mucous), sequestered, and bound activity. (Reproduced with permission from ICRP Publication 66: Human Respiratory Tract Model for Radiological Protection. *Ann ICRP*. 1994; 24(1–3). Copyright © 1994 International Commission on Radiological Protection.)

The HRTM is used to calculate the radiation dose to the lungs from an inhaled radioisotope. The dose to the rest of the body from the radioactivity transferred from the respiratory system to the blood requires knowledge of the metabolic kinetics or a physiologically based biokinetic model for that radioisotope or element. To calculate the lung dose from inhaled radioactive particles we

1. determine the regional deposition of the particles,
2. apportion the deposition within the regional compartments,
3. calculate the activity in each compartment, including the activity transported into the compartment from other compartments,
4. calculate compartmental mean residence time (MRT), including activity lost from each compartment by mechanical transport, dissolution, and radioactive decay,
5. calculate the total number of disintegrations in each compartment,
6. calculate the total energy emitted in each compartment,
7. calculate the total energy absorbed by the target tissues, using values from Tables G and H in ICRP 66 (abstracted in Table 8-17),
8. divide absorbed energy by mass of target tissues (Table 8-14), which is abstracted from Table 5, ICRP 66,
9. multiply the dose absorbed in each tissue by the appropriate radiation and tissue weighting factors, w_R and w_T, and
10. calculate lung dose $= w_R \sum H_T w_T$.

Dose Coefficient

The HRTM allows us to calculate the dose *only to the lung* from inhaled radioactivity. The activity absorbed into the body fluids and swallowed into the GI tract supplies the input data to physiologically based pharmacokinetic models that allow us to calculate the doses to the other organs and tissues, and to calculate the effective dose from the inhaled activity.

Through the use of the respective physiologically based pharmacokinetic models, we can calculate the committed equivalent dose and the committed effective dose from inhalation and ingestion of 1 Bq (or 1 μCi) of every radionuclide. These calculations yield the DC for each of the radionuclides, which, when multiplied by the intake, give the estimated dose to

TABLE 8-20 Dose Coefficients for Selected Radionuclides

RADIONUCLIDE	CLASS	INTAKE ROUTE	TARGET	DC (Sv/Bq)[a]	DC (Sv/Bq)[b]	DC (Sv/Bq)[c]
^{3}H (water vapor)		Inhalation	Whole body (effective)	1.73N11	1.8N11	—
^{32}P		Ingestion	Red marrow	8.09N9	—	—
^{32}P		Ingestion	Whole body (effective)	2.37N9	2.4E9	—
^{90}Sr–^{90}Y	D (F)	Inhalation	Bone surface	7.27N7	—	—
^{90}Sr–^{90}Y	D (F)	Inhalation	Whole body (effective)	6.47N8	2.4N8	3.0N8
^{137}Cs	(F)	Inhalation	Whole body (effective)	8.63N9	4.8N9	6.7N9
^{137}Cs		Ingestion	Whole body (effective)	1.35N8	1.3N8	—
^{226}Ra		Ingestion	Bone Surface	6.83N6	2.8N7	—
^{239}Pu	W (M)	Inhalation	Bone surface	2.11N3	4.7N5	3.2N5

Abbreviation: DC, dose coefficient.

Note: N11 = 10^{-11}.

Class refers to solubility class. Classes D and W are used in FGR 11, where Class D aerosols are rapidly cleared from the deep respiratory tract with a clearance half-time on the order of a day or a fraction of a day. Class W aerosols are cleared on the order of weeks.

Classes F and M are used in ICRP 119 where F (fast) assumes 100% absorbed into the blood in ≤10 minutes; M (moderate)—10% absorbed into blood in ≤10 minutes, 90% absorbed in ≤140 days.

All ICRP 119 values are for the whole body.

[a]Values are from FGR 11.

[b]Values are from ICRP 119 for 1-μm particles.

[c]Values are from ICRP 119 for 5 μm particles.

Source: [a]Eckerman KF, et al. Federal Guidance Report No. 11, 1988 (FGR 11) [b]ICRP, 2012. Compendium of Dose Coefficients based on ICRP Publication 60. ICRP Publication 119. *Ann. ICRP*. 41(Suppl).

the exposed person. DCs for all the radionuclides have been published by the U.S. EPA, the ICRP, and the IAEA. Some of these DCs are given in Table 8-20.

Derived Air Concentration

The ALI, which is a secondary standard that is based on the primary dose limit, only gives the annual intake limit; it does not deal with the rate of intake or with the atmospheric or environmental concentrations of a radionuclide that lead to the intake. It also is not amenable to direct measurement. For engineering design purposes, for control of routine operations, and for demonstration of compliance with regulations, we must know the environmental concentrations of the radionuclides with which we are dealing. To this end, the *derived air concentration* (DAC) is used by the U.S. NRC as a regulatory limit for airborne contaminants. The DAC is simply that average atmospheric concentration of the radionuclide that would lead to the ALI in a reference person as a consequence of exposure at the DAC for a 2000-hour working year. Since a reference worker inhales 20-L air per minute, or 2400 m^3 during the 2000 hours per year spent at work, the DAC is

$$\mathrm{DAC} = \frac{\mathrm{ALI}\,\dfrac{\mathrm{Bq}}{\mathrm{yr}}}{2400\,\dfrac{\mathrm{m}^3}{\mathrm{yr}}}. \tag{8.38}$$

Thus, for airborne ^{137}Cs, whose inhalation ALI is listed in ICRP 30 as 6×10^6 Bq, the DAC is

$$\mathrm{DAC} = \frac{6 \times 10^6\,\dfrac{\mathrm{Bq}}{\mathrm{yr}}}{2400\,\dfrac{\mathrm{m}^3}{\mathrm{yr}}} = 2.5 \times 10^3\,\frac{\mathrm{Bq}}{\mathrm{m}^3},$$

which is rounded off to 2×10^3 Bq/m^3.

According to ICRP 60 criteria, the annual dose limit is 0.02 Sv/yr. For 5-μm AMAD, class F ^{137}Cs particles, the DC is listed[5] as 6.7×10^{-9} Sv/Bq, the ALI is calculated as

$$\mathrm{ALI} = \frac{0.02\ \mathrm{Sv}}{6.7 \times 10^{-9}\,\dfrac{\mathrm{Sv}}{\mathrm{Bq}}} = 3 \times 10^6\,\mathrm{Bq},$$

and the DAC is

$$\mathrm{DAC} = \frac{3 \times 10^6\,\mathrm{Bq}}{2400\ \mathrm{m}^3} = 1.3 \times 10^3\,\frac{\mathrm{Bq}}{\mathrm{m}^3}.$$

Another unit which is utilized by the U.S. NRC is the DAC-hour. The DAC-hour is the product of the concentration of radioactive material in air, expressed as a fraction or multiple of the DAC, and the time of exposure to that nuclide in hours. Exposure to 2000 DAC-hours would deliver 5 rems (0.05 Sv). Effectively, one DAC-hour will deliver 2.5 mrem (25 μSv) under current U.S. regulations.

[5]The dose coefficient used here is from ICRP 119 for 5-μm particles with class F from Table 8-20.

Gaseous Radioactivity

Immersion in a cloud of radioactive gas leads to external exposure from the activity in the surrounding air and to internal exposure due to the inhaled gas. For the case of biochemically inert gases argon, krypton, and xenon, the external submersion dose limits the atmospheric concentration, as shown by the calculations for ^{41}Ar in the following paragraphs.

Argon-41, a biochemically inert gas, is transformed to ^{41}K by the emission of a 1.2-MeV beta particle and a 1.3-MeV gamma ray. The half-life of ^{41}Ar is 110 minutes, or 0.076 days. For the case of submersion, it is assumed that a person is exposed in an infinite hemisphere of the gas. For this exposure condition, ICRP 68 lists the effective DC for ^{41}Ar as 5.3×10^{-9} Sv/d/ per Bq/m^3. The reference working year is 250 days of 8 hours each. For an effective annual dose of 0.02 Sv (2 rems), the mean concentration of ^{41}Ar is calculated by

$$0.02\ \text{Sv} = 5.3 \times 10^{-9} \frac{\text{Sv/d}}{\text{Bq/m}^3} \cdot 250\ \text{d} \cdot C \frac{\text{Bq}}{\text{m}^3} \tag{8.39}$$

$$C = 1.5 \times 10^4 \frac{\text{Bq}}{\text{m}^3} \left(4 \times 10^{-7} \frac{\mu\text{Ci}}{\text{mL}} \right).$$

When a gas is inhaled, it may dissolve in the body fluids and fat after diffusion across the capillary bed in the lung. In the case of an inert gas, absorption into the body stops after the body fluids and fat are saturated with the dissolved gas. The saturation quantity of dissolved ^{41}Ar in the body fluids due to inhalation of contaminated air at the DAC, based on submersion, must be calculated in order to determine the internal dose. The first step in this calculation is the determination of the molar concentration of ^{41}Ar that corresponds to 1.5×10^4 Bq/m^3 (4×10^{-7} μCi/mL). The specific activity of ^{41}Ar is calculated with Eq. (4.30):

$$\text{SA}_i = 3.7 \times 10^{10} \left(\frac{A_{\text{Ra}} \cdot T_{\text{Ra}}}{A_i \cdot T_i} \right) \frac{\text{Bq}}{\text{g}}$$

$$= 3.7 \times 10^{10} \left(\frac{226 \cdot 1.6 \times 10^3\ \text{yrs} \cdot 365 \frac{\text{d}}{\text{yr}}}{41 \cdot 0.076\ \text{d}} \right) = 1.57 \times 10^{18} \frac{\text{Bq}}{\text{g}},$$

and the molar concentration of the ^{41}Ar is calculated as

$$\frac{1.5 \times 10^4 \frac{\text{Bq}}{\text{m}^3}}{1.57 \times 10^{18} \frac{\text{Bq}}{\text{g}}} \cdot \frac{1\ \text{mol}}{41\ \text{g}} = 2.33 \times 10^{-16} \frac{\text{mol}\ ^{41}\text{Ar}}{\text{m}^3}.$$

The molar concentration of air at standard temperature and pressure is

$$\frac{1\ \text{mol}}{22.4 \frac{\text{L}}{\text{mol}} \cdot 10^{-3} \frac{\text{m}^3}{\text{L}}} = 44.6 \frac{\text{mol air}}{\text{m}^3}.$$

Since argon constitutes 0.94 volume percent of the air, the molar concentration of naturally occurring argon in the air is

$$9.4\times10^{-3}\,\frac{\text{mol Ar}}{\text{mol air}}\cdot 44.6\,\frac{\text{mol air}}{\text{m}^3\ \text{air}}=0.42\,\frac{\text{mol Ar}}{\text{m}^3\ \text{air}}.$$

The amount of argon corresponding to the ^{41}Ar DAC based on submersion dose is thus seen to be insignificant relative to the argon already in the air. The molar concentration of argon in the air may therefore be assumed to be unchanged by the addition of 1.5×10^4 Bq/m^3 (4×10^{-7} μCi/mL) ^{41}Ar to the air. With this amount of ^{41}Ar in the air, the specific activity of the argon in the air is

$$\frac{1.5\times10^4\,\dfrac{\text{Bq}}{\text{m}^3}}{0.42\ \text{mol}\,\dfrac{\text{Ar}}{\text{m}^3}}=3.57\times10^4\,\frac{\text{Bq}}{\text{mol Ar}}\left(9.65\times10^{-7}\,\frac{\text{Ci}}{\text{mol Ar}}\right).$$

Now we will calculate the concentration of argon in the body fluids when the dissolved argon is in equilibrium with the argon in the air. According to Henry's law, the amount of a gas dissolved in a liquid is proportional to the partial pressure of the gas above the liquid:

$$P_{\text{gas}}=KN=K\frac{n_{\text{g}}}{n_{\text{g}}+n_{\text{s}}},\tag{8.40}$$

where

P_{gas} = partial pressure of the gas,
K = Henry's law constant,
N = mole fraction of the dissolved gas,
n_{g} = molar concentration of the dissolved gas, and
n_{s} = molar concentration of the solvent.

The solubilities of several gases in water at 38°C, expressed in terms of Henry's law constant, are given in Table 8-21. At body temperature, K, for argon is 3.41×10^7, and the partial pressure of argon in the atmosphere is

$$P_{\text{Ar}}=0.0094\cdot760=7.15\ \text{mm Hg}.$$

The total body water in a 70-kg reference person is 43 L. Therefore, the molar concentration of water, the solvent in Eq. (8.40), is

$$n_{\text{s}}=\frac{1000\,\dfrac{\text{g}}{\text{L}}}{18\,\dfrac{\text{g}}{\text{mol}}}=55.6\,\frac{\text{mol}}{\text{L}}.$$

Equation (8.40) may now be solved for the concentration of dissolved argon.

TABLE 8-21 Solubility of Several Gases in Water at 38°C

GAS	K ($\times 10^7$)
H_2	5.72
He	11.0
N_2	7.51
O_2	4.04
Ar	3.41
Ne	9.76
Kr	2.13
Xe	1.12
Rn	0.65
CO_2	0.168
C_2H_2	0.131
C_2H_4	1.21
N_2O	0.242

Note: $K = \frac{\text{partial pressure of gas in mm Hg}}{\text{mole fraction of gas in solution}}$.

$$7.15 = 3.41 \times 10^7 \frac{n_g}{n_g + 55.6}$$

$$n_g = 1.17 \times 10^{-5} \frac{\text{mol}}{\text{L}}.$$

Since the specific activity of the dissolved argon is 3.57×10^4 Bq/mol (9.65×10^{-7} Ci/mol), the argon activity concentration in the body fluids is

$$3.57 \times 10^4 \ \frac{\text{Bq}}{\text{mol}} \cdot 1.17 \times 10^{-5} \frac{\text{mol}}{\text{L}} = 0.42 \frac{\text{Bq}}{\text{L}} \quad \left(1.1 \times 10^{-5} \frac{\mu\text{Ci}}{\text{L}}\right),$$

and the total activity in the 43 L of body fluids contained in reference man is

$$43\ \text{L} \cdot 0.42 \frac{\text{Bq}}{\text{L}} = 18.1\ \text{Bq} \quad (4.9 \times 10^{-4}\ \mu\text{Ci}).$$

Argon is more soluble in fat than in water. At equilibrium, the partition coefficient, which is the concentration ratio of argon in fat to argon in water, is 5.4:1 at body temperature. The amount of argon in the 10 kg of fat in the reference worker is

$$5.4 \cdot 0.42 \frac{\text{Bq}}{\text{kg}} \cdot 10\ \text{kg} = 22.7\ \text{Bq} \quad (6.1 \times 10^{-4}\ \mu\text{Ci}).$$

The total argon activity in the reference person is the sum of the argon in the body fluids and in the fat:

$$\text{total body burden of } {}^{41}\text{Ar} = 18.1 \text{ Bq} + 22.7 \text{ Bq} = 40.8 \text{ Bq } (1.1 \times 10^{-3} \,\mu\text{Ci}).$$

If the argon is assumed to be uniformly distributed throughout the body, then the whole-body dose from the absorbed ^{41}Ar is calculated from

$$\dot{D}(\text{body} \leftarrow \text{body}) = \frac{q \text{ Bq} \cdot 1 \frac{\text{tps}}{\text{Bq}} \cdot E_a \frac{\text{MeV}}{\text{t}} \cdot 1.6 \times 10^{-13} \frac{\text{J}}{\text{MeV}} \cdot 3.6 \times 10^3 \frac{\text{s}}{\text{h}} \cdot 1 \frac{\text{Sv}}{\text{Gy}}}{70 \text{ kg} \cdot 1 \frac{\text{J/kg}}{\text{Gy}}} \quad (8.41)$$

If we substitute $q = 40.8$ Bq, and use an interpolated absorbed fraction of the photon energy from Table 6-6 of 0.31, and an average beta energy of Ar-41 of 0.46 MeV

$$E_a = \varphi \cdot E_\gamma + \overline{E}_\beta = \left(0.31 \cdot 1.3 \frac{\text{MeV}}{\text{t}}\right) + \left(0.46 \frac{\text{MeV}}{\text{t}}\right) = 0.86 \frac{\text{MeV}}{\text{t}}$$

into Eq. (8.41), we find

$$\dot{D}(\text{body} \leftarrow \text{body}) = 2.9 \times 10^{-10} \frac{\text{Sv}}{\text{h}} \quad \text{or} \quad 5.8 \times 10^{-7} \frac{\text{Sv}}{\text{yr}}.$$

The lungs are also irradiated by the ^{41}Ar within the airways, whose volume (according to ICRP 68) is 3.862 L. Since the air concentration is 15 Bq/L, there are

$$3.863 \frac{\text{L}}{\text{airway}} \cdot 15 \frac{\text{Bq}}{\text{L}} = 57.9 \text{ Bq}$$

in the air inside the lungs. The absorbed fraction of energy in the body from the Ar-41 in the lungs must be recalculated. Using the absorbed fraction from MIRD pamphlet 5 of 4.53×10^{-3} (body ← lung), we obtain

$$E_a = \varphi \cdot E_\gamma + \overline{E}_\beta = \left(4.53 \times 10^{-6} \cdot 1.3 \frac{\text{MeV}}{\text{t}}\right) + \left(0.46 \frac{\text{MeV}}{\text{t}}\right) = 0.46 \frac{\text{MeV}}{\text{t}}$$

The dose is computed with Eq. (8.41), using the total body mass, less the mass of the lungs (1.2 kg):

$$\dot{D}(\text{body} \leftarrow \text{lungs}) = \frac{57.9 \text{ Bq} \cdot 1 \frac{\text{tps}}{\text{Bq}} \cdot 0.46 \frac{\text{MeV}}{\text{t}} \cdot 1.6 \times 10^{-13} \frac{\text{J}}{\text{MeV}} \cdot 3.6 \times 10^3 \frac{\text{s}}{\text{h}} \cdot 1 \frac{\text{Sv}}{\text{Gy}}}{(70 - 1.2) \text{ kg} \cdot 1 \frac{\text{J/kg}}{\text{Gy}}}$$

So we obtain a dose rate of 2.2×10^{-10} Sv/h and a dose rate of 4.5×10^{-7} Sv/yr from the lungs to the body.

Finally, we must compute the dose from (lung ← lung). Using the absorbed fraction from MIRD pamphlet 5 of 4.5×10^{-3} (lung ← lung), we obtain

$$E_a = \varphi \cdot E_\gamma + \bar{E}_\beta = \left(4.5\times10^{-6} \cdot 1.3\frac{\text{MeV}}{\text{t}}\right) + \left(0.46\frac{\text{MeV}}{\text{t}}\right) = 0.46\frac{\text{MeV}}{\text{t}},$$

which results in the following dose rate

$$\dot{D}(\text{lungs} \leftarrow \text{lungs}) = \frac{57.9\ \text{Bq} \cdot 1\frac{\text{tps}}{\text{Bq}} \cdot 0.46\frac{\text{MeV}}{\text{t}} \cdot 1.6\times10^{-13}\frac{\text{J}}{\text{MeV}} \cdot 3.6\times10^{3}\frac{\text{s}}{\text{h}} \cdot 1\frac{\text{Sv}}{\text{Gy}}}{1.2\ \text{kg} \cdot 1\frac{\text{J/kg}}{\text{Gy}}}$$

to the lungs of 1.28×10^{-8} Sv/h and 2.56×10^{-5} Sv/yr.

The effective annual dose due to inhaling ^{41}Ar at the concentration based on the submersion dose is the sum of the doses to the whole body.

$$H = \sum w_T H_T.$$

Substituting the appropriate weighting factors, we have

$$H = (0.12 \cdot 2.7 \times 10^{-5}) + (0.88 \cdot (5.8 \times 10^{-7} + 4.5 \times 10^{-7})) = 4.1 \times 10^{-6}\ \text{Sv}.$$

The internal dose due to an atmosphere containing the limiting concentration for submersion is thus seen to be very much less than the submersion dose. The submersion dose is therefore the limiting dose. The same thing is true for the radioisotopes of krypton and xenon. For these radionuclides, therefore, the limiting atmospheric concentrations are based on the submersion dose.

ICRP 130 Revised Human Respiratory Tract Model

The HRT was revised in ICRP 130 to account for more recent data, so that the models were more realistic representations of the physiology of uptake, retention, and excretion. It provides for interpretation of bioassay measurement, monitoring programs, and retrospective dose assessment, and replaces ICRP 30, 54, 68, and 78. Some of the main changes to the HRTM are summarized here. Changes included revisions to the clearance of deposited material and absorption into blood. Generally, the calculation of dose is the same as for ICRP 66. The absorption classifications were changed from three to four:

- type V (fast)—100% instantly absorbed. Treated as injected into blood.
- type F (fast)—100% absorbed into blood with $t_{1/2} = 30$ minutes when $s_r = 30\ \text{d}^{-1}$. Approximately 100% rapid absorption in bb and AI, 80% in BB, and 25% in ET_2. Remaining materials in BB and ET_2 cleared by particle transport in alimentary tract.
- type M (moderate)—20% absorbed with $t_{1/2} = 6$ hours and 80% with $t_{1/2} = 140$ days when s_r=3 d^{-1}. Rapid absorption of 20% bb, 5% BB, 0.5% ET_2, 0.4% ET_1. Eighty percent in AI goes to blood.
- type S (slow)—1% absorbed with $t_{1/2} = 6$ hours and 99% with $t_{1/2} = 7000$ days when $s_r = 3\ \text{d}^{-1}$. Rapid absorption of 1% bb, 0.25% BB, 0.03% ET_2, 0.02% ET_1. Thirty percent in AI goes to blood.

TABLE 8-22 Absorption Values for ICRP 130 Modified HRTM

TYPE		F (fast)	M (moderate)	S (slow)
Fraction dissolved rapidly	f_x	1	0.2	0.01
Dissolution rates:				
Rapid (d^{-1})	S_r	30*	3†	3†
Slow (d^{-1})	S_s	–	0.005	0.0001

*Element specific rapid dissolution rates are adopted for Type F forms of many elements.

†The element-specific value for Type F is also used for Types M and S if it is less than 3 d^{-1}.

Gases and vapors were also simplified in ICRP 130. The default values for gases and vapors are 100% deposition in the respiratory tract, type F absorption, with 0% ET_1, 20% ET_2, 10% BB, 20%bb, and 50% AI. ICRP 130 no longer uses the SR-0, SR-01, SR-02 classification.

The absorption parameters for F, M, and S were altered, as detailed in Table 8-22, and deposition of aerosols in various regions has been altered. A detailed description of the reference worker is provided for inhalation dose calculations, as follows:

- Normal nose breathing
- Non-smoking
- Adult male at light work
- Light work is
  - 2.5-hour sitting, 0.54 m^3/h breathing
  - 5.5-hour light exercise, 1.5 m^3/h breathing

Although ICRP 130 purports to have made only minor changes to the HRTM, they are significant, and do impact many calculations (see Tables 8-23 and 8-24. Figure 8-19 provides an overview of the model. Internal dosimetry modeling has progressed to a point where the use of more complex voxel phantoms and multi-compartment modeling require the use of computers to perform repiratory tract dosimetry.

Gastrointestinal Tract

In cases of ingested radionuclides or radionuclides transferred to the GI tract from the lungs, and especially for those nuclides that are poorly absorbed from the GI tract, the GI tract or portions of it may be the tissue or organ that receives the greatest dose. The dose to the GI tract is calculated on the basis of the four-compartment dosimetric model shown in Figure 8-20 (ICRP 30). According to this model, the radionuclide enters the stomach (ST) and then passes sequentially through the small intestine (SI), from which most absorption into the body fluids occurs. It then passes through the upper large intestine (ULI) and the lower large intestine (LLI). Finally, the remaining activity is excreted in the feces. The clearance rate for transfer from the small intestine into the body fluids is given by

$$\lambda_B = \frac{f_1 \lambda_{SI}}{1 - f_1}, \tag{8.42}$$

where f_1 = fraction of the stable element reaching the body fluids after ingestion.

TABLE 8-23 ICRP 66 and ICRP 130 Comparison

PARTITION OF DEPOSIT IN EACH REGION BETWEEN COMPARTMENTS					
ORIGINAL HRTM (FROM ICRP, 1994A, TABLE 17B)			REVISED HRTM		
Region or deposition site	Compartment	Fraction of deposit in region assigned to compartment	Region or deposition site	Compartment	Fraction of deposit in region assigned to compartment
ET_1	ET_1	1	ET_1	ET_1	1
ET_2	ET_2'	0.9995	ET_2	ET_2'	0.998
	ET_{seq}	0.0005		ET_{seq}	0.002
BB	BB_1	$0.993–f_s$	BB	BB′	0.998
	BB_2	f_s		BB_{seq}	0.002
	BB_{seq}	0.007			
bb	bb_1	$0.993–f_s$	bb	bb′	0.998
	bb_2	f_s		bb_{seq}	0.002
	bb_{seq}	0.007			
AI	AI_1	0.3	AI	ALV	1
	AI_2	0.6			
	AI_3	0.1			

In making dose calculations for the purpose of calculating a DC and an ALI, we assume the radionuclide to be uniformly distributed throughout the contents of the respective segments of the GI tract and the weight of the contents of each segment to be as listed in Figure 8-19. Furthermore, the movement of the contents between compartments is assumed to follow first-order kinetics, with compartmental clearance rates as shown in Figure 8-19. The time rate of change of the contents of each of the four compartments can be calculated on the basis of mass balance. The increase or decrease in the quantity of radionuclide in any of the compartments is simply equal to the difference between what goes in and what goes out:

$$\text{rate of change of contents} = \text{rate in} - \text{rate out}. \tag{8.43}$$

If we have a constant input rate, $\dot{I}$ per day, as in the case of continuous ingestion of radioactivity in food or continuous inhalation of a radioactive aerosol that is cleared from the lung into the GI tract, then the mass balance equation for the stomach becomes

$$\left(\frac{dq}{dt}\right)_{St} = \dot{I} - \lambda_{St} q_{St} - \lambda_R q_{St}, \tag{8.44}$$

TABLE 8-24 Reference Values of Parameters for the Compartment Model to Represent Time-Dependent Particle Transport from the Human Respiratory Tract

CLEARANCE RATES							
ORIGINAL HRTM (FROM ICRP, 1994A, TABLE 17A)				REVISED HRTM			
From	To	Rate (d^{-1})	Half-time	From	To	Rate (d^{-1})	Half-time
AI_1	bb_1	0.02	35 d	ALV	bb′	0.002	–
AI_2	bb_1	0.001	700 d	ALV	INT	0.001	–
AI_3	bb_1	0.0001	–	INT	LN_{TH}	0.00003	–
AI_3	LN_{TH}	0.00002	–				
bb_1	BB_1	2	8 h	bb′	BB′	0.2	4 d
bb_2	BB_1	0.03	23 d	bb_{seq}	LN_{TH}	0.001	700 d
bb_{seq}	LN_{TH}	0.01	70 d				
BB_1	ET'_2	10	100 min	BB′	ET'_2	10	100 min
BB_2	ET'_2	0.03	23 d	BB_{seq}	LN_{TH}	0.001	700 d
BB_{seq}	LN_{TH}	0.01	70 d				
ET'_2	Gastrointestinal tract	100	10 min	ET'_2	Oesophagus	100	10 min
ET_{seq}	LN_{ET}	0.001	700 d	ET_{seq}	LN_{ET}	0.001	700 d
ET_1	Environment	1	17 h	ET_1	Environment	0.6	–
				ET_1	ET'_2	1.5	–

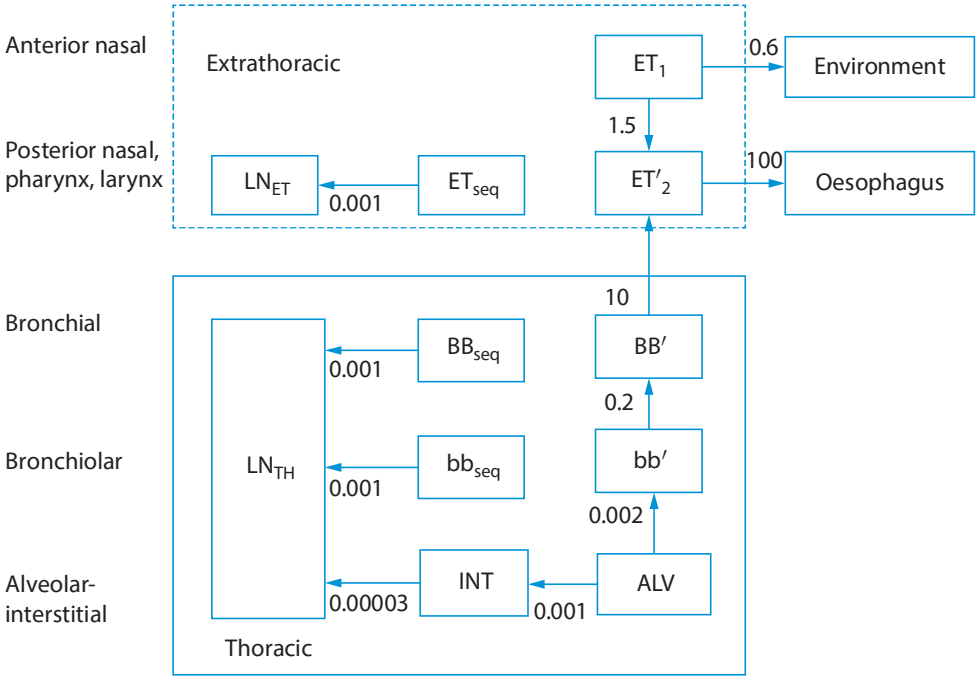


Figure 8-19. Modified Human Respiratory Tract Model of ICRP 130. Rates shown are in units of d^{-1}. (Reproduced with permission from ICRP, 2015. Occupational intakes of Radionuclides: Part 1. ICRP Publication 130. Ann. ICRP 44(2). Copyright © 2015 International Commission on Radiological Protection.)

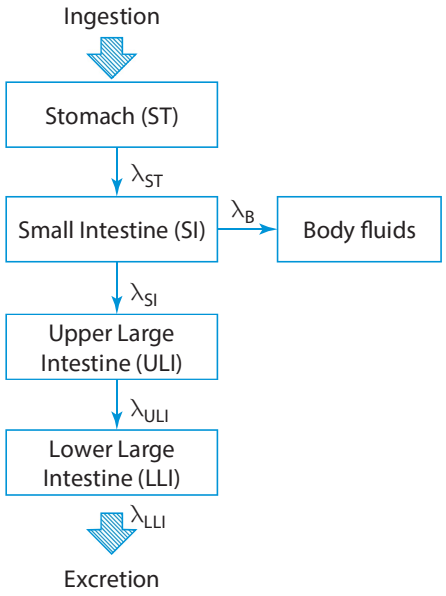


Figure 8-20. Dosimetric model of the gastrointestinal tract. The clearance rate for transfer from the small intestine into the body fluids is given by Eq. (8.42). (Reproduced with permission from ICRP Publication 30, Part 1: Limits for Intakes of Radionuclides by Workers. *Ann ICRP*. 1979; 2(3/4):33. Copyright © 1979 International Commission on Radiological Protection.)

where q may be measured either in SI units or in traditional units and λ is the turnover rate per day. When the amount of activity entering into the stomach is equal to the amount leaving, we have a steady-state condition, and $(dq/dt)_{St}$ becomes equal to zero. Under this condition, Eq. (8.44) becomes

$$\dot{I} = \lambda_{St} q_{St} + \lambda_R q_{St}. \tag{8.45}$$

The stomach contents empty into the small intestine, whose kinetics are similar to those of the stomach. The time rate of change of the contents, therefore, is described by the difference between what enters from the stomach and what leaves the small intestine. Material is cleared from the small intestine by two pathways:

1. by peristalsis into the upper large intestine, and
2. by molecular diffusion into the blood vessels in the inner surface of the small intestine.

The difference between what goes into the small intestine and what leaves it is expressed mathematically by

$$\left(\frac{dq}{dt}\right)_{SI} = \lambda_{St} q_{St} - \lambda_{SI} q_{SI} - \lambda_R q_{SI} - \lambda_B q_{SI}, \tag{8.46}$$

where λ_B is the transfer rate of the radionuclide from the small intestine into the blood and is given by Eq. (8.42). The dosimetric model of the GI tract assumes that only water is absorbed into the bloodstream from the large intestine. The rate of change of the radioactivity in the upper large intestine, therefore, is given by

$$\left(\frac{dq}{dt}\right)_{ULI} = \lambda_{SI} q_{SI} - \lambda_{ULI} q_{ULI} - \lambda_R q_{ULI}, \tag{8.47}$$

and for the lower large intestine, from which the radioactivity leaves the body, we have

$$\left(\frac{dq}{dt}\right)_{LLI} = \lambda_{ULI} q_{ULI} - \lambda_{LLI} q_{LLI} - \lambda_R q_{LLI}. \tag{8.48}$$

With the aid of Eqs. (8.44) to (8.48) and the appropriate specific AFs, we can calculate the dose per unit intake of radioactivity to the walls of the GI tract and to other organs and tissues for steady-state conditions and thus can compute the intake that will result in the dose limit, the ALI.

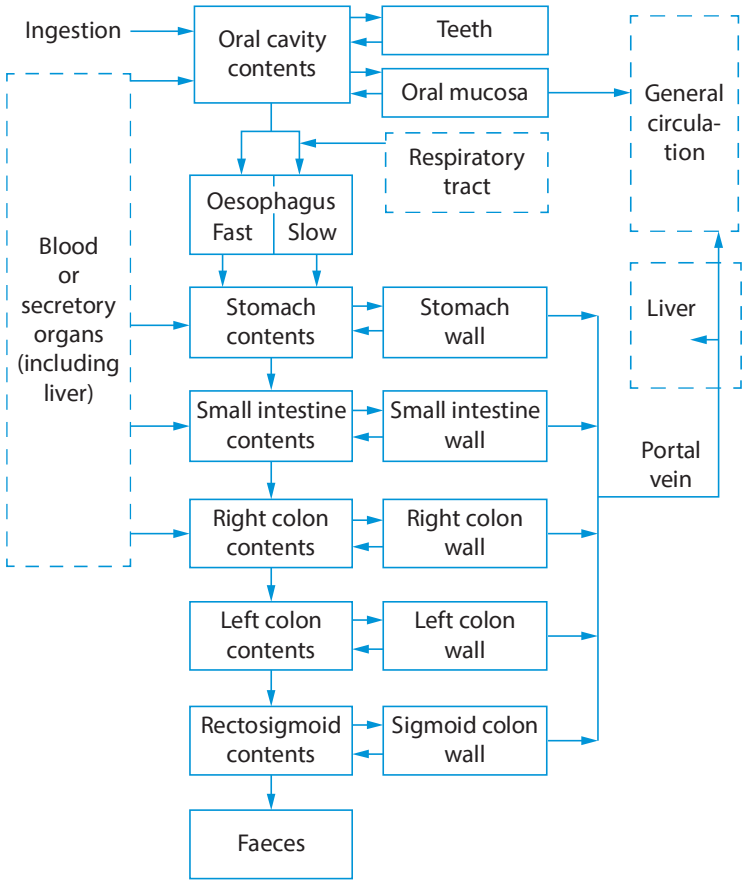
ICRP 130 Human Alimentary Tract Model

The ICRP 30 human alimentary tract model (HATM) was replaced in ICRP 100. ICRP 130 updates and expands the model to include all alimentary tract regions, fractional absorptions in tissues, and retention information. The HATM in ICRP 130 is more physiologically realistic, and can be utilized for bioassay. As with the HRTM, voxel phantoms (ICRP 110) and Monte Carlo models are used to calculated dose coefficients (DC). The calculations of doses are very loosely built upon the basic techniques used in ICRP 30, but are far more sophisticated in the quantity and quality of information input to the models, as seen in Figure 8-21. As with the HRTM, the HATM requires using computer modeling to calculate doses.

Dosimetric Model for Bone

ICRP 2 Methodology

To gain an insight into the evolution of safety standards for bone seekers, it is instructive to examine the ICRP 2 recommendations for intake limits, which were based on the critical organ concept. That is, on the organ that received the greatest dose from the intake of a radionuclide. For bone-seeking radionuclides, the intake limits were based on the application of a simple dosimetric model to data derived mainly from humans. The skeleton was treated as though it were a single tissue that weighed 7 kg. Because we had a great deal of experience with human exposure to radium and because radium is a "bone seeker"—that is, it is deposited in the bone—the maximum permissible body burdens of all bone seekers were established by comparing the dose equivalent of the bone seeker with that delivered to the bone by radium. On the basis of data on humans, 0.1-μg radium, corresponding to 3.7 kBq, in equilibrium with its decay products, was recommended as the maximum permissible body burden of ^{226}Ra. Using the then quality factor of 10 for alpha particles, the calculated dose equivalent to the bone from 0.1 μg ^{226}Ra and its daughters was 0.56 rem (5.6 mSv) per week.



SECTION OF GI TRACT	MASS OF WALLS (g)	MASS OF CONTENTS (g)	MEAN RESIDENCE TIME (d)	λ (d^{-1})
Stomach (ST)	150	250	1/24	24
Small intestine (SI)	640	400	4/24	6
Upper large intestine (ULI)	210	220	13/24	1.8
Lower large intestine (LLI)	160	135	24/24	1

Figure 8-21. Human Alimentary Tract Model from ICRP 130. Dashed boxes show relationship with HRTM. Default transfer coefficients are shown in the table. (Reproduced with permission from ICRP, 2015. Occupational intakes of Radionuclides: Part 1. ICRP Publication 130. Ann. ICRP 44(2). Copyright © 2015 International Commission on Radiological Protection.)

Radium is deposited relatively uniformly in the bone. Other bone seekers, however, were found to be deposited in a patchy, nonuniform manner that results in doses to some parts of the bone as much as five times greater than the average bone dose. For this reason, the ICRP introduced the *relative damage factor*, *N*, as a multiplier of the quality factor, QF. This factor has a value of 5 for all corpuscular (alpha or beta) radiation except for those cases where the corpuscular radiations are due to a chain whose first member is radium. When radium is the first member of the chain, then $N = 1$, since the distribution of the radioisotope will be determined by the radium. For example, the value of the relative damage factor *N* for

$$^{228}\text{Th} \xrightarrow{\beta} {}^{224}\text{Ra} \xrightarrow{\alpha}$$

is 5 for each particle, while the same particles are weighted with a relative damage factor of 1 in the chain

$$^{228}\mathrm{Ra} \xrightarrow{\beta} {}^{228}\mathrm{Ac} \xrightarrow{\beta} {}^{228}\mathrm{Th} \xrightarrow{\beta} {}^{224}\mathrm{Ra} \xrightarrow{a}.$$

The energy dissipated in the bone by ^{226}Ra and the daughters that remain in the bone is 11 MeV per transformation. Applying the QF value of 10 brings the effective energy to 110 MeV per transformation. Since 99% of the radium body burden is in the skeleton, ICRP 2, using data on humans as a basis, calculated a maximum permissible body burden of any other bone seeker:

$$q = \frac{3.7\times10^{3}\,\mathrm{Bq}\cdot 0.99}{f_2}\cdot\frac{110\dfrac{\mathrm{MeV}}{\mathrm{t}}}{E\dfrac{\mathrm{MeV}}{\mathrm{t}}} = \frac{4\times10^{5}}{f_2 E}\,\mathrm{Bq}, \tag{8.49}$$

where E is the effective corpuscular energy per transformation of any other bone seeker and f_2 is the fraction of the total body burden of the bone seeker that is in the skeleton. For the case of ^{90}Sr, for example, we have:

^{90}Sr-^{90}Y are pure beta emitters whose average energy is 0.194 MeV (^{90}Sr) + 0.93 MeV (^{90}Y) = 1.12 MeV/transformation

Q (quality factor) = 1,
N = 5, and
f_2 = 0.99.

The effective energy is 5·1.12 = 5.6 MeV per transformation. From Eq. (8.49) we find the maximum permissible body burden to be

$$q = \frac{4\times10^{5}}{0.99\cdot 5.6} = 7.2\times10^{4}\,\mathrm{Bq}\ \ (2\,\mu\mathrm{Ci}).$$

The effective half-life for ^{90}Sr in the skeleton is found in ICRP 2 to be 6400 days, which corresponds to an effective clearance rate, $\lambda_E = 1.08 \times 10^{-4}$ per day. Since 9% of the ingested Sr is deposited in the bone, the MPC in drinking water that will maintain the body burden at 7.2×10^4 Bq (2 μCi) is found through the use of activity-balance calculations. If we assume that the drinking water is the only source of intake of ^{90}Sr and that the ^{90}Sr containing water is the person's sole source of water, then we can calculate the concentration of radiostrontium in the water that would lead to a steady-state ^{90}Sr activity of 2 μCi. Under steady-state conditions,

$$\text{activity deposited} = \text{activity eliminated}, \tag{8.50a}$$

that is,

$$C\frac{\mu\mathrm{Ci}}{\mathrm{mL}}\cdot 2.2\times10^{3}\frac{\mathrm{mL}}{\mathrm{d}}\cdot f = \lambda_E\ \mathrm{d}^{-1}\cdot q\,\mu\mathrm{Ci}. \tag{8.50b}$$

In SI units, Eq. (8.50b) becomes

$$C\frac{\text{Bq}}{\text{mL}}\cdot 2.2\times 10^{3}\frac{\text{mL}}{\text{d}}\cdot f=\lambda_{\text{E}}\text{d}^{-1}\cdot q\ \text{Bq}, \tag{8.50c}$$

where

C = maximum permissible concentration (MPC, which was an ICRP 2 concept),
f = fraction of the intake that is deposited in the critical organ,
λ_{E} = effective elimination rate constant, and
q = steady-state activity in the critical organ.

Substituting the appropriate values into Eq. (8.50b) and solving for C yields

$$C=\frac{1.08\times 10^{-4}\,\text{d}^{-1}\cdot 2\ \mu\text{Ci}}{2.2\times 10^{3}\dfrac{\text{L}}{\text{d}}\cdot 9\times 10^{-2}}=1\times 10^{-6}\frac{\mu\text{Ci}}{\text{mL}}\quad\left(3.7\times 10^{-2}\frac{\text{Bq}}{\text{mL}}\right).$$

Ingestion of water at the rate assumed in the calculation above will result in the maximum permissible body burden *when equilibrium is attained* (Fig. 8-22). Because of the very long effective half-life of ^{90}Sr in the bone, the maximum allowable body burden is not attained during the 50-year occupational exposure time assumed for the purpose of computing values for the radiation safety guide. After 50 years of continuous ingestion at the above rate, the amount of ^{90}Sr in the skeleton will be

$$q=q_{\text{equil.}}(1-e^{-\lambda_{\text{E}}t})$$
$$q=2\ \mu\text{Ci}\left(1-e^{-\left(1.08\times 10^{-4}\cdot(50)\cdot 365\right)}\right)$$
$$q=1.7\mu\text{Ci}\quad(6.2\times 10^{4}\text{Bq}),$$

or only 86% of the maximum body burden. It is thus clear that the average body burden, and consequently the average dose rate to the skeleton during a 50-year period of maximum permissible ingestion, will be considerably less than the maximum permissible body burden. The mean body burden during a period of ingestion, T, starting at time zero when there is

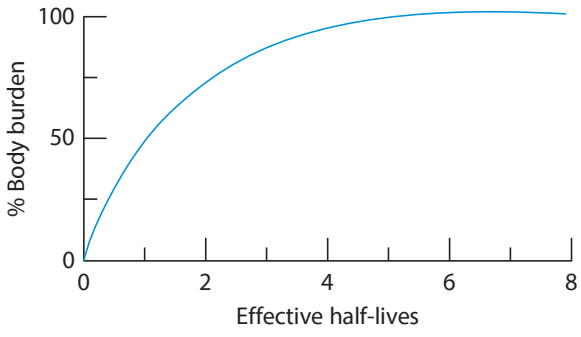


Figure 8-22. Buildup of a radioisotope in the body resulting from continuous intake.

no radioisotope of the species in question in the body, and assuming the effective elimination rate for the radioisotope to be λ_E, is given by

$$\bar{q} = \frac{1}{T}\int_0^T q_{\text{equil.}}(1 - e^{-\lambda_E t})\,dt. \tag{8.51}$$

Integrating Eq. (8.51), we obtain

$$\bar{q} = q_{\text{equil.}}\left[1 + \frac{1}{\lambda_E T}(e^{-\lambda_E T} - 1)\right]. \tag{8.52}$$

For ^{90}Sr, whose $\lambda_E = 0.0395\ \text{yr}^{-1}$, we have for a 50-year exposure period

$$\bar{q} = 1.13\ \mu\text{Ci} \qquad (4.18 \times 10^4\ \text{Bq}).$$

Several other radionuclides (Table 8-25) do not attain their equilibrium values in the body during 50 years of continuous ingestion at the maximum recommended concentrations.

TABLE 8-25 Radioisotopes That Do Not Reach Equilibrium in 50 Years

Z	ISOTOPE	T_E (yrs)	% EQUILIBRIUM AFTER 50 YEARS
38	^{90}Sr	18	86
88	^{226}Ra	44	56
89	^{227}Ac	20	83
90	^{230}Th	200	16
90	^{232}Th	200	16
91	^{231}Pa	200	16
93	^{237}Np	200	16
94	^{238}Pu	62	43
94	^{239}Pu	200	16
94	^{240}Pu	190	16
94	^{241}Pu	12	94
94	^{242}Pu	200	16
95	^{241}Am	140	22
95	^{243}Am	200	16
96	^{243}Cm	30	69
96	^{244}Cm	17	87
96	^{245}Cm	200	16
96	^{246}Cm	190	16
98	^{249}Cf	140	22
98	^{250}Cf	10	97

ICRP 30 Dosimetric Model

While the ICRP 2 recommendations were based on a dosimetric model that considered the "bone" as a single tissue consisting of a homogeneous mixture of its chemical compounds, the ICRP 30 dosimetric model considers the various different tissues within the bone that are at risk. Bone is modeled as three separate tissues:

- Cortical (or compact) bone, which is the hard outer portion of the bone, is assigned a mass of 4 kg in the ICRP 30 model. (ICRP 89 lists the masses of the skeleton's components according to sex and age.)
- Trabecular bone, which is the soft spongy inside the cortical bone, is assigned a mass of 1 kg in the ICRP 30 model.
- Red (or active) marrow, which is located in the spaces within the trabecular bone, has an assigned mass of 1.5 kg.

The most radiosensitive tissues are the 120 g of endosteum that lie within the first 10 μm of the adjacent bone surfaces and the 1.5 kg of red bone marrow. Since the AF (absorbed fraction) of the energy emitted by radionuclides within the bone depends on where the radionuclides are deposited, the newer bone model classifies the bone-seeking radionuclides as *volume seekers* and *surface seekers*. Whether any specific radionuclide is a volume or surface seeker is determined by the metabolism of the element. In this regard, the ICRP 30 dosimetric model established two general categories:

1. Isotopes of the alkaline earth elements whose half-lives exceed 15 days are assumed to be uniformly distributed throughout the volume of the bone.
2. Shorter-lived radionuclides are assumed to be distributed on the bone surfaces, since they are unlikely to have distributed themselves within the bone volume before they decay.

For dosimetric purposes, six nonexclusive categories of bone seekers are used in the ICRP 30 bone model:

1. photon emitters,
2. alpha-emitting volume seekers,
3. alpha-emitting surface seekers,
4. beta-emitting surface seekers whose mean beta energy is at least 0.2 MeV,
5. beta-emitting surface seekers whose mean beta energy is less than 0.2 MeV, and
6. beta-emitting volume seekers.

These categories are not mutually exclusive because a radionuclide, such as a beta–gamma emitter, belongs in two categories. In this case, each different type of radiation is considered separately. The AFs (absorbed fractions) for the various particle emitters are given in Table 8-26. The AFs for photons are given in Appendix D.

Using the physiologically based biokinetic model for a bone-seeking radionuclide, we can calculate the dose to the bone or bone surface and the doses to the other organs and tissues due to the intake, by ingestion or inhalation, of 1 Bq or 1 μCi of activity. Then, using either the ICRP and IAEA criterion of 0.02-Sv effective dose limit, or U.S. NRC criterion of 5-rems (0.05-Sv) effective dose limit or 50-rems (0.5-Sv) organ dose limit, we can calculate the secondary ALI and the tertiary DAC or maximum concentration in water. If we were to use the

TABLE 8-26 Recommended Absorbed Fractions for Dosimetry of Radionuclides in Bone

SOURCE	TARGET	*A*, Vol.	α, BS	β, Vol	$\beta, \bar{E} \geq 0.2$ MeV, BS	$\beta, \bar{E} < 0.2$ MeV, BS
Trabecular	Surface (BS)	0.025	0.25	0.025	0.025	0.25
Cortical	Surface	0.01	0.25	0.015	0.015	0.25
Trabecular	Red marrow	0.05	0.5	0.35	0.5	0.5
Cortical	Red marrow	0.0	0.0	0.0	0.0	0.0

Abbreviation: BS, bone surface.

Source: Reproduced with permission from ICRP Publication 30, Part 1: Limits for Intakes of Radionuclides by Workers. *Ann ICRP*. 1979; 2(3/4):42. Copyright © 1979 International Commission on Radiological Protection.

ICRP criterion of a mean annual effective dose of 0.02 Sv and the DC of 2.4×10^{-9} Sv/Bq for 5-μm moderately soluble ^{45}Ca particles, then the inhalation ALI would be

$$\text{ALI(effective)} = \frac{0.02\ \text{Sv}}{2.4\times10^{-9}\ \frac{\text{Sv}}{\text{Bq}}} = 8.33\times10^{6}\,\text{Bq} = 225\ \mu\text{Ci}.$$

For example, the U.S. NRC's inhalation ALI for soluble (class D) ^{90}Sr, using the DCs for the bone surface and for whole-body effective dose listed in Table 8-20, we have

$$\text{ALI(bone surface)} = \frac{0.5\ \text{Sv}}{7.27\times10^{-7}\ \frac{\text{Sv}}{\text{Bq}}} = 6.9\times10^{5}\,\text{Bq} = 1.9\times10^{1}\,\mu\text{Ci}$$

$$\text{ALI (effective)} = \frac{0.05\ \text{Sv}}{6.47\times10^{-8}\ \frac{\text{Sv}}{\text{Bq}}} = 7.7\times10^{5}\,\text{Bq} = 2.1\times10^{1}\,\mu\text{Ci}.$$

The smaller of the two ALIs is designated as the limit, and thus the dose to the bone surface is the limiting dose. Since the limits are rounded to one significant figure, the ALI for inhalation of 1-μm, class D ^{90}Sr particles is listed in 10 CFR 20 as 2×10^{1}, and would be applicable to both stochastic and nonstochastic cases. However, 10 CFR 20, Table 1, notes that the dose to the bone surface is the deciding criterion.

ICRP 130 Skeletal Dosimetric Model

The skeletal (bone) dosimetry model of ICRP 30 was updated in ICRP 130, with more detailed anatomical features and energy absorption parameters. ICRP 110 voxel phantoms were utilized. Some skeletal dose response functions (DRFs) can be found in ICRP 116, Annex D and E. Again, computations using the model are complex, and will require the use of computer programs.

UNITED STATES NUCLEAR REGULATORY PROGRAM

National Council on Radiation Protection and Measurements

The ICRP is not a regulatory agency. It is a scientific body that makes recommendations for radiation safety standards. In accordance with the policy laid down by the ICRP, its recommendations are adapted to the needs and conditions in the various countries by national bodies. In the United States, this function is served by the NCRP. This organization, which was originally known as the Advisory Committee on X-ray and Radium Protection (founded in 1929), consists of a group of technical experts who are specialists in radiation safety and scientists who are experts in the disciplines that form the basis for radiation safety. The concern of the NCRP is only with the scientific and technical aspects of radiation safety. To accomplish its objectives, the NCRP is organized into a main council, whose members are selected on the basis of their scientific expertise, and a number of subcommittees. Each of the subcommittees is responsible for preparing specific recommendations in its field of competence. The recommendations of the subcommittees require approval of the council before they are published. Finally, the approved recommendations are published by the council, with titles such as Report No. 147, *Structural Shielding Design for Medical X-Ray Imaging Facilities*. It should be emphasized that the NCRP is not an official government agency, although its recommendations are very seriously considered by regulatory agencies.

Atomic Energy Commission

In the United States, regulatory responsibility for radiation safety in the nuclear energy program originally was given by the U.S. Congress to the United States Atomic Energy Commission (AEC) through the enactment of the Atomic Energy Act of 1946 and the Atomic Energy Act Amendments of 1954 and the Energy Policy Act of 2005. The AEC continued to function until 1974, when its responsibilities were divided between two other agencies. The Atomic Energy Acts of 1946 and 1954 regulated the possession, use, and production of the following:

Source materials—Uranium and thorium, and their ores containing $\geq$0.05% U or Th,
Special nuclear materials (SNM)—Plutonium, ^{233}U, and uranium enriched in either ^{233}U or ^{235}U,
By-product material—Originally defined by the USAEC as "any material, except SNM, produced or made radioactive incident to making or using SNM." The Energy Policy Act of 2005 expanded the definition of "by-product material" to include certain discrete sources of radium, certain accelerator-produced radioactive material, and certain discrete sources of *n*aturally *o*ccurring *r*adioactive *m*aterial (NORM), and other radioactive material that the AEC's successor, the NRC, determines could pose a threat to public health and safety or the common defense and security.

Previously, these materials, as well as U or Th in concentrations $<$0.05% and radioisotopes produced by accelerators that were not on government contracts, had been regulated by the states.

The AEC exercised its regulatory authority through the issuance of radiation safety standards and regulations, the licensing of applicants who wished to use any of the materials

that the AEC was authorized to regulate, a system of inspection to verify that a licensee was in fact complying with the radiation safety regulations, and a system of penalties and fines for those licensees who were found not in compliance with the regulations. The AEC's regulations were published in 10 CFR 20, and contained the standards for safe use of radiation sources. These standards, which were designed to protect radiation workers, included a dose limit called the maximum permissible dose, and maximum permissible concentrations (MPCs) of radionuclides in air and water within the context of occupational exposure. MPCs in the gaseous and aqueous emissions to the environment from licensed facilities were also published in 10 CFR 20. The permissible concentrations in the emissions were much lower than those for occupational exposure, since the emitted radionuclides could now expose the general population. The published maximum permissible doses and maximum permissible environmental concentrations were *upper limits only*. In all instances, planning for radiation protection was and still is based on radiation doses that are ALARA. Furthermore, the tabulated MPCs for radionuclides emitted from the licensed facility notwithstanding, radiation safety in any particular case was required to be based on the most sensitive segment of the exposed population and on the environmental pathway that would lead to the greatest dose to the critical population group. For example, in the case of atmospheric ^{131}I in a region where dairy cattle graze, the critical population group is the milk-drinking infant population, and the critical exposure pathway is air to grass to cow to milk to infant. These considerations lead to a reduction of the tabulated occupational MPC of ^{131}I by a factor of 700.

Environmental Protection Agency

There are numerous other sources of radiation—such as medical and industrial X-ray machines; NORMs, which include uranium and thorium and their progeny; and accelerator-produced radionuclides. The acronyms NORM and NARM (natural and accelerator-produced radioactive materials) are frequently applied to these radiation sources. These radiation sources might be injurious to health but nevertheless were not regulated under the Atomic Energy Acts of 1946 and 1954. Accordingly, the Federal Radiation Council (FRC) was formed in 1959 to provide a uniform federal policy on human exposure to radiation. The FRC was charged to "Advise the President with respect to radiation matters directly or indirectly affecting health, including guidance for all federal agencies in the formulation of radiation standards and in the establishment and execution of programs of cooperation with states . . . ." In 1970, under the terms of the Energy Reorganization Act, the FRC was abolished and its functions were transferred to the newly established U.S. EPA. In addition to its other environmental protection responsibilities, the EPA was charged with the task of setting radiation safety policy and basic standards. To accomplish these tasks, the EPA submits *Radiation Protection Guides* to the president of the United States. If the president approves, these guides then become legally binding, and all the federal regulatory agencies that deal with radiation must issue regulations that are compatible with those in the guides. Promulgation of radiation safety regulations is the responsibility of the several regulatory agencies, including the EPA itself, which regulates radioactive discharges into the atmosphere and into waters, establishes drinking water standards, and regulates recovery and disposal of radioactive wastes not regulated under the Atomic Energy Act. EPA regulations are published in Title 40, CFR (40 CFR 9, 141, 142). The EPA limits for radioactivity in drinking water are listed in Table 8-27.

TABLE 8-27 U.S. Environmental Protection Agency Radioactivity Limits for Drinking Water

RADIONUCLIDE	CONCENTRATION LIMIT
Gross alpha, excluding Rn and U	15 pCi/L
Beta–gamma emitters	4 mrems/yr
Combined $^{226}Ra + ^{228}Ra$	5 pCi/L
Tritium	20,000 dpm/L
^{90}Sr	8 pCi/L
Uranium, natural	30 μg/L

Nuclear Regulatory Commission

According to the Atomic Energy Act, the AEC had two responsibilities. One was to develop nuclear energy and useful applications for by-product materials. The second was to regulate these activities so that they were carried out safely. Thus, a single agency was charged with the duty to develop and promote nuclear energy and also to regulate its safe use. Many persons in policymaking positions thought that these two responsibilities were mutually incompatible and that there was an inherent conflict of interest in carrying them out. To remedy this situation, the AEC was abolished in 1974 under the authority of the Energy Reorganization of 1974, and two new agencies were established in its place. The development and promotion of nuclear energy was assigned to the Energy Research and Development Administration (ERDA), which later became the Department of Energy (DOE). Responsibility for radiation safety in the use of source material, special nuclear material, and by-product material was assigned to the Nuclear Regulatory Commission (NRC). The NRC's regulations are published in 10 CFR. The standards for protection against radiation are published in Part 20 of these regulations, 10 CFR 20. The DOE regulations are published in 10 CFR 835. A basic tenet of these safety regulations is that the licensee must maintain strict control over all licensed sources at all times.

Dose Limits

A dose limit is the upper permissible bound for radiation dose; it is a dose level that may not be exceeded. From 1957 until 1991, the AEC and then NRC radiation safety regulations published in 10 CFR 20 were based on ICRP 2 recommendations for radiation workers. To keep up with scientific and engineering advances, the original regulations were amended numerous times. The publication of ICRP 26 in 1977 and ICRP 30 in 1979, which are based on "uniform risk" concept for fatal radiogenic cancers and for serious hereditary effects rather than the "critical organ" concept of ICRP 2, led to dose limits and calculational methodologies that differred significantly from those used in 10 CFR 20. These changes in the philosophical basis for dose limitation and in calculational methodology led to a need for revisions in derived limits, such as the ALI (which was only implied in the former 10 CFR 20), and in secondary limits, such as the DAC and effluent concentrations. Furthermore, in 1987, the EPA based its guidance on ICRP 26 and 30 recommendations. Since the NRC is required to comply with the EPA's guidance, the NRC revised 10 CFR 20 to make it compatible with the EPA's guidance. In this revision, however, the use of the traditional radiation units (rads, rems, and curies) was retained. The dose limits for radiation workers and for members of the general public that are listed in the revised Part 20 are summarized in Table 8-28. It is essential to understand that when setting dose limits, the NRC assumes that licensees will

TABLE 8-28 10 CFR 20 Annual Occupational Dose Limits

DOSE LIMIT TO	DOSE LIMIT
Whole body	5 rems effective dose
Lens of the eye	15 rems
Any other organ or tissue	50 rems
Limbs below elbow or knee	50 rems
Skin, averaged over 10 cm^2	50 rems
Minors	0.1 adult dose
Conceptus	0.5 rem
Members of the general public	0.1 rem

design routine operations so that workers will receive substantially smaller doses than the limit. Limits were to be approached only under unusual circumstances, and that only a small fraction of the exposed population would approach this limit.

Regulatory limits of the NRC generally do not apply to medical radiation exposure. However, if X-ray tests are included in a physical examination required by an employer as a condition of employment, then the X-ray dose is considered as occupational exposure. Chest X-rays are almost always included in these physical examinations. The effective dose from such medical radiation doses must be included in the worker's occupational dose history.

Dose Constraints

A constraint is a level below the maximum limit, which may be exceeded only under certain conditions. For example, a constraint on airborne emissions [10 CFR 20.1101(d)] requires that emissions be so limited that the dose to a member of the public from the emissions be $\leq$10 mrems in a year. However, when it is exceeded, the NRC requires the licensee to take certain actions, including appropriate timely corrective actions and a report to the NRC.

Agreement States

Under the terms of the Atomic Energy Act of 1954, the NRC may transfer to approved states the authority to license and regulate uranium, thorium, and certain quantities of special nuclear material. To be approved by the NRC, a state must agree to promulgate and to enforce radiation safety standards that are at least as rigorous as the NRC's standards, and must also have the resources and the legal authority to exercise these responsibilities. The NRC evaluates the technical licensing and inspection of the agreement states; it also conducts training courses and workshops, involves the agreement states in NRC rulemaking and other regulatory efforts, and coordinates with agreement states in the reporting of events and responses to allegations reported to the NRC involving agreement states.

Kentucky became the first agreement state in 1962. As of 2016, there are 37 agreement states.

Computational Methodology

ICRP 30 Methodology

The NRC uses ICRP 30 methodology to calculate radiation dose, ALIs, and DACs for internal emitters. However, the NRC chose to use the traditional radiation units rather than the

SI units. The appropriate ALI (either inhalation or ingestion, and either stochastic or non-stochastic) is calculated by the U.S. NRC from

$$\text{ALI, } \mu\text{Ci/yr} = \frac{\text{dose limit, } \dfrac{\text{rems}}{\text{yr}}}{\text{DCF, } \dfrac{\text{rems}}{\mu\text{Ci}}}. \tag{8.53}$$

The DCF expressed in traditional units of rems/μCi is related to the DC expressed in SI units of Sv/Bq by the following:

$$\begin{aligned}\text{DCF rems/}\mu\text{Ci} &= \text{DC}\,\frac{\text{Sv}}{\text{Bq}} \cdot 3.7\times10^{4}\,\frac{\text{Bq}}{\mu\text{Ci}} \cdot 100\,\frac{\text{rems}}{\text{Sv}} \\ &= 3.7\times10^{6}\times\text{DC}\,\frac{\text{Sv}}{\text{Bq}}.\end{aligned} \tag{8.54}$$

Thus, for ^{137}Cs, for inhalation of class D particles, we have, using the SI value for the DC from Table 8-20 in Eq. (8.54):

$$\text{DCF} = 3.7\times10^{6}\cdot 8.63\times10^{-9}\,\frac{\text{Sv}}{\text{Bq}} = 3.2\times10^{-2}\,\frac{\text{rems}}{\mu\text{Ci}}.$$

The SALI for inhaled class D ^{137}Cs particles is calculated with Eq. (8.53):

$$\text{SALI(inhalation)} = \frac{5\,\dfrac{\text{rems}}{\text{yr}}}{3.2\times10^{-2}\,\dfrac{\text{rems}}{\mu\text{Ci}}} = 1.56\times10^{2}\,\frac{\mu\text{Ci}}{\text{yr}}.$$

Since the values published in 10 CFR 20 are rounded off to one significant figure, the SALI for inhaled class D ^{137}Cs aerosol is listed as 2×10^2 μCi/yr.

The occupational DAC in traditional units (using a breathing rate of 1.2 m^3/h) is calculated from

$$\text{DAC, } \mu\text{Ci/mL} = \frac{\text{ALI(unrounded), } \dfrac{\mu\text{Ci}}{\text{yr}}}{2\times10^{3}\,\dfrac{\text{h}}{\text{yr}} \cdot 1.2\,\dfrac{\text{m}^3}{\text{h}} \cdot 10^{6}\,\dfrac{\text{mL}}{\text{m}^3}}. \tag{8.55}$$

For ^{137}Cs class D aerosols, Eq. (8.55) gives the calculated DAC as

$$\text{DAC} = \frac{1.57\times10^{2}\,\dfrac{\mu\text{Ci}}{\text{yr}}}{2\times10^{3}\,\dfrac{\text{h}}{\text{yr}} \cdot 1.2\,\dfrac{\text{m}^3}{\text{h}} \cdot 10^{6}\,\dfrac{\text{mL}}{\text{m}^3}} = 6\times10^{-8}\,\frac{\mu\text{Ci}}{\text{mL}},$$

when rounded to one significant figure. It should be noted that the rounding to one significant figure is for listing the final value only, not for carrying out the successive calculations. If the numbers rounded to one significant figure were to be used successively, rounding errors would accumulate and might lead to erroneous final results.

Particle Size and DAC

Safety standards for radioactive aerosols that are listed in 10 CFR 20 are based on ICRP 30 criteria for occupational inhalation. That is, they are for particles of 1-μm AMAD and $\sigma_g \leq 4.5$. For particles whose AMAD differs from 1 μm, adjustment of the published DAC may be made according to the following relationship, given in ICRP 30, between the dose from the *i*th particle size and the dose from 1-μm particles:

$$\frac{H_{50}(i)}{H_{50}(1\ \mu\text{m})} = f_{NP}\frac{D_{NP}(i)}{D_{NP}(1\ \mu\text{m})} + f_{TB}\frac{D_{TB}(i)}{D_{TB}(1\ \mu\text{m})} + f_P\frac{D_P(i)}{D_P(1\ \mu\text{m})}, \tag{8.56}$$

where

H_{50} = committed dose equivalents from the 1-μm and *i*th-μm AMAD particles;

f_{NP}, f_{TB}, and f_P = fractions of the committed dose equivalent due to deposition in the NP, TB, and P respiratory compartments. These values are listed in the supplements to Parts 1 and 2 of ICRP 30. Values can also be found in Table 8.7 for 1-μm particles (use Figure 8.8 for other sizes); and

D_{NP}, D_{TB}, and D_P = deposition fractions in the respective respiratory compartments for a given particle size.

EXAMPLE 8-8

The AMAD of UO_2 particles in one of the production departments of a uranium-processing facility was found to be 9.6 μm. The DAC given in 10 CFR 20 for class Y (UO_2 is a class Y compound) for 1-μm AMAD particles is 2×10^{-11} μCi/mL (and 7×10^{-1} Bq/m^3 in ICRP 30). What is the DAC corrected for the particle size?

Solution

We will calculate the size-corrected DAC with the aid of Eq. (8.56). The regional deposition probabilities for the two different size distributions, which are found in Figure 8-8, are listed below:

AMAD	D_{NP}	D_{TB}	D_P
1.0	0.30	0.08	0.25
9.6	0.87	0.08	0.05

The fraction of the committed dose equivalent due to the particles deposited in each of the respiratory compartments for class Y ^{238}U compounds is found in ICRP 30, supplement to Part 1, page 378 (Figure 8.8 may also be used to estimate deposition), to be

$f_{NP} = 0$,
$f_{TB} = 0$, and
$f_P = 1$.

If we insert the respective values into Eq. (8.56), we have

$$\frac{H_{50}(9.6\,\mu m)}{H_{50}(1\,\mu m)} = 0 \cdot \frac{0.87}{0.3} + 0 \cdot \frac{0.08}{0.08} + 1 \cdot \frac{0.05}{0.25} = 0.2.$$

Since the committed dose from the 9.6-mm particle is only 20% of that from 1-mm particles, the recommended DAC may be increased by as much as a factor of 5.

Effluents Released into the Environment

Since the dose limit for members of the general public is much lower than for radiation workers, radionuclide concentration in air and water that is discharged from NRC licensed facilities must be lower than those applied to occupational exposure. Accordingly, in addition to the secondary limits for occupational exposure that are published in Appendix B, Table 1 of 10 CFR 20, limits on the concentrations of air and water effluents from licensed facilities are published in Table 2 of Appendix B. Monthly average concentrations of radionuclides that are released to sanitary sewers are listed in Table 3 of Appendix B. The concentration values given in Table 2 are equivalent to radionuclide concentrations, which, if inhaled or ingested continuously, would result in a total effective dose equivalent (TEDE) of 0.1 rem to a member of the general public. For those airborne radionuclides whose occupational DAC is limited by submersion (external dose), the occupational DAC is divided by 219 to obtain the limiting atmospheric concentration before release to the public environment. The number 219 includes two factors: (1) a factor of 50 that relates the occupational dose limit of 5 rems/yr to the limit of 0.1 rems/yr to a member of the public and (2) a factor of 4.38 that relates the total exposure time of 8760 h/yr to the occupational exposure time of 2000 h/yr. Thus, for ^{41}Ar, whose occupational DAC is 3×10^{-6} μCi/mL, the effluent concentration is

$$\text{effluent conc. (air, submer.)} = \frac{\text{DAC}, \frac{\mu Ci}{mL}}{219}; \tag{8.57}$$

substituting the given values, we obtain

$$\text{effluent conc. } (^{41}\text{Ar}) = \frac{3 \times 10^{-6} \frac{\mu Ci}{mL}}{219} = 1 \times 10^{-8} \frac{\mu Ci}{mL}.$$

To calculate the effluent concentrations for those airborne nuclides that are limited by the internal dose and consequently have an ALI, the inhalation ALI is reduced by several factors. A factor of 1/50 relates the 5-rem occupational dose limit to the 0.1-rem limit for the general public, a factor of 1/3 to account for the difference in exposure time and inhalation rate between a worker and a member of the general public, and finally a factor of 1/2 to account for the age difference between workers and the general public. This reduced ALI is divided by the air inhaled by a worker during a 2000-hour working year:

$$\begin{aligned}\text{Effluent conc. (air, inhal.)} &= \frac{\frac{1}{50}\cdot\frac{1}{3}\cdot\frac{1}{2}\times\text{inhalation ALI}, \frac{\mu\text{Ci}}{\text{yr}}}{2.4\times10^{9}\ \frac{\text{mL}}{\text{yr}}} \\ &= \frac{\text{inhalation ALI}, \frac{\mu\text{Ci}}{\text{yr}}}{300\cdot\left(2.4\times10^{9}\ \frac{\text{mL}}{\text{yr}}\right)} \\ &= \frac{\text{inhalation ALI}, \frac{\mu\text{Ci}}{\text{yr}}}{7.2\times10^{11}\ \frac{\text{mL}}{\text{yr}}}. \end{aligned} \tag{8.58}$$

For ^{137}Cs, whose unrounded occupational ALI is 156 μCi, the effluent air concentration listed in Table 2 of Appendix B is calculated as

$$\text{Effluent air conc. } (^{137}\text{Cs}) = \frac{156\ \mu\text{Ci}}{7.2\times10^{11}\,\text{mL}} = 2\times10^{-10}\ \frac{\mu\text{Ci}}{\text{mL}}.$$

The concentration limits in 10 CFR 20 for radionuclides in liquid effluents discharged into waterways are based on two considerations:

1. The contaminated water will be the sole source of potable water for members of the general public.
2. The limiting annual dose through this exposure pathway is 0.1 rem. The occupational ALI for ingestion was therefore reduced by a factor of 50 to account for the difference between the occupational dose limit and the general public dose limit, and by a factor of 2 to account for the age difference between the working and general populations. Since the annual water intake by a reference person is 7.3×10^5 mL, the activity concentration of liquid effluents is calculated from

$$\begin{aligned}\text{Effluent conc. (water)} &= \frac{\frac{1}{50}\cdot\frac{1}{2}\cdot\text{ingestion ALI}, \frac{\mu\text{Ci}}{\text{yr}}}{7.3\times10^{5}\ \frac{\text{mL}}{\text{yr}}} \\ &= \frac{\text{ingestion ALI}\ \frac{\mu\text{Ci}}{\text{yr}}}{7.3\times10^{7}}. \end{aligned} \tag{8.59}$$

For the case of ^{137}Cs, for example, Table 1 of 10 CFR 20 says that the ingestion ALI = 100 μCi. The listing in Table 2 for the effluent concentration in water is obtained as follows:

$$\text{effluent conc. (water)} = \frac{\text{ingestion ALI}}{7.3\times10^{7}} = \frac{100\ \frac{\mu\text{Ci}}{\text{yr}}}{7.3\times10^{7}\ \frac{\text{mL}}{\text{yr}}}$$

$$= 1.4\times10^{-6}\ \frac{\mu\text{Ci}}{\text{mL}}.$$

Since the table's values are given to one significant figure, the effluent concentration limit is listed as 1×10^{-6} μCi/mL.

Dose Tracking

Part 20 of 10 CFR specifies that the 5-rem EDE limit includes the sum of the external dose and the dose from internally deposited radionuclides. This means that

$$\left[\frac{\text{external dose}}{5\ \text{rems}}\right] + \left[\frac{\text{intake}}{\text{ALI}}\right]_{\text{ingestion}} + \left[\frac{\text{intake}}{\text{ALI}}\right]_{\text{inhalation}} \leq 1. \tag{8.60}$$

For *regulatory purposes,* in order to demonstrate compliance with the regulations, personal dosimeter measurements are used for tracking external doses, and either environmental sampling, in vitro bioassay, or whole-body counting (in vivo bioassay) methods may be used for internal dose tracking. Environmental sampling may be used for the determination of

- intakes and comparison with ALIs,
- exposure to airborne radionuclides and comparison with DAC-hour limits, and
- CEDE and comparison with dose limits.

EXAMPLE 8-9

A worker wears a personal lapel sampler for an entire 8-hour shift to monitor 1-μm AMAD ^{60}Co particles. The sampler draws 2 L/m, and the measured activity on the filter is 10,000 dpm. The ALI for class Y ^{60}Co particles is 30 μCi and the DAC is 1×10^{-8} μCi/mL. Calculate the worker's

(a) intake,
(b) exposure, DAC-hours, and
(c) CEDE.