

Table 21-2. STOPPING POWER, RANGE, AND RADIATION YIELD FOR ELECTRONS IN MUSCLE TISSUE

ENERGY (MeV)	COLLISION (MeV cm <sup>2</sup> g <sup>-1</sup> )	STOPPING POWER RADIATIVE (MeV cm <sup>2</sup> g <sup>-1</sup> )	TOTAL (MeV cm <sup>2</sup> g <sup>-1</sup> )	CSDA RANGE (g cm <sup>-2</sup> )	RADIATION YIELD
0.0100	2.231E+01	3.835E-03	2.231E+01	2.543E-04	9.366E-05
0.0125	1.876E+01	3.863E-03	1.877E+01	3.771E-04	1.127E-04
0.0150	1.628E+01	3.880E-03	1.629E+01	5.205E-04	1.310E-04
0.0175	1.445E+01	3.892E-03	1.445E+01	6.838E-04	1.485E-04
0.0200	1.303E+01	3.901E-03	1.303E+01	8.662E-04	1.655E-04
0.0250	1.097E+01	3.913E-03	1.098E+01	1.286E-03	1.980E-04
0.0300	9.547E+00	3.924E-03	9.551E+00	1.776E-03	2.290E-04
0.0350	8.498E+00	3.934E-03	8.502E+00	2.332E-03	2.587E-04
0.0400	7.692E+00	3.946E-03	7.696E+00	2.951E-03	2.874E-04
0.0450	7.052E+00	3.959E-03	7.056E+00	3.631E-03	3.151E-04
0.0500	6.531E+00	3.973E-03	6.535E+00	4.368E-03	3.421E-04
0.0550	6.099E+00	3.988E-03	6.102E+00	5.160E-03	3.683E-04
0.0600	5.733E+00	4.004E-03	5.737E+00	6.006E-03	3.939E-04
0.0700	5.151E+00	4.040E-03	5.155E+00	7.848E-03	4.435E-04
0.0800	4.706E+00	4.079E-03	4.710E+00	9.881E-03	4.912E-04
0.0900	4.355E+00	4.122E-03	4.359E+00	1.209E-02	5.373E-04
0.1000	4.071E+00	4.168E-03	4.075E+00	1.447E-02	5.821E-04
0.1250	3.552E+00	4.294E-03	3.557E+00	2.106E-02	6.889E-04
0.1500	3.203E+00	4.431E-03	3.207E+00	2.848E-02	7.899E-04
0.1750	2.951E+00	4.579E-03	2.956E+00	3.662E-02	8.865E-04
0.2000	2.763E+00	4.734E-03	2.768E+00	4.537E-02	9.795E-04
0.2500	2.501E+00	5.070E-03	2.506E+00	6.442E-02	1.157E-03
0.3000	2.329E+00	5.438E-03	2.335E+00	8.513E-02	1.327E-03
0.3500	2.211E+00	5.832E-03	2.216E+00	1.071E-01	1.492E-03
0.4000	2.125E+00	6.252E-03	2.131E+00	1.302E-01	1.653E-03
0.4500	2.061E+00	6.694E-03	2.068E+00	1.540E-01	1.812E-03
0.5000	2.012E+00	7.158E-03	2.019E+00	1.785E-01	1.970E-03
0.5500	1.972E+00	7.642E-03	1.980E+00	2.035E-01	2.128E-03
0.6000	1.941E+00	8.141E-03	1.949E+00	2.290E-01	2.285E-03
0.7000	1.895E+00	9.186E-03	1.904E+00	2.809E-01	2.602E-03
0.8000	1.863E+00	1.028E-02	1.874E+00	3.339E-01	2.921E-03
0.9000	1.842E+00	1.143E-02	1.853E+00	3.876E-01	3.244E-03
1.0000	1.827E+00	1.262E-02	1.839E+00	4.418E-01	3.571E-03
1.2500	1.806E+00	1.578E-02	1.822E+00	5.784E-01	4.408E-03
1.5000	1.799E+00	1.916E-02	1.818E+00	7.158E-01	5.272E-03
1.7500	1.799E+00	2.271E-02	1.821E+00	8.532E-01	6.162E-03
2.0000	1.801E+00	2.642E-02	1.828E+00	9.903E-01	7.074E-03
2.5000	1.812E+00	3.421E-02	1.846E+00	1.263E+00	8.956E-03
3.0000	1.824E+00	4.241E-02	1.866E+00	1.532E+00	1.090E-02
3.5000	1.836E+00	5.095E-02	1.887E+00	1.798E+00	1.289E-02

From ICRU, 1984.

 $I = 75.3 \text{ eV}$ ; Density =  $1.040E+00 \text{ g/cm}^3$  $\Delta E$  = energy loss in the medium per unit distance (in MeV cm<sup>2</sup> g<sup>-1</sup>) $\mu_{en}/\rho$  = mass energy absorption coefficient (cm<sup>2</sup> g<sup>-1</sup>) $E_0$  = initial photon energy**ABSORBED DOSE***Dose and Dose Rate*

Absorbed dose is defined as the mean energy,  $e$ , imparted by ionizing radiation to matter of mass  $m$  (ICRU 1980)

$$D = e/m$$

(11)

where

Tables 21-3 and 21-4 give the attenuation coefficients for photons in air and the mass energy absorption coefficients for photons in air and in muscle tissue. Both tables are reproduced from Hubbell (1969).

**Table 21-4. MASS ENERGY ABSORPTION COEFFICIENTS FOR AIR AND WATER**

PHOTON ENERGY (MeV)	AIR $\mu_{en}/\rho$ ( $m^2 kg^{-1}$ )	MUSCLE, STRIATED (ICRU) $\mu_{en}/\rho$ ( $m^2 kg^{-1}$ )
0.01	0.46	0.49
0.015	0.13	0.14
0.02	0.052	0.055
0.03	0.015	0.016
0.04	0.0067	0.0070
0.05	0.0040	0.0043
0.06	0.0030	0.0032
0.08	0.0024	0.0026
0.10	0.0023	0.0025
0.15	0.0025	0.0027
0.20	0.0027	0.0029
0.30	0.0029	0.0032
0.40	0.0029	0.0032
0.50	0.0030	0.0033
0.60	0.0030	0.0033
0.80	0.0029	0.0032
1.00	0.0028	0.0031
1.50	0.0025	0.0028
2.00	0.0023	0.0026
3.00	0.0021	0.0023

From Hubbell, 1982.

$D$  = absorbed dose

$e$  = mean energy deposited in mass

$m$  = mass.

The unit for absorbed dose is the Gray (Gy) and is equal to  $1 J kg^{-1}$ . The older unit of dose is the rad and is equal to  $100 erg g^{-1}$ . The conversion for these units is  $100 rad = 1 Gy$ .

For uncharged particles (gamma rays and neutrons), kerma is sometimes used. It is the sum of the initial kinetic energies of all the charged ionizing particles liberated in unit mass. The units of kerma are the same as for dose.

Exposure is often confused with absorbed dose. Exposure is defined only in air for gamma rays or photons and is the charge of the ions of one sign when all electrons liberated by photons are completely stopped in air of mass  $m$ .

$$X = Q/m \quad (12)$$

where

$X$  = exposure

$Q$  = total charge of one sign

$m$  = mass of air.

The unit of exposure is the Coulomb per kilogram of air. The older unit of exposure is the Roentgen which is equal to  $2.58 \times 10^{-4} C kg^{-1}$  of air.

Exposure and dose are used interchangeably in some publications even though this is not correct. The reason is that the older numerical values of dose in rad and exposure in Roentgen are similar. Although they are similar numerically they are fundamentally different in that exposure is ionization (only in air) and dose is absorbed energy in any specified medium.

$$1 \text{ Roentgen} = 0.87 \text{ rad (in air)}$$

The SI units are not numerically similar,

$$1 C kg^{-1} = 33.85 Gy$$

Dose rate is the dose expressed per unit time interval. The dose rate delivered to the thyroid by  $^{99m}Tc$  for a nuclear medicine scan, for example, is diminishing with time due to the 6.0-hour half-life of the nuclide. The total dose is a more pertinent quantity in this case because it can be related directly to risk and compared with the benefit of the thyroid scan.

The dose rate from natural body  $^{40}K$  in all cells, on the other hand, is relatively constant throughout life and is usually expressed as the annual dose rate.

#### *Dose Equivalent*

The linear energy transfer (LET) from alpha and beta particles is much greater than for gam-

ma rays. In considering the health or cellular effects of each particle or ray, it is convenient to normalize the various types of radiation. For a particular biologic end point, such as cell death in an experiment with mouse fibroblasts, it is common to calculate a relative biologic effectiveness (RBE). This is defined as the ratio of the gamma dose to the dose from radiation under study which yields the same end point.

Such refinement in the normalization of end points (cancer) in the human is not possible with the available data. An attempt to normalize human health effects is made through the values for linear energy transfer of the various types of radiation in water. The ratio of the LET for gamma to the radiation in question is defined as a quality factor,  $Q$ , and the normalized dose is called the dose equivalent. The unit for the dose equivalent is the Sievert and the older unit the rem.

$$H = D Q \quad (13)$$

where

$H$  = dose equivalent in Sievert (older unit rem)

$D$  = dose in Gray (older unit rad)

$Q$  = quality factor.

Table 21-5 is reproduced from NCRP (1987).

**Example 3.** Find the dose equivalent (in Sievert) for a dose to lung from an internal emitter of 0.01-Gy alpha particles and 0.01 Gy from external gamma-ray radiation.

alpha  $H = 0.01 (20) = 0.20$  Sv

gamma  $H = 0.01 (1) = 0.01$  Sv

#### Effective Dose Equivalent and Cancer Risk

The term effective dose equivalent (EDE) was introduced formally by ICRP in 1977 to be able to add or directly compare the cancer and genetic risk from different partial body or whole body doses. A partial body dose to the lung, for ex-

ample, was thought to give 0.002 cancers over a lifetime per Sievert whereas a whole body dose would result in 0.0165 total cancers and early genetic effects over the same lifetime interval. The ratio 0.002/0.016 was defined as a weighting factor,  $w_t$ , for lung and is numerically equal to 0.12.

The effective dose equivalent (EDE),  $H_E$ , is defined as

$$H_E = w_t D Q \quad (14)$$

This concept was useful in the case of occupational exposure because EDE values from different sources can be simply summed to yield a direct estimate of total cancer and genetic risk.

Table 21-6 is taken from NCRP (1987) and gives the values of  $w_t$  for various organs.

The occupational guideline for EDE is 50 mSv per annum (NCRP, 1987; ICRP, 1977). This requires that the sum of all EDE be less than or equal to this value, namely,

$$H_E = \sum w_t H \leq 50 \text{ mSv} \quad (15)$$

#### Committed Dose Equivalent

A problem arises with internal emitters in that once ingested there is an irreversible dose that is committed because of the biokinetics of the particular element. The absorbed dose depends on the biologic and physical half-times of the element in the body. For this reason the concepts of committed dose equivalent and committed effective dose equivalent were derived to accommodate the potential for dose to be delivered over long times after incorporation in the body. The committed dose is taken over a 50-year interval after exposure and is equal to

**Table 21-6. RECOMMENDED VALUES OF THE WEIGHTING FACTORS,  $w_t$ , FOR CALCULATING EFFECTIVE DOSE EQUIVALENT AND THE RISK COEFFICIENTS FROM WHICH THEY WERE DERIVED**

TISSUE	RISK COEFFICIENT (Sv <sup>-1</sup> )	$w_t$
Gonads	0.0040	0.25
Breast	0.0025	0.15
Red bone marrow	0.0020	0.12
Lung	0.0020	0.12
Thyroid	0.0005	0.03
Bone surfaces	0.0005	0.03
Remainder	0.0050	0.30
Total	0.0165	1.0

Values from ICRP, 1977.

**Table 21-5. RECOMMENDED VALUES OF  $Q$  FOR VARIOUS TYPES OF RADIATION**

TYPE OF RADIATION	APPROXIMATE $Q$
X rays, gamma rays, beta particles and electrons	1
Thermal neutrons	5
Neutrons (other than thermal), protons, alpha particles, charged particles of unknown energy	20

NCRP, 1987.

$$H_{T,50} = \int_{t_0}^{t_0+50} H_T dt \quad (16)$$

where

$H_{T,50}$  = the 50-year dose to tissue  $T$ , for a single intake at time  $t_0$

$H_T$  = is the dose equivalent rate in organ or tissue  $T$  at time  $t$ .

NCRP (1987) recognizes that for radionuclides with half-lives ranging up to about three months the committed dose equivalent is equal to the annual dose for the year of intake. For longer lived nuclides the committed dose equivalent will be greater than the annual dose equivalent and must be calculated on an individual basis. ICRP Publication 30 (ICRP, 1978) provides the details of this calculation for all nuclides.

### Negligible Individual Risk Level

The current radiobiologic principle commonly accepted is that of linear, nonthreshold cancer induction from ionizing radiation. Thus, regardless of the magnitude of the dose a numerical cancer risk can be calculated. For this reason the National Council on Radiation Protection and Measurements proposed the Negligible Individual Risk Level (NIRL) and defined it as

a level of annual excess risk of fatal health effects attributable to irradiation below which further effort to reduce radiation exposure to the individual is unwarranted.

NCRP emphasized that the NIRL is not to be confused with an acceptable risk level, a level of significance, or a limit.

The NCRP recommended an annual effective dose equivalent limit for continuous exposure of members of the public of 1 mSv (0.1 rem). This value is in addition to that received from natural background radiation (about 2 mSv). In this context the NIRL was taken to be 0.01 mSv (1 mrem).

### HUMAN STUDIES OF RADIATION TOXICITY

There are five major studies of the health detriment resulting from exposure of humans to ionizing radiation. Other studies of large worker populations exposed to very low levels of radiation and environmental populations exposed to radon are ongoing but these are not expected to provide new data on the risk estimates from ionizing radiation. These latter worker or environmental populations are studied to ensure

that there is no inconsistency in the radiation risk data in extrapolating from the higher exposures.

The basic studies on which the quantitative risk calculations are founded include the radium exposures, the atom bomb survivors, the underground miners exposed to radon, patients irradiated with X rays for ankylosing spondylitis, and children irradiated with X rays for tinea capitis (ringworm).

### Radium Exposures ( $^{226,228}\text{Ra}$ )

Radium was discovered in the early part of the twentieth century. Its unique properties suggested a potential for the healing arts. It was incorporated into a wide variety of nostrums, medicines, and artifacts. The highest exposure occurred in the United States in the radium dial painters who ingested from 10s to 1000s of micrograms (microcuries). These exposed groups including patients, chemists, dial painters, and so forth have been studied for over 60 years to determine the body retention of radium and the health effects of long-term body burdens.

The only late effect of ingestion of  $^{226,228}\text{Ra}$  seen is osteogenic sarcoma. It is significant that no study has ever identified a statistically significant excess of leukemia following even massive doses of radium. This implies that the target cells for leukemia residing in bone marrow are outside the short range of the radium series alpha particles (70  $\mu\text{m}$ ).

Several thousand people were exposed to radium salts either as part of the modish therapies using radium in the era from 1900 to 1930 or occupationally in the radium dial painting industry around 1920. Radium therapy was accepted by the American Medical Association and around 1915 advertisements were common for radium treatment of rheumatism and as a general tonic and in the treatment of mental disorders. Solutions were available for drinking containing 2  $\mu\text{g}/60 \text{ cm}^3$  as well as ampoules for intravenous injection containing 5 to 100  $\mu\text{g}$  radium (Woodard, 1980). Luminous paint was developed before World War I and in 1917 there were many plants in New England and New Jersey painting watch dials, clocks, and military instruments (Woodard, 1980).

The first large studies on osteogenic sarcoma in radium-exposed people were done by Martland (1931) and Aub *et al.* (1952), who found 30 cases of bone sarcoma; Evans (1969) with 496 cases of sarcoma out of 1064 studied at the Massachusetts Institute of Technology; and Rowland *et al.* (1978), 61 cases out of 1474 female dial painters (Woodard 1980).

Radium, once ingested, is somewhat similar to calcium in its metabolism and is incorporated on bone surfaces into the mineralized portion of

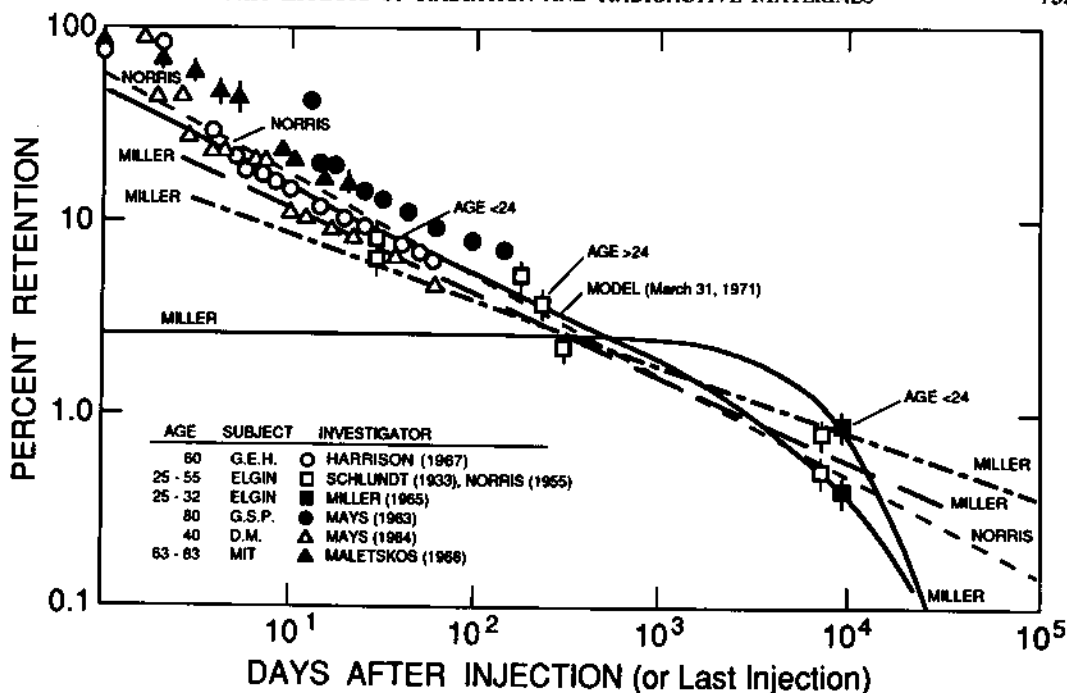


Figure 21-1. Whole body radium retention in humans. Summary of all available data for adult man. (From Marshall *et al.*, 1972.)

bone. The long half-life of  $^{226}\text{Ra}$  allows distribution throughout the mineral skeleton over life. The target cells for osteogenic sarcoma reside in marrow on endosteal surfaces at about  $10\ \mu\text{m}$  from the bone surface. At long times post exposure, target cells are beyond the range of alpha particles from radium not on bone surfaces.

The loss of radium from the body by excretion was determined to follow a relatively simple power function (Norris, 1955).

$$R = 0.54 t^{-0.52} \quad (17)$$

where

$R$  = total body retention  
 $t$  = time in days.

Other models to fit the data were developed as more information became available, the most recent being that of Marshall *et al.* (1972). The entire body of radium data and the various models are shown in Figure 21-1. It can be seen that the Norris function fits the observed data well except at very long times post exposure. A simplified form of the more complex later model of Marshall *et al.* (1972) which fits the human data over all observed times is

$$R = 0.8t^{-0.5} (0.5 e^{-\lambda t} + 0.5 e^{-4\lambda t}) \quad (18)$$

where

$R$  = whole body retention  
 $\lambda$  = rate of bone apposition or resorption  
 $= 0.0001\ \text{day}^{-1}$   
 $t$  = time in days.

For most purposes the Norris formula is applicable. It can be seen from Figure 21-1 for the Norris equation that, even one year after exposure, only about 2 percent of the radium is retained in the body but after 30 years about 0.5 percent still remains.

The risk of osteogenic bone cancer following radium exposure has been summarized in the National Academy of Sciences Report BEIR IV (NAS, 1988).

Equations were proposed by Rowland *et al.* (1978) for the annual risk of sarcoma (including the natural risk) expressed as a function of either radium intake or dose from  $^{226,228}\text{Ra}$ . Risk per unit intake:

$$I = [0.7 \times 10^{-5} + (7 \times 10^{-9})D^2] \exp[-(1.1 \times 10^{-3})D] \quad (19)$$

where

$I$  = total bone sarcomas per person year at risk