

# Chapter 21

## TOXIC EFFECTS OF RADIATION AND RADIOACTIVE MATERIALS

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### INTRODUCTION

Ionizing radiation, of all branches of toxicology, provides the most quantitative estimates of health detriments for humans. There are five large studies that provide data on the health effects of radiation on people. These include external X- and gamma-ray radiation and internal alpha radioactivity. The studies encompass the radium exposures (including radium dial painters), atom bomb survivors, patients irradiated with X rays for ankylosing spondylitis, children irradiated with X rays for tinea capitis (ringworm), and uranium miners exposed to radon and its short-lived daughter products. The only health effect seen with statistical significance to date, subsequent to radiation exposure, is cancer. The various types and the quantitative risks are described in subsequent sections.

All of the studies provide a consistent picture of the risk of exposure to ionizing radiation. There are sufficient details in the atom bomb, occupational, and medical exposures to estimate the risk from lifelong low-level environmental exposure. Natural background radiation is substantial and only within the past 10 to 15 years has the extent of the radiation insult to the global population from natural radiation and radioactivity been appreciated.

### BASIC RADIATION CONCEPTS

There are four main types of radiation: alpha particles, beta particles (negatively charged) and positrons (positively charged), gamma rays, and X rays. An atom can decay to a product element by loss of a heavy (mass = 4) charged (+2) alpha particle, consisting of two protons and two neutrons. An atom can decay by loss of a negatively or positively charged electron (beta particle or positron). Gamma radiation results when the nucleus releases excess energy, usually after an alpha, beta or positron transition. X rays occur whenever an inner shell orbital electron is

removed and rearrangement of the atomic electrons results with the release of the element's characteristic X-ray energy.

There are several excellent textbooks available that describe the details of radiological physics (Evans, 1955; Andrews, 1974; Turner, 1986).

#### *Energy*

Alpha particles and beta rays (or positrons) have kinetic energy due to their motion. The energy is equal to

$$E = \frac{1}{2} mV^2 \quad (1)$$

where

$m$  = mass of the particle  
 $V$  = velocity of the particle.

Alpha particles have a low velocity compared with the speed of light and calculations of alpha particle energy do not require any corrections for relativity. Most beta particles (or positrons) do have high velocity and the basic expression must be corrected for their increased relativistic mass (the rest mass of the electron is 0.511 MeV). The total energy is equal to

$$E = \frac{0.511}{(1 - v^2/c^2)^{1/2}} + 0.511 \quad (2)$$

where

$v$  = velocity of the beta particle  
 $c$  = speed of light.

Gamma and X rays are pure electromagnetic radiation with energy equal to

$$E = h\nu \quad (3)$$

where

$h$  = Planck's constant ( $6.626 \times 10^{-34}$  J sec)  
 $\nu$  = frequency of the radiation.

The conventional energy units for ionizing radiation are the electron volt (eV) or multiples of this basic unit, million electron volts (MeV), and kiloelectron volts (keV). The conversion to the international system of units (System International or SI) is currently taking place in the United States and the more fundamental energy unit of the Joule (J) is slowly replacing the older unit. The relationship is

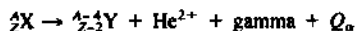
$$1 \text{ eV} = 1.6 \times 10^{-19} \text{ J}$$

Authoritative tables of nuclear data such as those of Lederer *et al.* (1978) and Browne *et al.* (1986) contain the older units.

### Alpha Particles

Alpha particles are helium nuclei (consisting of two protons and two neutrons) with a charge of +2 that are ejected from the nucleus of an atom. When the alpha particle loses energy, slows to the velocity of a gas atom, and acquires two electrons from the vast sea of free electrons present in most media, it becomes part of the normal background helium in the environment.

The formula for alpha decay is



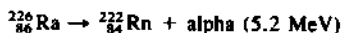
where

Z = atomic number

A = atomic weight.

The energy available in this decay is  $Q_\alpha$  and is equal to the mass difference of the parent and the two products. The energy is shared among the particles and the gamma ray, if one is present.

An example of alpha decay is given by the natural radionuclide  ${}^{226}\text{Ra}$ ,



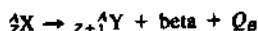
The energy of alpha particles for most emitters lies in the range of 4 to 8 MeV. More energetic alpha particles exist but are seen only in the very short-lived emitters such as those formed by reactions occurring in particle accelerators. These are not considered in this chapter.

Although there may be several alpha particles with very similar energy emitted by a particular element such as radium, each particular alpha is monoenergetic. No continuous spectrum of energies exists but only discrete energies.

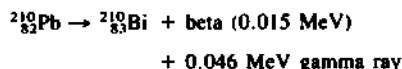
### Beta Particles, Positrons, and Electron Capture

Beta particle decay occurs when a neutron in the nucleus of an element effectively transforms

into a proton and an electron. Subsequent ejection of the electron occurs and the maximum energy of the beta particle equals the mass difference between the parent and the product nuclei. A gamma ray may also be present to share the energy,  $Q_\beta$ .



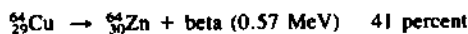
An example of beta decay is given by the natural radionuclide  ${}^{210}\text{Pb}$



Unlike alpha decay, where each alpha particle is monoenergetic, beta particles are emitted with a continuous spectrum of energy from zero to the maximum energy available for the transition. The reason for this is that the total available energy is shared in each decay or transition by two particles, the beta and an antineutrino. The total energy released in each transition is constant but the observed beta particles then appear as a spectrum. The residual energy is carried away by the antineutrino, which is a particle with essentially zero mass and charge and cannot be observed without extraordinarily complex instrumentation. The beta particle, on the other hand, is readily observed with conventional nuclear counting equipment.

Positron emission is similar to beta particle emission, but results from the effective nucleon transformation of a proton to a neutron plus a positively charged electron. The atomic number decreases rather than increases as in beta decay.

An example of positron decay is given by the natural radionuclide  ${}^{64}\text{Cu}$  which decays by beta emission 41 percent of the time, positron emission 19 percent of the time, and electron capture 40 percent of the time.



The energy of the positron appears as a continuous spectrum similar to that in beta decay where the total energy available for decay is again shared between the positron and a neutrino. In the case of positron emission, the maximum energy of the emitted particle is the mass difference of the parent and product nuclide minus the energy needed to create two electron masses (1.02 MeV) whereas the maximum energy of the beta particle is the mass difference itself. This happens because, in beta decay, the increase in the number of orbital electrons due to

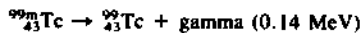
the increase in atomic number of the product nucleus cancels the mass of the electron lost in emitting the beta particle. This does not happen in positron decay and there is an orbital electron lost due to the decrease in atomic number of the product as well as loss of the electron mass in positron emission.

Electron capture competes with positron decay and the resulting product nucleus is the same. In electron capture an orbiting electron is acquired by the nucleus and the transformation of a proton plus the electron to form a neutron takes place. In some cases the energy available is released as a gamma-ray photon but this is not necessary and a monoenergetic neutrino may be emitted. If the 1.02 MeV required for positron decay is not available, then positron decay is not kinetically possible and electron capture will be the only mode observed.

### Gamma-Ray (Photon) Emission

Gamma-ray emission is not a primary process except in rare instances but occurs in combination with alpha, beta, or positron emission or electron capture. Whenever the ejected particle does not utilize all of the available energy for decay, the nucleus contains the excess energy and is in an excited state. The excess energy is released as photon or gamma-ray emission coincident with the ejection of the particle.

One of the rare instances of pure gamma-ray emission is Tc.



In many cases, the photon will not actually be emitted by the nucleus but the excess excitation energy will be transferred to an orbital electron. This electron is then ejected as a monoenergetic particle with energy equal to that of the photon minus the binding energy of the orbital electron. This process is known as internal conversion. In tables of nuclear data such as those of Lederer *et al.* (1978), the ratio of the conversion process to the photon is given as  $e/\nu$ . For example, the  $e/\nu$  ratio for  $^{99m}\text{Tc}$  is 0.11 and therefore the photon is emitted 90 percent of the time and the conversion electron 10%.

## INTERACTION OF RADIATION WITH MATTER

All ionizing radiation loses energy when passing through matter by producing ion pairs (an electron and a positively charged atom residue) or by raising atomic electrons to an excited state. The average energy to produce an ion pair is given the notation  $W$ , and is numerically equal to 33.85 eV. This energy is roughly two times

the ionization potential of most gases or other elements because it includes the energy lost in the excitation process. It is not clear what part the excitation plays, for example, in damage to targets in the cellular DNA. Ionization, on the other hand, can break bonds in DNA, causing strand breaks and easily understood damage.

All particles and rays interact through their charge or field with atomic or free electrons in the medium through which they are passing. There is no interaction with the atomic nucleus except at energies above about 8 MeV which is required for interactions that break apart the nucleus (spallation).

Alpha and beta particles and gamma rays lose energy by ionization and excitation in somewhat different ways and this is described in the following sections.

### Alpha Particles

The alpha particle is a heavy charged particle with a mass that is 7300 times that of the electrons with which it interacts. A massive particle interacting with a small particle has the interesting property that it can give a maximum velocity during energy transfer to the small particle of only two times the initial velocity of the heavy particle. In terms of the maximum energy that can be transferred per interaction, this is

$$E_{(\text{maximum electron})} = 4/7300 E_{(\text{alpha particle})} \quad (4)$$

Although alpha particles can lose perhaps 10 to 20 percent of their energy in traveling 10  $\mu\text{m}$  in tissue (1 cm in air), each interaction can only impart the small energy given in the maximum in equation 4. Thus, alpha particles are characterized by a high energy loss per unit path length and thus high ionization density along the track length. This is called a high linear energy transfer (LET).

An exact expression for the energy loss in matter,  $dE/dx$  or stopping power, was derived by Hans Bethe (1953) with modifications added by Bloch and others. For alpha energies between 0.2 and 10 MeV the Bethe-Bloch expression can be simplified to

$$dE/dx = 3.8 \times 10^{-25} C NZ/E \ln\{548 E/I\} \text{ MeV } \mu\text{m}^{-1} \quad (5)$$

where

- $N$  = number of atoms  $\text{cm}^{-3}$  in the medium
- $Z$  = atomic number of the medium
- $I$  = ionization potential of the medium
- $E$  = energy of the alpha particle
- $C$  = charge correction for alpha particles with energy below 1.6 MeV.

A simple rule of thumb derived by Bloch may be used to estimate the ionization potential of a compound or element,

$$I = 10 (Z) \quad (6)$$

or the Bragg additivity rule (Attix *et al.*, 1968) may be used for compounds when the individual values of ionization potential for the elements are available. A tabulation of values of ionization potential is given in ICRU 37 (ICRU, 1984) and the stopping power in all elements has been calculated by Ziegler (1977).

When alpha particles are near the end of their range the charge is not constant at +2, but can be +1 or even zero as the particle acquires or loses electrons. A correction factor, *C*, is needed for energies between 0.2 and 1.5 MeV to account for this effect. Whaling (1958) has published values for the correction factor by which equation 4 should be multiplied. These factors vary from 0.24 at 0.2 MeV, 0.75 at 0.6 MeV, 0.875 at 1.0 MeV, up to 1.0 at 1.6 MeV.

For the case of tissue, equation 5 reduces to

$$dE/dX_{\text{tissue}} = [0.126C/E] \ln \{7.99 E\} \text{ MeV } \mu\text{m}^{-1} \quad (7)$$

**Example 1.** Find the energy loss (stopping power) of an 0.6 and a 5 MeV alpha particle in tissue.

$$\begin{aligned} dE/dX &= 0.126 (0.75)/0.6 \ln (7.99 \times 0.6) \\ &= 0.25 \text{ MeV } \mu\text{m}^{-1} \\ &= 0.126 (1.0)/5.0 \ln (7.99 \times 5.0) \\ &= 0.093 \text{ MeV } \mu\text{m}^{-1} \end{aligned}$$

### Beta Particles

The equations for beta particle energy loss in matter cannot be simplified as in the case of alpha particles, because of three factors.

1. Even at low energies of a few tenths of an MeV, beta particles are traveling near the speed of light and relativistic effects (mass increase) must be considered.

2. Electrons are interacting with particles of the same mass in the medium (free or orbital electrons) and so large energy losses per collision are possible.

3. Radiative or bremsstrahlung energy loss occurs when electrons or positrons are slowing down in matter. Such a loss also occurs with alpha particles but the magnitude of this energy loss is negligible.

Including the effects of the above three factors, the energy loss for electrons and positrons has been well quantitated. Tabulations of energy loss in various media have been prepared with the ionization energy loss and the radiative loss

detailed. Tables 21-1 and 21-2 are reproduced from ICRU 37 (1984) to show the energy loss in air and muscle.

**Example 2.** What is the energy loss in tissue for an electron with an initial energy of 1.75 MeV? What is its range and what fraction of the initial energy is given up as bremsstrahlung as the electron slows from 1.75 MeV to rest?

From Table 21-2, the stopping power at the initial energy of 1.75 MeV is 1.82 MeV cm<sup>2</sup> g<sup>-1</sup>, the range is 0.85 g cm<sup>-2</sup>, and the fraction of the energy given up as bremsstrahlung in slowing to rest is 0.006.

### Gamma Rays

Photons do not have a mass or charge as do alpha and beta particles. The interaction between a photon and matter is therefore not controlled by the Coulomb fields but by interaction of the electric and magnetic field of the photon with the electron in the medium.

There are three modes of interaction with the medium.

**The Photoelectric Effect.** The photon interaction with an orbital electron in the medium is complete and the full energy of the photon is given to the electron.

**The Compton Effect.** Part of the photon energy is transferred to an electron and the photon scatters (usually at a small angle from its original path) (Evans, 1955) with reduced energy.

The governing expressions are

$$E' = E \frac{0.511}{1 + 1/\alpha - \cos \Theta} \quad (8)$$

$$T = E \alpha (1 - \cos \Theta) / [1 + \alpha(1 - \cos \Theta)]$$

where

*E, E'* = initial and scattered photon energy in MeV

*T* = kinetic energy of the electron in MeV

$\alpha = E/0.511$

$\Theta$  = angle of photon scatter from its original path.

**Pair Production.** This occurs whenever the photon energy is greater than the rest mass of two electrons, 2(0.511 MeV) = 1.02 MeV. The electromagnetic energy of the photon can be converted directly to an electron-positron pair with any excess energy above 1.02 MeV appearing as kinetic energy given to these particles.

The loss of photons and energy loss from a photon beam as it passes through matter is described by two coefficients. The attenuation coefficient determines the fractional loss of photons per unit distance (usually in normalized units of g/cm<sup>2</sup> which is the linear distance times the density of the medium). The mass energy

Table 21-1. STOPPING POWER, RANGE, AND RADIATION YIELD FOR ELECTRONS IN AIR

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	1.975E+01	3.897E-03	1.976E+01	2.883E-04	1.082E-04
0.0125	1.663E+01	3.921E-03	1.663E+01	4.269E-04	1.299E-04
0.0150	1.445E+01	3.937E-03	1.445E+01	5.886E-04	1.506E-04
0.0175	1.283E+01	3.946E-03	1.283E+01	7.726E-04	1.706E-04
0.0200	1.157E+01	3.954E-03	1.158E+01	9.781E-04	1.898E-04
0.0250	9.753E+00	3.966E-03	9.757E+00	1.451E-03	2.267E-04
0.0300	8.492E+00	3.976E-03	8.496E+00	2.001E-03	2.618E-04
0.0350	7.563E+00	3.986E-03	7.567E+00	2.626E-03	2.955E-04
0.0400	6.848E+00	3.998E-03	6.852E+00	3.322E-03	3.280E-04
0.0450	6.281E+00	4.011E-03	6.285E+00	4.085E-03	3.594E-04
0.0500	5.819E+00	4.025E-03	5.823E+00	4.912E-03	3.900E-04
0.0550	5.435E+00	4.040E-03	5.439E+00	5.801E-03	4.197E-04
0.0600	5.111E+00	4.057E-03	5.115E+00	6.750E-03	4.488E-04
0.0700	4.593E+00	4.093E-03	4.597E+00	8.817E-03	5.049E-04
0.0800	4.198E+00	4.133E-03	4.202E+00	1.110E-02	5.590E-04
0.0900	3.886E+00	4.175E-03	3.890E+00	1.357E-02	6.112E-04
0.1000	3.633E+00	4.222E-03	3.637E+00	1.623E-02	6.618E-04
0.1250	3.172E+00	4.348E-03	3.177E+00	2.362E-02	7.826E-04
0.1500	2.861E+00	4.485E-03	2.865E+00	3.193E-02	8.968E-04
0.1750	2.637E+00	4.633E-03	2.642E+00	4.103E-02	1.006E-03
0.2000	2.470E+00	4.789E-03	2.474E+00	5.082E-02	1.111E-03
0.2500	2.236E+00	5.126E-03	2.242E+00	7.212E-02	1.311E-03
0.3000	2.084E+00	5.495E-03	2.089E+00	9.527E-02	1.502E-03
0.3500	1.978E+00	5.890E-03	1.984E+00	1.199E-01	1.688E-03
0.4000	1.902E+00	6.311E-03	1.908E+00	1.456E-01	1.869E-03
0.4500	1.845E+00	6.757E-03	1.852E+00	1.722E-01	2.048E-03
0.5000	1.802E+00	7.223E-03	1.809E+00	1.995E-01	2.225E-03
0.5500	1.769E+00	7.708E-03	1.776E+00	2.274E-01	2.401E-03
0.6000	1.743E+00	8.210E-03	1.751E+00	2.558E-01	2.577E-03
0.7000	1.706E+00	9.258E-03	1.715E+00	3.135E-01	2.929E-03
0.8000	1.683E+00	1.036E-02	1.694E+00	3.722E-01	3.283E-03
0.9000	1.669E+00	1.151E-02	1.681E+00	4.315E-01	3.638E-03
1.0000	1.661E+00	1.271E-02	1.674E+00	4.912E-01	3.997E-03
1.2500	1.655E+00	1.588E-02	1.671E+00	6.408E-01	4.906E-03
1.5000	1.661E+00	1.927E-02	1.680E+00	7.900E-01	5.836E-03
1.7500	1.672E+00	2.284E-02	1.694E+00	9.382E-01	6.784E-03
2.0000	1.684E+00	2.656E-02	1.711E+00	1.085E+00	7.748E-03
2.5000	1.712E+00	3.437E-02	1.747E+00	1.374E+00	9.716E-03
3.0000	1.740E+00	4.260E-02	1.783E+00	1.658E+00	1.173E-02
3.5000	1.766E+00	5.115E-02	1.817E+00	1.935E+00	1.377E-02

From ICRU, 1984.

 $I = 85.7$  eV; density =  $1.205E-03$  g/cm<sup>3</sup> (20°C).

absorption coefficient determines the fractional energy deposition per unit distance traveled. The loss of photons from the beam is given by

$$I/I_0 = \exp(-\mu/\rho d) \quad (9)$$

where

$I$  = intensity of the photon beam (numbers of photons)

$I_0$  = beam intensity

$\mu/\rho$  = attenuation coefficient in the medium for the energy considered (in cm<sup>2</sup> g<sup>-1</sup>)

$d$  = thickness of the medium in g cm<sup>-2</sup> (thickness in cm  $\times$  density).

The energy actually deposited in the medium per unit distance is given by

$$\Delta E = (\mu_{en}/\rho)E_0 \quad (10)$$

where

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 =  $1.040\text{E}+00 \text{ g/cm}^3$  $\Delta E$  = energy loss in the medium per unit distance (in MeV cm<sup>2</sup> g<sup>-1</sup>) $\mu_{\text{en}}/\rho$  = 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

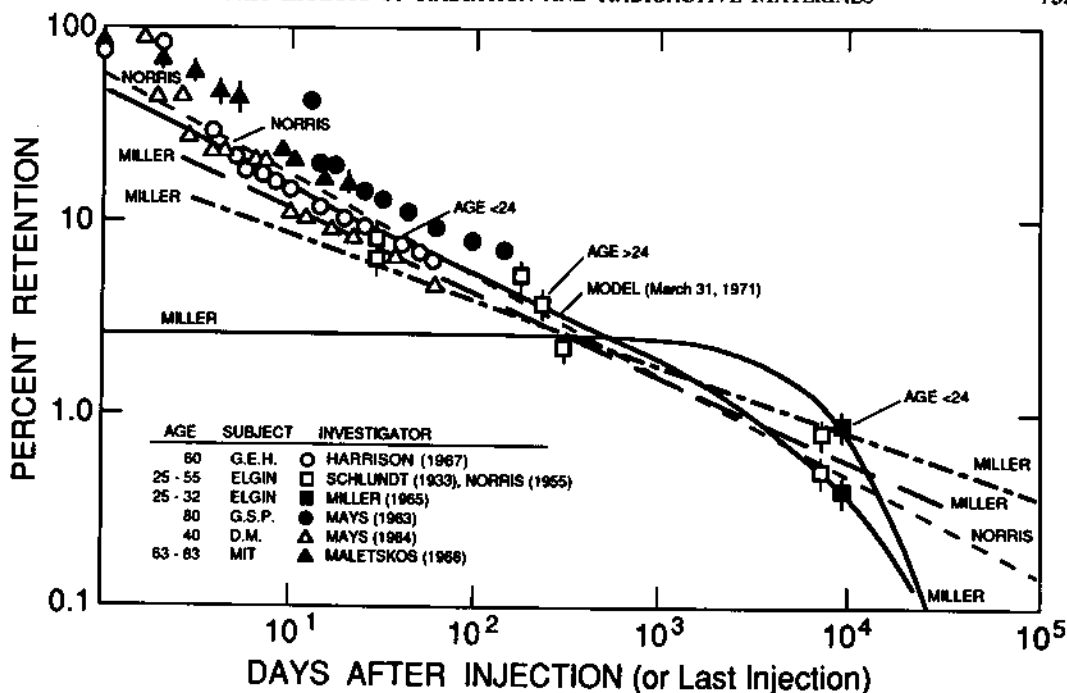


Figure 21-1. Whole body radium retention in humans. Summary of all available data for adult man. (From Marshall *et al.*, 1972.)

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

$D$  = total systemic intake of  $^{226}\text{Ra}$  plus 2.5 times the total systemic intake of  $^{228}\text{Ra}$ , both in microcuries.

Risk per unit dose:

$$I = [10^{-5} + 9.8 \times 10^{-6} D^2] \exp(-1.5 \times 10^{-2} D) \quad (20)$$

where

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

$D$  = total mean skeletal dose in Gray from  $^{226}\text{Ra}$  plus 1.5 times the mean skeletal dose from  $^{228}\text{Ra}$ .

Raabe *et al.* (1980) modeled bone sarcoma risk in the human, dog, and mouse and have determined that there is a practical threshold dose and dose rate (a dose low enough so that bone cancer will not appear within the human life span). The dose rate is 0.04 Gy per day or a total dose of 0.8 Gy to the skeleton. This practical threshold for bone cancer has useful implications in considering health effects from exposures to environmental radioactivity.

#### Radium Exposure ( $^{224}\text{Ra}$ )

In Europe,  $^{224}\text{Ra}$  was used for more than 40 years in the treatment of tuberculosis and ankylosing spondylitis. The treatment of children was abandoned in the 1950s but the relief of debilitating pain from ankylosing spondylitis in adults has prolonged its use.  $^{224}\text{Ra}$  is different from  $^{226}\text{Ra}$  in that it has a short half-life (3.62 days) and the alpha dose is delivered completely while the radium is still on bone surfaces.

Spieß and Mays (1970) and Mays (1988) studied the health of 900 German patients given  $^{224}\text{Ra}$  therapeutically. The calculated average mean skeletal dose was 4.2 Gy (range 0.06 to 57.5 Gy) with injection time spans ranging from 1 to 45 months. There were two groups, juveniles and adults, and the bone sarcoma response was not significantly different for the two. There were 54 patients who developed bone sarcoma, the last one occurring in 1983.

In a second cohort, Wick *et al.* (1986) studied 1432 adult patients treated for ankylosing spondylitis with an average skeletal dose of 0.65 Gy. This study was originally started by Otto Hug and Fritz Schales and has been continued following their deaths. Two patients in this group have developed osteogenic sarcoma with none in the control group.

Spieß and Mays (1973) found that the observed effectiveness of the  $^{224}\text{Ra}$  in their cohort in producing bone sarcomas increased if the time span of the injections was long. In-

jections were given in 1, 10, or 50 weekly fractions. They developed an empirical expression to estimate the added risk from this protracted injection schedule,

$$I = \{0.003 + 0.014 [1 - \exp(0.09m)]\} D \quad (21)$$

where

$I$  = cumulative incidence of bone sarcomas after most tumors have appeared (25 years)

$m$  = span of injections in months

$D$  = average skeletal dose in Gy.

Chemelevsky (1986) analyzed the Spiess data and developed an equation for the total cumulative sarcoma risk from  $^{224}\text{Ra}$ ,

$$R = (0.0085D + 0.0017D^2) \exp(-0.025D) \quad (22)$$

where

$R$  = cumulative risk of bone sarcoma

$D$  = average skeletal dose in Gy.

These two equations for risk predict 5.7 and 5.8 bone sarcomas in the second series of (spondylitis) patients, with two actually observed.

Chemelevsky (1986) also showed that, in the Spiess study, linearity (sarcoma response with dose) could be rejected. For example, equation 22 results in a lifetime risk of sarcoma of 0.02  $\text{Gy}^{-1}$  at an average skeletal dose of 10 Gy but 0.01  $\text{Gy}^{-1}$  at 1 Gy. Also, there was no difference in sarcoma response between juveniles and adults. These data are presented in Figure 21-2.

Again, no excess leukemia was found in either series of  $^{224}\text{Ra}$  patients.

#### Atomic Bomb Survivors

On August 6, 1945, the United States military dropped an atomic bomb on the city of Hiroshi-

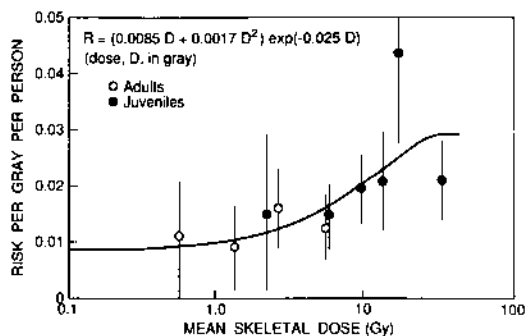


Figure 21-2. Lifetime risk per Gray versus mean skeletal dose in  $^{224}\text{Ra}$  exposed subjects. (From Chemelevsky *et al.*, 1986.)

ma, Japan. Three days later a second bomb was dropped on Nagasaki which effectively ended World War II. The weapons were of two different types the first being  $^{235}\text{U}$  and the second a  $^{239}\text{Pu}$  device.

Within one kilometer of the explosions in both cities, a total of 64,000 people were killed by the blast, thermal effects, and as a result of the instantaneous gamma and neutron radiation released by the weapons. Others between 1 and 2 kilometers received radiation doses up to several Gray.

Within a few years it was decided to follow the health of the people in both cities over their lifetime to determine quantitatively the effects of external ionizing radiation.

The study of prospective mortality of atom bomb survivors was initiated by the Atomic Bomb Casualty Commission (ABCC) in 1950 and is ongoing by the Radiation Effects Research Foundation (RERF). The main study, called the Life Span Study (LSS), included 92,228 people within 10,000 meters of the hypocenter (the point on earth directly below the detonation point in air) and 26,850 people who were not in either city at the time of bombing (ATB). The most recent report of the RERF (1988) is a follow-up of the cancer mortality of a subcohort (DS86 subcohort) of 75,991 persons over the period 1950–85.

In 1978, questions arose that the original dose estimates for persons in the LSS might be somewhat in error and that an effort should be made to improve the dose estimates. This study is now complete and the dosimetry is published in a United States–Japan joint reassessment of dose called DS86—Dosimetry System 1986 (RERF, 1986).

Dose estimation by reconstruction of the event is always problematical but direct computation of dose to about 18,500 persons in the LSS with detailed shielding information is complete. The remaining DS86 dose values for 57,000 individuals without detailed shielding information are also incorporated into the mortality study by various estimation techniques. Of the 75,991 persons in the DS86 subcohort, 16,207 were within 2000 meters of the hypocenter and these are the individuals who received a substantial exposure.

Previous reports of cancer risk estimates were based on the air dose (gamma ray plus neutron tissue kerma in air) adjusted for shielding by structures or terrain. The 1987 and 1988 reports also include DS86 organ dose estimates and these are about 80 percent of the shielded kerma.

The dose from fallout at Hiroshima and Nagasaki has not been included in the health effects studies. Fallout was found in certain restricted

localities in Nagasaki and in Hiroshima. The absorbed dose from gamma rays at Nagasaki for persons continuously in the fallout area from one hour on ranged from 0.12 to 0.24 Gy. The absorbed doses at Hiroshima ranged from 0.006 to 0.02 Gy. Because the region of fallout was quite limited, it would appear that the total contribution of fallout to survivor dose was probably negligible in Hiroshima but may have been significant for a limited number of survivors in Nagasaki where an exposure of one-fifth the maximum extends over some 1000 hectares. Estimates of internal dose from ingested  $^{137}\text{Cs}$  yield about 0.0001 Gy integrated over 40 years (Harley, 1987; RERF, 1987).

Complete mortality data and the dose estimates are reported in RERF Technical Reports 5-88 (RERF, 1988) and so it is possible to calculate the lifetime cancer risk as of the follow-up through 1985. This is done for this chapter in Table 21–7. The dose used in the calculation is the shielded kerma dose so that it is more comparable with the older publications. The organ dose estimates are about 80% of the shielded kerma dose and so the risk estimates would increase by about 20 percent if organ dose were utilized (Shimizu *et al.*, 1988). However, when the organ dose equivalent is calculated, the organ dose equivalent in Sievert is almost identical to the organ dose in Gray. This is due to the small neutron component in the DS86 dosimetry (about 1 percent of the organ gamma-ray dose). When multiplied by a value of 20 for  $Q$  the neutron dose increases the total organ dose equivalent by 20 percent.

No statistically significant excess cancer of the gall bladder, pancreas, uterus, or prostate or of malignant lymphoma has been seen in the LSS to date.

The previous cancer risk estimates, reproduced in Table 21–6, were published by ICRP in 1977 and were based on earlier follow-up of the atom bomb survivors and utilized air dose. The calculations shown here in Table 21–7 are preliminary and the increase in risk is due to differences between the older dose and the DS86 estimates as well as the added number of cancers observed since the previous update in the mortality studies. The leukemia risk is about a factor of three higher than that projected by ICRP in 1977, and the lung cancer risk about a factor of 2 higher.

It is of interest to consider the effects of smoking as it is the most important factor in assessing lung cancer risk. The analysis performed by Shimizu *et al.* (1988) examined the interaction of smoking and radiation in detail. The results showed no interaction indicating that smoking and the atom bomb radiation act independently

**Table 21-7. CANCER MORTALITY AND LIFETIME CANCER RISK AT SELECTED SITES FOR ATOM-BOMB SURVIVORS WITH AVERAGE SHIELDED KERMA OF 0.295 Gy<sup>†</sup>**

SITE	NUMBER OF SUBJECTS* (0.01+) Gy	TOTAL CA MORTALITY*	ATTRIBUTABLE RISK (%) <sup>†</sup>	RADIATION CANCERS	LIFETIME <sup>‡</sup> RISK (Gy <sup>-1</sup> )
All sites	41,719	3435	10.4	357	0.029
All sites (except leukemia)	41,719	3291	8.0	265	0.022
Leukemia	40,701	144	56.6	81	0.0067
Multiple myeloma	(40,701) <sup>§</sup>	23	32.9	8	0.0007
Colon	39,859	129	15.2	20	0.0017
Esophagus	(39,859) <sup>§</sup>	93	12.8	12	0.0010
Lung	40,382	385	11.6	45	0.0038
Stomach	39,961	1153	6.4	74	0.0063
Female breast	25,252	98	22.4	22	0.0029
Bladder	40,060	84	23.4	20	0.0017
Ovary	24,581	51	18.6	9	0.0012

\* From RERF Report TR-12-87 Part 1, Tables 2 and 3.

† From RERF Report TR-12-87 Part 1, Table 6. Attributable risk is the percent of cancer deaths caused by radiation.

‡ Lifetime risk Gy<sup>-1</sup> is calculated as (radiation cancers)/(number of subjects) (average dose).

§ Estimated as the leukemia or colon population.

¶ Average organ dose equivalent in Sievert is essentially identical to shielded air dose in Gray. See text.

rather than multiplicatively in lung cancer induction.

It is also possible to model the risk over full life if a projection model is assumed. RERF has preferred a constant relative risk model (radiation mortality is a constant fraction of the baseline age specific mortality per Gray) for this purpose. There is evidence in the atom bomb mortality and in several other studies discussed later (ankylosing spondylitis patients, uranium miners) that the constant relative risk model is not appropriate, but that the risk coefficient decreases with time subsequent to exposure. In most cases this also means that the absolute excess cancer risk (risk above that expected) declines with time. This is a biologically plausible model suggesting the loss or repair of the damaged stem cell population.

The National Academy of Sciences BEIR V Committee (NAS, 1990) utilized the atom bomb mortality data through 1985 and the DS86 dosimetry to model the lifetime risk of all cancer, leukemia, female breast, respiratory, and digestive cancer. The expressions for respiratory, breast, and digestive cancer all include a term for reduction in relative risk with time since exposure. For example, the model for respiratory cancer is

$$\gamma(d) = \gamma_0 [1 + f(d) g(\beta)] \quad (23)$$

where

$\gamma(d)$  = an individual's age specific lung cancer risk for dose  $d$

$\gamma_0$  = the age specific background risk of death due to lung cancer

$f(d)$  =  $0.636 \times (\text{dose in Gray})$

$g(\beta) = \exp[-1.437 \ln(T/20) + \beta_2 I(S)]$

$T$  = years after exposure

$I(S) = 1$  if female, 0 if male.

The integration of this model yields a lifetime risk of respiratory cancer in males of 0.002, 0.0124, and 0.039 for exposure to 1 Gray at ages 5, 25, and 55, or a risk of 0.019 for a stationary male population with United States mortality rates. Given the existing risk in the Japanese cohort of  $0.0038 \text{ Gy}^{-1}$ , the BEIR V lifetime risk estimate is clearly conservative.

The lifetime risk of cancer from exposure to atom bomb radiation estimated by the National Academy of Sciences BEIR V Committee is four times that of the BEIR III Committee values (NAS, 1980). The reason for this difference is due partly to more complete follow-up of the population and the difference in the new DS86 dosimetry, but primarily to the models used for risk expression following exposure. Given the small number of radiation induced cancer in the Japanese population studied, the models cannot be derived with a high degree of certainty.

The estimates of lifetime risk of cancer will undoubtedly increase somewhat with time, but given the present age of the population, the final values are unlikely to be higher than about a factor of three from the original 1977 values in Table 21-6.

#### *Tinea Capitis (Ringworm) Irradiation*

During the period 1905-60, X-ray epilation in the treatment of tinea capitis was performed reg-

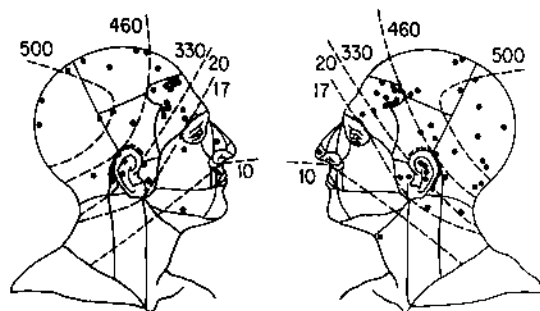
ularly in children. The treatment was introduced by Sabouraud in 1904 and was standardized by Kienbock (1907) and Adamson (1910). Over the half century it was used, as many as 200,000 children worldwide may have been irradiated (Albert *et al.*, 1986).

No follow-up studies of the long-term effects of irradiation were performed until Albert and Omran (1968) reported on 2200 children irradiated at the Skin and Cancer Unit of New York University Hospital during 1940–59. Subsequent publications on this group have appeared at regular intervals (Shore *et al.*, 1976, 1984).

Since the New York University (NYU) study, a follow-up of 11,000 children irradiated in Israel was performed (Ron and Modan, 1984).

The mean age of children irradiated in both the New York and Israeli studies was between 7 and 8 years. Dose reconstruction in the New York University series was performed using a head phantom containing the skull of a 7-year-old child covered with tissue-equivalent material (Schulz and Albert, 1963; Harley *et al.*, 1976, 1983). The doses to organs in the head and neck for a typical Adamson-Kienbock five-field treatment of the scalp are shown in Table 21–8 and the dose to the skin is shown in Figure 21–3.

In the NYU series there were eight thyroid adenomas and no thyroid cancer. In the Israeli series there were 29 thyroid cancers. In the NYU series there are 80 skin lesions predominantly basal cell carcinoma in 41 persons. Lightness of skin is an important factor in the appearance of skin cancer (Shore *et al.*, 1984). Skin cancer was found only in caucasians even though 25 percent of the study population were blacks. This and the fact that there appears to be a much lower dose response on the hair covered scalp than on the face and neck (Harley *et al.*, 1983) suggests that



**Figure 21–3.** X-ray dose in rads for the Adamson-Kienbock five-field tinea capitis treatment and locations of basal cell lesions. (From Shore *et al.*, 1984.)

promotional effects of UV radiation play an important part in skin cancer.

A summary of the tumors of the head and neck and hemopoietic/lymphopoietic tumors for the NYU studies is shown in Table 21–9 and for the Israeli studies in Table 21–10. In the Israeli study the estimate of the dose to the thyroid is 0.09 Gy compared with 0.06 Gy in the NYU study.

A risk projection model was used to estimate the lifetime risk of basal cell carcinoma (BCC) for facial skin and for the hair covered scalp following X-ray epilation in caucasians. The model used was a cumulative hazard plot which assumes that the BCC appearance rate in the exposed population remains constant over time (Harley *et al.*, 1983). The result of this risk projection for BCC is shown in Table 21–11.

The small numbers of tumors other than skin cancers in the NYU study makes it of dubious value to estimate the lifetime risk per Gy although a clear excess is appearing. The tinea capitis studies are prospective and sound numerical values should be forthcoming as the populations age. These are particularly important studies because children were the exposed group and because only partial body irradiation was involved. The temporal pattern of appearance of these tumors is also important. The dose was delivered over a short time interval (minutes at NYU and 5 days in Israel) and lifetime patterns will be indicative of the underlying carcinogenic mechanisms.

The skin and thyroid cancers are of importance in documenting health effects from ionizing radiation. However, both types of cancer are rarely fatal. NCRP (1985) reports that about 10 percent of thyroid cancer is lethal. It is estimated that fatality rate of skin cancer is 1 percent (NCRP, 1990). The lifetime risk per Gray derived by NCRP for total thyroid cancer incidence (0.003 for female and 0.0014 for males for ex-

**Table 21–8. AVERAGE DOSE TO ORGANS IN THE HEAD AND NECK FROM MEASUREMENTS PERFORMED WITH A PHANTOM FOR A CHILD'S HEAD**

ORGAN	AVERAGE DOSE AT 25 CM TREATMENT DISTANCE (rads)
Scalp	220–540
Brain	140
Eye	16
Internal ear	71
Cranial marrow	385
Pituitary	49
Parotid gland	39
Thyroid	6
Skin (eyelid)	16
Skin (nose)	11
Skin (mid-neck)	9

**Table 21-9. TUMORS IN THE NEW YORK UNIVERSITY SERIES OF CHILDHOOD IRRADIATIONS FOR TINEA CAPITIS**

TUMOR	IRRADIATED CASES	CONTROL CASES
Neurogenic		
Brain	6	0
Acoustic neuroma	2	0
Neck	2	0
Parotid		
Skin		
Basal cell	41	3
Cylindroma	4	0
Other	3	2
Bone (skull/jaw)	4	0
Mouth/larynx		
Papilloma	7	2
Thyroid adenoma		
Male	3	0
Female	5	0
Hemopoietic		
Lymphopoietic		
Leukemia	4	1
Hodgkins disease	5	2
Other lymphoma	2	0

**Table 21-10. TUMORS IN THE ISRAELI STUDY BY SEX**

TUMOR	IRRADIATED CASES	CONTROL CASES
Leukemia		
Male	6	4
Female	4	1
Thyroid malignancies		
Male	6	1
Female	23	5
CNS (malignant/benign)		
Male	21	3
Female	9	0

From Ron and Modan, 1984.

**Table 21-11. LIFETIME RISK ESTIMATES FOR BASAL CELL CARCINOMA (BCC) AND THYROID CANCER FOLLOWING X-RAY IRRADIATION FOR TINEA CAPITIS**

	TOTAL INCIDENCE (Risk Gy <sup>-1</sup> )	MORTALITY (Risk Gy <sup>-1</sup> )
Skin malignancies (NYU Study)		
BCC (facial skin)	0.32	
BCC (hair covered scalp)	0.01	
Thyroid malignancies (Israeli study)		
Male	0.01	0.001
Female	0.04	0.004

**Table 21-12. EXCESS CANCER IN 6158 ANKYLOSING SPONDYLITIS PATIENTS GIVEN A SINGLE X-RAY TREATMENT AS OF THE LAST FOLLOW-UP**

SITE	OBS	ESTIMATED EXPOSURE	DOSE (Gy) <sup>†</sup>	LIFETIME (Risk Gy <sup>-1</sup> )
Leukemia	31	6.5	2.9	0.0011
Lung*	101	69.5	1.8-6.8 <sup>‡</sup>	0.0008-0.0028
Esophagus	28	12.7	4.2	0.0006
Breast	26	16.0	6.8 <sup>§</sup>	0.0015 <sup>  </sup>

\* Lung cancer appearing less than five years after exposure is not included as this is less than the minimum latency for tumor expression.

† The doses are taken from Lewis *et al.* 1988.

‡ The dose to the pulmonary lung and main bronchi was estimated as 1.8 Gy and 6.8 Gy, respectively. The majority of lung cancer is bronchogenic and the dose estimates for the main bronchi are probably most pertinent.

§ The dose to the breast is taken as the dose to the main bronchi.

|| The number of women having one X-ray treatment was 1008.

ternal X or gamma radiation for persons under 18 years of age) is about a factor of 10 lower than that reported by Ron and Modan in the tinea capitis irradiations. However, the tinea irradiations were given to children with mean age of about 7 years and in the Israeli study there is apparently an increased sensitivity due to ethnicity.

The effect of ethnicity and sex is also suggested by NCRP (1985) for thyroid cancer. The incidence rates of spontaneous thyroid cancer for persons of Jewish origin in Europe and North America is three to four times that for other racial groups. There is an obvious susceptibility of women for thyroid cancer and adenomas in both the NYU and Israeli tinea capitis studies.

### *Ankylosing Spondylitis*

About 14,000 persons, mostly men, were treated with X rays for ankylosing spondylitis at 87 radiotherapy centers in Great Britain and Northern Ireland between 1935 and 1954. Court Brown and Doll (1957) were the first to report that these patients had a leukemia risk substantially in excess of that for the general population. Subsequent publications have developed the time pattern of appearance not only of leukemia but of solid tumors (Court Brown and Doll, 1959, 1965; Smith and Doll, 1978, 1982; Smith, 1984; Darby *et al.*, 1985, 1987).

A group was selected consisting of 11,776 men and 2335 women all of whom had been treated with X rays either once or twice. About half of the total group received a second X-ray treatment or treatment with thorium. The reports on the ankylosing spondylitis patients attempt to consider health effects from only the first X-ray treatment. For this reason an individual receiving a second treatment is included in their follow-up only until 18 months after the second course (a short enough time so that any malig-

nancies in this interval cannot be ascribed to the second X-ray treatment).

The appearance of excess leukemia is now well documented and solid tumors are also apparent in the population. The part of the body in the direct X-ray beam (spine) received the highest dose but it is thought that other sites received substantial radiation from scatter or from the beam itself.

The importance of this study is in the health effects of partial body exposure and in the temporal pattern of appearance of solid tumors in irradiated adults. Smith and Doll (1978, 1982) and Darby *et al.* (1985, 1987) in the most recent follow-up publications concerning these patients have shown that the excess risk for solid tumors is diminishing with time since exposure, with maximum appearance 5 to 20 years after exposure. This has significant implications for risk projection modeling. Many projection models assume a constant rate of appearance either as an absolute number of tumors per person per unit exposure (constant absolute risk) or as a fraction of the baseline age specific cancer mortality rate (constant relative risk). The emerging pattern is that constant risk models, either absolute or relative, are not correct for certain cancers such as lung cancer.

The dosimetry was redone in 1988 (Lewis *et al.*, 1988) and, although better estimates of dose are now available, it is still the dose which is most uncertain for the cohort. No details of the X-ray machines used to deliver the exposures, such as output, kilovoltage, or half-value layer, are reported.

The excess cancers and the estimate of lifetime cancer risk at three sites in the ankylosing spondylitis cohort are shown in Table 21-12. For the purpose of calculating lifetime risks as of the time of follow-up, the number of persons used here as the individuals at risk is the number

actually receiving only one X-ray treatment (6158). This assumes that those followed for 18 months subsequent to the second treatment do not contribute significantly to the malignancies.

The relatively low risk for leukemia (compared with atom bomb survivors) has been suggested to be due to cell sterilization at the high dose delivered. It is also possible that the low risk is due to partial irradiation of the skeletal red marrow. The volume of bone marrow irradiated in the spine, rib, and pelvis is much less than 50 percent of that in whole body irradiation.

The deaths due to causes other than neoplasms in the total cohort is about 30 percent higher than expected. This higher total mortality is of significance in risk modeling as the premature deaths due to competing causes decreases the observed fractional cancer mortality. Thus, the lifetime risk in this population probably underestimates the risk when projecting the effects of exposure in a healthy population.

### Uranium Miners

Radon is ubiquitous on earth. It is found outdoors and in all dwellings as a result of the decay of the parent  $^{226}\text{Ra}$  which is present in all of earth's minerals.

Although the lung cancer risk from radon exposure in underground miners is firmly documented, and quantitative risk estimates are available, the current interest lies in whether this risk carries over into environmental situations. Radon levels in homes that are comparable to those in mines surely confer risks to the residents. The question remains, can the risks in mines for exposures at higher concentrations over short time periods be used to model risks at lower environmental levels over a lifetime?

**Underground Mines.** There are four large studies of underground miners exposed to high concentrations of radon and radon daughters and the documentation of excess lung cancer is convincing. The carcinogen in the case of radon is actually the alpha emitting short-lived daughters of radon,  $^{218}\text{Po}$  and  $^{214}\text{Po}$ . The decay scheme for the entire uranium series, including radon and the daughter species, is shown in Figure 21-6. The daughters are solids and deposit on the bronchial airways during inhalation and exhalation according to the laws of diffusion. As the airway lining (bronchial epithelium) is only 40  $\mu\text{m}$  thick, the alpha particles emitted are able to reach and transfer a significant amount of energy to all of the cells implicated in lung cancer induction. Although the daughters are the carcinogen, the term radon will be used interchangeably for radon daughters as without the parent radon the daughters could not exist for longer than a few hours.

The measurements in mines were usually of the daughter species rather than radon and the term working level (WL) was defined for occupational exposure. It indicated the total potential energy content in one liter of air for complete decay of the short-lived daughters.\* The exposure attributed to miners was developed in working level months (WLM) which is the numerical value of WL times the time exposed in multiples of the working month of 170 hours (Holaday *et al.*, 1957).

The follow-up studies from four large underground mining cohorts in Canada, Czechoslovakia, Sweden, and the United States have all produced data to show that the excess lung cancer risk from exposure to radon is about two to three per 10,000 persons per WLM exposure (Radford and Renard, 1984; Hornung and Meinhardt, 1987; Sevc *et al.*, 1988; Muller *et al.*, 1989). Expressed in another way, radon exposure increases the normal age specific lung cancer risk by about 1 percent for each WLM exposure. The latter way of expressing risk brings in the thought that many epidemiologists prefer, that the lung cancer risk is proportional to the normal baseline risk. This means, for example, that the lifetime excess lung cancer risk from radon would be different for smokers and nonsmokers (NAS, 1988).

The actual data from the underground studies are not clear cut with regard to the effect of smoking and it is apparent from more recent data that radon exposure does not simply multiply the baseline risks of the population by a constant factor. This is discussed in the section on risk.

The excess lung cancer risk in each of the exposure cohorts for the four major mining populations as of the date of the last published follow-up are summarized in Figure 21-4 (Harley, 1989). It can be seen in this figure that the range of risks for the same exposure varies by about a factor of six among the different studies. The highest values of excess lung cancer shown are in the Czechoslovakian mines and the lowest in the U.S. Colorado mines.

The differences are probably accounted for by errors in measuring and estimating total exposure. However, the Czech mine atmosphere is reported to contain arsenic as well as radon and the arsenic may contribute to the excess lung cancers observed.

A maximum value of 50 percent lung cancer

\* One working level is any combination of short-lived daughters in one liter of air that will result in  $1.3 \times 10^5$  MeV of alpha energy when complete decay occurs. One working level is approximately equal to concentrations of 7400 Bq  $\text{m}^{-3}$  (200 pCi/liter) of radon in a home and 11000 Bq  $\text{m}^{-3}$  (300 pCi/liter) in a mine.

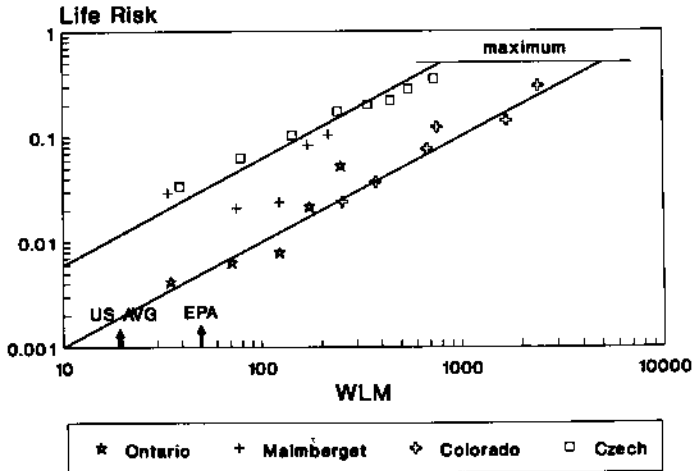


Figure 21-4. Lifetime lung cancer in four underground mining populations as a function of radon exposure. (From Harley, 1989.)

risk is indicated in Figure 21-4. This is the highest value ever observed in a mining population and was reported in the mines in Saxony at the turn of the century (Muller, 1989). These mines are thought to have had about 100,000 Bq  $m^{-3}$  of radon. It is noteworthy that concentrations this high have been reported in a few homes in the United States.

In Figure 21-4, the lowest exposures were in the Ontario mines and a mean exposure of 35 WLM has given an excess lung cancer risk of about 0.4 percent to date.

When radon gas decays to the solid daughter products, some 8 to 15 percent of the  $^{218}Po$  does not attach to the normal aerosol particles and this ultrafine species is deposited with 100 percent efficiency on the upper bronchial airways. The rest of the daughters attach to the aerosol of about 100  $\mu m$  average diameter (George and Breslin, 1980) and only a few percent of this aerosol deposits on these airways.

Measurements in mines have mostly been of the short-lived radon daughters as these are the easiest to measure rapidly. The alpha dose from radon gas itself is very low in comparison with that from the daughters as the daughters deposit and accumulate on the airway surfaces.

The first few branching airways of the bronchial tree are the region where almost all the lung cancers appear. This is true in general and not only for miners exposed to radon daughters. The alpha dose from radon daughters must therefore be calculated in these airways and not in the pulmonary or gas exchange regions. Although the dose to the pulmonary region should not be neglected it is about one-fifth that to the airways.

Several calculations regarding the absorbed alpha dose exist for radon daughters (NCRP,

1984; ICRP, 1987; Harley, 1987, 1989; James, 1987). The authors make different assumptions about the atmospheric and biologic parameters that go into the dose calculation, yet the results are comparable. The most significant variable is the particle size of the ambient aerosol. Very small particles deposit more efficiently in the airways so if small particles, such as from open flame burning (Tu and Knutson, 1988), contribute to the atmosphere, then the dose delivered to the bronchial epithelium can be higher per unit WLM exposure than the dose predicted from an average particle size. Conversely a hygroscopic particle can increase in size in the humid environment of the bronchial airways and deposition will be diminished. The particle size of the aerosol in mines is somewhat larger than that for environmental conditions (200 versus 100 nm; George *et al.*, 1975). Figure 21-5 shows the alpha dose per unit exposure as it is related to the variables (particle size, unattached fraction, nasal deposition) known to affect dose.

As carcinogenesis is related to absorbed alpha dose, Figure 21-5 shows that particle size is an important determinant of risk. The average dose per unit exposure in WLM for miners is also indicated in Figure 21-5 to show that it is somewhat smaller than that for average environmental conditions.

Radon can deliver more or less carcinogenic potential by about a factor of 2 over the range of realistic indoor conditions (average particle size ranging from 80 to 300 nm).

The allowable effective dose equivalent for continuous exposure of the population in the United States is 1 mSv/year (100 mrem/yr, NCRP, 1984, 1987). This limit would be delivered by exposure to 20 Bq  $m^{-3}$  of radon or

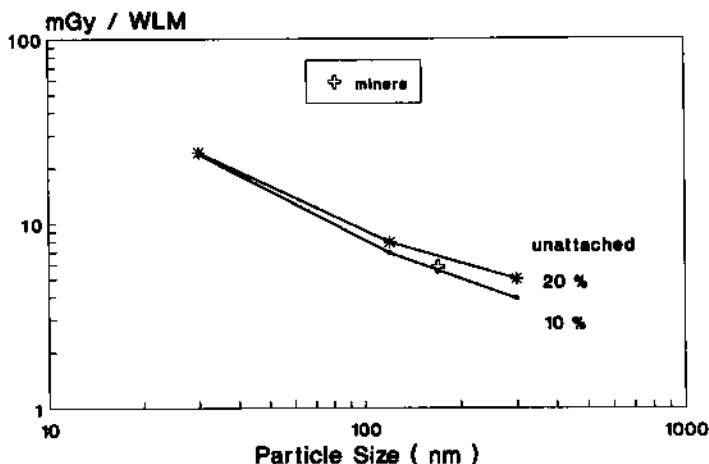


Figure 21-5. Radon daughter bronchial dose as a function of particle size and percent unattached fraction. (From Harley, 1989.)

one-half the actual average measured indoor concentration in most countries where measurements have been made. Thus, the guidelines for exposure cannot be set in the usual way from dosimetric considerations.

**Lifetime Environmental Lung Cancer Risk Projections.** There are at present three sets of publications that provide the risk projection calculations for exposure to radon daughters. The following sections describe each in detail.

**National Council on Radiation Protection and Measurements.** In 1984, the U.S. National Council on Radiation Protection and Measurements (NCRP, 1984) developed a model to project the risk derived from the miner studies to whole life risk in the environment. It is a modified absolute risk model that reduces the risk subsequent to exposure with a half-life of 20 years. Risk is not accumulated until after age 40, the time when lung cancer normally appears in the population. There is no indication that early exposure produces any significant shift to younger ages, even for young miners exposed at significantly higher concentrations.

**National Academy of Sciences.** The U.S. National Academy of Sciences report in 1988 (BEIR IV) developed a model based on examination of the raw data from five mining cohorts (NAS, 1988). The data indicated that the highest risk appears from 5 to 15 years following exposure. After 15 years, the risk is one-half that of the 5- to 15-year risk (per unit exposure) and persists to the end of life. Again, no significant risk appears before 40, the usual age for the appearance of lung cancer. The NAS model also has a correction for attained age (at age >65 the risk is 0.4 of that for ages 55 to 64). The BEIR IV Committee assumed a relative risk model (risk is a fraction of the normal age specific lung

cancer risk per unit radon exposure with risk dependent on time from exposure. This means that the risk for smokers and nonsmokers differs because of their different baseline lung cancer values. Although the miner epidemiology did not support this strictly multiplicative relationship, the NAS chose the relative risk model as a conservative one. Their analysis supports the risk reduction subsequent to exposure.

**ICRP.** The International Commission on Radiation Protection (ICRP, 1987) developed two risk projection models, one based on a constant relative risk and the other a constant absolute risk model. Although neither the constant relative or constant absolute risk model is correct, because of the temporal reduction pattern of lung cancer subsequent to cessation of exposure, the numerical values obtained for the lifetime risk of lung cancer from radon exposure are not significantly different from other models.

Later follow-up of the Czechoslovakian underground uranium miners presented by Kunz and Sevc (1988) indicates that the excess lung cancer risk may actually reduce to zero 35 years after exposure. If this factor were included in the NAS model (zero risk after 35 years) it would reduce their values by about a factor of 2.

The risk values obtained from the three models are shown in Table 21-13.

**Environmental Epidemiology.** There are at least 22 published studies attempting to define or detect the effect of radon exposure in the environment. These have been summarized by Borak and Johnson (1988), and by Neuberger (1989). The most recent study was performed in 1989 by the New Jersey Department of Health (NJDOH) in the United States (Schoenberg and Klotz, 1989). This is the most rigorous to date and is a case-control study with 433 lung cancer

**Table 21-13. LUNG CANCER RISK FOR CONTINUOUS EXPOSURE TO 1 WLM YR<sup>-1</sup> (150 Bq m<sup>-3</sup> OR 4 pCi LITER<sup>-1</sup>) AS PREDICTED BY VARIOUS MODELS**

	LIFETIME RISK (%)	MODEL TYPE	COMMENT
NCRP	0.9	Modified absolute	Risk decreases with time from exposure
ICRP	1.6	Constant relative	No reduction in risk with time from exposure for either model
	1.1	Constant absolute	
BEIR IV	3.4 (2.2)* men	Modified relative	Risk decreases with time from exposure
	1.4 (0.9)* women		

\* Beir IV values modified to express risk for 35 years after exposure rather than entire lifetime.

cases and 402 controls with year-long measurements of radon in the homes where individuals lived for 10 or more years. This study devoted a considerable effort to quality control concerning the exposure measurements. The results of this study are slightly positive, suggesting an association of radon and lung cancer, even at concentrations of 80 Bq m<sup>-3</sup>, but the results are not statistically significant.

Of the total studies, 13 are ecologic and 9 are case control. Ecologic studies depend on relating the disease response of a population to some measure of a suspected causative agent. There are usually insufficient data on all of the variables involved in the disease to infer any reliable associations. Ecologic studies are the weakest type of epidemiologic exploration.

Unless a biologic marker for radon-induced lung cancer is found, it is unlikely that environmental epidemiology will be effective. The effects of radon in the environment are subtle compared with the overwhelming lung cancer mortality from smoking.

Four concepts emerge from the radon research so far, however, and these are

1. The mining epidemiology indicates that short exposure to high levels of radon and daughters produce a clear excess of lung cancer.
2. Particle size can change the actual dose delivered by radon to bronchial tissue with small sized particles giving a substantially higher dose per unit exposure. Passive tobacco smoke and open flames indoors produce a higher dose.
3. Smokers are at higher risk from radon per unit exposure than nonsmokers.
4. Urban areas almost universally have low radon and apartment dwellers removed from the ground source have particularly low radon exposure at home.

The miner data show clearly that there is a risk of lung cancer from exposure to high con-

centrations of radon delivered over short time periods. Comparable exposures delivered over a lifetime in the home have not produced statistically significant increases in lung cancer mortality. The risk can still exist, but the confounding effect of other carcinogens such as smoking and urbanization make it impossible to extract the more subtle impact of radon in existing studies.

#### *Natural Radioactivity and Radiation Background*

The occupational, accidental, and wartime experiences detailed in the preceding sections have provided the bases for all of the current radiation risk estimates. For many years, the radioisotopes deposited internally were compared with <sup>226</sup>Ra in order to evaluate the maximum permissible body burden for a particular emitter. The present limits for external and internal radiation are based on dose estimates which in turn can now be related to cancer risks. One standard of comparison has always been the exposure from natural background and this source is assessed here.

Background radiation from all sources is described in detail in NCRP report 94 (1987) and some of the information is summarized here.

The risk estimates in the previous sections must be placed in context with the radiation dose received by all humans from natural background radiation. There is a substantial dose received annually from cosmic radiation and from external terrestrial radiation present from uranium, thorium, and potassium in the earth's crust. Internal emitters are present in the body as a consequence of dietary consumption and inhalation. For example, potassium is a necessary element in the body and is homeostatically controlled. Radioactive <sup>40</sup>K is a constant fraction of all natural potassium. Potassium delivers the largest internal dose from diet of 0.15 mSv per year. However, the data are scant on the dietary intake of other radionuclides in the United States population. Given the usual distribution of intakes across a large population, it is probable that

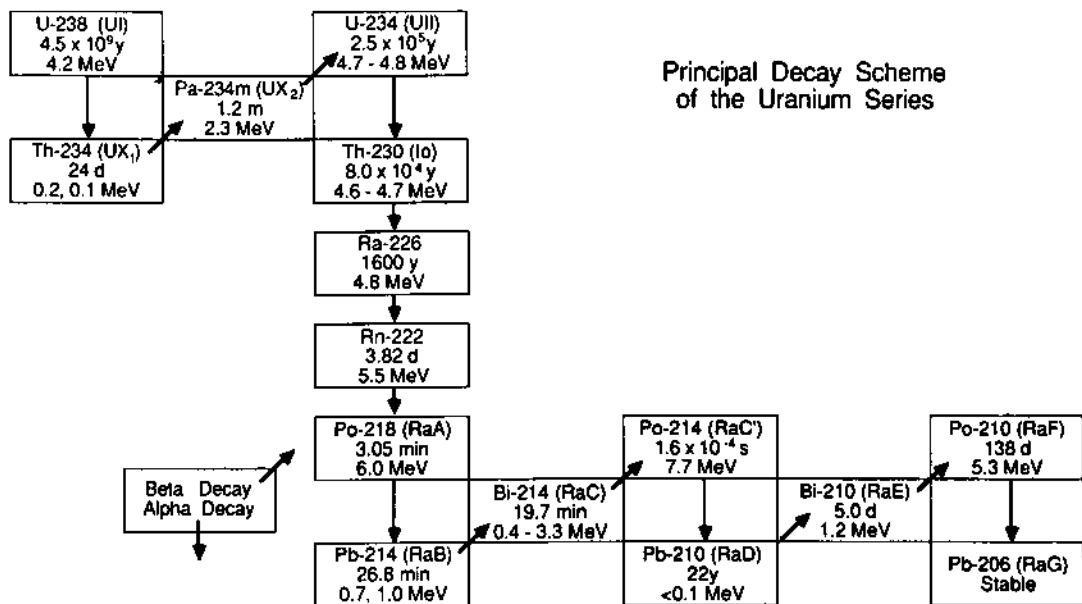


Figure 21-6. Uranium-238 decay series. (From NCRP, 1987.)

other emitters, notably  $^{210}\text{Pb}$ , could deliver a significant dose to a fraction of the population.

The largest dose received by the population is from the inhaled short-lived daughters of radon. These are present in all atmospheres because radon is released rather efficiently from the  $^{226}\text{Ra}$  in rock and soil. The short-lived daughters  $^{218}\text{Po}$ ,  $^{214}\text{Pb}$ ,  $^{214}\text{Bi}$ ,  $^{214}\text{Po}$ , have an effective half-life of 30 minutes, but the 3.8-day parent radon supports their presence in the atmosphere. Figure 21-6 shows the entire uranium series decay.

Average outdoor concentrations in the United States have been measured as  $7 \text{ Bq m}^{-3}$  and indoors as 40 to  $80 \text{ Bq m}^{-3}$ . A structure such as a house prevents the rapid upward distribution of radon into the atmosphere and substantial levels can be built up indoors. The source of radon is the ground and so levels in living areas above ground are generally one-third to one-fifth the concentrations measured in basements. An effective barrier across the soil/building interface also inhibits radon entry to buildings. Ventilation with outdoor air reduces indoor radon. For this reason, industrial buildings with more substantial foundations and higher ventilation rates tend to have lower radon concentrations than single family (or detached) houses. Apartments above ground level have radon concentrations about half the average of single family dwellings.

It is of significance that an average radon concentration indoors of  $40 \text{ Bq m}^{-3}$  results in a dose

equivalent to bronchial epithelium of 24 mSv/year or an effective dose equivalent (EDE) of 2 mSv per year.

The dose equivalents for the major natural internal emitters are shown in Table 21-14. These are reproduced from NCRP (1987).

The annual effective dose equivalent for all of the external and internal emitters from natural background are summarized in NCRP Report 94 (1987) and these are shown in Table 21-15.

The lifetime dose from the natural emitters is shown in Table 21-16, assuming an average exposure from birth to a full life of 85 years. It should be recognized that the actual dose accumulated by an individual depends upon dietary habits, location (Denver for example, at an altitude of 1.6 kilometers has double the average cosmic ray exposure) and the dwelling. Apartment dwellers would accumulate approximately half the dose from inhaled radon daughters as a person living in a single-family dwelling.

Table 21-16 is informative in considering the effects of radiation exposure from sources other than natural. For example, in assessing an occupational dose, which might add, say, 10 mSv effective dose equivalent, natural background would be a strong confounder. Any health detriment would have to be calculated rather than observed directly. No study would be able to detect an increase in health effects from 10 mSv above the average whole life natural background of 260 mSv.

**Table 21-14. DOSE EQUIVALENT RATES TO VARIOUS TISSUES FROM NATURAL RADIONUCLIDES CONTAINED IN THE BODY**

RADIONUCLIDE	DOSE EQUIVALENT RATE (mSv yr <sup>-1</sup> )			
	Bronchial Epithelium	Soft Tissue	Bone Surfaces	Bone Marrow
<sup>14</sup> C	—	0.10	0.08	0.30
<sup>40</sup> K	—	1.80	1.40	2.70
<sup>87</sup> Rb	—	0.03	0.14	0.07
<sup>238</sup> U, <sup>234</sup> Th	—	0.046	0.03	0.004
<sup>230</sup> Th	—	0.001	0.06	0.001
<sup>226</sup> Ra	—	0.03	0.90	0.15
<sup>222</sup> Rn	—	0.07	0.14	0.14
<sup>222</sup> Rn daughters	24	—	—	—
<sup>210</sup> Pb, <sup>210</sup> Po	—	1.40	7.00	1.40
<sup>232</sup> Th	—	0.001	0.02	0.004
<sup>228</sup> Ra- <sup>224</sup> Ra	—	0.0015	1.20	0.22
<sup>220</sup> Rn	—	0.001	—	—
Total	24	3.50	11.00	5.00

**Table 21-15. ESTIMATED TOTAL EFFECTIVE DOSE EQUIVALENT RATE FOR A MEMBER OF THE POPULATION IN THE UNITED STATES AND CANADA FROM VARIOUS SOURCES OF BACKGROUND RADIATION**

SOURCE	TOTAL EFFECTIVE DOSE EQUIVALENT RATE (mSv yr <sup>-1</sup> )					
	Lung	Gonads	Bone Surf	Bone Marrow	Other Tissues	Total
<i>W<sub>1</sub></i>	0.12	0.25	0.03	0.12	0.48	1.0
Cosmic	0.03	0.07	0.008	0.03	0.13	0.27
Cosmogenic	0.001	0.002	—	0.004	0.003	0.01
Terrestrial	0.03	0.07	0.008	0.03	0.14	0.28
Inhaled	2.0	—	—	—	—	2.0
In Body	0.04	0.09	0.03	0.06	0.17	0.40
Total	2.1	0.23	0.05	0.12	0.44	3.0

From NCRP, 1987.

**Table 21-16. LIFETIME EFFECTIVE DOSE EQUIVALENT (IN mSv FROM BIRTH TO AGE 85) FROM NATURAL RADIONUCLIDE EXPOSURE**

	LUNG	BONE MARROW	WHOLE BODY
Effective Dose Equivalent	180	10	260

From NCRP, 1987.

**Environmental Releases (Chernobyl)**

Large-scale accidents will undoubtedly occur that release substantial radioactivity into the environment. The accident at the Windscale nuclear power reactor in 1957 was a local incident in Great Britain. The nearby population has been studied for over 30 years without appearance of significant health effects.

The accident at the Chernobyl nuclear power

plant was another such occasion but, in this case, the radioactivity was widespread over Europe. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1988) has summarized the committed dose from measurements made in the affected countries and these are shown in Table 21-17. The dose in this case is largely from long-lived <sup>137</sup>Cs (half-life 30 years) both deposited on the ground and taken up by vegetation and directly deposited on vegeta-

Table 21-17. TOTAL CESIUM-137 DEPOSIT AND DOSE COMMITMENTS IN THE NORTHERN HEMISPHERE

REGION	AREA (10 <sup>3</sup> km <sup>2</sup> )	POPULATION (10 <sup>6</sup> )	DISTANCE FROM CHERNOBYL (km)	CESIUM-137 DEPOSITION (kBq/m <sup>2</sup> ) WEIGHTED BY		CS-137 DEPOSIT (PBq)	EFFECTIVE DOSE EQUIVALENT COMMITMENT			
				Area	Population		Per caput (μSv)		Collective (man Sv)	
							First Year	Total		First Year
Europe										
North <sup>a</sup>	1,249	22.8	1,300	8.2	7.0	10.2	210	970	4,700	22,000
Central <sup>b</sup>	1,253	178.0	1,200	7.0	6.0	8.8	280	930	49,000	166,000
West <sup>c</sup>	936	137.7	2,000	1.3	1.0	1.2	48	150	6,600	21,000
Southeast <sup>d</sup>	829	101.6	1,500	8.2	7.2	6.8	380	1200	39,000	121,000
Southwest <sup>e</sup>	596	47.2	2,900	0.03	0.03	0.02	4	7	180	340
USSR	22,190	279.1	—	1.4	5.0	30.9	260	810	72,000	226,000
Asia										
Southwest <sup>f</sup>	4,611	114.9	2,200	1.0	1.0	4.6	70	190	8,000	22,000
South <sup>g</sup>	6,786	108.2	5,400	0.08	0.08	0.5	6	15	6,100	16,000
Southeast <sup>h</sup>	2,575	240.6	7,800	0.03	0.03	0.08	2	6	510	1,400
East <sup>i</sup>	11,720	1268	6,600	0.04	0.04	0.5	3	8	3,600	9,600
America										
North <sup>j</sup>	20,560	347.0	9,000	0.02	0.02	0.4	1	4	490	1,300
Caribbean <sup>k</sup>	216	30.1	9,200	0.018	0.018	0.004	1	3	40	100
Central <sup>l</sup>	517	26.9	10,700	0.012	0.012	0.006	0.7	2	20	60
South <sup>m</sup>	2,520	49.7	10,100	0.013	0.013	0.03	1	2	50	120



**Table 21-18. LIFETIME CANCER MORTALITY PER GRAY FROM FIVE MAJOR EPIDEMIOLOGIC STUDIES (IN PARENTHESES, RISK PER SIEVERT FOR ALPHA EMITTERS)**

STUDY	ALL SITES	LEUKEMIA	LUNG	FEMALE BREAST	BONE	THYROID	SKIN
Atom bomb whole body gamma	0.029	0.0067	0.0038	0.0029			
Uranium miner bronchial epithelium alpha			0.04 (0.0020)				
Ankylosing spondylitis spine X ray		0.0011	0.0008- 0.0028	0.0015			
Tinea capitis head X ray						0.0010 <sup>§</sup> 0.0040 <sup>  </sup>	0.0030 <sup>‡</sup>
Radium ingestion bone alpha ( <sup>226</sup> Ra)					0.0040* (0.0002)		
Radium ingestion bone alpha ( <sup>226</sup> Ra)					0.02 <sup>†</sup> (0.0010)		

\* The lifetime risk is calculated for an average skeletal dose of 10 Gy, assuming the risk to persist for 50 years and using equation 20.

† The risk is nonlinear and is about 0.01 Gy<sup>-1</sup> at 100 Gy, for example.

‡ The lifetime risk is calculated for an average skeletal dose of 10 Gy using equation 22. The risk is nonlinear and is about 0.01 Gy<sup>-1</sup> for a skeletal dose of 1 Gy.

§ The mortality for skin cancer is estimated as 1 percent of the incidence; see text.

|| Thyroid mortality for males. Estimated as 10 percent of incidence.

|| Thyroid mortality for females. Estimated as 10 percent of incidence.

tion. Cesium behaves similarly to potassium in the body and so the dose is essentially to the whole body.

There are plans to follow-up the health effects in the local population affected by Chernobyl.

### SUMMARY OF HUMAN CANCER RISKS FROM RADIATION

The details of the five major studies have been given in the preceding sections. The data are summarized in Table 21-18. This shows the lifetime cancer risks which are significant. The risks are given in units of per Gray (or per Sievert where appropriate for alpha emitters).

Within the table, leukemia and cancers of the lung and female breast are the most critical. Osteogenic sarcoma is seen in the radium exposures. There is no clear linear dose response for <sup>224,226</sup>Ra. This has been attributed to the existence of an apparent threshold.

The cancer risk to individual organs from dif-

ferent study groups is in general agreement regardless of radiation type or whole or partial body exposure.

### ADDENDUM

In 1990 the International Commission on Radiation Protection (ICRP) circulated a draft document (ICRP, 1990) which is to be adopted and published by the Commission in 1991. This document includes new estimates of risk for both fatal and nonfatal cancer and new guidelines for exposure to workers from external and internal radiation.

The ICRP document is a response to the increase in cancer risk from ionizing radiation observed in atomic-bomb survivors. Mental retardation is a recent finding in the atomic-bomb survivor cohort and this is now included in the risk estimates. There are no conclusive data from A-bomb survivors on cancer in the thyroid, bone, liver, and skin. Therefore, the risks to

**Table 21-19. NOMINAL RISK COEFFICIENTS FOR STOCHASTIC EFFECTS\***

BIOLOGICAL EFFECT (10 <sup>-2</sup> Sv <sup>-1</sup> )	EXPOSED POPULATION	RISK COEFFICIENT
Fatal cancer	Adult workers	4.0
Fatal cancer	Whole population	5.0
Serious hereditary effects	Adult workers	0.6
	Whole population	1.0
Mental retardation	8-15 week conceptus	30 IQ points Sv <sup>-1</sup>

\* From ICRP, 1990.

**Table 21-20. NOMINAL PROBABILITY COEFFICIENTS AND TISSUE WEIGHTING FACTORS FOR INDIVIDUAL TISSUES AND ORGANS\***

TISSUE OR ORGAN	CANCER LETHALITY FRACTION K	NOMINAL PROBABILITY COEFFICIENT (10 <sup>-2</sup> Sv <sup>-1</sup> )				TISSUE WEIGHTING FACTOR w <sub>T</sub>
		WHOLE POPULATION		WORKING POPULATION		
		Fatality coeff F	Weighted effect coeff F (2-K)	Fatality coeff F	Weighted effect coeff F (2-K)	
Bone marrow <sup>†</sup>	0.99	0.45	0.91	0.36	0.73	0.12
Bladder	0.50	0.20	0.30	0.16	0.24	0.04
Bone surface	0.70	0.05	0.07	0.04	0.05	0.01
Breast	0.50	0.25	0.38	0.20	0.30	0.05
Colon	0.55	0.95	1.38	0.76	1.10	0.18
Gonads	—	—	1.00	—	0.60	0.13
Liver	0.95	0.20	0.21	0.16	0.17	0.03
Lung	0.95	0.90	0.95	0.72	0.76	0.13
Oesophagus	0.95	0.35	0.37	0.28	0.29	0.05
Skin	0.01	0.02	0.04	0.016	0.03	0.01
Stomach	0.90	1.10	1.21	0.88	0.97	0.16
Thyroid	0.10	0.08	0.15	0.06	0.11	0.02
Remainder <sup>‡</sup>	0.80	0.45	0.54	0.36	0.43	0.07
Total (rounded)		5.00	7.49 7.5	4.00	5.79 6.0	1.0

\* From ICRP, 1990.

† The values of w<sub>T</sub> relate to a population of equal numbers of both sexes and a wide range of ages. The coefficients relate only to adults.

‡ Relates to red bone marrow and includes extra weighting by a factor of 2 for short mean latency of leukaemia.

§ The dose equivalent in the remainder is the estimated mean dose equivalent over the whole body excluding the specified tissues and organs.

these tissues are developed from other epidemiological studies discussed in this chapter.

The overall risk per unit exposure for adult workers and the whole population given in the draft document are shown in Table 21-19. The risk of fatal cancer is adopted as 0.04 per Sievert (4 percent per Sievert) for adult workers and 0.05 per Sievert (5 percent per Sievert) for the whole adult population.

ICRP had been criticized for excluding the effects of nonfatal cancer in previous documents. An attempt to correct this omission is made in the present document. A cancer lethality fraction, K (the fraction of total cancer that is lethal), is used as a weighting factor for nonfatal cancers in deriving the total effect risk coefficient. The cancer risk per Sievert, F, and the total weighted risk per Sievert (weighted effect coefficient), F(2-K), are shown in Table 21-20. The reasoning given for the weighted effect coefficient is given as follows.

The total number of cancers (fatal plus nonfatal) Sv<sup>-1</sup> will be F/K. The total number of nonfatal cancers is (1-K)F/K and multiplying by the

weighting factor K yields a "health detriment" of (1-K)F. The total health detriment is then

$$F + F(1-K) = F(2-K).$$

The total risk including this weighting for nonfatal cancer is proposed as 7.5 Sv<sup>-1</sup> for the whole adult population and 6.0 Sv<sup>-1</sup> for the working population.

The tissue weighting factors corresponding to those derived formerly in ICRP (1977) and shown in Table 21-6, are the organ fraction of the total.

In assessing radiation risk from low-dose, low-dose rate, low LET radiation using risk coefficients such as in Table 21-20 derived from high dose, high-dose rate exposures, a dose rate reduction factor (DREF) needs to be applied. NCRP (1980) and UNSCEAR (1988) have shown that the human data cover a range for the DREF of 2 to 10. That is, the risk coefficients in Table 21-20 are conventionally divided by the DREF factor. ICRP has previously used 2.5 as the adopted DREF, however, the draft document proposes to adopt a DREF of 2.0.

The new occupational guidelines for radiation developed from this ICRP document are proposed as 100 mSv in 5 years with a limit of 50 mSv in any single year. This is compared with the 1977 limit of 50 mSv per year.

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