N69-12437

3. IONIZING RADIATION

Operational evaluation of the radiation hazard to man is dependent on the nature and timing of the radiation flux incident on the vehicle, the composition and three-dimensional thickness of the shielding, the geometrical position of the human behind the shielding, the self-shielding of the body, the current-to-dose conversion factors in the body, and the pathophysiology of radiation damage. The following are data which may be used for calculation of specific organ doses, radiation shielding, and mission risk-hazard analysis for man. Emphasis will be placed on the interaction between radiation and man. The physical nature of space radiation and the molecular aspects of radiobiology will be covered only as they contribute directly to the evaluation of radiobiological risks and operational problems.

SPACE RADIATIONS

The physical nature of the radiation environment has received much study and review. Space radiation can be defined as falling into three categories: primary cosmic or galactic radiation; the geomagnetically trapped radiations of the Van Allen belts; and solar wind and flare events. Cosmic radiation is now preferably called galactic radiation to distinguish it from trapped radiation and solar-particle fluxes. The current hypotheses assume that these nuclei have traveled for eons in galactic space, gradually being accelerated in turbulent magnetic fields or arise from supernovas or other events near the center of our own galaxy. The galactic radiation spectrum has been described in great detail but is continually changing as new data are obtained from geophysical satellites and other sources (87, 90, 162, 243). Table 3-16 presents the composition of the primary cosmic-ray flux outside the atmosphere at northern latitudes. The truly primary galactic radiation (i. e., the flux containing no secondaries produced locally in a compact absorber), consists of 85 percent hydrogen nuclei (protons, 14 percent helium nuclei (alpha particles), and 1 percent of the heavier nuclei up to iron (Z = 26) as regular constituents and so-called superheavy nuclei beyond iron occasionally recorded. These particles transfer energy to matter by ionization along their pathway, by terminal absorption ("thindowns"), and by inelastic collision ("star formation"), which result in highly localized areas of very dense ionization. The intensity of galactic radiation in the vicinity of the Earth varies with the 11-year solar cycle, with magnetic storms, and possibly with the appearance of supernovae near the solar system.

Trapped particle radiation is a problem in the Van Allen belts, where protons and electrons are injected and trapped in the Earth's magnetic field. Though the topological conditions and direction of injection are still obscure, it is currently thought that the trapped particles arise from protons and electrons from the primary cosmic-ray beam backscattered from the Earth's atmosphere - the source of the bulk of this component is neutrons decaying into protons and electrons; protons from the solar wind; and primary cosmic-ray protons, which are assumed to be of minor importance.

Estimation of exposures during space flight in these toroidal belts is most difficult because of the complicated geomagnetic and particle interactions determining the geometric distribution of the radiation. Magnetic storms can influence the nature of the belts (163, 235 , 296). The inner belts contain not only protons, but sizable fluxes of soft electrons of natural origin and from fission electrons injected into the trapping region by high altitude nuclear weapons testing.

A summary of the particles' energies has been recently presented from which the following data are taken directly (145). In going vertically up from sea level at the geomagnetic equator, the flux of trapped protons in the inner belt is found to start rising sharply at 1.16 Earth radii (about 1,000 km altitude above sea level) and reaches a first maximum at 1.5 Earth radii (3,185 km), with an integral flux of slightly more than 10,000 protons/cm^2 sec of 40 MeV minimum energy. In the heart of the inner belt, a flux of 2,000 protons/cm^2 sec per unit solid angle in the energy interval from 40 to 110 MeV, corresponds to a range interval in aluminum from 2 to 12 g/cm^2. Since the radiation sensors on Relay I discriminated fluxes only in the energy intervals from 1.1 to 14 MeV, from 18.2 to 35 MeV, and from 40 to 110 MeV, the slope in the last interval can only be inferred indirectly by extrapolation plotting of the three flux fractions. The flux then drops slightly and reaches a second flat maximum smaller than the first one at 2.2 Earth radii and finally drops below 100 protons/cm^2 sec at 2.8 Earth radii. The corresponding altitude profiles toward higher magnetic latitudes show gradually smaller fluxes up to geomagnetic latitude 37.5°, which marks the boundary at which the fluxes of high-energy protons and electrons drop to insignificant levels.

A phenomenon of special importance for satellite missions in near-Earth orbits is the so-called South Atlantic anomaly. It is a region where the mirror points of the trajectories of trapped protons in the inner belt dip down more closely to the Earth than at any other longitude, due to an asymmetry of the geomagnetic field. Dose rates below 1.5 g/cm^2 shielding come close to 100 mrads/hr at altitudes as low as 120 miles, as direct dose-rate measurements on the Gemini IV mission indicate. Since the point of intersection of a satellite orbit with the geographic equator continuously drifts westward due to the rotation of the Earth, any mission comprising a large enough number of revolutions passes through the anomaly on some orbits. Although the time of a single passage is less than 15 min and the accumulated passage time on a mission of many orbits remains well below 10 percent of the total time in orbit, the proton exposure in the anomaly accounts for more than 90 percent of the total exposure. The accumulated exposure in the anomaly will be a limiting factor for long-duration, low-orbital missions.

A large fraction of the artifically injected electron fluxes shows lifetimes of at least several years, and it might still take 30 years for the natural radiation levels to be restored. It now seems reliably established that electrons injected into regions of $L<1.2$ Earth radii, corresponding to an altitude of 1,275 km above sea level at the geomagnetic equator, are rapidly removed by the Earth's atmosphere. Electrons injected into L regions beyond 1.7 (4,460-km altitudes) are also removed comparatively rapidly by magnetic fluctuations due to solar activity. These are the same fluctuations that cause

the large variations of natural flux levels noted above. In the intermediate altitude region between 1.2 and 1.7 L, however, artificially injected electrons become semipermanently trapped with 1/e decay times of the order of 5 to 10 years. In January 1963, the flux at 1.5 L in the plane of the geomagnetic equator was 5×10^8 electrons/cm^2 sec for the energy band from about 0.5 to 5 MeV and 1.5×10^7 electrons/cm^2 sec for the band higher than 5 MeV. The combined exposure hazard from protons and electrons in the heart of the artificial electron belt, which happens to coincide with the heart of the natural inner proton belt, was predominantly due to electrons 6 months after creation of the artificial belt. Whereas the decay times for the fluxes in the outer regions and the inner fringes of the artificial belt are on the order, respectively, of months and weeks, they grow to the order of at least several years in the center of the belt. In the natural outer belt of electrons, fluxes show large irregular fluctuations. At 4.0 L (sea-level altitude of 3 Earth radii at the magnetic equator), peak electron intensities closely approach the maximum values found in the heart of the artificial belt at 1.5 L.

Models of the trapped radiation environment are available as are computer codes for their use in establishing dose rates (91, 184, 284, 285).

Solar plasma comes from the Earth in three ways: the solar wind, solar corpuscular streams, and solar flare emissions or plasma shells. Solar wind is a perpetual outflow from the solar corona all over the sun. The solar wind is distinguished from the other two flows which are called ejected flows. The magnetic fields and low energy particles of the solar wind have been recently reviewed (163, 195, 235). The solar wind does not appear to represent a significant human hazard.

The solar corpuscular stream is an intermittent emission continuing for weeks and months, apparently from the same region of the sun. These spiral streams interact with the solar wind and the cosmic galactic radiation (163). The solar flare emissions are of much shorter duration, usually lasting about an hour or less. The solar ejected flows vary from one event to the next in the differential energy spectrum and intensity of the proton and alpha particles. Analyses of these spectral data are available (14, 30, 93, 142, 162, 242, 295). The proton:alpha flux ratio for equal rigidity intervals in four events were 1:1 and in 3 events were >5:1. Calculations of the exponential rigidity-flux relationship and rigidity-kinetic energy relationship for the time-integrated spectra are available (184). Values of P_o or characteristic rigidity are uniformly distributed between 45 and 150 mV for all events of solar cycle 19 when the largest solar particle events were recorded. (See discussion of Table 3-33). The typical characteristic rigidity is 100 mV and may be used for first-order dose computations. Data are now available on large events during the quiet sun period (237).

Reviews of Soviet studies on the geophysical aspects of space radiations are available (35, 255).

NOMENCLATURE AND DOSAGE FACTORS

The basic terms for expressing the exposure field and absorbed dose of radiation are seen in Figure 3-1. RBE (relative biological effectiveness)

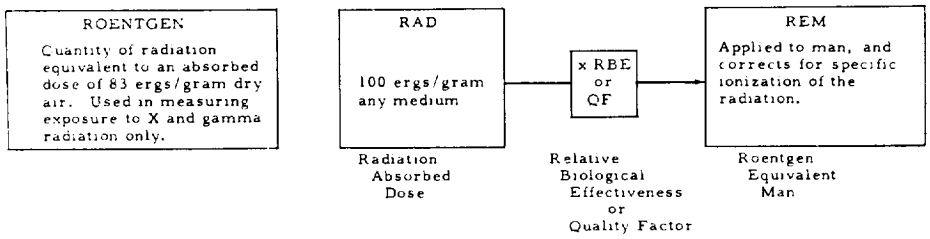


Figure 3-1

Radiation Terms

expresses the effectiveness of a particular type of radiation in producing the same specific biological response as 250 kVp X-radiation or gamma radiation having a linear energy transfer (LET) equivalent to 3.5 kilovolts per micron of water and delivered at the rate of about 10 rads per minute. When an RBE is used not for a specific biological endpoint but for general considerations of health protection, it is referred to as QF or quality factor (41). The exact degree of relative effectiveness is dependent also on the criterion of effect, the tissue or organ of interest, the dose rate, and whether the response is early or delayed (41, 76). High-LET radiations (arbitrarily taken as those radiations having a mean LET greater than 3.5 keV/μ) are more effective per rad than the conventional x and gamma rays normally used as standard radiations. For late or delayed effect of low dose rate, the QF-LET relationship shown in Table 3-2 has been proposed (122). To a first approximation,

Table 3-2

Values of QF_L for Late or Delayed Effects as a Function of Average LET

(After ICRP(122))

LET_∞ (keV/μ IN WATER)	QF
X rays and electrons of any LET	1
3.5 or less	1
3.5–7	1–2
7–23	2–5
23–53	5–10
53–175	10–20

this relationship may be represented as:

$$QF_L = 0.8 + 0.16\,\bar{L} \qquad (1)$$

where $\bar{L}$ is the mean LET in keV/μ. No official committee or organization has made specific recommendations with respect to the QF-LET relationship for early effects. Accurate calculation of the biological effect should refer to detailed LET spectra where available.

In general, early responses to large doses delivered at high dose rates are less dependent on LET than are late responses to low doses at low dose rates. The relationship generally follows the equation:

$$QF_E = 0.9 + 0.05\,\bar{L} \qquad (2)$$

For gross evaluation it is suggested that the general QF_E-LET relationships shown in Table 3-3 be applied to early responses from high-intensity space-radiation exposure (145). For computer programming of more exact dosage

Table 3-3

Suggested QF_E Values for Early Effects of High-Intensity Space-Radiation Exposure

(After Langham (ed.)-NAS-NRC[145])

	COMPONENT	QF
Skin responses	Low LET ($\leq$ 3.5 keV/μ)	1
	High LET ($>$ 3.5 keV/μ)	3
Prodromal syndrome	Total flux	1
Hematological responses	Total flux	1
Lethality, hematological syndrome	Total flux	1
Lethality, intestinal syndrome	Low LET ($\leq$3.5 keV/μ)	1
	High LET ($>$ 3.5 keV/μ)	3
Atrophy of germinal epithelium	Low LET ($\leq$ 3.5 keV/μ)	1
	High LET ($>$ 3.5 keV/μ)	3

schedules under operational conditions, more specific energy-LET-QF relationships can be used when available. It must be kept in mind that RBE or QF is dose-rate dependent (41 , 76). In general, radiation of low LET tends to be more sensitive to dose rate factors than does radiation of high LET. Particles of high LET may have higher RBE or QF at low dose rates than at higher dose rates. Specific examples will be noted below. In all cases, the biological dose equivalent for late and early responses should be calculated separately.

Examples of RBE or QF values for late effects at low dose rates used in ground laboratories are seen in Table 3-4. In some animal studies, RBE's of over 35 have been reported for genetic changes after neutron irradiation (251). At high dose rates, the QF_E of Table 3-3 may be used for neutrons and the other radiations of Table 3-4 (134).

Table 3-4

Typical QF_L for Late Effects at Low Dose Rate in Ground-Based Exposure

Type of radiation	RBE or QF
X-rays	1
Gamma rays	1
Beta particles, 1.0 mev	1
" " 0.1 mev	1
Neutrons, thermal energy	2.8
" 0.0001 mev	2.2
" 0.005 mev	2.4
" 0.02 mev	5
" 0.5 mev	10.2
" 1.0 mev	10.5
" 10.0 mev	6.4
Protons, greater than 100 mev	1 - 2
" 1.0 mev	8.5
" 0.1 mev	10
Alpha particles, 5 mev	15
" " 1 mev	20

For more specific calculations of organ doses in experimental ground-based studies, the energy-LET-QF relationship will be covered in more detail below.

Reference Equivalent Space Exposure (RES)

Quantitative evaluation of the factors that modify radiation responses is singularly the greatest uncertainty in establishing human response criteria for space radiation exposure. The most obvious modifying factors are radiation quality, dose rate (as influenced both by protraction and fractionation), and dose distribution (both topical and depth). For space applications it has been suggested that "dose equivalent" in "rems" used in conventional occupational radiation protection, be replaced by "reference equivalent space exposure" (RES) in "reference equivalent units" designated "reu" (145). Conceptually, the method of evaluation is the same as that employed in conventional radiation protection. The space radiation dose (D) is multiplied by a radiation quality factor (QF) and subsequently by other appropriate modifying factors to give the reference equivalent space exposure:

$$RES\ (reu) = D\ (rads) \times QF \times (f_1 \cdot f_2 \cdots f_n), \qquad (3)$$

where $f_1 \cdots f_n$ are the appropriate modifying factors for early vs. late effects, dose protraction, and dose distribution of the particular response being evaluated. In principle, this procedure is applicable to evaluation of both early and late radiation responses. However, inadequate knowledge of the quantitative influence of the relevant modifying factors such as dose dis-

tribution and dose protraction and their interdependence necessitates the choice of a set of values for each specific situation on the basis of rather arbitrary simplications and generalizations.

Dose Distribution Factors

Dose distribution in space radiation exposure will be highly non-uniform with respect to depth, area, volume, and region or organ systems exposed. More detailed evaluation will be made for each organ system. At present, only very arbitrary simplifying generalizations will be made.

With respect to depth-dose distribution the acquired dose is calculated or measured at the average depth or anatomical site (volume) of interest for the particular response. The point of interest for skin responses is at a depth of 0.1 mm; for hematological depression, a depth of 5 cm; for hematopoietic and gastrointestinal lethality, a depth of 11 cm; and for prodromal response and general physiological injury a 15-cm diameter sphere in the mid-epigastric region. At these depths the penetration factor (f_p) will be considered to be one.

With regard to region or volume exposed, it has been suggested for prodromal, hematological and early lethal responses a dose involving a major portion of the trunk be considered as capable of eliciting full response and be assigned a "volume factor" (f_v) of one (145). Exposure of the extremities exclusive of the trunk would be much less effective. Based on fraction of total body mass and active bone marrow, an arbitrary choice of a f_v of 1/5 might be suggested for the extremities when only the hematological response is considered.

Skin and germinal epithelium responses must be considered specifically. A severe response of even a small skin area, regardless of location, could be highly uncomfortable particularly under a space suit. Furthermore, dose values are usually established for skin areas of $\sim$35 to 100 cm^2; and early erythema and desquamation are somewhat area-dependent up to $\sim$300 to 400 cm^2. The area-effect over this range amounts to an increase in effective dose of $\sim$25 percent. It is suggested, therefore, that an area-effectiveness factor (f_a) of $\sim$1.25 be applied to the doses given when exposure involves skin areas greater than $\sim$150 cm^2.

In the case of the germinal epithelium response is considered a local effect. Because of the limited size and localization of the testicles, it is reasonable to assume that they either will or will not be in the exposure field in which case f_v will either be unity or zero. In mentioning the germinal epithelium it is emphasized that the response in this case is considered of no significance in evaluating risk of early performance decrement but may have social or emotional significance to the astronaut.

Dose Protraction

The effect of dose protraction has been studied to some degree for most of the early signs and symptoms (32 , 41 , 73 , 148 , 172 , 191 , 201 , 205, 228, 231 , 238). Some suggestions are possible regarding general "dose-effectiveness" factors (f_r) that are useful for exposure periods up to three or four weeks. To derive the factors, it has been assumed that the decrease per rad in biological effect associated with a dose-rate decrease can be compensated for by an increase in total dose required to produce the given effect (145). Thus the ratio of total doses required for low dose-rate versus high dose-rate exposure will determine the "dose-rate-effectiveness" factors. This is not the same as taking a ratio of dose rates, however, since for some tissues the latter may change by a factor of 10 or more while being accompanied by a change in effect of only about 2. There is also considerable difference in protraction period over which the change from maximum to minimum effect occurs in the various tissues and systems.

Table 3-5 attempts to encompass all of these variables for exposure periods varying from a few hours to a few weeks with respect to total doses

Table 3-5

Suggested Dose-Rate or "Rate-Effectiveness" Factors (f_r) for Early Responses Following Exposure to Low-LET Radiations at High Intensity. (See text for specific definitions)

(After Langham (ed.)-NAS-NRC[145])

	Duration of Exposure to Produce Same Response Level		
	Skin Erythema and Desquamation	Prodromal Signs	Hematological Depression and Lethality
A. High Dose-Rate			
Duration of Exposure for Maximum Effectiveness	1 - 2 hours or less	2 - 4 hours or less	1 - 2 days or less
B. Low Dose-Rate			
Duration of Exposure for Minimum Effectiveness	4 - 6 days or longer	2 - 4 days or longer	3 - 4 weeks
Ratio of Total Doses to Produce Same Response Level (B/A)	3	2.5	2
Rate-Effectiveness Factor (f_r)	1/3	1/2.5	1/2

high enough to elicit the more significant early responses. The rate-effectiveness factors (f_r) are given as the reciprocals of the ratios of total doses.

In general, "maximum dose" implies no repair is possible. For the prodromal syndrome, fractionated doses longer than 7 days apart can be considered as single doses with no residual effects from the prior doses. For the hematological syndrome, the range of population sensitivity will spread with prolongation beyond 3-4 weeks and an increasingly larger fraction will show no effects as the doses are separated by longer intervals. The terms

"high" dose rate and "low" dose rate are difficult to define for all situations. What is considered a high and a low dose rate for early responses might be quite different from high and low dose rates for progressive and late responses. Furthermore, a high and a low dose rate for early skin responses might be different from those for early hematological and prodromal responses. In general, Table 3-5 attempts to take these variations into consideration for the important early responses. As an example of the use of this table, a given prodromal sign (e.g., nausea) may have a 10 percent probability of occurrence following an exposure of 50 rads delivered over 2 to 4 hours (dose rate 12 to 25 rads/hr), while an exposure of 125 rads (2.5 x 50) would produce the same probability of response if the dose was protracted over 2 to 4 days (dose rate 30 to 60 rads/day). It is suggested, therefore, that the space radiation dose (D) be multiplied by the appropriate rate-effectiveness factor (f_r) from Table 3-5 to evaluate RES when exposures are protracted over periods comparable to those specified.

Radiation recovery rates are influenced by LET and for this reason, the f_r values given in Table 3-5 are specified for low-LET radiations. Technically, the rate-effectiveness factors should be applied only to the low-LET components of space radiation. Correction of f_r for LET appears unnecessary under shielding conditions that result in only a small fraction of the absorbed dose at the site of interest being delivered at high LET. Information on early skin responses suggests, however, that the slopes of the time-dose response curves decrease with increasing LET. For early skin responses under exposure conditions of very light to nominal shielding, where from $\sim$ 10 to $\sim$ 75 percent of the absorbed dose at 0.1-mm depth from solar flare events may be due to densely ionizing components, adjustments should be made by assuming f_r is unity for that fraction of the dose delivered at or above some arbitrary cut-off for high LET e.g., $\sim$ 15 keV/μ.

Application of the information given in this section to an evaluation of a risk of early performance decrement may be illustrated (using skin erythema as the early response) by a hypothetical mission during the triplet solar flare event of July 10-16, 1959. No attempt is made to make the assumptions conform necessarily to the actual conditions. If it is assumed that the average effective shielding of the spacecraft was 2 g/cm^2, the accumulated skin dose (D) during the 6-day period of the triplet flare would have been 674 rads. Let it be assumed also that the QF_E was 1.45, the skin area involved was a major portion of the front surface of the body, the dose received was measured at a depth of 0.1 mm, and 25 percent was delivered at a LET of >15 keV/μ. Under these conditions, QF_E = 1.45, f_a = 1.25, f_p = 1, and f_r = (1/3 x 0.75 + 1 x 0.25) = 0.5. The reference equivalent space exposure, evaluated from Equation 1 would be:

$$RES = 674 \times 1.45 \times 1.25 \times 1 \times 0.5 = 610 \text{ reu.}$$

Since 1 reu is equivalent in effectiveness to 1 rad of reference radiation, comparison of RES with the reference radiation dose-response relationship given in Table 3-47 suggests that the probability of an erythema response under the specified conditions would have been of the order of 50 percent.

Since most exposures to radiation in space flight are expected to be at low dose rates, the dose factors noted for early effects following high dosage of radiation must be modified. Under space flight conditions a gradually accumulating injury to the bone marrow may be expected. Since injury and recovery may be concurrent for long periods of time, the dose distribution and protraction factors applied to acute injury cannot be directly applied to this situation. Suggestions for dosage factors covering these "progressive performance decrements" will be presented in a separate section below. Dosage factors for late or delayed injury following acute exposures will also be covered as a separate entity.

INTERACTION OF RADIATION WITH BIOLOGICAL MATERIALS

In order to calculate the rate of energy transfer, QF or RBE, tissue range, and stopping power of space radiations, data are needed on the interaction with tissue. Since the bulk of absorption takes place in bone or muscle, much of the data have been generated for these model tissues. Table 3-6 represents the model composition of muscle and bone which can be used in the many calculations noted above.

To apply the general purpose nucleon transport codes to the calculation of usable current-to-dose conversion factors of sufficient generality of application, the nuclear density must be known. A tissue of composition $C_{21}H_{140}O_{57}N_3$ with a density of 1 gm/cm^3, results in the nuclear densities seen in Table 3-7. The average ionization potentials can be used in the stopping-power formulas for the computation of particle ranges in tissue.

Energy-LET and Quality Factors

The stopping power and rate of energy loss given as MeV/cm or keV/micron (LET) for protons in standard muscle and bone is given in Table 3-8. The calculations of DE/DZ, DE/DX, and LET (127) were made from the ICRU data of Table 3-6 (190), and the energy loss equations of references 11 , 17 , and 218. The resulting information can be applied to the dosimetric measurement of the protons found in the space environment and can be used in the design of tissue-equivalent radiation detectors. Similar data on proton path length, straggling factors, percent scattering, and probability of inelastic nuclear interaction are available for muscle and other materials (125).

When particles of high energy pass through solid materials, star formation occurs with the release of many nucleons and mesons of different type. These stars are not as frequently generated in biological materials because of the low Z values. The secondary particles from wall materials can pass through biological tissue. Tables 3-9 and 3-10 represent the stopping power and range of muons, pions, kaons, and protons in muscle and bone for a wide range of energies. Powers of 10 are indicated by the symbol E; e.g., 1.234E02 means 1.234×10^2. The mean excitation energy, I_{adj}, (in eV) is indicated by the symbol I. These data for protons replace previous data

Table 3-6

Composition of ICRU Muscle and Bone

Data compiled by Janni[127] from the Report of the International Commission on Radiological Units and Measurements (ICRU)[190]

a. Muscle*

Element	Atomic number	Atoms/ molecule	Percent by weight	Atomic weight
H	1	10.11905	10.20	1.01
C	6	1.02415	12.30	12.01
N	7	0.24986	3.50	14.01
O	8	4.55625	72.90	16.00
Na	11	0.00348	0.08	23.00
Mg	12	0.00082	0.02	24.33
P	15	0.00646	0.20	30.98
S	16	0.01559	0.50	32.07
K	19	0.00767	0.30	39.11
Ca	20	0.00017	0.01	40.09

b. Bone**

Element	Atomic number	Atoms/ molecule	Percent by weight	Atomic weight
H	1	6.3491	6.40	1.01
C	6	2.31474	27.80	12.01
N	7	0.19275	2.70	14.01
O	8	2.5625	41.00	16.00
Mg	12	0.00822	0.20	24.32
P	15	0.22599	7.00	30.97
S	16	0.00628	0.20	32.07
Ca	20	0.36677	14.70	40.08

* Density of muscle is 1 gram per gm/cm^3; electron density 3.313×10^{23} electrons/gm

** Density of bone is 1.85 gram per gm/cm^3; electron density 3.193×10^{23} electrons/gm

Table 3-7

Composition and Mean Excitation Potentials for Model Tissue

(After NBS[189], Kinney and Zerby[139])

Element	Nucleon density (nuclei/cm^3) $\times 10^{24}$	Mean excitation potential, eV
H	6.265×10^{-2}	17.5
O	2.55075×10^{-2}	99.0
C	9.3975×10^{-3}	74.44
N	1.3425×10^{-3}	86.0

Table 3-8

Stopping Power and Rate of Energy Loss of Protons
in Standard Muscle and Bone

(After Janni[127])

a. ICRU Muscle

PROTON ENERGY MEV	DE/DZ MEV CM2/GM	DE/DX MEV/CM	L E T KEV/MICRON
0.50	408.62	408.62	40.86
0.60	365.03	365.03	36.50
0.70	331.02	331.02	33.10
0.80	302.30	302.30	30.23
0.90	279.13	279.13	27.91
1.00	259.74	259.74	25.97
2.00	158.70	158.70	15.87
3.00	117.37	117.37	11.74
4.00	94.26	94.26	9.43
5.00	79.28	79.28	7.93
6.00	68.72	68.72	6.87
7.00	60.83	60.83	6.08
8.00	54.67	54.67	5.47
10.00	45.73	45.73	4.57
20.00	26.08	26.08	2.61
30.00	18.76	18.76	1.88
40.00	14.87	14.87	1.49
50.00	12.44	12.44	1.24
60.00	10.77	10.77	1.08
80.00	8.62	8.62	0.86
100.00	7.28	7.28	0.73
200.00	4.49	4.49	0.45
300.00	3.51	3.51	0.35
400.00	3.03	3.03	0.30
500.00	2.74	2.74	0.27
800.00	2.33	2.33	0.23
1000.00	2.21	2.21	0.22

Table 3-8 (continued)

b. ICRU Bone

PROTON ENERGY MEV	DE/DZ MEV CM2/GM	DE/DX MEV/CM	L E T KEV/MICRON
0.50	369.42	683.43	68.34
0.60	329.59	609.75	60.97
0.70	298.75	552.69	55.27
0.80	273.26	505.53	50.55
0.90	252.56	467.25	46.72
1.00	235.24	435.19	43.52
2.00	144.91	268.08	26.81
3.00	107.65	199.15	19.91
4.00	86.68	160.36	16.04
5.00	73.02	135.10	13.51
6.00	63.41	117.31	11.73
7.00	56.21	103.99	10.40
8.00	50.58	93.58	9.36
10.00	42.38	78.41	7.84
20.00	24.29	44.94	4.49
30.00	17.51	32.40	3.24
40.00	13.90	25.72	2.57
50.00	11.64	21.54	2.15
60.00	10.09	18.67	1.87
80.00	8.08	14.95	1.49
100.00	6.83	12.64	1.26
200.00	4.22	7.80	0.78
300.00	3.31	6.12	0.61
400.00	2.85	5.27	0.53
500.00	2.58	4.77	0.48
800.00	2.20	4.06	0.41
1000.00	2.08	3.85	0.39

Table 3-9

Stopping Power, MEV/CM^2/G

(See text for explanation of symbols)

(Adapted from Berger and Seltzer[24])

	MUON		PION		KAON		PROTON	
ENERGY MEV	MUSCLE I= 66.2	BONE I= 85.1	MUSCLE I= 66.2	BONE I= 85.1	MUSCLE I= 66.2	BONE I= 85.1	MUSCLE I= 66.2	BONE I= 85.1
2.0	2.901E 01	2.680E 01	3.638E 01	3.353E 01	1.029E 02	9.356E 01	1.679E 02	1.513E 02
4.0	1.651E 01	1.533E 01	2.070E 01	1.918E 01	5.740E 01	5.232E 01	9.876E 01	8.980E 01
6.0	1.193E 01	1.110E 01	1.491E 01	1.385E 01	4.158E 01	3.827E 01	7.049E 01	6.424E 01
8.0	9.520E 00	8.870E 00	1.185E 01	1.102E 01	3.293E 01	3.038E 01	5.509E 01	5.037E 01
10.0	8.023E 00	7.483E 00	9.939E 00	9.258E 00	2.746E 01	2.538E 01	4.622E 01	4.249E 01
14.0	6.256E 00	5.842E 00	7.680E 00	7.164E 00	2.088E 01	1.935E 01	3.520E 01	3.245E 01
18.0	5.243E 00	4.901E 00	6.380E 00	5.957E 00	1.703E 01	1.581E 01	2.869E 01	2.651E 01
22.0	4.587E 00	4.290E 00	5.533E 00	5.170E 00	1.448E 01	1.346E 01	2.437E 01	2.255E 01
26.0	4.127E 00	3.862E 00	4.936E 00	4.615E 00	1.267E 01	1.179E 01	2.127E 01	1.970E 01
30.0	3.787E 00	3.545E 00	4.494E 00	4.204E 00	1.131E 01	1.053E 01	1.893E 01	1.756E 01
34.0	3.526E 00	3.302E 00	4.153E 00	3.886E 00	1.025E 01	9.549E 00	1.711E 01	1.588E 01
38.0	3.319E 00	3.108E 00	3.882E 00	3.634E 00	9.403E 00	8.762E 00	1.564E 01	1.452E 01
42.0	3.152E 00	2.952E 00	3.662E 00	3.429E 00	8.705E 00	8.115E 00	1.443E 01	1.341E 01
46.0	3.015E 00	2.825E 00	3.480E 00	3.259E 00	8.122E 00	7.574E 00	1.341E 01	1.247E 01
50.0	2.901E 00	2.718E 00	3.326E 00	3.114E 00	7.627E 00	7.115E 00	1.255E 01	1.167E 01
60.0	2.682E 00	2.513E 00	3.034E 00	2.842E 00	6.663E 00	6.220E 00	1.086E 01	1.011E 01
70.0	2.531E 00	2.368E 00	2.827E 00	2.649E 00	5.962E 00	5.569E 00	9.630E 00	8.972E 00
80.0	2.421E 00	2.266E 00	2.672E 00	2.504E 00	5.429E 00	5.073E 00	8.687E 00	8.098E 00
90.0	2.337E 00	2.188E 00	2.556E 00	2.390E 00	5.010E 00	4.683E 00	7.942E 00	7.408E 00
100.0	2.272E 00	2.128E 00	2.465E 00	2.306E 00	4.671E 00	4.369E 00	7.338E 00	6.847E 00
110.0	2.222E 00	2.081E 00	2.391E 00	2.238E 00	4.393E 00	4.109E 00	6.838E 00	6.383E 00
120.0	2.182E 00	2.044E 00	2.331E 00	2.183E 00	4.159E 00	3.892E 00	6.418E 00	5.992E 00
130.0	2.150E 00	2.014E 00	2.282E 00	2.137E 00	3.961E 00	3.707E 00	6.059E 00	5.659E 00
140.0	2.123E 00	1.989E 00	2.241E 00	2.099E 00	3.790E 00	3.549E 00	5.749E 00	5.371E 00
150.0	2.102E 00	1.968E 00	2.207E 00	2.067E 00	3.642E 00	3.411E 00	5.478E 00	5.119E 00
160.0	2.084E 00	1.951E 00	2.178E 00	2.040E 00	3.513E 00	3.290E 00	5.240E 00	4.898E 00
170.0	2.069E 00	1.937E 00	2.154E 00	2.017E 00	3.397E 00	3.181E 00	5.029E 00	4.701E 00
180.0	2.057E 00	1.926E 00	2.133E 00	1.998E 00	3.296E 00	3.086E 00	4.840E 00	4.526E 00
190.0	2.047E 00	1.916E 00	2.115E 00	1.981E 00	3.205E 00	3.002E 00	4.671E 00	4.368E 00
200.0	2.039E 00	1.908E 00	2.099E 00	1.966E 00	3.123E 00	2.925E 00	4.518E 00	4.226E 00
220.0	2.026E 00	1.896E 00	2.074E 00	1.942E 00	2.983E 00	2.794E 00	4.252E 00	3.979E 00
240.0	2.018E 00	1.889E 00	2.055E 00	1.924E 00	2.866E 00	2.686E 00	4.030E 00	3.772E 00
260.0	2.012E 00	1.885E 00	2.041E 00	1.910E 00	2.768E 00	2.593E 00	3.841E 00	3.596E 00
280.0	2.009E 00	1.882E 00	2.031E 00	1.900E 00	2.683E 00	2.515E 00	3.679E 00	3.445E 00
300.0	2.008E 00	1.881E 00	2.023E 00	1.893E 00	2.613E 00	2.449E 00	3.538E 00	3.314E 00
320.0	2.008E 00	1.882E 00	2.017E 00	1.888E 00	2.551E 00	2.386E 00	3.414E 00	3.197E 00
340.0	2.009E 00	1.883E 00	2.013E 00	1.885E 00	2.498E 00	2.337E 00	3.306E 00	3.095E 00
360.0	2.010E 00	1.885E 00	2.010E 00	1.883E 00	2.451E 00	2.293E 00	3.209E 00	3.005E 00
380.0	2.013E 00	1.888E 00	2.009E 00	1.882E 00	2.409E 00	2.254E 00	3.123E 00	2.925E 00
400.0	2.015E 00	1.891E 00	2.008E 00	1.881E 00	2.372E 00	2.220E 00	3.046E 00	2.853E 00

Table 3-9 (continued)

ENERGY MEV	MUON MUSCLE I= 66.2	MUON BONE I= 85.1	PION MUSCLE I= 66.2	PION BONE I= 85.1	KAON MUSCLE I= 66.2	KAON BONE I= 85.1	PROTON MUSCLE I= 66.2	PROTON BONE I= 85.1
420.0	2.018E 00	1.894E 00	2.008E 00	1.882E 00	2.338E 00	2.189E 00	2.976E 00	2.788E 00
440.0	2.022E 00	1.898E 00	2.008E 00	1.883E 00	2.308E 00	2.162E 00	2.913E 00	2.729E 00
460.0	2.025E 00	1.902E 00	2.009E 00	1.884E 00	2.282E 00	2.137E 00	2.855E 00	2.675E 00
480.0	2.029E 00	1.906E 00	2.011E 00	1.886E 00	2.257E 00	2.114E 00	2.802E 00	2.625E 00
500.0	2.032E 00	1.910E 00	2.012E 00	1.888E 00	2.236E 00	2.094E 00	2.754E 00	2.580E 00
520.0	2.036E 00	1.914E 00	2.014E 00	1.890E 00	2.216E 00	2.076E 00	2.710E 00	2.539E 00
540.0	2.040E 00	1.918E 00	2.016E 00	1.892E 00	2.199E 00	2.060E 00	2.667E 00	2.500E 00
560.0	2.044E 00	1.922E 00	2.019E 00	1.895E 00	2.183E 00	2.045E 00	2.630E 00	2.465E 00
580.0	2.048E 00	1.926E 00	2.021E 00	1.898E 00	2.168E 00	2.031E 00	2.596E 00	2.427E 00
600.0	2.052E 00	1.930E 00	2.024E 00	1.901E 00	2.155E 00	2.018E 00	2.563E 00	2.397E 00
620.0	2.056E 00	1.934E 00	2.027E 00	1.904E 00	2.142E 00	2.007E 00	2.534E 00	2.370E 00
640.0	2.060E 00	1.939E 00	2.029E 00	1.907E 00	2.131E 00	1.996E 00	2.506E 00	2.344E 00
660.0	2.063E 00	1.943E 00	2.032E 00	1.910E 00	2.121E 00	1.986E 00	2.480E 00	2.320E 00
680.0	2.067E 00	1.947E 00	2.035E 00	1.913E 00	2.111E 00	1.977E 00	2.455E 00	2.297E 00
700.0	2.071E 00	1.951E 00	2.038E 00	1.916E 00	2.102E 00	1.969E 00	2.432E 00	2.276E 00
720.0	2.075E 00	1.955E 00	2.041E 00	1.919E 00	2.094E 00	1.961E 00	2.411E 00	2.256E 00
740.0	2.079E 00	1.958E 00	2.044E 00	1.922E 00	2.087E 00	1.954E 00	2.390E 00	2.238E 00
760.0	2.082E 00	1.962E 00	2.047E 00	1.925E 00	2.080E 00	1.948E 00	2.371E 00	2.220E 00
780.0	2.086E 00	1.966E 00	2.050E 00	1.928E 00	2.074E 00	1.941E 00	2.353E 00	2.203E 00
800.0	2.089E 00	1.970E 00	2.053E 00	1.932E 00	2.068E 00	1.936E 00	2.337E 00	2.188E 00
820.0	2.093E 00	1.974E 00	2.056E 00	1.935E 00	2.063E 00	1.931E 00	2.321E 00	2.173E 00
840.0	2.096E 00	1.977E 00	2.059E 00	1.938E 00	2.058E 00	1.926E 00	2.305E 00	2.159E 00
860.0	2.100E 00	1.981E 00	2.062E 00	1.941E 00	2.053E 00	1.921E 00	2.291E 00	2.146E 00
880.0	2.103E 00	1.984E 00	2.065E 00	1.944E 00	2.049E 00	1.917E 00	2.277E 00	2.133E 00
900.0	2.107E 00	1.988E 00	2.068E 00	1.947E 00	2.045E 00	1.914E 00	2.265E 00	2.121E 00
920.0	2.110E 00	1.991E 00	2.070E 00	1.950E 00	2.041E 00	1.910E 00	2.253E 00	2.110E 00
940.0	2.113E 00	1.995E 00	2.073E 00	1.953E 00	2.038E 00	1.907E 00	2.241E 00	2.099E 00
960.0	2.116E 00	1.998E 00	2.076E 00	1.956E 00	2.035E 00	1.904E 00	2.231E 00	2.089E 00
980.0	2.119E 00	2.001E 00	2.079E 00	1.959E 00	2.032E 00	1.902E 00	2.220E 00	2.080E 00
1000.0	2.123E 00	2.004E 00	2.082E 00	1.962E 00	2.030E 00	1.899E 00	2.211E 00	2.071E 00
1200.0	2.151E 00	2.034E 00	2.108E 00	1.989E 00	2.013E 00	1.885E 00	2.136E 00	2.000E 00
1400.0	2.176E 00	2.060E 00	2.132E 00	2.014E 00	2.008E 00	1.881E 00	2.088E 00	1.955E 00
1600.0	2.198E 00	2.083E 00	2.153E 00	2.036E 00	2.009E 00	1.883E 00	2.057E 00	1.925E 00
2000.0	2.235E 00	2.122E 00	2.189E 00	2.074E 00	2.019E 00	1.896E 00	2.024E 00	1.894E 00
2400.0	2.266E 00	2.154E 00	2.219E 00	2.105E 00	2.035E 00	1.912E 00	2.011E 00	1.883E 00
2800.0	2.292E 00	2.180E 00	2.245E 00	2.132E 00	2.052E 00	1.930E 00	2.008E 00	1.882E 00
3200.0	2.314E 00	2.203E 00	2.267E 00	2.155E 00	2.068E 00	1.948E 00	2.010E 00	1.885E 00
3600.0	2.334E 00	2.224E 00	2.287E 00	2.176E 00	2.084E 00	1.964E 00	2.016E 00	1.892E 00
4000.0	2.351E 00	2.242E 00	2.305E 00	2.194E 00	2.099E 00	1.980E 00	2.023E 00	1.900E 00
5000.0	2.388E 00	2.280E 00	2.342E 00	2.232E 00	2.133E 00	2.015E 00	2.045E 00	1.923E 00

Table 3-10

Range, G/CM^2

(See text for explanation of symbols)

(Adapted from Berger and Seltzer[24])

ENERGY MEV	MUON		PION		KAON		PROTON	
	MUSCLE I= 66.2	BONE I= 85.1	MUSCLE I= 66.2	BONE I= 85.1	MUSCLE I= 66.2	BONE I= 85.1	MUSCLE I= 66.2	BONE I= 85.1
2.0	3.813E-02	4.154E-02	3.042E-02	3.324E-02	1.124E-02	1.250E-02	7.233E-03	8.162E-03
4.0	1.338E-01	1.447E-01	1.067E-01	1.157E-01	3.839E-02	4.230E-02	2.335E-02	2.594E-02
6.0	2.785E-01	3.005E-01	2.223E-01	2.403E-01	7.989E-02	8.749E-02	4.767E-02	5.265E-02
8.0	4.677E-01	5.036E-01	3.741E-01	4.035E-01	1.344E-01	1.466E-01	8.008E-02	8.820E-02
10.0	6.976E-01	7.502E-01	5.593E-01	6.024E-01	2.012E-01	2.189E-01	1.198E-01	1.315E-01
14.0	1.268E 00	1.362E 00	1.022E 00	1.099E 00	3.700E-01	4.014E-01	2.200E-01	2.403E-01
18.0	1.971E 00	2.113E 00	1.597E 00	1.715E 00	5.835E-01	6.315E-01	3.467E-01	3.775E-01
22.0	2.769E 00	2.989E 00	2.273E 00	2.438E 00	8.392E-01	9.068E-01	4.985E-01	5.417E-01
26.0	3.711E 00	3.974E 00	3.040E 00	3.259E 00	1.135E 00	1.225E 00	6.747E-01	7.320E-01
30.0	4.724E 00	5.057E 00	3.891E 00	4.169E 00	1.470E 00	1.585E 00	8.745E-01	9.474E-01
34.0	5.820E 00	6.227E 00	4.818E 00	5.160E 00	1.842E 00	1.984E 00	1.097E 00	1.187E 00
38.0	6.991E 00	7.477E 00	5.815E 00	6.225E 00	2.250E 00	2.422E 00	1.342E 00	1.451E 00
42.0	8.229E 00	8.799E 00	6.877E 00	7.359E 00	2.692E 00	2.897E 00	1.608E 00	1.738E 00
46.0	9.527E 00	1.018E 01	7.998E 00	8.557E 00	3.168E 00	3.408E 00	1.896E 00	2.048E 00
50.0	1.088E 01	1.163E 01	9.175E 00	9.813E 00	3.677E 00	3.953E 00	2.205E 00	2.379E 00
60.0	1.447E 01	1.546E 01	1.233E 01	1.318E 01	5.084E 00	5.461E 00	3.064E 00	3.303E 00
70.0	1.832E 01	1.957E 01	1.575E 01	1.683E 01	6.674E 00	7.164E 00	4.044E 00	4.355E 00
80.0	2.236E 01	2.389E 01	1.939E 01	2.072E 01	8.435E 00	9.048E 00	5.139E 00	5.530E 00
90.0	2.657E 01	2.839E 01	2.322E 01	2.481E 01	1.035E 01	1.110E 01	6.345E 00	6.823E 00
100.0	3.091E 01	3.302E 01	2.721E 01	2.907E 01	1.242E 01	1.331E 01	7.656E 00	8.228E 00
110.0	3.536E 01	3.778E 01	3.133E 01	3.348E 01	1.463E 01	1.568E 01	9.069E 00	9.742E 00
120.0	3.991E 01	4.263E 01	3.557E 01	3.800E 01	1.697E 01	1.818E 01	1.058E 01	1.136E 01
130.0	4.452E 01	4.756E 01	3.991E 01	4.264E 01	1.944E 01	2.081E 01	1.218E 01	1.308E 01
140.0	4.921E 01	5.255E 01	4.433E 01	4.736E 01	2.202E 01	2.357E 01	1.388E 01	1.489E 01
150.0	5.394E 01	5.761E 01	4.883E 01	5.216E 01	2.471E 01	2.645E 01	1.566E 01	1.680E 01
160.0	5.872E 01	6.271E 01	5.339E 01	5.703E 01	2.751E 01	2.943E 01	1.753E 01	1.880E 01
170.0	6.353E 01	6.786E 01	5.801E 01	6.196E 01	3.041E 01	3.253E 01	1.948E 01	2.088E 01
180.0	6.838E 01	7.303E 01	6.268E 01	6.694E 01	3.340E 01	3.572E 01	2.151E 01	2.305E 01
190.0	7.325E 01	7.824E 01	6.738E 01	7.197E 01	3.647E 01	3.900E 01	2.361E 01	2.530E 01
200.0	7.815E 01	8.347E 01	7.213E 01	7.704E 01	3.963E 01	4.238E 01	2.579E 01	2.763E 01
220.0	8.799E 01	9.399E 01	8.172E 01	8.728E 01	4.619E 01	4.938E 01	3.035E 01	3.251E 01
240.0	9.788E 01	1.046E 02	9.141E 01	9.763E 01	5.303E 01	5.668E 01	3.519E 01	3.768E 01
260.0	1.078E 02	1.152E 02	1.012E 02	1.081E 02	6.014E 01	6.426E 01	4.027E 01	4.311E 01
280.0	1.178E 02	1.258E 02	1.110E 02	1.186E 02	6.748E 01	7.210E 01	4.560E 01	4.880E 01
300.0	1.277E 02	1.364E 02	1.209E 02	1.291E 02	7.503E 01	8.016E 01	5.114E 01	5.472E 01
320.0	1.377E 02	1.470E 02	1.308E 02	1.397E 02	8.278E 01	8.844E 01	5.690E 01	6.087E 01
340.0	1.476E 02	1.577E 02	1.407E 02	1.503E 02	9.071E 01	9.691E 01	6.285E 01	6.723E 01
360.0	1.576E 02	1.683E 02	1.506E 02	1.609E 02	9.879E 01	1.056E 02	6.900E 01	7.379E 01
380.0	1.675E 02	1.789E 02	1.606E 02	1.715E 02	1.070E 02	1.143E 02	7.531E 01	8.053E 01
400.0	1.775E 02	1.895E 02	1.705E 02	1.822E 02	1.154E 02	1.233E 02	8.180E 01	8.746E 01

Table 3-10 (continued)

ENERGY MEV	MUON MUSCLE I= 66.2	MUON BONE I= 85.1	PION MUSCLE I= 66.2	PION BONE I= 85.1	KAON MUSCLE I= 66.2	KAON BONE I= 85.1	PROTON MUSCLE I= 66.2	PROTON BONE I= 85.1
420.0	1.874E 02	2.000E 02	1.805E 02	1.928E 02	1.239E 02	1.324E 02	8.845E 01	9.455E 01
440.0	1.973E 02	2.106E 02	1.905E 02	2.034E 02	1.325E 02	1.416E 02	9.524E 01	1.018E 02
460.0	2.072E 02	2.211E 02	2.004E 02	2.140E 02	1.412E 02	1.509E 02	1.022E 02	1.092E 02
480.0	2.170E 02	2.316E 02	2.104E 02	2.246E 02	1.500E 02	1.603E 02	1.092E 02	1.168E 02
500.0	2.269E 02	2.421E 02	2.203E 02	2.352E 02	1.589E 02	1.698E 02	1.164E 02	1.244E 02
520.0	2.367E 02	2.526E 02	2.303E 02	2.458E 02	1.679E 02	1.794E 02	1.238E 02	1.323E 02
540.0	2.465E 02	2.630E 02	2.402E 02	2.564E 02	1.770E 02	1.890E 02	1.312E 02	1.402E 02
560.0	2.563E 02	2.734E 02	2.501E 02	2.670E 02	1.861E 02	1.988E 02	1.388E 02	1.482E 02
580.0	2.661E 02	2.838E 02	2.600E 02	2.775E 02	1.953E 02	2.086E 02	1.464E 02	1.564E 02
600.0	2.759E 02	2.942E 02	2.699E 02	2.880E 02	2.046E 02	2.185E 02	1.542E 02	1.647E 02
620.0	2.856E 02	3.045E 02	2.798E 02	2.986E 02	2.139E 02	2.284E 02	1.620E 02	1.731E 02
640.0	2.953E 02	3.149E 02	2.896E 02	3.091E 02	2.232E 02	2.384E 02	1.700E 02	1.816E 02
660.0	3.050E 02	3.252E 02	2.995E 02	3.195E 02	2.326E 02	2.485E 02	1.780E 02	1.902E 02
680.0	3.147E 02	3.354E 02	3.093E 02	3.300E 02	2.421E 02	2.586E 02	1.861E 02	1.988E 02
700.0	3.244E 02	3.457E 02	3.191E 02	3.404E 02	2.516E 02	2.687E 02	1.943E 02	2.076E 02
720.0	3.340E 02	3.559E 02	3.289E 02	3.509E 02	2.611E 02	2.789E 02	2.025E 02	2.164E 02
740.0	3.436E 02	3.662E 02	3.387E 02	3.613E 02	2.707E 02	2.891E 02	2.109E 02	2.253E 02
760.0	3.533E 02	3.764E 02	3.485E 02	3.717E 02	2.803E 02	2.993E 02	2.193E 02	2.343E 02
780.0	3.629E 02	3.866E 02	3.583E 02	3.821E 02	2.899E 02	3.096E 02	2.277E 02	2.433E 02
800.0	3.724E 02	3.967E 02	3.680E 02	3.924E 02	2.996E 02	3.199E 02	2.363E 02	2.524E 02
820.0	3.820E 02	4.069E 02	3.777E 02	4.028E 02	3.092E 02	3.303E 02	2.448E 02	2.616E 02
840.0	3.915E 02	4.170E 02	3.875E 02	4.131E 02	3.190E 02	3.407E 02	2.535E 02	2.708E 02
860.0	4.011E 02	4.271E 02	3.972E 02	4.234E 02	3.287E 02	3.511E 02	2.622E 02	2.801E 02
880.0	4.106E 02	4.372E 02	4.069E 02	4.337E 02	3.384E 02	3.615E 02	2.710E 02	2.895E 02
900.0	4.201E 02	4.472E 02	4.165E 02	4.440E 02	3.482E 02	3.719E 02	2.798E 02	2.989E 02
920.0	4.296E 02	4.573E 02	4.262E 02	4.543E 02	3.580E 02	3.824E 02	2.886E 02	3.083E 02
940.0	4.391E 02	4.673E 02	4.359E 02	4.645E 02	3.678E 02	3.929E 02	2.975E 02	3.178E 02
960.0	4.485E 02	4.774E 02	4.455E 02	4.747E 02	3.776E 02	4.034E 02	3.065E 02	3.274E 02
980.0	4.580E 02	4.874E 02	4.551E 02	4.850E 02	3.875E 02	4.139E 02	3.155E 02	3.370E 02
1000.0	4.674E 02	4.973E 02	4.647E 02	4.952E 02	3.973E 02	4.244E 02	3.245E 02	3.466E 02
1200.0	5.610E 02	5.964E 02	5.602E 02	5.964E 02	4.963E 02	5.301E 02	4.166E 02	4.450E 02
1400.0	6.534E 02	6.940E 02	6.545E 02	6.963E 02	5.958E 02	6.364E 02	5.114E 02	5.462E 02
1600.0	7.448E 02	7.906E 02	7.479E 02	7.951E 02	6.954E 02	7.426E 02	6.080E 02	6.494E 02
2000.0	9.252E 02	9.807E 02	9.321E 02	9.897E 02	8.941E 02	9.544E 02	8.043E 02	8.591E 02
2400.0	1.103E 03	1.168E 03	1.114E 03	1.181E 03	1.091E 03	1.164E 03	1.003E 03	1.071E 03
2800.0	1.278E 03	1.352E 03	1.293E 03	1.370E 03	1.287E 03	1.373E 03	1.202E 03	1.284E 03
3200.0	1.452E 03	1.535E 03	1.470E 03	1.556E 03	1.481E 03	1.579E 03	1.401E 03	1.496E 03
3600.0	1.624E 03	1.716E 03	1.646E 03	1.741E 03	1.674E 03	1.783E 03	1.600E 03	1.708E 03
4000.0	1.795E 03	1.895E 03	1.820E 03	1.924E 03	1.865E 03	1.986E 03	1.798E 03	1.919E 03
5000.0	2.217E 03	2.337E 03	2.250E 03	2.376E 03	2.338E 03	2.487E 03	2.289E 03	2.442E 03

which were erroneously calculated (17). The data for protons are slightly different from those of Table 3-8 because of different assumptions regarding the molecular properties of the tissues in question. Similar data are available for other materials (24).

Similar stopping powers, ranges, and radiation yield(bremsstrahlung efficiency) for electrons are seen in Tables 3-11 and 3-12. These can be used for secondary electrons or primary electrons in the Van Allen belts. These tables replace previous data which were erroneously calculated (25). Similar data are available on other non-biological materials (24). The angular distribution of thick-target bremsstrahlung including multiple electron scattering is now under study (248). Figure 3-13 compares the rate of energy loss for electrons and protons in tissue. Neutron mean free paths in ICRU muscle vs. energy are seen in Figure 3-14.

In the past, stopping power and range for ions $>Z = 18$ have been poorly investigated either theoretically or experimentally. The Omnitron accelerator may make available ions up to 500 MeV/atomic mass unit (amu) and Z numbers through 92. A program has been recently written which computes range, energy, and stopping power data. These are available for hydrogen, helium 4, carbon 12, neon 20, argon 40, krypton 84, xenon 131, and uranium-238 incident upon water, aluminum, copper, silver, lead, and uranium (262). Programs are available in Chippewa, Fortran, and Fortran IV. The calculations have been corroborated by comparison with experimental data available on ions of $Z < 10$ (197). Figures 3-15a, b, and c represent only water, aluminum, and lead targets most pertinent to the space radiation problem. The stopping power has been plotted as a function of ion residual range. The symbols on each curve match points corresponding to various energies. The Ne-C and Xe-U crossovers are low energy, and although possibly a physical reality, occur in regions of low confidence of the calculations. Discontinuities and irregularities in several of the curves are caused by different equations and assumptions used for calculation of $Z < 10$ and for 4 separate energy regions of $Z > 10$ (262).

As noted in the discussion of Figure 3-15a, b, and c, calculation of LET's for the primary galactic cosmic-ray flux is difficult. Table 3-16 shows theoretical considerations of dose and energy distribution in tissue from the constituents of the primary cosmic-ray flux. The sections, energies, and angles of emissions, as well as relative frequencies of alternate decay schemes for the various types of secondaries, are not well enough known to allow more than a tentative designation for this table. Data are available for some of the nuclei above iron in the primary cosmic ray spectrum (90). A large percentage of the absorbed dose appears to be derived from particles giving very heavy ionization.

Figure 3-17 presents examples of primary cosmic-ray data graphically. Contrary to conditions for electrons, where the LET reaches its maximum at the very end of the particle track, the maximum LET occurs for nuclear particles a very short distance before the end (2 micra $_T$ for protons, 46 micra $_T$ for Ca nuclei) and then drops steeply to zero. As the exact shape of this final descent of the LET to zero is not known, the exact height of the

Table 3-11

Electrons in Muscle

(After Berger and Seltzer[24])

ENERGY	STOPPING POWER			RANGE	RADIATION YIELD
	COLLISION	RADIATION	TOTAL		
MEV	MEV CM2/G	MEV CM2/G	MEV CM2/G	G/CM2	
0.010	2.292E 01	4.971E-03	2.292E 01	2.467E-04	1.236E-04
0.015	1.670E 01	4.874E-03	1.670E 01	5.061E-04	1.674E-04
0.020	1.334E 01	4.810E-03	1.335E 01	8.435E-04	2.072E-04
0.025	1.123E 01	4.766E-03	1.123E 01	1.254E-03	2.443E-04
0.030	9.763E 00	4.735E-03	9.768E 00	1.733E-03	2.794E-04
0.035	8.686E 00	4.705E-03	8.691E 00	2.276E-03	3.128E-04
0.040	7.859E 00	4.702E-03	7.863E 00	2.882E-03	3.449E-04
0.045	7.202E 00	4.710E-03	7.207E 00	3.547E-03	3.761E-04
0.050	6.669E 00	4.726E-03	6.673E 00	4.269E-03	4.066E-04
0.055	6.225E 00	4.749E-03	6.230E 00	5.045E-03	4.365E-04
0.060	5.851E 00	4.777E-03	5.856E 00	5.873E-03	4.659E-04
0.065	5.531E 00	4.808E-03	5.536E 00	6.752E-03	4.948E-04
0.070	5.254E 00	4.843E-03	5.259E 00	7.679E-03	5.234E-04
0.075	5.012E 00	4.881E-03	5.017E 00	8.653E-03	5.516E-04
0.080	4.799E 00	4.921E-03	4.804E 00	9.672E-03	5.795E-04
0.085	4.609E 00	4.952E-03	4.614E 00	1.073E-02	6.071E-04
0.090	4.440E 00	4.995E-03	4.445E 00	1.184E-02	6.344E-04
0.095	4.287E 00	5.041E-03	4.292E 00	1.298E-02	6.615E-04
0.100	4.149E 00	5.087E-03	4.154E 00	1.417E-02	6.884E-04
0.150	3.261E 00	5.609E-03	3.267E 00	2.792E-02	9.497E-04
0.200	2.811E 00	6.169E-03	2.817E 00	4.451E-02	1.201E-03
0.250	2.543E 00	6.781E-03	2.550E 00	6.323E-02	1.446E-03
0.300	2.366E 00	7.420E-03	2.373E 00	8.359E-02	1.687E-03
0.350	2.244E 00	8.088E-03	2.252E 00	1.052E-01	1.926E-03
0.400	2.155E 00	8.754E-03	2.164E 00	1.279E-01	2.162E-03
0.450	2.088E 00	9.432E-03	2.097E 00	1.514E-01	2.397E-03
0.500	2.036E 00	1.011E-02	2.046E 00	1.755E-01	2.629E-03
0.550	1.996E 00	1.078E-02	2.007E 00	2.002E-01	2.859E-03
0.600	1.964E 00	1.146E-02	1.976E 00	2.253E-01	3.086E-03
0.650	1.939E 00	1.214E-02	1.951E 00	2.508E-01	3.311E-03
0.700	1.918E 00	1.282E-02	1.931E 00	2.766E-01	3.534E-03
0.750	1.901E 00	1.350E-02	1.915E 00	3.026E-01	3.754E-03
0.800	1.887E 00	1.418E-02	1.902E 00	3.288E-01	3.973E-03
0.850	1.876E 00	1.487E-02	1.891E 00	3.552E-01	4.190E-03
0.900	1.867E 00	1.556E-02	1.882E 00	3.817E-01	4.405E-03
0.950	1.859E 00	1.625E-02	1.875E 00	4.083E-01	4.619E-03
1.000	1.852E 00	1.694E-02	1.869E 00	4.350E-01	4.831E-03
1.100	1.843E 00	1.834E-02	1.861E 00	4.886E-01	5.252E-03
1.200	1.836E 00	1.975E-02	1.856E 00	5.424E-01	5.668E-03
1.300	1.832E 00	2.116E-02	1.854E 00	5.963E-01	6.081E-03

Table 3-11 (continued)

ENERGY	STOPPING POWER			RANGE	RADIATION YIELD
	COLLISION	RADIATION	TOTAL		
MEV	MEV CM2/G	MEV CM2/G	MEV CM2/G	G/CM2	
1.400	1.830E 00	2.259E-02	1.853E 00	6.503E-01	6.490E-03
1.500	1.829E 00	2.402E-02	1.853E 00	7.043E-01	6.895E-03
1.600	1.829E 00	2.546E-02	1.854E 00	7.582E-01	7.299E-03
1.700	1.830E 00	2.687E-02	1.857E 00	8.121E-01	7.698E-03
1.800	1.831E 00	2.833E-02	1.859E 00	8.660E-01	8.096E-03
1.900	1.833E 00	2.981E-02	1.862E 00	9.197E-01	8.492E-03
2.000	1.835E 00	3.130E-02	1.866E 00	9.733E-01	8.887E-03
2.200	1.839E 00	3.432E-02	1.874E 00	1.080E 00	9.674E-03
2.400	1.844E 00	3.739E-02	1.882E 00	1.187E 00	1.046E-02
2.600	1.850E 00	4.050E-02	1.890E 00	1.293E 00	1.124E-02
2.800	1.855E 00	4.354E-02	1.899E 00	1.398E 00	1.203E-02
3.000	1.861E 00	4.673E-02	1.907E 00	1.504E 00	1.280E-02
3.500	1.874E 00	5.495E-02	1.929E 00	1.764E 00	1.476E-02
4.000	1.886E 00	6.346E-02	1.950E 00	2.022E 00	1.673E-02
4.500	1.898E 00	7.230E-02	1.970E 00	2.277E 00	1.871E-02
5.000	1.908E 00	8.129E-02	1.990E 00	2.530E 00	2.072E-02
5.500	1.918E 00	9.045E-02	2.008E 00	2.780E 00	2.274E-02
6.000	1.927E 00	9.977E-02	2.026E 00	3.028E 00	2.477E-02
6.500	1.935E 00	1.092E-01	2.044E 00	3.273E 00	2.682E-02
7.000	1.942E 00	1.188E-01	2.061E 00	3.517E 00	2.887E-02
7.500	1.949E 00	1.285E-01	2.078E 00	3.758E 00	3.093E-02
8.000	1.956E 00	1.384E-01	2.094E 00	3.998E 00	3.299E-02
8.500	1.962E 00	1.483E-01	2.110E 00	4.236E 00	3.506E-02
9.000	1.967E 00	1.593E-01	2.127E 00	4.472E 00	3.715E-02
9.500	1.973E 00	1.695E-01	2.142E 00	4.706E 00	3.925E-02
10.000	1.978E 00	1.798E-01	2.158E 00	4.939E 00	4.135E-02
20.000	2.043E 00	3.986E-01	2.441E 00	9.289E 00	8.266E-02
30.000	2.079E 00	6.311E-01	2.710E 00	1.317E 01	1.214E-01
40.000	2.103E 00	8.701E-01	2.973E 00	1.670E 01	1.570E-01
50.000	2.123E 00	1.113E 00	3.236E 00	1.992E 01	1.894E-01
60.000	2.138E 00	1.360E 00	3.498E 00	2.289E 01	2.189E-01
80.000	2.163E 00	1.859E 00	4.021E 00	2.822E 01	2.710E-01
100.000	2.182E 00	2.363E 00	4.545E 00	3.289E 01	3.152E-01
200.000	2.241E 00	4.927E 00	7.167E 00	5.027E 01	4.654E-01
300.000	2.275E 00	7.521E 00	9.796E 00	6.215E 01	5.542E-01
400.000	2.299E 00	1.013E 01	1.243E 01	7.119E 01	6.139E-01
500.000	2.318E 00	1.274E 01	1.506E 01	7.849E 01	6.574E-01
600.000	2.334E 00	1.536E 01	1.769E 01	8.461E 01	6.908E-01
800.000	2.358E 00	2.061E 01	2.296E 01	9.451E 01	7.391E-01
1000.000	2.377E 00	2.585E 01	2.823E 01	1.023E 02	7.727E-01

Table 3-12

Electrons in Bone

(After Berger and (Seltzer[24])

ENERGY	STOPPING POWER			RANGE	RADIATION YIELD
	COLLISION	RADIATION	TOTAL		
MEV	MEV CM2/G	MEV CM2/G	MEV CM2/G	G/CM2	
0.010	2.101E 01	6.373E-03	2.101E 01	2.711E-04	1.726E-04
0.015	1.536E 01	6.282E-03	1.537E 01	5.533E-04	2.341E-04
0.020	1.231E 01	6.206E-03	1.231E 01	9.195E-04	2.898E-04
0.025	1.037E 01	6.172E-03	1.038E 01	1.364E-03	3.418E-04
0.030	9.030E 00	6.159E-03	9.036E 00	1.882E-03	3.913E-04
0.035	8.041E 00	6.153E-03	8.047E 00	2.469E-03	4.387E-04
0.040	7.281E 00	6.169E-03	7.287E 00	3.123E-03	4.846E-04
0.045	6.678E 00	6.196E-03	6.684E 00	3.841E-03	5.292E-04
0.050	6.186E 00	6.231E-03	6.193E 00	4.619E-03	5.730E-04
0.055	5.778E 00	6.271E-03	5.785E 00	5.455E-03	6.159E-04
0.060	5.434E 00	6.316E-03	5.440E 00	6.347E-03	6.581E-04
0.065	5.139E 00	6.365E-03	5.145E 00	7.292E-03	6.998E-04
0.070	4.883E 00	6.417E-03	4.890E 00	8.290E-03	7.409E-04
0.075	4.660E 00	6.472E-03	4.666E 00	9.337E-03	7.814E-04
0.080	4.463E 00	6.528E-03	4.469E 00	1.043E-02	8.216E-04
0.085	4.288E 00	6.571E-03	4.294E 00	1.157E-02	8.612E-04
0.090	4.131E 00	6.631E-03	4.138E 00	1.276E-02	9.003E-04
0.095	3.990E 00	6.693E-03	3.997E 00	1.399E-02	9.392E-04
0.100	3.862E 00	6.757E-03	3.869E 00	1.526E-02	9.778E-04
0.150	3.041E 00	7.442E-03	3.049E 00	3.001E-02	1.351E-03
0.200	2.623E 00	8.154E-03	2.631E 00	4.778E-02	1.706E-03
0.250	2.374E 00	8.941E-03	2.383E 00	6.781E-02	2.050E-03
0.300	2.210E 00	9.765E-03	2.219E 00	8.960E-02	2.388E-03
0.350	2.092E 00	1.063E-02	2.103E 00	1.128E-01	2.723E-03
0.400	2.011E 00	1.148E-02	2.022E 00	1.371E-01	3.054E-03
0.450	1.949E 00	1.235E-02	1.961E 00	1.622E-01	3.380E-03
0.500	1.901E 00	1.321E-02	1.914E 00	1.880E-01	3.701E-03
0.550	1.864E 00	1.407E-02	1.878E 00	2.144E-01	4.019E-03
0.600	1.835E 00	1.493E-02	1.850E 00	2.412E-01	4.333E-03
0.650	1.811E 00	1.578E-02	1.826E 00	2.684E-01	4.642E-03
0.700	1.791E 00	1.664E-02	1.808E 00	2.959E-01	4.948E-03
0.750	1.775E 00	1.750E-02	1.793E 00	3.237E-01	5.250E-03
0.800	1.762E 00	1.836E-02	1.780E 00	3.517E-01	5.550E-03
0.850	1.751E 00	1.925E-02	1.770E 00	3.799E-01	5.847E-03
0.900	1.742E 00	2.012E-02	1.762E 00	4.082E-01	6.141E-03
0.950	1.734E 00	2.098E-02	1.755E 00	4.366E-01	6.433E-03
1.000	1.728E 00	2.185E-02	1.750E 00	4.652E-01	6.722E-03
1.100	1.720E 00	2.359E-02	1.743E 00	5.224E-01	7.294E-03
1.200	1.714E 00	2.533E-02	1.740E 00	5.798E-01	7.856E-03
1.300	1.711E 00	2.708E-02	1.738E 00	6.374E-01	8.411E-03

Table 3-12 (continued)

ENERGY	STOPPING POWER			RANGE	RADIATION YIELD
	COLLISION	RADIATION	TOTAL		
MEV	MEV CM2/G	MEV CM2/G	MEV CM2/G	G/CM2	
1.400	1.710E 00	2.883E-02	1.739E 00	6.949E-01	8.959E-03
1.500	1.709E 00	3.059E-02	1.740E 00	7.524E-01	9.501E-03
1.600	1.710E 00	3.236E-02	1.742E 00	8.098E-01	1.004E-02
1.700	1.711E 00	3.400E-02	1.745E 00	8.672E-01	1.056E-02
1.800	1.713E 00	3.580E-02	1.749E 00	9.244E-01	1.109E-02
1.900	1.715E 00	3.761E-02	1.753E 00	9.815E-01	1.161E-02
2.000	1.717E 00	3.944E-02	1.757E 00	1.039E 00	1.212E-02
2.200	1.723E 00	4.317E-02	1.766E 00	1.152E 00	1.315E-02
2.400	1.728E 00	4.697E-02	1.775E 00	1.265E 00	1.418E-02
2.600	1.734E 00	5.084E-02	1.785E 00	1.377E 00	1.520E-02
2.800	1.741E 00	5.483E-02	1.795E 00	1.489E 00	1.622E-02
3.000	1.747E 00	5.883E-02	1.805E 00	1.600E 00	1.724E-02
3.500	1.761E 00	6.907E-02	1.830E 00	1.875E 00	1.980E-02
4.000	1.775E 00	7.963E-02	1.854E 00	2.147E 00	2.237E-02
4.500	1.787E 00	9.053E-02	1.878E 00	2.415E 00	2.495E-02
5.000	1.798E 00	1.016E-01	1.900E 00	2.679E 00	2.754E-02
5.500	1.809E 00	1.129E-01	1.922E 00	2.941E 00	3.014E-02
6.000	1.818E 00	1.243E-01	1.943E 00	3.200E 00	3.274E-02
6.500	1.827E 00	1.359E-01	1.963E 00	3.456E 00	3.534E-02
7.000	1.835E 00	1.477E-01	1.983E 00	3.709E 00	3.795E-02
7.500	1.843E 00	1.596E-01	2.003E 00	3.960E 00	4.056E-02
8.000	1.850E 00	1.716E-01	2.022E 00	4.209E 00	4.317E-02
8.500	1.857E 00	1.838E-01	2.040E 00	4.455E 00	4.578E-02
9.000	1.863E 00	1.969E-01	2.060E 00	4.699E 00	4.839E-02
9.500	1.869E 00	2.093E-01	2.078E 00	4.940E 00	5.101E-02
10.000	1.874E 00	2.218E-01	2.096E 00	5.180E 00	5.362E-02
20.000	1.945E 00	4.885E-01	2.434E 00	9.599E 00	1.040E-01
30.000	1.983E 00	7.718E-01	2.755E 00	1.346E 01	1.499E-01
40.000	2.010E 00	1.063E 00	3.073E 00	1.689E 01	1.909E-01
50.000	2.029E 00	1.360E 00	3.389E 00	1.999E 01	2.276E-01
60.000	2.045E 00	1.660E 00	3.705E 00	2.281E 01	2.606E-01
80.000	2.070E 00	2.267E 00	4.337E 00	2.780E 01	3.172E-01
100.000	2.089E 00	2.880E 00	4.969E 00	3.210E 01	3.642E-01
200.000	2.147E 00	5.994E 00	8.141E 00	4.766E 01	5.172E-01
300.000	2.180E 00	9.144E 00	1.132E 01	5.803E 01	6.033E-01
400.000	2.204E 00	1.231E 01	1.451E 01	6.581E 01	6.599E-01
500.000	2.222E 00	1.548E 01	1.770E 01	7.204E 01	7.003E-01
600.000	2.237E 00	1.865E 01	2.089E 01	7.724E 01	7.310E-01
800.000	2.260E 00	2.501E 01	2.727E 01	8.559E 01	7.747E-01
1000.000	2.278E 00	3.137E 01	3.365E 01	9.218E 01	8.048E-01

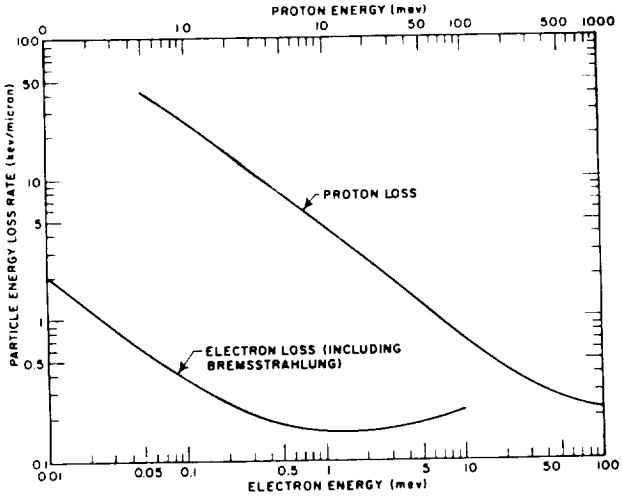


Figure 3-13

Energy Loss Per Unit Path Length for Protons and Electrons in Model Biological Materials

(After Janni et al[128])

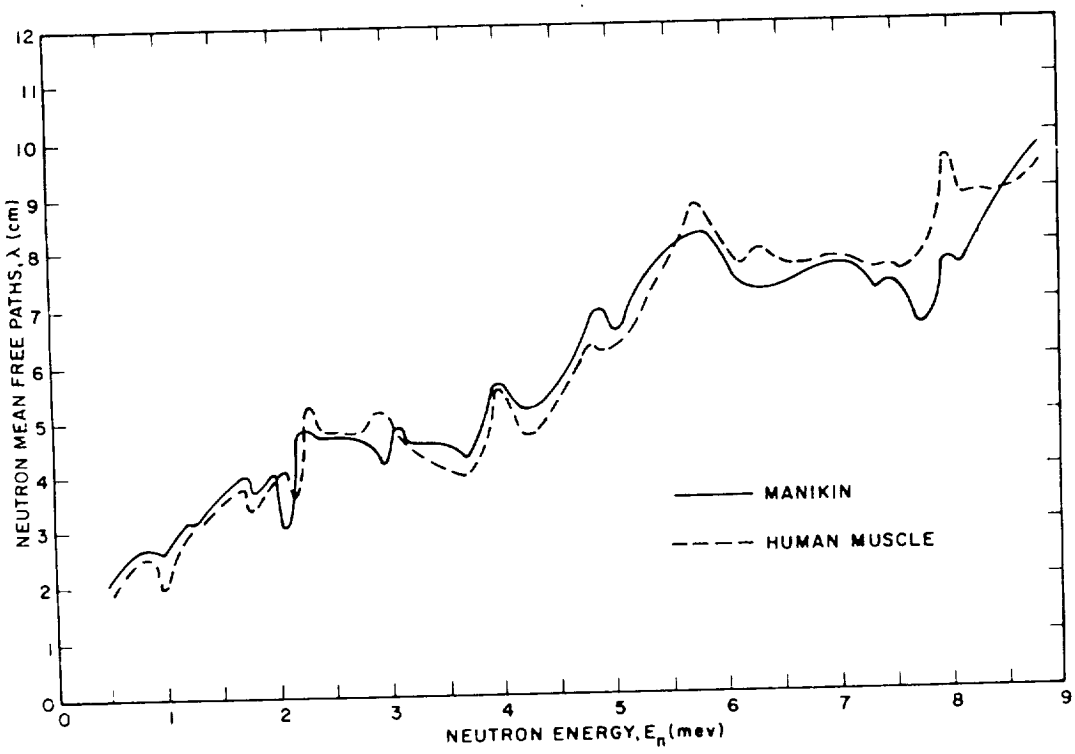


Figure 3-14

Neutron Mean Free Paths for ICRU Muscle Tissue and a Tissue Equivalent Manikin

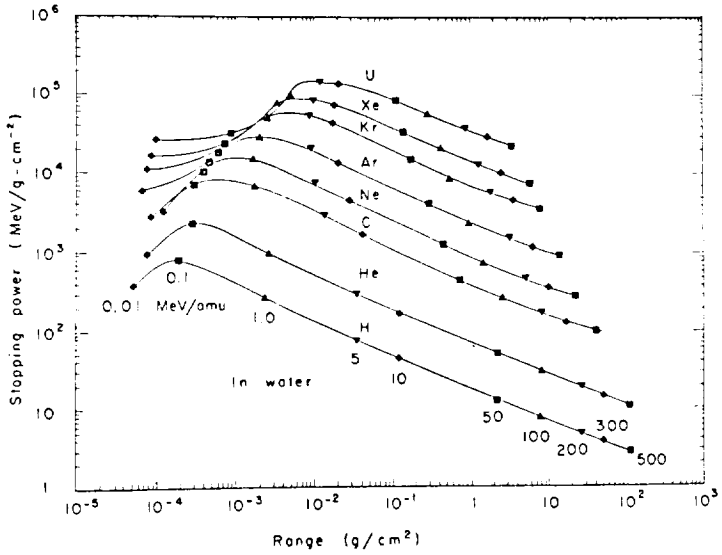
(After Janni et al[128])

Figure 3-15

Stopping-Power Curves as a Function of Range for Various Ions as Calculated by a UCRL Computer Program. Various Ion Energies in Units of MeV/amu Are Designated on Each Curve by the same Symbols as Noted for H. (See text for details).

(After Steward and Wallace[262])

a, In Water



b. In Aluminum

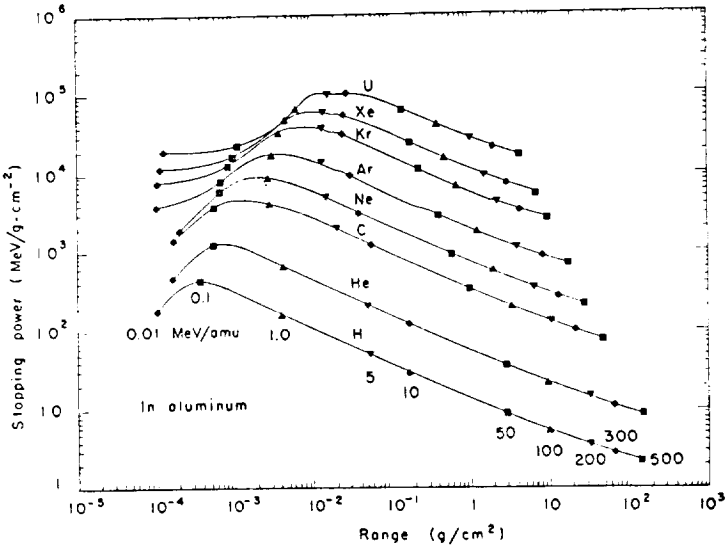


Figure 3-15 (continued)

c. In Lead

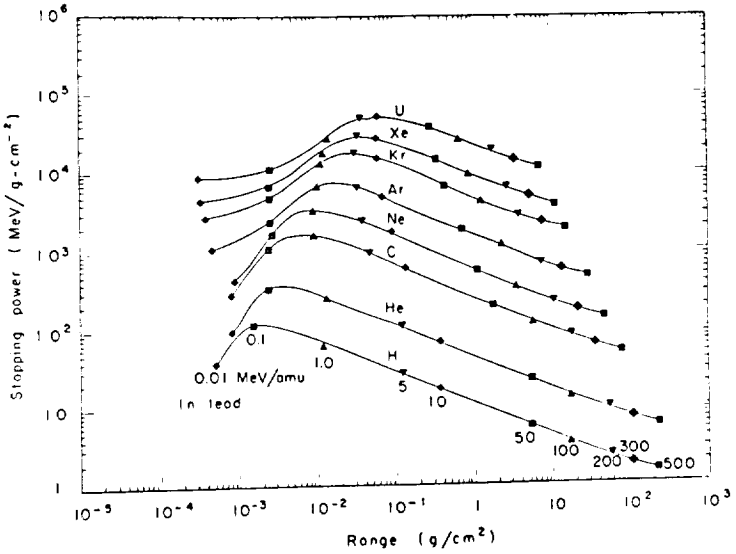


Table 3-16

Composition of the Primary Cosmic-Ray Flux Outside the Atmosphere in Northern Latitudes

(After Langham-NAS-NRC[145])

	TYPE NUCLEUS					
	H PROTONS	He ALPHA PARTICLES	CNO	Mg	Ca	Fe
Z[a]:	1	2	7	12	20	26
Particle flux[b]	4,460	633	32	8.4	2.9	1.4
Absorbed dose contribution (mrads/24 hr)	4	2.3	1.4	0.99	0.13	0.28
LET (keV/μ tissue)						
Minimum	0.21	0.84	10.5	30.3	84	142
Maximum	57.8	252	1,230	1,780	2,570	3,500
Absorbed dose to centrally traversed cell (rads)[c]						
Minimum	0.07	0.24	0.36	1	2.85	4.8
Maximum	20	85	420	610	870	1,200

[a]Z numbers from 7 to 26 are group representatives.
[b]Particle intensity: particles traversing sphere of 1 cm² cross section per hour from all directions.
[c]Dose per particle calculated for a 10-μ cell at center of track.

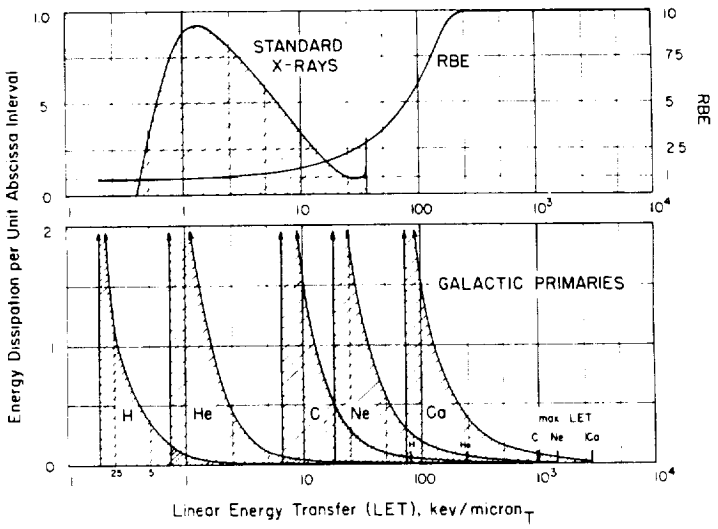


Figure 3-17

LET Distributions of Standard X–Rays and of Heavy Galactic Primaries in Tissue and RBE/LET Function.

(After Schaefer[239] and Cormack and Johns[57])

spike at the upper end of the LET distribution remains uncertain. For this reason the spikes in the lower graph of Figure 3-17 are drawn with the same arbitrary height for all five components. Accordingly, the spikes should be interpreted merely as denoting the position of the steep terminal rise of the curve on the LET scale. A quantitative assessment of the extremely small fraction of the energy dissipation at the maximum LET would require a separate and entirely different approach. The fractional dose at the maximum LET represents, radiobiologically, an essentially unknown quantity best described dosimetrically with the term "microbeam" (145, 247).

This track of a very heavy charged particle in tissue is characterized by a very small central core of ionization caused by the particle itself, surrounded by a much wider area of ionization caused by the secondary particles ejected from the central core. Thus most of the volume of the track is attributable to secondary radiation, and a living cell in the path of the track would probably be affected predominantly by proton and electron radiation. It has been suggested that only about 5 to 10 millirads/day of the 40 millirads/day galactic ray dose at solar minimum have a LET above 30 keV/μ (238, 239).

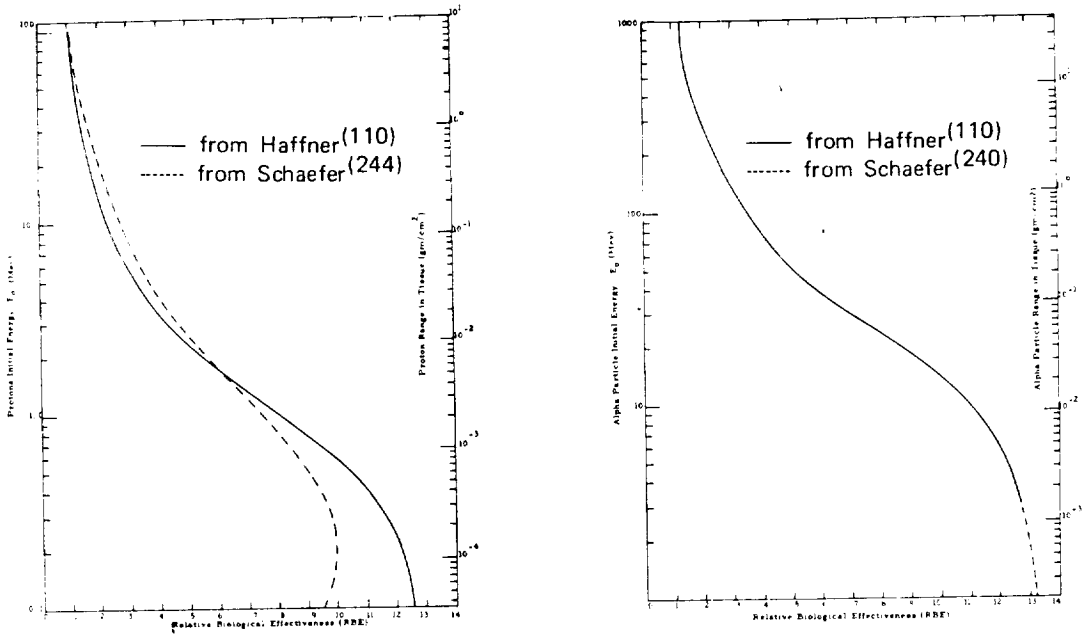
A large part of galactic radiation exposure falls into the region of low and very low LET values. For the proton component in particular, the bulk of the energy dissipation takes place at LET values even below the lowest LET of standard x-rays. In fact, the LET distribution of galactic protons closely resembles the one for Co-60 gamma rays. This is to be expected since for both radiations a large part of the energy dissipation is produced by secondary relativistic particles of single charge. Since LET depends only on charge and speed, but not on mass, there is no difference in the energy loss between electrons and protons of the same speed.

For calculation of local, effective tissue doses especially under conditions of low shielding, a reasonably satisfactory dependence of the RBE or QF on the

local Linear Energy Transfer (LET), has been obtained and experimentally verified (to some extent) by Rossi (215, 243). The relationship between RBE or QF and local LET for protons, alphas, and electrons of specific energy can be determined (110, 128, 240, 244). Figure 3-18a and b shows that for alphas and protons, RBE values vary from unity at high particle energies

Figure 3-18

RBE Versus Energy or Particle Range



a. Protons in Tissue

b. Alpha Particles in Tissue

increasing to 12 at low energies where electron acquisition becomes important. The upper critical energies, at which the LET = 4 keV/micron and, therefore, the RBE equals 1, are 10.8 MeV for protons and 249 MeV for alpha particles. The composite RBE values in infinite tissue (50 percent bone and 50 percent muscle) for protons and alpha particles between their initial and final energies are 2.1 and 2.2, respectively. Above 0.5 MeV, the two independent calculations are in good agreement (110, 240). Below 0.5 MeV (6 microns residual range), the Haffner calculations predict somewhat higher RBE values. Probably saturation effects (there are only so many atoms per unit path length for the particles to ionize), which the Haffner calculation did not take into account, are responsible for the differences. From the overall shielding viewpoint the differences are unimportant.

The RBE may be determined instantaneously or as the mean RBE of a particle during dissipation of its entire energy. The differences are seen in Figure 3-19.

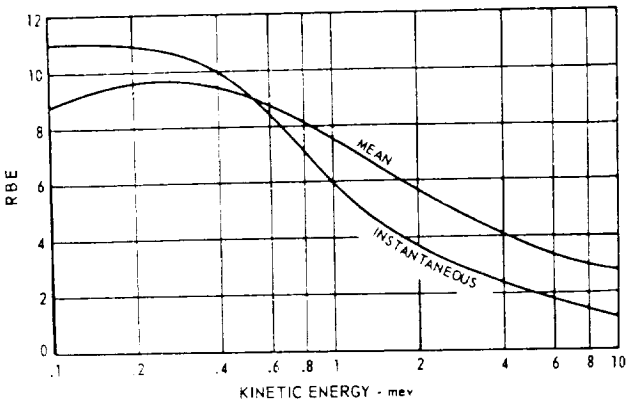


Figure 3-19

Mean Versus Instantaneous RBE Values for Protons.

Instantaneous and mean RBEs are shown for protons of different energies, corresponding to the instantaneous linear energy transfer at energy E, and the mean RBE for dissipation of the entire energy from E down to zero.

(After Grahn[103] from the data of Schaefer[244])

At higher LET's, the RBE saturates and begins to decrease. The curve varies for different biological systems. After acute doses, mammalian cells tend to respond as seen in Figure 3-20.

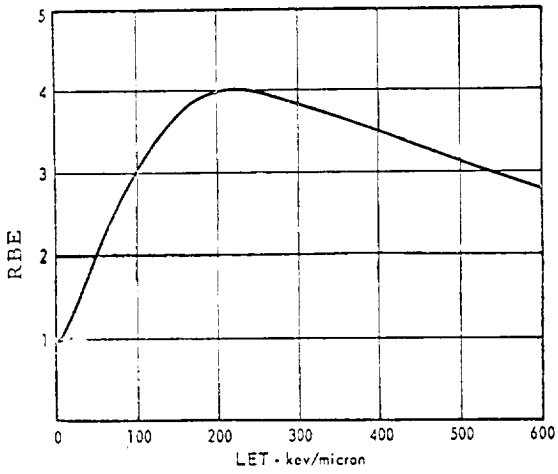


Figure 3-20

The Relative Biological Effectiveness (RBE) is Plotted as a Function of Linear Energy Transfer (LET) for Survival of Mammalian Cells Following Exposure to Charged Particles at About 8.3 mev/nucleon

(After Grahn[103] Based on data from Sondhaus[258])

The proton RBE for many biological endpoints has been studied for different energies in many biological systems (135, 145, 154). As predicted by LET considerations and the calculations of Figure 3-18, the empirical RBE (lethality) for protons and alpha particles >500 MeV is equal to or less than 1 (72, 259, 267). Depending on the particular type of radiation injury used as criterion, RBE values of 0.6 to 0.9 have been determined (135, 145). RBE values compared to Co^{60} gamma rays of 1 have been reported for iridocyclitis and erythema, and of 2, for epilation and desquamation in monkeys irradiated focally with 14, 39, 185, and 730 MeV protons (209, 298). However, in similarly irradiated animals, 730 MeV protons induced cataracts in 12 to 18 months at doses as low as 750 rads, whereas lower energy protons (14, 40, and 187 MeV) were ineffective even at doses as high as 2000 rads. This

observation of cataractogenesis with high energy but not with low energy protons is of interest, since damage to the lens is generally considered to be less pronounced with increasing energies of fast neutrons (158). More recent data, however, indicate that several hundred rads of protons in all of the energies under consideration can probably produce cataracts (18, 19).

As a given particle degrades in tissue, the RBE will rise as its energy transfer per micron rises. At the same time, a heterogeneous beam of protons will have an average RBE that tends to drop with increasing depth in tissue as the lower energy component becomes fully absorbed and the higher energy component continues its traverse. For gamma radiation, average body dose (ABD) is higher than midline tissue dose (MTD); and for protons, it is lower in large animals (267). Figure 3-21 represents calculations for depth-dependence of RBE for C-nuclei, alphas, and protons of 3 different rigidities: P_o = 50, 125, and 300 mv. The conservative RBE-LET relationship recommended by the ICRP was employed with a constant RBE of 10 being used for all values of LET above 150 keV/μ. The alpha and C-nuclei show a pronounced decrease of local RBE extending down to tissue depths well beyond 1 gm/cm^2.

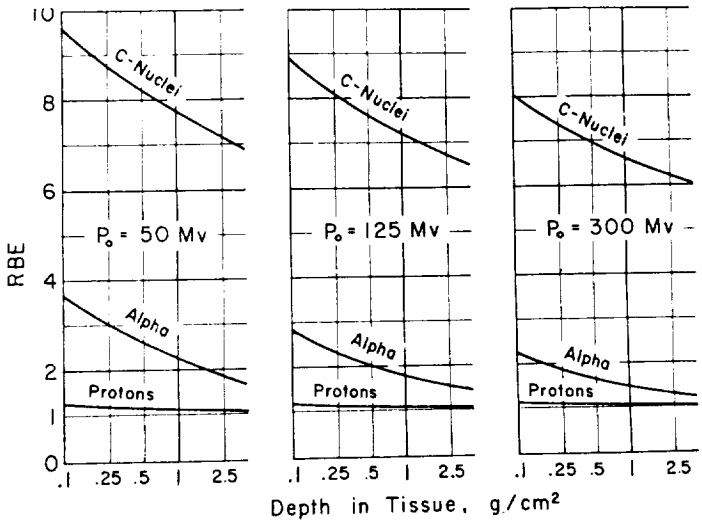


Figure 3-21

Local RBE or QF in Tissue at Increasing Depth

(After Schaefer[242])

The concept of RBE cannot be applied in those cases where special types of effects are produced by passage of very high LET particles and star formation in cosmic ray events (215). It is seen in Figure 3-17 that the proton contribution to the total dose from galactic primaries should be assigned an RBE well below 1.0. For the alpha component, the bulk of the energy dissipation falls well within the LET limits of standard x-rays, assuring that for this dose contribution an RBE of 1.0 is appropriate. The picture changes as one proceeds to heavier components. For C nuclei, a sizable portion of

the energy dissipation extends into the LET region for which the RBE factor exceeds 2.0. If the mean RBE for the absorbed dose from the C component is computed by numerical methods from the LET and RBE curves, a value of 1.56 is obtained. The corresponding mean RBE values for Ne and Ca nuclei are 2.86 and 6.64, respectively. Weighting these mean RBE values for the individual components according to their respective shares in the total dose and using a mean RBE of 0.75 for the proton contribution, one arrives at an overall mean RBE of 1.82 for the total dose from galactic primaries. This value might appear unexpectedly low in the light of other estimates in the literature (239). The discrepancy is due mainly to the fact that a ceiling value of 10 for the RBE was used whereas in other estimates values up to 20 have been assumed. Justification or preference for either value is largely subjective, though the higher values may be reasonable for protracted exposure and delayed effects.

It is possible to calculate the dose delivered to cells in a microbeam track core and the approximate range of LET of the particles. This has been attempted in Table 3-16. One cannot, however, assign LET or QF values to these microbeams (145). Attempts have been made to circumvent this problem but not enough data are available to substantiate the hypotheses (66).

The RBE or QF for electrons of all energies above .03 MeV is assumed to be unity (215).

The quality, nature, flux, and distribution of secondary radiations are dependent on the characteristics (flux, energy, mass, and charge) of the primary beam and on the materials and geometry of the spacecraft shielding as well as on the tissues of the crew. Present assessments of RBE or QF for secondary radiation doses for long-duration missions are estimates only, derived by complex analysis and evaluation of each organ system individually (290). Insofar as the QF's of various secondary radiations are concerned, it seems reasonable to assume a value of unity for secondary electrons and electromagnetic radiations, since their LET characteristics do not differ greatly from those of conventional x and beta rays. The QF of secondary protons may be assumed to be the same as for primary protons of comparable LET. As with protons, QF values for secondary neutrons are complicated by the dependence of effectiveness on energy, biological response (whether early or delayed), the organ or tissue under consideration (ocular lens vs. bone marrow, for example), and, to some degree, on depth within the body. Presently the most accepted values are those proposed by the NCRP (188). These values are shown graphically in Figure 3-22 and represent late effects of protracted exposure at a depth of 3 mm in tissue. The broken-line portion of the curve is an extension based on the observations of the secondary-proton spectrum (289). QF values applicable to early effects have not been specified by the NCRP or similar committees, but experimental observations on animals suggest values appreciably lower than those for production of late effects (41). Reported RBE values of fission neutrons (mean energy 0.5 to 1 MeV) for production of 30-day lethality and other early responses in experimental animals range from $\sim$1 to $\sim$3 (145).

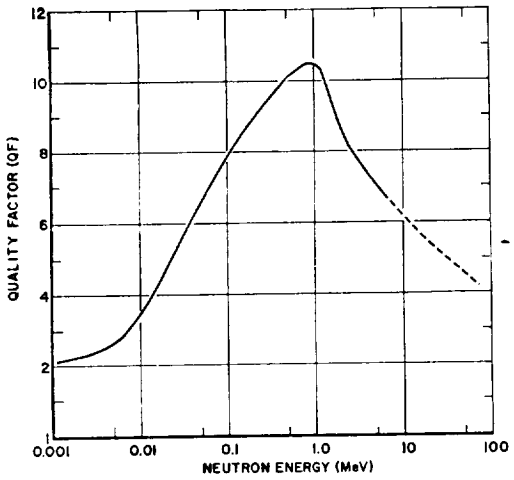


Figure 3-22

QF of Neutrons for Late Effects as a Function of Energy. Calculated for a Depth of 3 mm in Tissue Under Conditions of Protracted Exposure.

(After Langham-NAS-NRC[145] from the data of NCRP[188])

For the major space radiations, the LET decreases with increasing shield thickness and with increasing depth in the body, approaching (behind nominal shielding) that of conventional electromagnetic radiation at the depth of the bone marrow and the gastrointestinal tract. The greatest contribution of QF to the estimation of the biological dose equivalent therefore would be for early skin responses behind light shielding, as in the case of extravehicular operations.

Flux (Current)-To-Tissue Dose Conversion Factors for Nucleons

Conversion of particle flux data to tissue rads and rems is most difficult. The interaction of high-energy nucleons with matter initiates a complex avalanche of secondary particles with lower energy which proceeds through the medium, increasing in population and decreasing in total energy as energy is deposited in the medium. In general, a non-elastic interaction with a nucleus produces, first of all, several secondary nucleons which are due to direct interactions of the incident particle with the nuclear constituents and which have energies ranging from a few MeV up to a large fraction of the incident particle energy. There is left a highly excited, recoiling nucleus which rids itself of most of its excess energy by evaporating nucleons and heavy particles of relatively low energy of the order of a few MeV. Any energy left after evaporation presumably goes into the production of electromagnetic radiation. As a consequence of the significant contribution of the heavy particles and secondary protons to the rem dose, it is not reasonable to expect that the rem dose at any depth from incident protons can be calculated very accurately unless the secondary radiation created in the body is taken into consideration. For the case of incident neutrons this is obviously true, because only through secondary radiations is it possible for neutrons to deposit energy.

The data of Figure 3-23 and Table 3-24 were calculated by the Monte Carlo technique for the study of the transport of nucleons of energies up to 400 MeV through quite arbitrary geometrical configurations (138, 139, 169,

170, 295). The intranuclear cascade is treated by a subroutine version of Bertini's code (27) which is itself a Monte Carlo nucleon transport calculation on an intranuclear scale and gives the velocities and types of particles resulting from direct interaction processes. The evaporation portion of the cascade is handled by Dresner's subroutine (77). Protons below 50 MeV are allowed to proceed to the end of their range with no nuclear interaction, while neutrons below this energy are transported by neutron transport code (77). The data on Tables 3-6 and 3-7 were used for tissue factors in the calculations. In order to provide current-to-dose conversion factors which could be used to estimate upper and lower bounds on the doses for most practical cases of interest, the nucleons were made to impinge on a 30 cm tissue slab both normally in a broad beam and isotropically, with the expectation that these two extremes of incident angular distribution would represent the bounding cases.

The dose as a function of depth was calculated in units of rads and rems. Because of the uncertainties connected with the QF (quality factor) versus LET curves, the dose data were recorded in energy intervals in a manner that any preferred set of QF's could be employed to calculate the rem dose with relative ease. The energy deposition resulting from protons as they passed through the energy ranges 0-1, 1-5, 5-10,10-50, and >50 MeV was recorded separately. Average QF values, for each interval, of 8, 3, 1.25 and 1, respectively, were calculated from QF versus LET curves (190, 215). The values of the energy of the protons were correlated with the LET values by means of the stopping-power formulas. A constant value of 20 for the QF above a LET value of 1750 MeV/cm was used. This constituted a quite arbitrary assumption that a saturation effect takes place and can be represented by a constant QF at high LET values. It should be noted, that under all circumstances, the QF value of 20 is applied to the dose from the heavy evaporation particles and recoil nuclei in calculating the rem dose, since their LET value is generally above 1750 MeV/cm.

Figures 3-23a and b present the average total whole-body rad and rem doses for both normally incident neutrons and protons. Also shown is the average whole-body rad dose that would be received if the proton beam were totally absorbed. In comparison with the latter curve, it is easy to see that below 215 MeV, little error would be introduced if the whole-body rad dose were calculated on the basis that all the energy is totally absorbed. The reason for the primary proton dose having a discontinuity at 215 MeV is that above this energy the proton beam penetrates 30 cm of tissue and some of the energy is not deposited. The decrease in dose with increasing energy above 215 MeV is accounted for by the decrease in stopping-power with increasing energy in this energy range. Thus, less energy is deposited in the 30 cm of tissue as the energy increases.

The flux-to-dose conversion factors for average dose, 5 cm dose, and surface dose as well as maximum dose along the particle path can be calculated by an expression of the form:

$$\log_{10} D = A + BE + CE^2 \qquad (4)$$

where D is the dose in rem per nucleon per cm^2 and E is the energy in MeV. Table 3-24 contains the values of the coefficients.

Computer codes have been used to generate depth-dose curves for protons in spherical targets for cases where the usual slab techniques are inadequate (290). Depth dose due to an incident isotropic flux of monoenergetic protons, including the effect of primary and first-generation secondary protons can be determined in spheres of arbitrary size containing tissue-equivalent material. The sphere is chosen because it is, for present purposes, the simplest reference solid useful in showing the effects of the variables. Dose rate due to primary protons and three classes of proton secondaries are treated by the code. The secondary protons cover cascade, evaporation, and elastically scattered (hydrogen nuclei) particles.

In Figure 3-25, the dose rate contributed by each of the proton classes described above is recorded as a function of depth in the sphere. At each depth, the dose rate deposited by protons in each of eight energy intervals (0-1, 1-2, 2-5, 5-10, 10-20, 20-40, 40-80, and 80-∞ MeV) was tabulated separately for each energy interval and each class of protons. Only the total and primary dose contributions are indicated though others are available (290). Similar deposition data are available for 32 and 35 MeV protons (268).

Tissue current-to-dose conversion factors for neutrons with energies from 0.5 to 60 MeV have been recently programmed (123).

Flux to rad conversion functions may be combined with RBE-energy functions for protons (Figure 3-18a) to give rem dose/unit flux for each particle energy after acute doses (100, 110). Figure 3-26 represents such conversions.

Shielding and Tissue Range

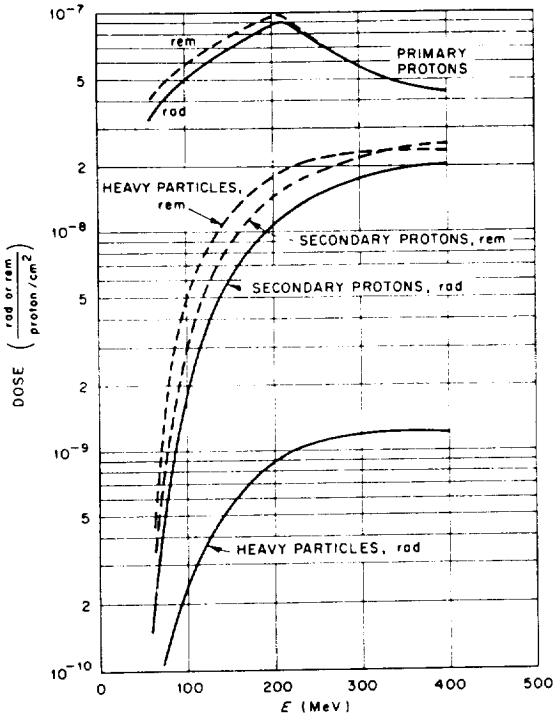
The design of shielding requires an understanding of the residual exposure of the human target behind the shield. The stopping power and ranges of various spacecraft materials for protons, mesons, electrons (24), and heavy ions (262), have been calculated and can be used with Tables 3-9 to 3-12 to determine radiation input to internal organs. Proton penetration codes are being continually refined (249, 250). Brehmsstrahlung from electrons hitting thick targets are receiving current re-evaluation (248).

Table 3-27 gives the minimum kinetic energy and rigidity of protons and alpha particles which can penetrate typical shielding projected for future missions. In the actual situation, of course, the thickness of shielding will be heterogenous in 4π geometry and will allow an influx of variable particles, kinetic energies and rigidities to hit the human target (280). For the Apollo vehicle, for instance, the range of shielding extends from 1.75 g/cm^2 to 212 g/cm^2, with lower thicknesses predominantly on the anterior side of the astronauts and larger ones on the posterior side afforded by the heat shield and fuel tanks, rocket engine, and other heavy equipment in the service module. In such systems, the dose distribution throughout the body becomes

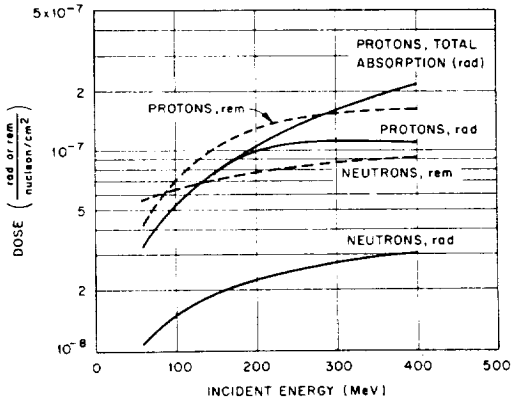
Figure 3-23

Flux-to-Dose Conversion for High Energy Protons and Neutrons

(After Kinney and Zerby[139])



a. Average Total Rad and Rem Doses Versus Incident Energy for Normally Incident Protons in Tissue (30 cm slab)



b. Average Total Rad and Rem Doses Versus Incident Energy for a Unit Flux of Isotropically Incident Protons and Neutrons in Tissue (30 cm slab)

Table 3-24

Coefficients of the Expansion for the Rem Dose Log D for Various Cases

(After Kinney and Zerby[139])

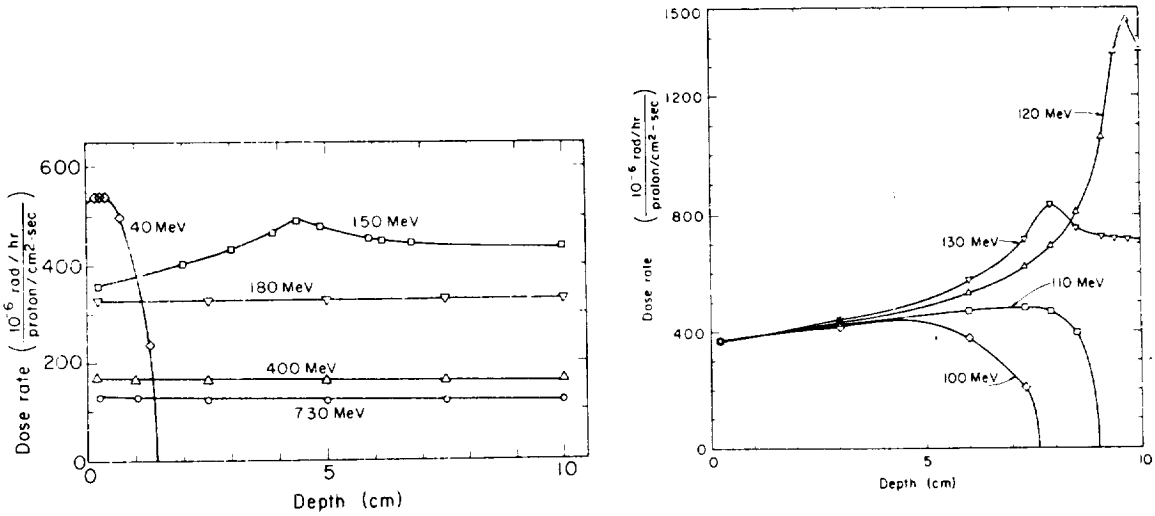
Normally incident protons	
Average dose	$-7.72+6.4\times10^{-3}E-1.1\times10^{-5}E^2$; $60<E<215$
	$-6.20-4.3\times10^{-3}E+5.5\times10^{-6}E^2$; $215<E<400$
5-cm-deep dose	$-6.27-4.6\times10^{-3}E+6.4\times10^{-6}E^2$; $80<E<400$
Surface dose	$-6.64-2.2\times10^{-3}E+2.9\times10^{-6}E^2$; $60<E<400$
Maximum dose	$-6.02-1.2\times10^{-3}E$; $60<E<215$
	$-6.62-1.1\times10^{-3}E$; $215<E<400$
Normally incident neutrons	
Average dose	$-7.43+2.7\times10^{-4}E$; $60<E<400$
5-cm-deep dose	-7.38; $60<E<400$
Surface dose	$-7.59+3.7\times10^{-4}E$; $60<E<400$
Maximum dose	$-7.35+3.8\times10^{-4}E$; $60<E<400$
Isotropically incident protons	
Average dose	$-7.79+7.9\times10^{-3}E-1.7\times10^{-5}E^2$; $60<E<215$
	$-7.07+1.2\times10^{-3}E-1.3\times10^{-6}E^2$; $215<E<400$
5-cm-deep dose	$-6.57-5.4\times10^{-4}E$; $80<E<400$
Surface dose	$-6.30-2.7\times10^{-3}E+3.7\times10^{-6}E^2$; $60<E<400$
Maximum dose	$-6.26-2.9\times10^{-3}E+4.1\times10^{-6}E^2$; $60<E<400$
Isotropically incident neutrons	
Average dose	$-7.26+5.6\times10^{-4}E$; $60<E<400$
5-cm-deep dose	$-7.18+3.9\times10^{-4}E$; $60<E<400$
Surface dose	$-7.26+4.5\times10^{-4}E$; $60<E<400$
Maximum dose	$-7.18+4.0<10^{-4}E$; $60<E<400$

Figure 3-25

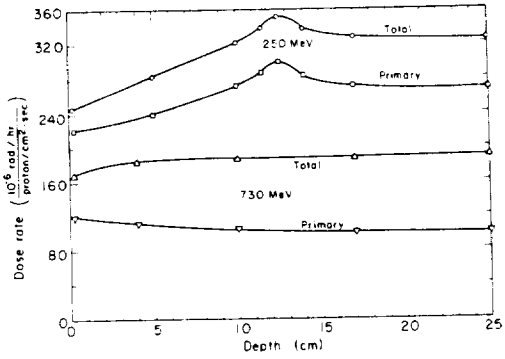
Proton-Depth Dose Patterns in Spheres of 10 and 25 cm Radius

(Total primary plus secondary proton depth-dose patterns due to monoenergetic isotropic fluxes of protons.)

(After Wallace et al[290])



a and b. 10-cm-radius Sphere of Tissue Equivalent Material (Two graphs used for clarity)



c. 25-cm-radius Sphere of Tissue-Equivalent Material

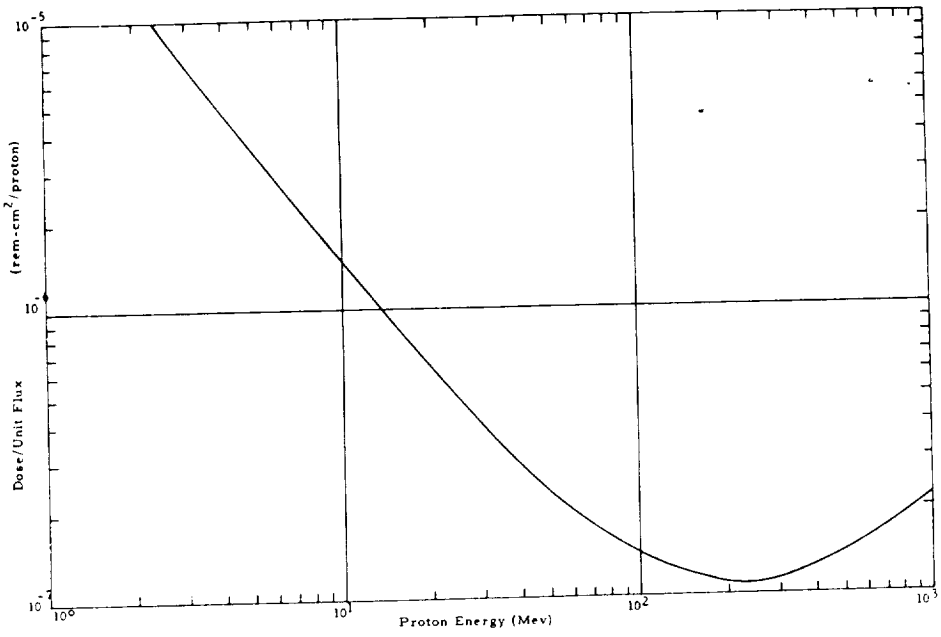


Figure 3-26

Flux to Rem Dose Conversion Function for Protons in Tissue

(After Haffner[110])

Table 3-27

Minimum Kinetic Energies and Rigidities for Protons and Alpha Particles Required for Penetration of Typical Shield Thicknesses

(After Schaefer[243])

Space System	Min. Shield. Equivalent, g/cm²	Kinetic Energy, Mev		Magnetic Rigidity, Mv	
		Protons	Alpha Particles	Protons	Alpha Particles
Space suit	0.1	8.4	33.6	125	252
Gemini vehicle	0.2	12.3	49.2	152	303
—	0.5	20.5	82.0	197	392
—	1.0	30.1	120	240	492
Apollo vehicle	1.5	37.8	151	269	545
Permanent lunar base	2.0	44.2	177	290	588

extremely complex. Figure 3-28 compares the typical flux of primaries which pass through typical shielding during exposure to solar flare and galactic protons. The relative levels vary with time because the magnetic field caused by the solar wind can screen off the galactic particles of low rigidity. The actual flux hitting the human target is not the primary flux noted above,

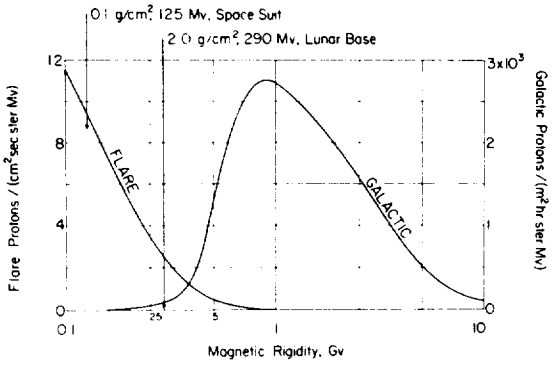


Figure 3-28

Comparison of the Differential Rigidity Spectra of Galactic Protons and of Protons of a Typical Solar Particle Beam

(Note the great disparity of ordinate units differing by a factor of 36 million). Galactic spectrum is based on the data of Freier and Waddington[92]; flare spectrum pertains to event of 17 July 1959.

(After Schaefer[243])

but the secondary and higher order products after absorption process in the barrier.

Tissue Range and Tissue Self-Shielding

For rough calculation of self-shielding or shielding by other humans, the simple range-energy relations of charged particles in matter can be used (293):

$$R = \delta E^n \qquad (5)$$

where R = range, gm/cm^2
E = energy, MeV
δ = constant - specific for each particle
n = constant - same for all charged particles

For bone and muscle, these constants are:

Bone $n = 1.779$, $\delta_{proton} = 2.30 \times 10^{-3}$
Muscle $n = 1.786$, $\delta_{proton} = 2.03 \times 10^{-3}$

Taking the human body to be 50% bone and 50% muscle (the bone is usually weighted more heavily in this way because of the importance of the marrow), the δ constant for protons becomes 2.17×10^{-3} and that for alpha particles 1.87×10^{-4}.

The range in tissue and stopping power vs. particle energy conversion can be seen in Tables 3-9 to 3-11. However, geophysical data are often expressed not as particle energy but in magnetic rigidity. Rigidity and depth of penetration or range are disparate magnitudes. Since rigidity is the momentum per unit charge, and alpha particles have twice the charge of

protons, equal integral fluxes of the same rigidity represent different fluxes in terms of momentum, energy, or range spectrum. Figure 3-29 may be used to convert rigidity spectra to tissue ranges. It shows that for a given rigidity the corresponding ranges of protons and alpha particles differ greatly.

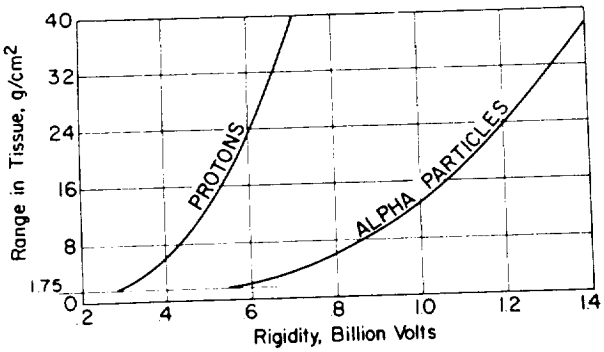


Figure 3-29

Range/Rigidity Function of Protons and Alpha Particles in Human Tissues

(After Schaefer[240])

Since the presence of shielding by tissue will alter the RBE of the emergent particles, the spectrum of the particles impinging the tissue must be considered in any operational configuration. If a particle integral energy spectrum is present of the form,

$$\Phi(E>E_o) = AE_o^{-a} \qquad (6)$$

where E and E_o represent the range of external spectral energies to be considered, RBE due to particles in a given energy interval is the integral of the product of the relationship of this power equation and the RBE factors of Figure 3-18. The time-integrated energy spectra of solar proton events appear to follow such a law down to ~10 MeV. Solar alpha particle spectra may follow a similar law. However, spectra behind tissue shields will be modified thus:

$$E_i = \left[E_o^{n_s} - (E')^{n_s}\right]^{\left(\frac{1}{n_s}\right)} \qquad (7)$$

where E_o = particle energy outside shield

E_i = particle energy inside shield

E' = particle cutoff energy of shield

n_s = constant for charged particles in the metallic shield material or in shielding tissue

Using the values of the constants n and δ in Equation (5), the particle cutoff energy E' for tissue shielding for any finite tissue thickness X may be calculated. Figures 3-30a and b give the emergent cutoff energy E' as a function of the initial energy E_o for protons at any tissue thickness. Figures 3-31a and b give similar values for alpha particles.

The feasibility of partial body shielding against ionizing radiation has been recently reviewed (198, 253).

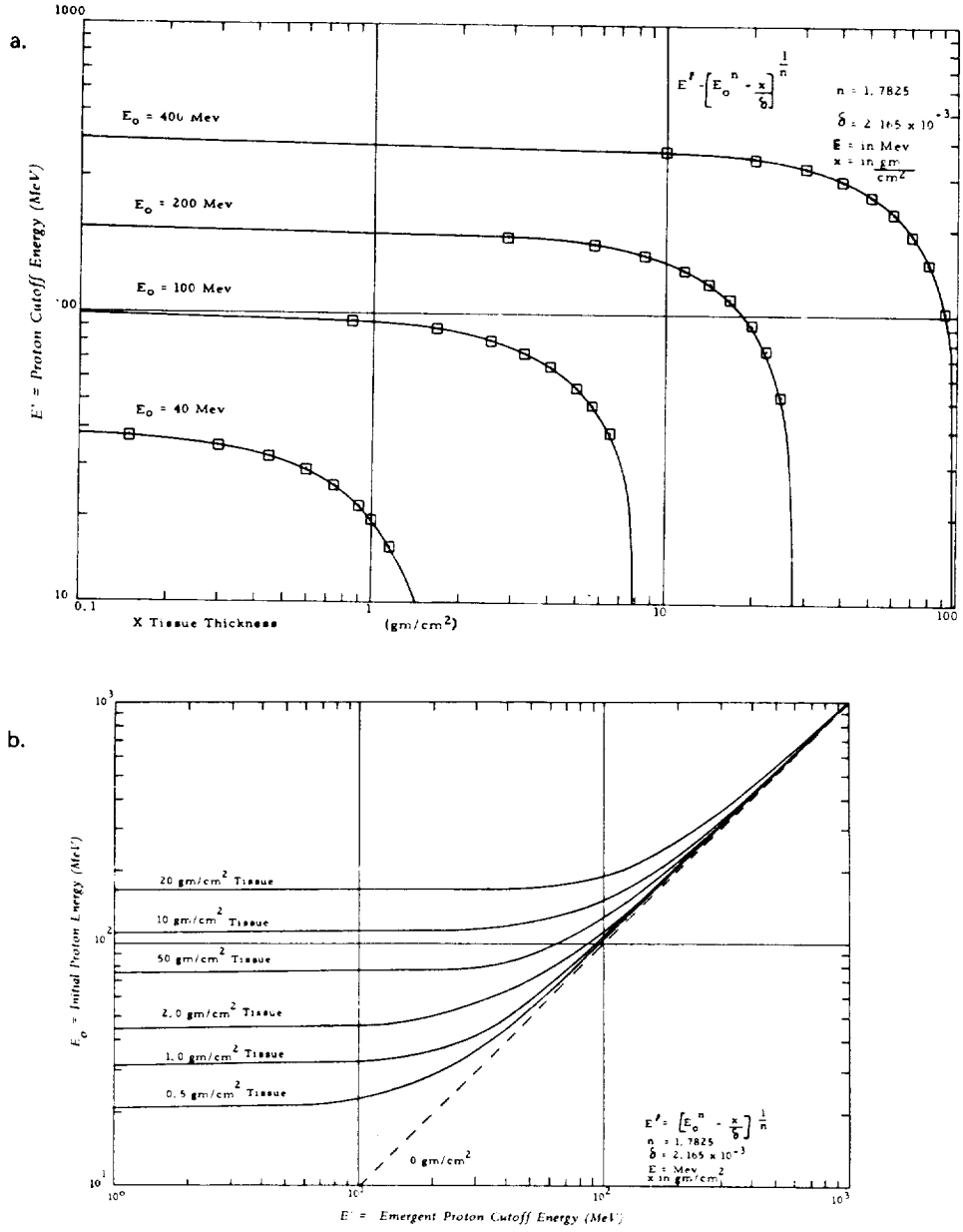
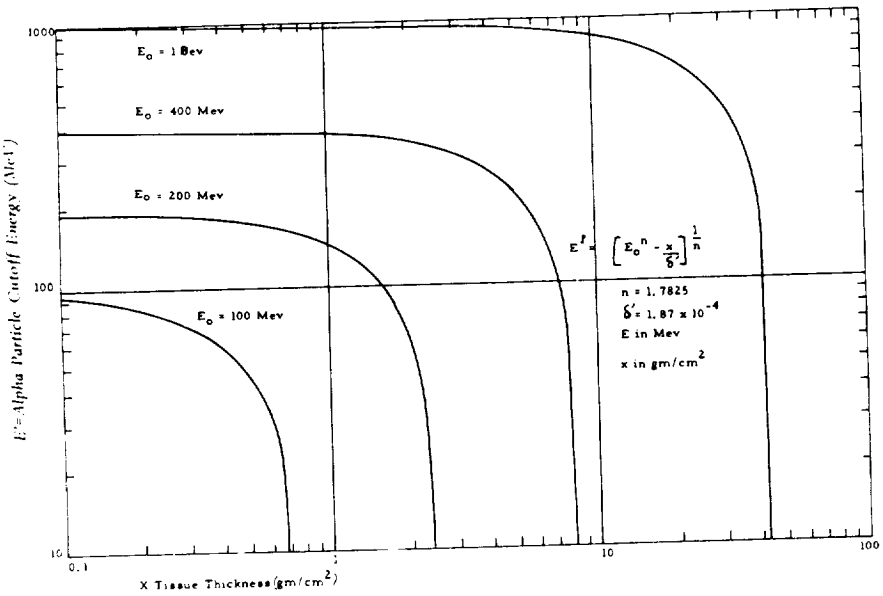


Figure 3-30

Emergent Proton Cutoff Energy at Any Depth of Tissue for Different Initial Energies (After Haffner[110])

a.



b.

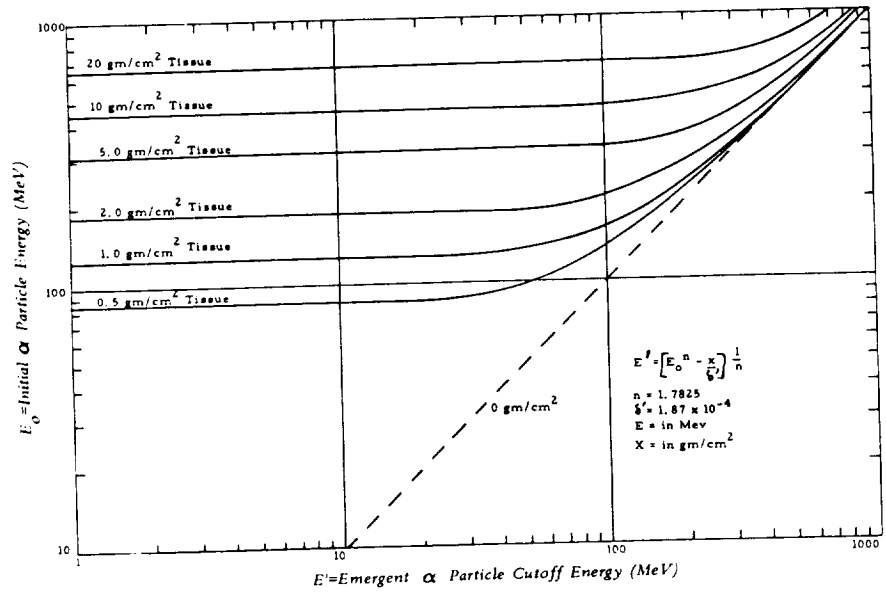


Figure 3-31

Emergent Alpha Particle Cutoff Energy at Any Depth of Tissue for Different Initial Energies (After Haffner[110])

Typical Depth-Dose Patterns in Space Under Shielding

Several examples will be given of the typical depth-dose under shielding of space radiation. Unfortunately, data are deficient on the charge and momentum spectra of solar flare particles measured directly in satellites. Current estimates are based on uncertain and incomplete data (162, 295).

Figure 3-32 shows the calculated dose from only protons at various depths in tissue for inner Van Allen belt and the Solar Proton Event of May 12, 1959, assuming cabin provides only 2 gm/cm^2 of shielding. The greater drop of tissue depth-dose from flare protons as compared to Inner Belt protons is a function of the differences in the integral energy spectra (see inset). The greater frequency of higher energy protons in the Inner Belt increases the dose rate in deep tissues. Note the importance of knowing the integrated energy spectrum of the proton radiation when considering the critical targets -- i. e., bone marrow, spleen, and intestinal locations beneath the surface. It is to be emphasized that no critical organ is located at a discrete depth below the surface. Since the body self-shielding is non-uniform, a more detailed analysis is necessary to determine the effect of a given radiation exposure on specific organs or organ systems.

Table 3-33 shows typical depth doses beneath the 14 largest events of solar cycle 19 under homogeneous shielding of different values. Since flare events occur essentially at random during the active part of the solar cycle, and since the magnitude of the individual flare dose does not show any correlation with the phase in the active half-cycle, the preferred way of formulating the flare hazard for the entire active period is to establish the probability of encountering, on a mission of given duration and for a randomly chosen launch date, an arbitrarily selected dose (145). It can be derived from Table 3-33 that 92 percent of the total dose is delivered in eight critical time intervals, none of them exceeding 10 days' duration, spaced at random over the 6 years. Sixty-four percent of the total is condensed still more to a few large increments on the ominous dates of February 23, 1956; July 10, 14, and 16, 1959; and November 12 and 15, 1960. As a consequence, the dose-probability plot is not a smooth curve but exhibits an irregular pattern. It should also be emphasized that the dosage received in the operational situation would be asymmetric and so the doses of Table 3-33 cannot be used directly to estimate probability of symptoms (253). Calculations have been made on the probability per week of different organs receiving varied doses under different shielding conditions when exposed to spectra typical of solar cycle 19 (80). Radiation shielding considerations and depth dose predictions have also been prepared for interplanetary flights (142).

Figure 3-34 compares degradation of depth dose for specific solar flares rigidities and inner Van Allen belt protons.

Figure 3-35 shows a rigidity spectrum of a typical solar flare of solar cycle 19 converted into a differential range spectrum in tissue for protons and alphas assuming a 1:1 flux ratio. The range spectra allow direct comparisons of fluxes that would reach the same depth in tissue or shielding material. For low and very low shielding the alpha flux drops much more

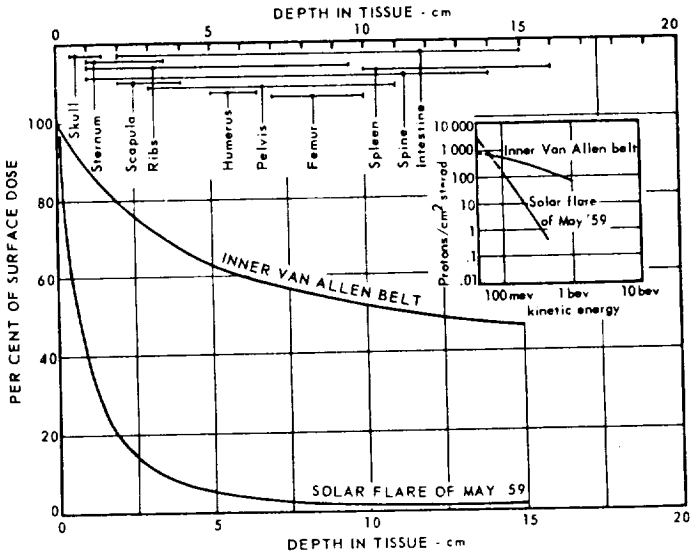


Figure 3-32

Depth Dose Under Solar Flare and Inner Van Allen Belt Exposure Under 2 gm/cm^2 (After Grahn[103] Adapted from Schaefer[246])

Table 3-33

Radiation Doses for the 14 Largest Solar-Particle Events of Solar Cycle 19 (After Langham (ed.)-NAS-NRC[145])

DATE OF EVENT / SHIELDING (g/cm²):	DOSE AT TISSUE SURFACE (rads) 1	2	5	10	DOSE AT 4-cm TISSUE DEPTH (rads) 1	2	4	6	10
Feb. 23, 1956	280	180	89	48	73	64	51	42	30
Mar. 23, 1958	148	54	10	2.1	6.4	4.5	2.55	1.53	0.66
July 7, 1958	150	54	9.5	1.93	6	4.3	2.35	1.4	0.59
Aug. 16, 1958	23.7	8.6	1.6	0.34	1.02	0.72	0.41	0.24	0.11
Aug. 22, 1958	45	14.7	2.24	0.38	1.33	0.91	0.49	0.27	0.11
Aug. 26, 1958	75	22.3	3	0.43	1.76	1.17	0.57	0.3	0.11
May 10, 1959	470	206	55	15.6	38	29.3	18.2	12.5	6.4
July 10, 1959	420	210	69	24.5	50	40	27.5	19.5	11.5
July 14, 1959	650	273	72	19.5	48	36	22.8	15.1	7.5
July 16, 1959	382	191	63	22.3	46	36	25	17.7	10.5
Nov. 12, 1960	484	263	100	43	75	62	46	34	20.8
Nov. 15, 1960	288	151	53	20.5	39.6	31.7	23	16.6	10.1
July 12, 1961	25.7	8.4	1.28	0.22	0.76	0.52	0.28	0.15	0.06
July 18, 1961	128	63	20.4	7.2	15	12	8.1	5.7	3.3
Grand total of all 50 events of Cycle 19	3,914	1,837	584	217	426	342	241	176	107

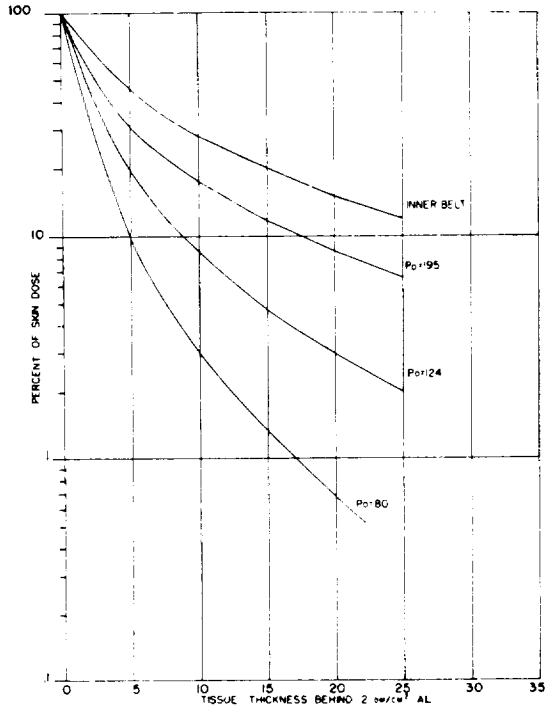


Figure 3-34

Variation of the Percent of Skin Dose as a Function of Tisse Depth for the Three Solar Flare Rigidity Spectra in the Range Expected

(After Jones et al[134])

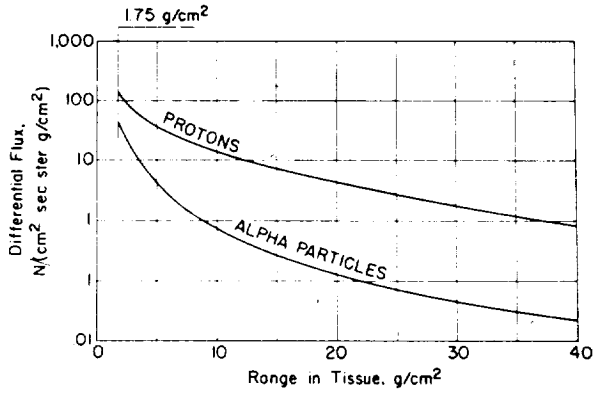


Figure 3-35

Differential Range Spectrum for Protons and Alpha Particles of Solar Flare 19 Assuming a 1:1 Flux Ratio of These Particles.

(After Schaefer [240])

steeply at greater depths than the proton flux. This indicates that possible objectionable exposures from solar flare alpha particles can occur only for low shielding as, for instance, for an astronaut outside the vehicle merely protected by his space suit with equivalent shielding of only 0.3 gm/cm^2 for suit and thermal-meteorite coverall.

Figure 3-16 gives spectral absorbed dose contribution of typical primary galactic cosmic ray particles and the dose per particle calculated for a 10 μ cell at the center of the track of heavy particles. The only available empirical data on absorption of galactic radiation with buildup of secondaries is in the atmosphere. It has been estimated that the corresponding buildup in a compact scatterer containing high-Z materials would be substantially higher. A total dose of 30 to 50 mrads/24 hr would seem a conservatively high estimate (145, 243). A major uncertainty is introduced by the neutron component of the secondary radiation. Neutrons play an important role in the development of nucleonic cascades, yet data obtained by various authors on the altitude profile of the neutron flux in the atmosphere and the corresponding transition of the local neutron energy spectrum seem to differ greatly. This particular limitation of present knowledge on the transition of the galactic beam is all the more important for assessments of tissue dosages because the neutron component would require elevated QF or RBE, thus substantially

enhancing the dose equivalents (see Figure 3-22). Discrepancies still exist with respect to the exact configuration of the lower side of the galactic energy spectrum extending down to thermal energies. However, it now seems well established that, contrary to the spectra of trapped or flare-produced particle fluxes with their very large fluxes at low energies, the galactic spectrum maintains its maximum flux at high energies with values decreasing toward lower energies. As a consequence, dose rates from the galactic field even under worst conditions (solar minimum; location outside the magnetosphere; low shielding) should not greatly exceed the estimate of 50 mrads/24 hr (145).

The actual doses received in space flight to date fall within these predicted ranges for quiet solar years. American and Soviet space flights have been well below the trapped radiation belts (275). The apogee and perigee for the highest prolonged Gemini flight were 100 and 215 miles respectively for 128 orbits. The highest Soviet flight was the Voshkod 2 with perigee and apogee at 107 miles and 308 miles respectively for 17 orbits. Beneath 3.2 mm of Al, the mean total radiation doses of the Soviet Cosmonauts in the Vostok series were measured as ranging from 8.4 to 17 ± 2 mrads/day (286). Ninety percent of the dose was from the primary cosmic radiation and 10 percent from the inner belts at an orbital vector of 65° and apogees of 409 km. In U. S. astronauts the highest dose rates in the South Atlantic anomaly were recorded as 107 mrad/hr in the portable ionization chamber and 363 mrads/hr in the fixed chamber of Gemini IV (14). Differential spectra of Gemini IV and VI are available (126). Over the 10 day period of Gemini VII, the dose rate per man averaged about 11 mrad/day (26 , 241). Emulsions on MA8 and MA9 showed that the inherent shielding of the capsule was sufficient to absorb electrons and secondary brehmsstrahlung from the artificial belt.

Long duration missions on the moon require the use of electrical power sources other than solar cells. Such power sources are radioisotopic and rely on plutonium 238, curium 244, strontium 90, and others as the heat generator (187, 234). Thermoelectric or thermionic converters are used to convert the heat into electrical power. Since a 100 MeV alpha particle requires only 0.8 gm/cm^{-2} of aluminum for absorption (0.3 cm thick or 0.12 inch), the shield around the unit will, for all practical purposes, absorb or attenuate the alpha particles. This leaves a radiation environment of neutrons and gamma rays from primary decay and secondary emission in the shield.

The external radiation characteristics of lunar, SNAP, radioisotopic thermoelectric generator (RTG) modules or units at a distance of 1 meter are as follows (96):

Neutrons	Snap 19	Snap 27
mrem hr^{-1}	1.5	125
neutrons cm^{-2} sec^{-1}	10.5	8.75 x 10^{2}
Gamma		
mrads hr^{-1}	2	11 ($\perp$ to long RTG axis) 1.7 ($\parallel$ to long RTG axis)
photons cm^{-2} sec^{-1}	14	77

For either the neutron or the gamma radiation, the mean energy is nearly 1 MeV for these Pu 238 RTG's. For a cluster of RTG's, the total radiation dose is the sum of the individual doses, accounting for the distance from each RTG. Data are available on the shielding and radiation fields for other SNAP and reactor systems proposed for spacecraft and lunar surface operations (10 , 22 , 129 , 141 , 187).

EARLY EFFECTS OF ACUTE RADIATION AT HIGH DOSE RATES

Table 3-36 represents the expected early effects of acute whole-body irradiation. It should be emphasized that these thresholds do not hold true for partial body and protracted radiation of the same total dosage. They do not cover exposure to simultaneous environmental stress of other types. The latency periods and relative duration of symptoms are dependent upon the penetration, quality factors, total dose, dose distribution, and intensity of the exposure. In very general terms, limiting systemic and/or tissue responses are:

1. Acute gastrointestinal or prodromal symptomatology, i. e., nausea, vomiting, diarrhea. These may appear within an hour or two and subside within a day at any dose above about 50-100 r at the midline.
2. Acute hematopoietic symptomatology, i. e., thrombocytopenia, leukopenia, hemorrhage, intercurrent infection. These symptoms will appear within a few days to a week and can reach a clinically aggravating level at doses of 50-150 r or more to the marrow within several weeks to a month.
3. Widespread erythema and skin blistering. Under certain circumstances, such as extravehicular operations, high intensity surface exposure with little deep tissue dosage may occur. Depending upon the quality of the radiation, erythema will appear within a few hours to days following exposures of 400 r to 800 r. Severe damage will occur at doses above 1400-2000 r. Due to the restrictions and abrasive contacts of the space suit, even a partial body moderate erythema could become extremely uncomfortable and somewhat incapacitating.
4. Degradation of general operational skills through direct and indirect physiologic and neurologic injury, i. e., lassitude, fatigability. At lower doses, acute systemic radiation injury is accompanied by nebulous symptoms of reduced performance capacity interfering with information processing, decision-making, emotional stability, and motivation often related to the prodromal symptomatology. At higher doses, vascular shock, cerebral edema, and hypoxia of the central nervous system all contribute to the entity often referred to as the "central nervous system syndrome."

Table 3-36

Expected Early Effects from Acute Whole-Body Radiation on Earth

(Modified from Glasstone[102])

Dose in Rads	Probable Effect
0 to 50	No obvious effect, except, possibly, minor blood changes and anorexia.
50 to 100	Vomiting and nausea for about 1 day in 10 to 20% of exposed personnel. Fatigue, but no serious disability. Transient reduction in lymphocytes and neutrophils.
100 to 200	Vomiting and nausea for about 1 day, followed by other symptoms of radiation sickness in up to 50% of personnel; < 5% deaths anticipated. A reduction of approximately 50% in lymphocytes and neutrophils will occur.
200 to 350	Vomiting and nausea in 50 to 90% of personnel on first day, followed by other symptoms of radiation sickness, e.g., loss of appetite, diarrhea, minor hemorrhage; 5 to 90% deaths within 2 to 6 weeks after exposure; survivors convalescent for about 3 months.
350 to 550	Vomiting and nausea in most personnel on first day, followed by other symptoms of radiation sickness, e.g., fever, hemorrhage, diarrhea, emaciation. Over 90% deaths within 1 month; survivors convalescent for about 6 months.
550 to 750	Vomiting and nausea, or at least nausea, in all personnel within four hours from exposure, followed by severe symptoms of radiation sickness, as above. Up to 100% deaths; few survivors convalescent for about six months.
1000	Vomiting and nausea in all personnel within 1 to 2 hours. Probably no survivors from radiation sickness.
5000	Incapacitation almost immediately (several hours). All personnel will be fatalities within one week.

Figure 3-37 presents the mean survival time of man vs. the acute rad dose of whole-body x-ray radiation. The major cause of death is indicated for the appropriate dose range. The possibility of GI tract death and CNS damage occurring at lower doses with protons and alpha particles will be covered below. Defects in all functions of these organ systems can be expected.

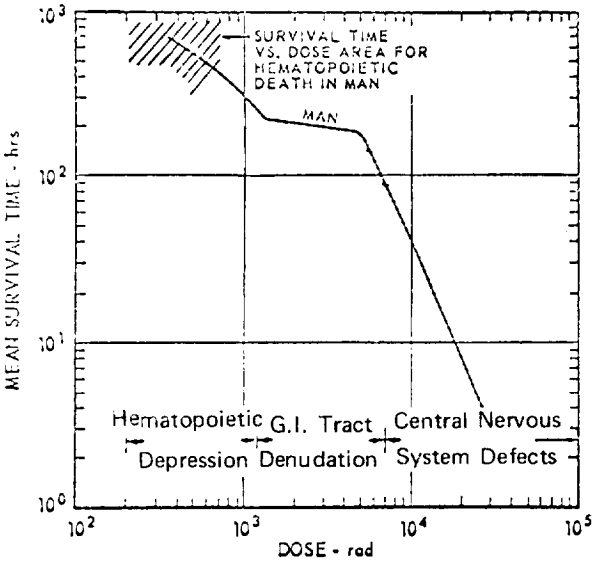


Figure 3-37

The Relationship Between Mean Survival Time and Acute Radiation Dose for Man

This curve is extrapolated from animal studies and very few human studies. It holds only for acute total body radiation. Note spread of data.

(After Grahn[103] Adapted from Langham[146])

Whenever possible, the above responses will be examined in a probabilistic manner. Not all individuals may show the symptoms mentioned above at the stated dose levels. In defining upper-limit emergency doses, the lowest limit will be the first determinant, modified by depth-dose protraction, distribution, and dose rate. For example, at high dose rate whole-body exposure to a penetrating radiation will undoubtedly cause the dose for prodromal responses to be determinant. A more protracted exposure will bring hematopoietic injury into the determining position, and when moderate to high doses of very low energy radiations prevail under certain unshielded exposure conditions, skin injury will be determinant.

Alteration of these response patterns by space radiation is not clear. Protons, like neutrons, are more effective in producing gastrointestinal symptoms following whole-body irradiation of several species (68, 69, 70, 72, 134, 154, 259, 267). How much protons will modulate the usual x-ray response in humans is not clear since the effect is postulated to be a complex function of relative secondary electron flux in bone, bone size, marrow geometry, ionization density, LET, dose distribution, and dose rate. Early reports that hemorrhage appears earlier and is more severe in large animals irradiated by proton than by x-ray (69, 70, 108, 178) have not always been substantiated (267). Symptoms of the nervous system are variable. Death is reported in animals exposed to 2000 rads of 187 MeV proton within 100 to 200 days after exhibiting central nervous system (CNS) symptoms (216). It has also been found that 6000 rads of 40 MeV protons to the whole-body

(given in two parts - upper and lower halves) caused convulsive seizures and death in about 48 hours following exposure, suggesting a CNS radiation effect. Possible latent or long-term effects based on this observation of a gradual onset of lethargy, anorexia, and ataxia exhibited among survivors of whole-body, proton-irradiated animals at 2 1/2 to 5 1/2 months irradiation have been suggested but not always found (267). In extrapolating from animal data, profound species differences must be kept in mind (39, 61). The above animal data should not be used as examples of dose-specific symptoms expected in man after proton irradiation but as background information about possible aberrant responses in humans to be guarded against when extrapolating to proton radiation effects from x-ray or gamma data obtained from radiation of humans.

Even less is known about high energy alpha particles. Differences in the timing and severity of response between high energy protons and alpha particles in small animals have been demonstrated (258). Extrapolation to humans is not as yet possible from these data.

Lethality

In studying human lethality after irradiation many LD_{50} doses have been proposed ranging up to 700 rads (28, 63, 102, 114, 154, 178, 179). The spontaneous 60-day death rate for persons between 25 and 44 years is .02% (148). Data obtained from irradiation of patients (160, 292), atom bomb casualties (39, 292), large animal studies (60), and evaluation committees (145, 185, 278) have been reviewed, and the best probabilistic early-lethality curve for acute total-body irradiation has been proposed as seen in Figure 3-38. This study suggests an LD_{50} value of 286± 25 for normal man. The suggested values for LD_{10} and LD_{90} are given in Table 3-39. These recommendations can be compared with previous LD_{50} estimates for humans indicated by different committees and groups as seen in Table 40.

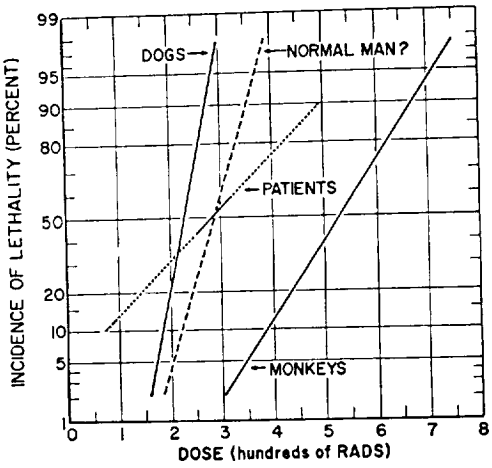


Figure 3-38

Derived Dose-Lethality Relationships for Dogs, Monkeys and Human Patients and a Postulated Relationship for Normal Man.

(After Langham (ed.)-NAS-NRC[145])

Table 3-39

Estimated High-Intensity Whole-Body Dose Levels for Production of Hematopoietic Lethality*

(After Langham (ed.)-NAS-NRC[145])

Response Probability Level (percent)	Dose (rads)**
10	220
50	285
90	350

*Symptoms begin within a few hours with the prodromal reaction, followed by progressive hematological depression terminating in death in 2 to 8 weeks.

** Point of interest for dose estimation: 11-cm depth; QF assumed to be unity.

Table 3-40

Estimates of LD_{50} of Man for High-Intensity Radiation Exposure

(After Langham (ed.)-NAS-NRC[145])

Source	Exposure (R)	Dose (rads)[a]	Reference
Patients, pathology of atomic bomb casualities	~450	~300[b]	Warren and Bowers (292)
Committee evaluation	400-600	260-400[b]	NAS-NRC (185)
Marshallese observations, large-animal studies	~350	~300	Bond and Robertson (39) Cronkite and Bone (60)
Committee evaluation	--	300-500[c]	United Nations (278)
Whole-body exposure of patients	370[b]	243±22	Lushbaugh et al (160)
Whole-body exposure of patients	380[b]	250±28	Langham (ed.)NAS-NRC (145)
Whole-body exposure of patients and accident cases	430[b]	285±25	Figure 3-38

[a] Average absorbed dose near midline of the body.
[b] Assuming radiation of the quality of ^{137}Cs gamma rays.
[c] Nature of radiation and point of dose assessment not specified.

Partial-body, nonuniformly-distributed doses are less effective than a uniform whole-body dose for production of lethality in the dosage range of the early hematological syndrome. Except when exposure involves a major portion of the trunk, this decrease in effectiveness is true, even when the dose is expressed in terms of integral absorbed energy (kilogram-rads). A dose to the extremities exclusive of the trunk would be much less effective. Based on fraction of total body mass and active bone marrow, an arbitrary choice of a volume factor (f_v) of 1/5 has been suggested for the extremities (145). Dose distribution effects on lethality are covered in section on Dose Distribution Factors and Progressive Performance Decrement).

In animals, the LD_{50} dose increased with decreasing energy (decreasing depth dose) (106). Sparing a portion of the bone marrow by nonuniform dose distribution (either by irradiating only a portion of the body or by decreasing depth-dose distribution) results in increased protection against early death (145). Under operational space conditions, the geometry of a high-intensity exposure through influence on depth-dose distribution may have a pronounced effect on lethal response if the shielding geometry of the spacecraft and position of the crewmen are such that dose delivery approximates unilateral exposure. Use of midline tissue dose to characterize exposure seems to give the most acceptable correlation with response for exposure geometries other than unilateral exposure (39). The quality factors of Table 3-3 may be applied. Protraction factors for lethal doses are given in Table 3-5, Figure 3-53, and the discussion of Progressive Performance Decrement.

Prodromal Symptoms

In spite of individual idiosyncracy, the dosage parameters for prodromal reaction follows a definite pattern (99 , 102 , 148 , 157). After a short latent period, a feeling of fatigue is followed by depression and emotional disturbance accompanied by anorexia, nausea, retching, salivation, and vomiting. Intestinal cramps and diarrhea occur prodromally at lethal doses. Symptoms reach a peak in about 4 to 6 hours and then improve rapidly. For 200 and 300 rads the peak may occur as quickly as 2 hrs after exposure. The degree of upset and duration of recovery depend on size and location of dose, on individual sensitivity, and, most importantly, on the dose rate. Radiation to the epigastrium is particularly productive of prodromal systems. The volume factor (f_v) for irradiation of extremities should be the same as that for lethality or hematologic response, or 1/5 that for whole-body radiation (145). Antinauseant and tranquilizer therapy are effective in reducing symptoms.

The time of onset of the prodromal complex of nausea and vomiting, empirically derived, may be seen in Figure 3-41.

The dose response is a probabilistic function expressed often as the 50% symptom dose or SD_{50}. The probability of symptomatic response may be expressed as a normal or logarithmic function:

$$Y = a + b(D) \qquad (8)$$

or

$$Y = a + b(\log D). \qquad (9)$$

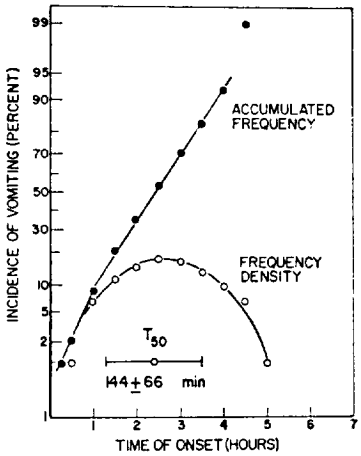


Figure 3-41

Anticipated Elapsed Time Between Irradiation and Onset of Severe Nausea and Vomiting in a Sample of 100 Persons Exposed to Radiation Doses in the Lethal Range.

(After Langham (ed.)-NAS-NRC[145] Adapted from Gerstner[99])

In either case, Y is the probability of the response as percent transformed into probit units, D is the radiation dose, a is the intercept, and b is the slope constant. Such dose-response relationships can be used to predict a dose that will elicit any given level of response in an infinitely large number of subjects, providing the fiducial limitations of the data are known. Data from atom bomb casualties, nuclear accident victims, and irradiation of cancer patients have recently been summarized (145).

The most well-controlled data were obtained from 165 patients treated for neoplastic disease by isotropic gamma radiation in the Cesium 137 total-body radiation facility at the Medical Division of the Oak Ridge Associated Universities (159, 160). The data were normalized to an effective rad dose of the prodromally sensitive epigastrium (100 r to the midline results in only 66 rads to a 10 cc epigastric volume). A dose rate of 1.5 r/min was used.

Table 3-42 gives the extrapolated SD_{10}, SD_{50}, and SD_{90} with account

Table 3-42

Estimated High-Intensity Radiation Dose Levels for Production of Early Prodromal Response

(After Langham (ed.)-NAS-NRC[145] from data of Lushbaugh et al[145,159,160])

	ABSORBED DOSE FOR PROBABILITY OF RESPONSE (rads)		
CLINICAL SIGN	10 PERCENT	50 PERCENT	90 PERCENT
Anorexia	40	100	240
Nausea	50	170	320
Vomiting	60	215	380
Diarrhea	90	240	390

Point of interest for dose estimate: a 26-cm diameter sphere in the mid-epigastric region; QF assumed to be unity.

made of the concurrent signs of disease by Abbott's method. Because of the nature of the patients and the uncertainty of the corrections for concurrent

illness, this table should be considered conservative with higher SD values and wider range of distribution than would be expected in a normal population. The data shown in Figure 3-41 and Table 3-42 are extrapolated from x-ray and gamma experiments and may not be quantitatively appropriate for space radiation, especially when depth dose variations are brought to bear. Appropriate modification of these probabilities for multi-energetic protons and alpha particles as well as for specific dose rate factors is not known from empirical data. Quality factors should follow suggestion of Table 3-3 since the radiation at midepigastric region should typically give a low enough LET to have a QF of 1.

Dose protraction is known to affect prodromal symptomatology (see Table 3-5). Increased sensitivity to large second doses has been noted as well as the prolongation of fatigability and listlessness for as long as 60 days after sublethal exposures (145). Predictions for chronic low level exposure will be covered below under Progressive Performance Decrement on p. (3-63).

Hematological Syndrome

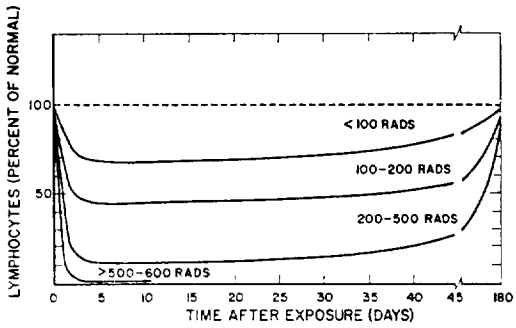
Hematological responses to radiation are largely dependent on damage to the marrow and lymphoid tissue (55 , 202). Damage to the chromosomal mechanisms of the cell inhibit normal division and reproduction of the elements, resulting in decrease in blood counts. Signs and symptoms develop in relation to depression of specific elements. Whole-body irradiation with doses in the lethal range (above about 200 to around 600 rads) causes a typical illness characterized by early transient nausea and vomiting (the prodromal syndrome) followed by a latent period of several days or a week or more, the length of which is inversely related to the dose (see Table 3-36). Signs and symptoms then develop relating to depression of blood elements; infections and fever, relating to granulocyte depression and impairment of immune mechanisms; and bleeding and possibly anemia, relating to platelet depression. Anemia from red-blood-cell depression does not usually occur. These symptoms may lead to death if the bone marrow is incapable of responding in time by adequate cellular regeneration.

The time-course of changes in most of the peripheral-blood elements is fairly well correlated with the dose of radiation to the bone marrow. The description is taken from recent reviews of the subject (55 , 145). Based on dose relationship of these changes, the following categories of prognosis in irradiated persons may be made: (1) survival almost certain (dose <100 rads); (2) survival probable (dose 100 to 200 rads); (3) survival possible (dose 200 to 500 rads); and (4) survival very improbable (dose greater than 500 to 600 rads). Figures 3-43 a to d indicate roughly the smoothed average time-course, based on the human cases from accidental exposure, for lymphocytes, neutrophils, and platelets in these four categories (37 , 44 , 62 , 109 , 115 , 117 , 124 , 145 , 159 , 254 , 288). The changes are represented as percentages of normal counts (the average levels for the population), and the curves portray generally the time-course of changes as a function of radiation dose.

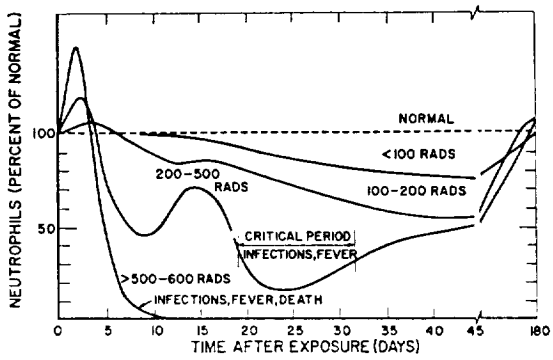
Figure 3-43

The Time Course of Blood Changes After Different Doses of Radiation of QF = 1.

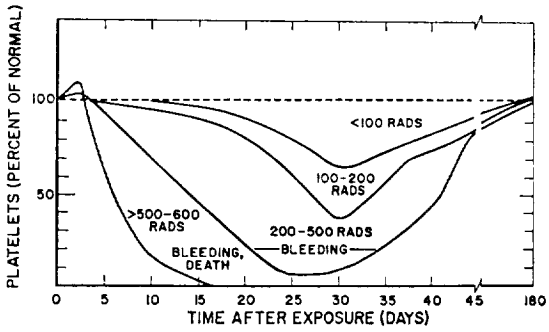
(After Langham (ed.)-NAS- NRC[145] from many sources (see text))



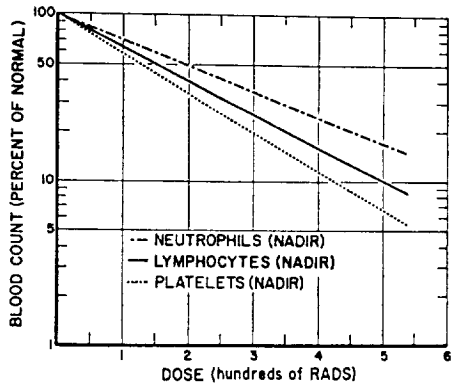
a. Smoothed Average Time-Course of Lymphocyte Changes in Human Cases from Accidental Radiation Exposure as a Function of Dose.



b. Smoothed Average Time-Course of Neutrophil Changes in Human Cases from Accidental Radiation Exposure as a Function of Dose.



c. Smoothed Average Time-Course of Platelet Changes in Human Cases from Accidental Radiation Exposure as a Function of Dose.



d. Idealized Average Dose-Response Relationship for Lymphocytes, Neutrophils, and Platelets in which the Nadir of Each Blood Element is Plotted Against Dose.

The lymphocyte count (Figure 3-43a) is significantly depressed in 1 to 2 days even with doses of less than 100 rads and approaches zero at that time with near-lethal doses. The return to normal is slow and may be incomplete months and even years later, as has been the case with the exposed Marshallese people (56). Neutrophils increase in the first few days after exposure to doses greater than 100 rads (Figure 3-43b). This initial leukocytosis is even greater with higher doses. The neutrophil count then declines steadily and either levels off or has an "abortive rise" after about 8 to 20 days unless a lethal dose has been given. Absence of this latter phenomenon following substantial radiation dose is a grave prognostic sign. Following the abortive rise, further depression ensues, the nadir being reached earlier with higher doses (8-10 days with lethal doses to 40-45 days with sublethal doses). If profound neutropenia occurs, serious or fatal febrile infections may develop. If the bone marrow is able to regenerate sufficiently before overwhelming infection and death occur, the neutrophil count will climb toward normal during the ensuing weeks, and recovery of the individual is likely. Complete return to normal counts may be delayed for months, or, with higher doses, the counts may actually rise above normal levels for a time.

Changes in platelet counts are shown in Figure 3-43c. Lowest levels are reached in about 24 to 32 days following all but lethal doses where levels may approach zero by 10 to 15 days. Bleeding is likely to develop when the platelets approach zero. With bone-marrow regeneration, platelet recovery may begin rapidly but be incomplete for months or years.

Figure 3-43d shows an idealized average dose-response relationship for lymphocytes, neutrophils, and platelets in which the nadir of each of the blood elements is plotted against dose.

Daily fluctuations in individual counts and normal variations among individuals limit the usefulness of blood counts as a direct biological dosimeter or precise detector of radiation damage. However, repeated hematological examinations afford valuable prognostic information. The lymphocyte count is valuable as an early criterion of radiation injury. If there are 1, 200 or more lymphocytes/mm^3 at 48 hr after exposure, it is unlikely that the individual has received a fatal dose. Lower lymphocyte counts at this time indicate more serious exposure. Neutrophil and platelet counts are of value in following subsequent progress of exposure cases and as general indicators of the seriousness of the exposure.

Although precise information is lacking on the threshold doses necessary to produce symptoms from blood-cell depression, it might be stated generally that only a few individuals would show hematological symptoms following a whole-body dose of 200 rads, but that a majority would show them following about 300 rads.

The key points of the curves in Figure 3-43 are summarized in Table 3-44.

Radiation doses to different parts of the bone marrow undoubtedly will vary widely under space flight conditions owing to variations in shielding

of the spacecraft, in energy and penetrability of the space radiations, and in distribution of active bone marrow throughout the body. The principal locations of active bone marrow are the pelvis, spine, ribs, and proximal ends of the bones of the extremities (8). The depth of the bone marrow varies from about 1 to 15 cm. For calculation and assessment of dose to the blood-forming tissues, the average effective depth of the active marrow is given as 5 cm. Although no definitive statements can be made at this time regarding the quantitative influence of nonuniform dose distribution on early hematological response, animal experiments show definitely that uniform whole-body exposure is more effective than nonuniform exposure for the production of hematological effects, even when expressed as integral absorbed dose in kilogram-rads (4 , 113 , 171).

Table 3-44

Estimated High-Intensity Radiation Dose Levels for Production of Hematological Depression*

(After Langham (ed.)-NAS-NRC(145))

	ABSORBED DOSE FOR REDUCTION FROM NORMAL (rads) **		
CIRCULATING ELEMENT	25 PERCENT	50 PERCENT	75 PERCENT
Platelets ***	50	120	250
Lymphocytes ***	60	150	300
Neutrophils ***	80	190	390

*Symptoms appear within 1 to 10 days after bone marrow exposure.
**Point of interest for dose estimation average depth of 5cm: QF assumed to be unity.
*** ~3,25, and 30 days, respectively, for lymphocytes, neutrophils, and platelets.

Quantitative approaches to the evaluation of effects and recovery from nonuniform exposure have been proposed (38, 202). Under nonuniform irradiation the unequal distribution of dose to the bone marrow should permit a higher rate of survival than if the same average dose were distributed uniformly. This is due to the exponential nature of dose effect on survival of bone-marrow stem cells. Furthermore, it is considered possible that stem cells from unirradiated marrow migrate to irradiated areas and hasten regeneration. However, migration of stem cells may not be as significant in humans as in small rodents. The strong dependence of early hematological effects on the region of the body irradiated suggests that emergency partial shielding of the bone marrow be considered. Local shielding of the pelvic region would probably be the most beneficial since about 40 percent of the bone marrow is located there. Such a procedure would markedly increase the chance of survival in cases of high dose exposure. The volume factor (f_V) for irradiation of extremities should be about 1/5 that for whole-body irradiation (145). (See Distribution, page 3-7.)

From several studies it is apparent that marrow exposures will be of varying fractions of weakly-penetrating space radiation at high LET and highly-penetrating radiation at low LET. (See Figures 3-25, 3-32, 3-34, and 3-35). For rough estimation, work with primates suggests that the overall LET is ≤ 3.5 keV/μ and that a QF of 1 should be used for the entire flux as seen in Table 3-3 (68 , 69 , 70 , 71 , 135 , 145 , 153 , 154).

Suggestions for dose protraction factors are given in Table 3-5.

Skin Reactions

Early responses of the skin to radiation exposure may be particularly important to manned space-flight operations, especially those involving extravehicular activity and light to moderate shielding. A few systematic clinical investigations have been relied upon heavily in deriving the limited dose-response relationships for human skin that are established at this time (7 , 21 , 78 , 81 , 131 , 161 , 206 , 213 , 214).

The levels of early skin response in order of increasing severity are usually designated as (1) erythema, (2) dry desquamation, (3) moist desquamation (vesiculation), (4) slough of skin layers, and (5) chronic ulceration. The first four levels of reaction are often followed by restoration of the irradiated skin to near-normal or pre-exposure conditions; however, clinically evident permanent changes occur regularly after doses that yield at least a dry desquamation response.

Table 3-45 represents the air dose-response of the skin for early symptoms following acute x-radiation.

Table 3-45

Radiation Damage to the Skin*

(After Grahn[103] Adapted from Saenger[232] and Cronkite et al[183])

Epilation -loss of hair	*Erythema (first degree burns)*	*Moist desquamation and blistering (second degree burns)*	*Ulceration (third degree burns)*
Rare at less than 200 r			
Partial epilation at 350-450 r	Response is dependent on energy, dose rate, area exposed, & complexion of the individual. Full effect in 1 to 3 weeks after:		
Complete epilation in 16-18 days at > 450 r	200-400 r (<150 kev)		
	500-600 r (200-400 kev)		
Permanent epilation at > 700 r	800-1000 r (>400 kev)		
	Response in first hours at 1000 r	Effect in 1-2 weeks at >1000 r	
			Rapidly progressive effect at > 2000 r

*These statements are based on air doses. Dose estimates are at 0.1 mm depth where 1 r $\cong$ 1 rad.

The ED's for skin responses have been evaluated in the light of uncertainties regarding effects of quality of radiation, local skin areas, endpoints, etc. The only reactions with adequate statistical data are erythema and moist desquamation for fields of less than 100 cm^2 (145). The estimated responses are seen in Figure 3-46. It is probable that the dose response curves for more severe erythema and dry desquamation would lie between these curves. The statistical projections are summarized in Table 3-47. The factors modifying radiation effects on the skin are numerous and more closely studied than many of the other responses. They include (1) quality (LET) of the radiation, (2) dose fractionation and total time over which irradiation occurs, (3) dose rate, (4) depth-dose distribution, (5) area of skin irradiated, (6) anatomical region exposed, and (7) presence of other irritants or trauma.

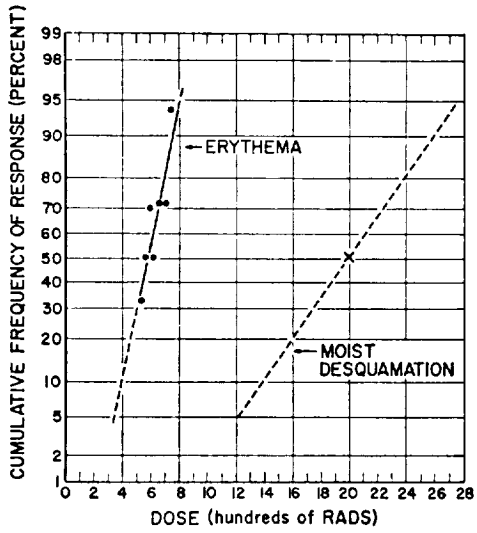


Figure 3-46

Dose-Frequency Relationship of Minimal Erythema[177] and Moist Desquamation[7,81,131,206] (Clinical Tolerance Response) for Acute Exposure 200kVp X-rays for Areas of <100 cm^2 and Dose Rates ≥ 30 rads/min

(After Langham (ed.)-NAS-NRC[145])

Table 3-47

Estimated Doses of High-Intensity Radiation (QF=1) at 0.1 mm Depth for Production of Erythema and Desquamation of the Skin

(After Langham (ed.)-NAS-NRC[145])

	ABSORBED DOSE FOR PROBABILITY OF RESPONSE (rads)		
CLINICAL SIGN	10 PERCENT	50 PERCENT	90 PERCENT
Erythema	400	575	750
Desquamation	1,400	2,000	2,600

An overall modifying factor (QF_E) weighted for LET should be applied to the dose values given here (See Table 3-3). An area effectiveness factor of 1.25 is suggested to reduce the dose values given here when exposure involves skin areas up to or greater than 150 cm^2.

Corneal lesions can occur after 2500 r of 100 kVp x-rays and lower energy cathode radiation (220).

Quality

The sensitivity of skin to low energy protons and alpha of high LET has already been covered. Primate studies with 1440 rads and 5200 rads of 32 MeV protons lacking sufficient range to penetrate to the marrow and gastrointestinal tract, show that the skin and subcutaneous tissues can bear the weight of the injury (67 , 154). During an initial quiescent period of 2 weeks, only epilation and erythema were noted on the skin, accompanied by anorexia and dehydration. In the next two weeks the erythema turned to ulceration of the skin and mucous membranes of the mouth with massive edema of the soft tissue. The lesions began to heal poorly at 5 weeks. Capillary and lymphatic damage appeared to be a prime factor. These signs progress in primates to severe fibrous contracture of the skin which leads to immobilization and often death from starvation (178, 195).

The threshold dose for the late alpha and proton effect in humans is not yet known. Animal studies with electrons of varying penetration suggest that the threshold dose at 0. 1 mm for death of the critical basal layers of the epidermis is about 1200 to 1700 rads for transdermal injury; 1800 to 2500 rads for atrophy and chronic inflammation. Comparative data for pertinent protons and alpha may be extrapolated from the RBE or QF - LET relationship of Table 3-3. Some reservations should be held because of the uncertainty regarding dose-rate-dependent quality factors in skin radiation (131, 145).

For high-intensity single exposure, observed RBE values for radiations of LET $\geq$3.5 keV/μ (largely soft x-rays and fast neutrons) for production of early skin reactions have been in the range of 2 to 3. In converting absorbed dose of space radiations to local dose equivalents for production of early skin responses, it should be kept in mind that for high-LET radiations, QF appears to increase more slowly with increasing dose fractionation than for conventional x-ray exposure (145) (see Figure 3-49). Some compromise between the recommendations of Tables 3-2 and 3-3 will have to be arrived at for fractionated dosage patterns leading to subacute injury (see below).

Dose Fractionation

The repair of sublethal injury in the surviving cells and the proliferation of the surviving cells between fractions determines the dependence of ED_{50} and TD_{50} (doses required to produce 50 percent erythema and moist desquamation response, respectively) on fractionation number and total time required for dose administration (89). The repair of sublethal cellular injury after moderate doses is complete or very nearly so in less than 24 hr. Cellular repopulation of irradiated tissues is a much slower process. Accordingly, the ED_{50} and TD_{50} are sensitive to the number of equal fractions into which the total dose is divided (administered at $\geq$ 24 hr intervals) and are less sensitive to the total time of its administration (7, 53, 54, 82, 161, 206, 213, 214, 264).

Figure 3-48 shows the effect of daily fractionation of dose on the ED_{50} and TD_{50} of 200- to 250 kVp x-rays (30 to 60 rads/min, 35 to a 100 cm^2 area). In none of the clinical studies are there applicable data for more than about 40 fractions. If the expressions shown in Figure 3-48 are to be extrapolated to long times or to large fraction numbers, a slope constant of perhaps 0.25 may be more appropriate (145).

Figure 3-49 suggests that radiations of low LET show a greater RBE sensitivity to dose fractionation than do radiations of higher LET.

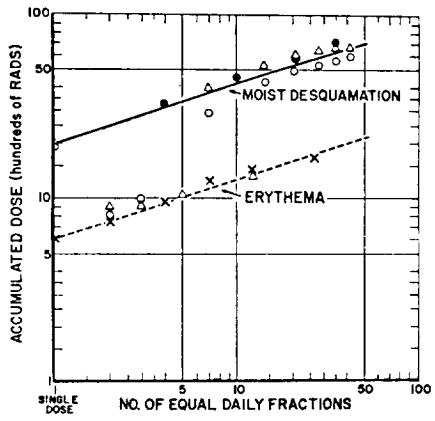


Figure 3-48

Effect of Dose Fractionation or Overall Exposure Time on Dose Producing 50 Percent Probability of Erythema (ED_{50})[161,213,214] and Moist Desquamation (TD_{50})[81,131,206] of Human Skin (200-250 kVp x-rays).

(After Langham (ed.)-NAS-NRC[145])

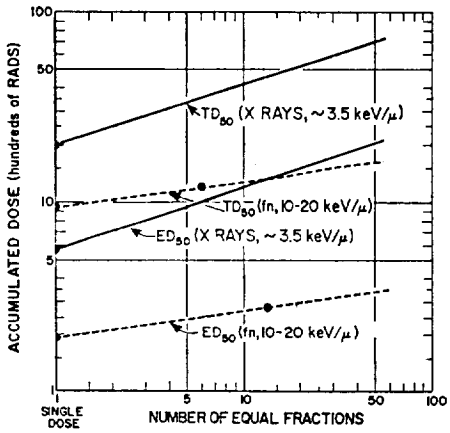


Figure 3-49

Dependence of Erythema (ED_{50}) and Moist Desquamation (TD_{50}) of Human Skin on Radiation Quality (LET). (fn = fast neutrons)

(After Langham (ed.)-NAS-NRC[145])

Dose Rate

A given dose of radiation administered continuously at relatively low dose rates is known to be less effective in producing early skin reactions than is the same dose given at a rate of $\geq$30 rads/min. Clinical estimates of the dependence of skin reaction on dose rate (for a single exposure only) are shown in Figure 3-50. The only empirical data are the symbols and solid lines (206). All other points are extrapolations (145).

Depth Distribution and Area Factors

It has been determined from study of effective depth-dose patterns with different isotopes that any dose to a depth of $\geq$0.9 mm should be considered as capable of producing full skin reactions (177).

The dose required to produce a given response is larger for an area of a few cm^2 than for one of a few hundred cm^2 with both x-rays (81, 131,

53 , 206) and electrons (256). Above ~ 400 cm^2, the size of the area irradiated has little effect on the dose required to produce a given level of response. Figure 3-51 illustrates the effect of area irradiated on skin-tolerance dose (TD^{50}) over the range of 30 to 600 cm^2 for 25 treatments given over a period

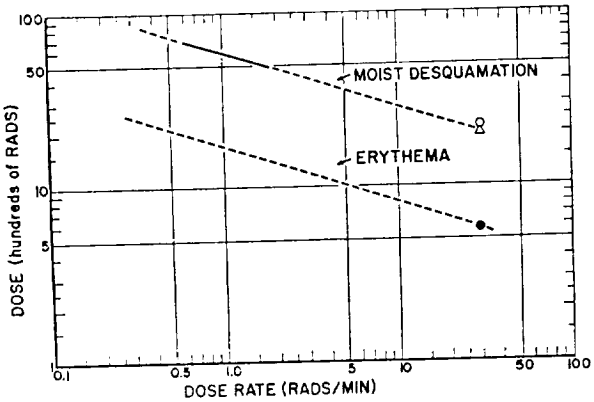


Figure 3-50

Dose-Rate Dependence (Single Exposure Only) of Erythema (ED_{50}) and Moist Desquamation (TD_{50}) of Human Skin (200-250 kVp x-rays).

(After Langham (ed.)-NAS-NRC[145] Adapted from Paterson[206]).

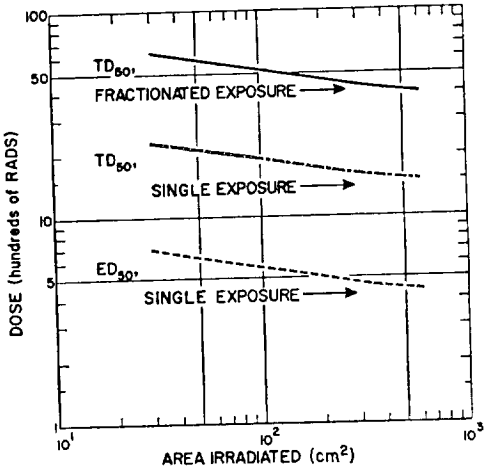


Figure 3-51

Effect of Area on Response of Human Skin to Radiation Exposure (Upper Curve, Skin Tolerance Dose, TD_{50}, for 25 Treatments Given Over 5 Weeks; Lower Curves, TD_{50} and ED_{50} for Single Exposure).

(After Langham (ed.)-NAS-NRC[145] from data of Paterson[206]).

of 5 weeks (upper curve). The lower curves demonstrate the possible effect of area irradiated on the single high- intensity exposure TD_{50} and ED_{50}. In this case, the TD_{50} and ED_{50} curves are drawn parallel to the upper curve through the established points of 2,000 and 575 rads for the high-intensity tolerance and erythema doses, respectively, for an exposed area of 100 cm^2. In view of the limited data, it has been suggested that the erythema and tolerance-dose values perhaps should be lowered by about 20 to 25 percent if applied to situations in which the potentially exposed skin area may exceed 100 to 200 cm^2 (145). These considerations are related only to effect of area exposed on the ED_{50} and TD_{50} and do not apply to effect of area of skin involved on the degree of discomfort and the probability of producing decrement in performance. It is suggested, therefore, that in calculating RES, the space-radiation dose (D) be multiplied by an area-effectiveness factor (f_a) of 1.25 to evaluate the reference equivalent space exposure (RES) when exposure involves skin areas greater than ~150 cm^2 (see Distribution, page 3-7).

The less sensitive regions of the body are the face, trunk, arms, and legs; more sensitive regions are the dorsa of the hands and feet, the scalp, the eyelids, and the perineum. Data are not available from which an estimate of the ED_{50} or TD_{50} for hands and feet can be made. The response threshold for skin of these regions appears to be about 15 to 25 percent less than that for skin on the trunk (145).

Irradiated skin exposed to chronic irritation or trauma, such as from tightly fitting clothing, will develop a more severe reaction than would non-traumatized skin. The increase in severity would be a function of the type of trauma and whether the area was being continually subjected to trauma during the course of the development and healing of the reaction. Exposure to elevated local temperature would also act to augment the reaction. It has been suggested that exposure of irradiated skin to trauma and irritants could effect a reduction in ED_{50} or TD_{50} by a factor of ~ 0.25 (145).

Germinal Epithelium

The sterilizing effect of radiation will not affect the success of a specific mission, but may have a second-order psychological bearing on crew effectiveness during the whole program. Due to the high radiosensitivity of the gametogenic epithelium, the gonads are probably the most sensitive organs of the human body. Table 3-52 represents the expected dose-response curve for male sterility obtained from experience with electromagnetic radiations.

Table 3-52

Radiation Damage to Gonads

(Adapted from Brown[42], Oakes and Lushbaugh[200] and Heller[116])

Dose	Response
15-100 rad	Progressive reduction in fertility with dose reduced sperm count (oligospermia) and increased frequency of abnormal sperm. Above 100 rads, azoospermia is usually evident at 10 weeks.
200-300 rad	Temporary, absolute sterility (azoospermia) for approximately 12-15 months after 10 weeks.
400-500 rad	Temporary sterility for 18-24 months.
500-600 rad	Probably permanent sterility, if individual survives.

The time course of decreased sperm count is related to the fact that post-spermatogonial cells are quite resistant to radiation. Cells beyond the second and third phases of spermatogenesis continue to develop. Below 300 rads, there is a normal sperm count for about 46 days with a sudden drop over the next 10 or 12 days to a nadir at about 80-120 days depending on dose (146). Above 300 rads, the sperm count falls after several weeks. Libido and potency are unaffected up to about 600 rads. The lower the dose, the more rapid the recovery. Beyond 500-600 rads, little or no recovery is seen (182). A review of gonadal response after clinical x-ray exposure is available (118).

The principal effect of radiation on the germinal epithelium is a direct one (95). Therefore, nonuniformity of dose distribution, both topically and in depth, would influence response of the germinal epithelium to space-radiation exposure. For purposes of effective dose calculation and measurement, the average depth of the testes is assumed to be 2.5 cm (145). It should be remembered that the testes are shielded in operational conditions by at least 3π steradians of body shielding. The fact that radiation effect on the

germinal epithelium is predominantly a direct one suggests the feasibility of local shielding, if necessary, to lessen the probability of response.

The QF-LET factors suggested in Table 3-3 appear appropriate for acute exposure of the germinal epithelium (20 , 145 , 199). It has been pointed out, however, that in small animals some testicular cells are very sensitive to high LET radiation; 6 rads of alpha particles causing a significant effect (270). Under conditions of fractionated or protracted exposure the QF-LET factors of Table 3-5 appear reasonable (260).

PROGRESSIVE PERFORMANCE DECREMENT

Where exposures are at low levels and where no early manifestations occur, continued or periodic exposures can lead to a progressive decay in health and in performance necessary to maintain flight operations. Quantification of this problem encompasses one of the most difficult areas for the prediction of biological effects.

Radiation injury has a comparatively slow time-course of expression and its manifestations will progressively emerge, then subside. Expression and recovery are concurrent. When the exposure is essentially continuous but at a low daily rate, injury and recovery will probably equilibrate and a steady state will be maintained for long periods. Such observations have been made in experimental animal populations and certainly would occur in man, but there are not yet sufficient data available to establish the kinetics of injury and recovery with any degree of confidence (228).

One theoretical approach to this problem has lead to the evolution of the "equivalent residual dose" (ERD) concept (32 , 73 , 148 , 172, 191, 231). This assumes a simple linear additive model for injury accumulation and concurrent recovery. The assumptions and constants employed in the ERD calculation have never been validated in man and are often, for specific biological end points, in conflict with much present day radiobiological data (201). The ERD concept is not based upon a correlation of physiological or cellular injury with lethality, and therefore it cannot determine in any realistic way a dose accumulation that can be related to an acute end point. Dose protraction studies in larger animals may shed more light on the problem (2 , 205 , 297).

The following discussion is taken directly from the recommendations of the Space Radiation Study Panel of the Life Sciences Committee, Space Science Board, NAS-NRC (145).

The possibility of radiation-induced progressive performance decrement will increase with increasing mission duration. The highest-intensity exposures will occur very infrequently and then only over a period of a few days, such as during solar flares. Most exposures to radiation in space flights are, therefore, expected to be at low dose rates, Since radiation effects decrease roughly proportionately with decreasing dose rate, the early effects described above for high-intensity exposures will become less marked

or even absent under sufficiently protracted exposure, even though the dose rate during an occasional episode may be relatively high. Under these conditions, as a result of a gradually accumulating injury to the blood-forming tissues, more subtle effects may occur that may be accompanied by a reduction of the spacecrew's ability to maintain normal flight operations. This injury may be characterized by vague symptoms of fatigability, headache, dyspnea, reduced resistance to general stress, increased incidence of low-grade infection, and decreased blood-oxygen transport.

In space radiation exposure, the expressions of injury and recovery are concurrent. When the exposure to reference-quality radiation is essentially continuous but at a low daily rate (perhaps 1 rad/day or less for man), the rate of injury and recovery may approach equilibrium, and a steady state may be maintained for long periods. Although the phenomenon of equilibrated injury and recovery has been quantitatively defined in experimental animal populations under specific conditions of exposure, the kinetics of injury and recovery for man cannot yet be given with any degree of confidence.

Prediction of man's response is difficult even when a regular pattern of protracted or fractionated exposure is involved. The erratic pattern of exposure that may occur in most projected space flights, and the accompanying moderate to serious depth-dose inhomogeneity, make extrapolation virtually impossible at present. Sufficient dose protraction (whether by low dose rate or by fractionation) will lessen or even preclude the occurrence of prodromal symptoms and early skin responses. Restricting one's self to injury to the blood-forming tissues, the important questions are to what extent damage to the bone marrow will be lessened and what time factors are involved.

In the absence of any well-substantiated method of estimating an individual's residual radiation damage from intermittent exposure and his capacity to tolerate additional doses, the Panel suggests that a dose-accumulation procedure as outlined below be utilized. The procedure allows for any changing effectiveness of accumulating dosage by taking dose rate into account. Although there are insufficient data to permit a high degree of precision, the Panel feels that some evaluation of dose-rate effects under the anticipated conditions of exposure is important in determining the radiation status of a spacecrew. A suggested approach is outlined below.

1. Radiation absorbed by a crew on a deep-space mission will typically result from occasional limited periods of exposure at elevated dose rates (which will vary from period to period and with time during each period) superimposed on a continuous low-dose-rate ambient cosmic-radiation background. The irregularities in dose rate can be smoothed for calculation and accumulated on a mean-daily-dose basis.

2. It is assumed that the effect per unit dose will decrease linearly with decreasing dose rate.

3. For bone-marrow responses, doses delivered at dose rates of 50 rads/day and above are assumed to produce maximum injury per rad, while exposures at rates of 1 rad/day and below are assumed to produce minimum injury per rad accumulated.

4. A dose-rate accumulation effectiveness ratio of 3 will be assumed between these limiting dose rates, and linear interpolations of the accumulation-rate factor (RF_A) may be made for all intermediate dose rates (Figure 3-53). RF_A should not be confused with f_r (Table 3-5), which applies only to early responses within ~30 days after high-intensity exposure.

5. The RF_A values taken from Figure 3-53 may be applied to the space-radiation dose (D) in the general equation (3) to derive a value for RES that allows for differences in progressive bone-marrow injury as a result of differences in dose-accumulation rate. It has been suggested that, for extended missions, the acceptable mission-accumulated exposures be set on the basis of the lowest dose rate (1 rad/day or less) (see Table 3-66). This recommendation is consistent with standard practice in occupational radiation protection. In contrast to early responses where the standard exposure situation has always been the high dose rate producing maximum effect, the standard for protracted low dose-rate exposures has always been that associated with minimum effectiveness. Therefore, RES for progressive bone-marrow injury will only be subject to upward adjustments by multiplying the space dose (D) by RF_A (which is always ≥ 1) to allow for increasing effect as dose rate increases above 1 rad/day. As dose rate and dose accumulation are protracted, expression of bone-marrow injury approaches that of late or delayed responses. It seems, therefore, that quality factors for late responses (QF_L) should be used for evaluating progressive bone-marrow injury. Quality factors for late responses are given in Table 3-2 and equation (1).

It was also recommended that the contribution of daily dose increments (assuming exposure of a major portion of the bone-marrow) to the accumulated RES for progressive bone-marrow injury be evaluated as

$$\text{RES (reu)} = \text{D (rads)} \times QF_L \times RF_A \qquad (10)$$

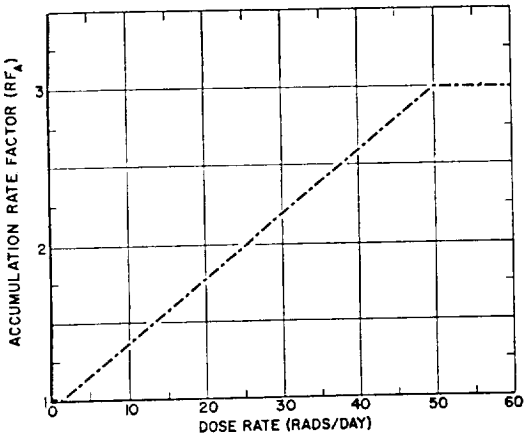


Figure 3-53

Accumulation Rate Factor (RF_A) for Progressive Bone-Marrow Injury as a Function of Mean Daily Dose Rate.

(After Langham (ed.) -NAS-NRC[145])

and subtracted from a pre-established acceptable mission reference-equivalent space exposure (RES_m) expressed in reu to give a chronological record for the remaining allowable mission exposure.

An example of the manner in which a record may be kept of the accumulated marrow exposure received by a flight crew during a hypothetical 1-year mission is shown in Table 3-54. In this example, it is assumed that the

Table 3-54

Example of a Dose Accumulation Record for a Hypothetical 1-Year Mission

(After Langham (ed.)-NAS-NRC[145])

ELAPSED TIME PERIOD (days)	MEAN DOSE RATE (rads/day)	MEASURED DOSE (D) RECEIVED (rads)[a]	RF_A	ESTIMATED RES RECEIVED[b] (reu)	ALLOWABLE RES_m REMAINING (reu)
0	—	—	—	—	250
0–1	10	10	1.4	14	236
2–150	< 1	15	1	15	221
151–152	20–30	45	2	90	131
153–364	< 1	20	1	20	111
365	15	15	1.5	23	89
TOTAL		105		162	

[a]At 5-cm depth, tissue-equivalent; total-body exposure.
[b]Q_{RF_L} assumed to be unity.

acceptable RES_m, established on the basis of a risk-versus-gain philosophy, was set at 250 reu. It is assumed also that exposure involved two traversals of the geomagnetically trapped radiation fields, continuous low-level background radiation (~ 0.1 rad/day), and interception of one major solar-flare even on the 151st day. The example illustrates how such a chronological record may give some feeling as to the progressive bone-marrow exposure status of the crew during an extended mission.

The uncertainties in evaluating the risks from space-radiation exposure increase disproportionately with increasing mission duration. For missions up to 30 to 60 days, risk evaluation is based on a reasonable amount of factual information. For missions beyond 1 year, evaluation becomes more and more a matter of judgment. In an effort to provide some guidance for long-duration missions, suggestions of annual exposure-accumulation factors are given in Table 3-55. The factors are given as a set of multiples of a 1-year exposure on the assumption that the 1-year exposure is in the range of 200 to 300 reference equivalent units (reu). The factors are selected on the judgment that any derived RES values will produce no clinical signs or symptoms of hematological injury (such as infection, hemorrhage, fatigue, or fever) if exposure is generally distributed or fractionated over the indicated time periods. The factors do not increase in direct proportion with time; they drop away from simple proportionality to allow for uncertainties of damage to recovery mechanisms and for possible cumulative effects of other stresses associated with space flight.

Table 3-55

Examples of Exposure-Accumulation Factors or Multiples of the 1-Year RES[a] for Missions of Specified Duration

(After Langham (ed.)-NAS-NRC[145])

MISSION DURATION (years)	MULTIPLE
1	1.0
2	1.8
3	2.4
4	2.8
5	3.0

[a]At 5-cm depth, tissue-equivalent; total-body exposure.

The NAS-NRC Panel believes that present knowledge permits a limited prediction regarding tolerance to progressive bone-marrow injury. Although it may be possible to judge from existing experience when a population is approaching the limits of its tolerance (the point where overt signs and symptoms may appear), there is no way at present to predict individual variations in sensitivity or distribution of sensitivities in the population. The variance of the population is an extremely important parameter of any quantitative prediction, and present knowledge is far from adequate. In general, the variance may be a function of age, dose rate, total accumulated dose, post-irradiation time period, radiation quality, presence of other physiologic stresses, and particular tissue or system involved. Since most or all of these factors will be variables in space flight, quantitative and accurate predictions will not be possible. With the above considerations in mind, the consensus of the Panel is that a safety factor or uncertainty factor (as the case may be) of 2 may be inherent in the exposure-accumulation multiples given in Table 3-55. As exposure and time accumulate, the safety factor becomes more of an uncertainty factor because of the gradual shift of the response pattern into the late-injury mode. The limiting consideration, therefore, is ultimately the extent to which long-term risks are acceptable.

Although it is not possible to avoid all risk of radiation injury, signs and symptoms of early and intermediate injury to the blood-forming system can be selectively avoided by controlling the exposure-accumulation rate. However, the probability of manifestation of late radiation injury and general life-shortening will increase in proportion to the total accumulated exposure.

LATE OR DELAYED EFFECTS OF RADIATION

Late or delayed manifestations of radiation damage are those that do not appear until after a latent period of months, years, or the remaining life span of the individual. These effects are nonspecific in that they cannot be correlated to any particular radiation exposure. Lack of correlation between a particular dose and ultimate manifestations of effect arises partly as a result of the relatively long latent period before appearance of injury and

partly because the effects from continuous exposures, multiple exposures, or both are additive, but not necessarily in a 1:1 ratio in their final expression.

Late responses in what would appear to be a reasonable order of relative importance to manned space flight are changes in the ocular lens, permanent impairment of skin, general life-shortening, increased incidence of leukemia and other neoplastic disease, and genetic manifestations. The genetic manifestations differ from the others in that they affect the progeny of the irradiated individual rather than his health or faculties. The others are manifestations of cumulative somatic injury.

In general, late (or delayed) effects are qualitatively the same, regardless of the nature of the radiation and whether exposure is continuous, intermittent, or from a single high-intensity dose. Although modified quantitatively by a variety of factors (including dose rate or protraction, depth-dose distribution, portion and region of the body irradiated, and nature and quality of the radiation), some delayed effects (life-shortening, increase of incidence of malignancy, and genetic manifestations) are considered nonthreshold phenomena and impose on an exposed individual a probability of response in proportion to the total accumulated dose. In this case, the associated actuarial risks provide both the necessity and the basis for limits to radiation exposure in long-duration missions and space-flight careers.

Ocular Lens

Either acute or chronic exposure of the ocular lens will result in opacities which may go on to true cataracts depending on dose (51, 52, 145, 168). The time of appearance is highly variable and may range from as early as 6 months to many years after exposure. The higher the dose, the earlier the appearance and the greater the degree of impairment. Because of the variability of clinical and accidental exposures, no definite response-probability values for production of lens opacities can be assigned to specific radiation doses. About 150 to 200 rads of reference-quality radiation is the minimum cataractogenic single-exposure dose, and some lessening of effect appears to result from dose protraction over a period of at least 2 to 12 weeks. This is summarized in Figure 3-56.

It appears also that a single dose of about 650 to 750 rads of reference radiation may have a cataractogenic probability approaching unity, 50 percent of which may be progressive, resulting ultimately in impaired vision. The slope of the time-dose response curve between single (1-day) exposure and exposure protracted over an average time of ~7 weeks suggests a ratio of protracted dose to single dose of ~2 for the doses required to produce the same level of response. The dose protraction factor for lens opacities is, thus, much less than for other tissues (79, 145, 161).

The incidence of cataract after a given dose level fractionated over 2 to 12 weeks is shown in the histogram of Figure 3-57. The available data suggest a log-normal dose-response distribution between the probability limits and predict that doses between 550 and 950 rads (average 800 rads) delivered over periods from 2 weeks to 3 months may produce an opacity in

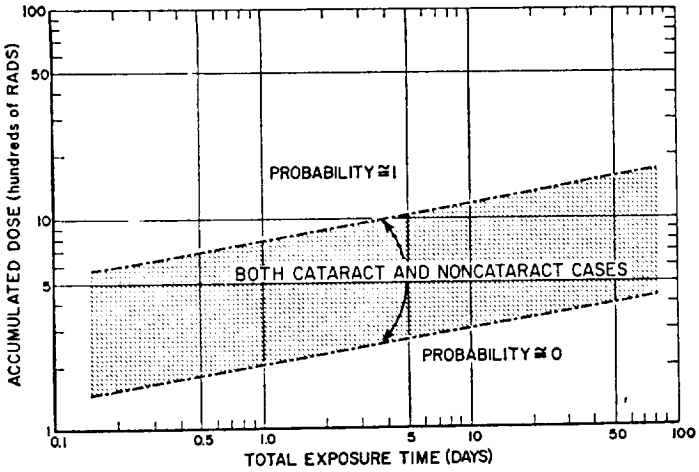


Figure 3-56

Time-Dose Relationship for Production of Late Radiation Changes in the Ocular Lens, Suggesting Probability Limits of $0 \leqq p \leqq 1$.

(After Langham (ed.)-NAS-NRC[145])

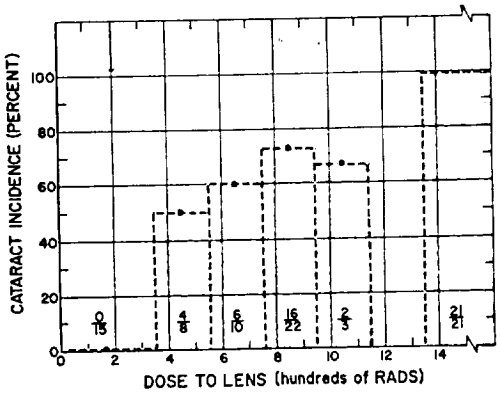


Figure 3-57

Incidence of Late Lenticular Changes in Patients Who Received Photon Exposures Fractionated over 2 to 12 Weeks.

(After Langham (ed.)-NAS-NRC[145] from data of Merriam and Focht[167])

about 70 percent of those exposed. Of these, about 30 percent may be progressive and eventually result in impaired vision. On this basis, one might estimate that about 20 percent of flight crews who receive lens exposures of ~800 rads of ionizing radiation (equivalent in effectiveness to 200-kVp x-rays) in an interval from a few weeks to about 3 months may develop clinically significant cataracts at some time during their lives. Table 3-58 represents a probabilistic summary of the dose response relationship after acute and protracted radiation.

Review of animal and human data suggests an RBE of at least 10 for late lenticular effects from recoil protons of neutrons under conditions of protracted

Table 3-58

Suggested Absorbed Doses[a] of Reference Radiation for Production of Late Changes in the Ocular Lens

(After Langham (ed.)-NAS-NRC[145])

PROBABILITY OF RESPONSE	HIGH-INTENSITY SINGLE (1-DAY) DOSE (rads)	PROTRACTED DOSE, C (rads)
Minimal ($p \geqq 0$)	150	300
Median ($p \cong 0.5$) d	300	600
Maximal ($p \leqq 1$)	650	1,300

a. Point of interest for dose estimation, 3-mm depth.
c. Dose protracted over 7 weeks or longer.
d. Assuming log-normal distribution of response.

exposure (1, 166). Demonstrable observations of an increase in the RBE of high-LET radiations with increasing dose protraction indicate that the slope of the time-dose curve approaches zero with increasing LET. It is advisable, therefore, to use the QF-LET relationship given in Table 3-2 and the local LET spectrum at the depth of the lens when considering delayed cataractogenic effects of space-radiation exposure. Curves have been recently published on specific QF values for different proton energies in lenticular radiation (75). It has been noted that protons and neutrons may tend to produce more vacuolar degeneration of the lens in animals than do other forms of radiation (211).

Topical nonuniformity of dose distribution is highly important and suggests the feasibility of locally shielding the eyes to lessen the probability of late lenticular changes from space radiations. Because of the rapid drop-off in dose and local LET of the principal sources of space-radiation exposure, nonuniform depth-dose distribution may also be significant. The average depth to the surface of the lens is estimated as ~3mm. Dose evaluation for late cataractogenic-effects, therefore, should take into consideration dose and local QF at a depth of 3mm (145).

The RES for late changes in the ocular lens may be evaluated by the expression

$$\text{RES (reu)} = \text{D (rads)} \times QF_L \times F_{pr} \qquad (11)$$

where D is the space-radiation dose and F_{pr} is the protraction factor equal to unity for protraction times of 7 weeks and greater and linearly increasing to 2 with decreasing time to 1 day. As an example, if the pre-established acceptable exposure risk for a mission of 7 weeks or longer ($F_{pr} = 1$) corresponds to minimal probability of lens response (RES = 300 reu), and that exposure is anticipated to a space radiation having a predicted mean QF_L of 3, the allowable space-radiation dose (D) would be 100 rads. If delivered in a 1-day exposure, however, the allowable dose would be only 50 rads.

It should be kept in mind that the greater the level of exposure, the greater will be the probability that any cataracts produced will be progressive and the shorter will be the latent period before development.

Chronic ulcerative lesions and opacification of the cornea can result from 100 kVp x-rays in the 2000 - 3000r range, from lower doses of soft x-rays (<75 kVp), and from β radiation of $< 1\text{-}2 \times 10^6$ eV. (50, 220). Retinal vascular occlusions have also been reported following x-radiation in excess of that necessary to cause lenticular changes (50).

Permanent or Late Skin Effects

In the review of early skin effects (see Table 3-45 and discussion of Figure 3-46), it was mentioned that clinically evident permanent changes in the skin occur regularly after radiation doses that yield at least a dry desquamation response. Furthermore, induction of skin malignancy is known

to be one of the late or delayed oncogenic effects of single high-intensity and protracted radiation exposure. The characteristic late radiation changes in the skin are referred to collectively as "chronic radiodermatitis" and consist of telangiectasia, loss of hair, pigmentation, parchment-like appearance with atrophy, keratosis, malignancy, and sensitivity to mild trauma. The latter, combined with decreased capacity for healing, predisposes to chronic ulceration. As with other late effects, manifestations of chronic radiodermatitis do not occur until many months or many years after exposure, and their frequency and severity are proportional to the accumulated dose to the exposed area.

Although minimal delayed radiation changes in the skin are not serious per se, there is a general feeling by some that severe or symptomatic radiodermatitis is a dangerous lesion because it is progressive and may eventuate in carcinoma of the skin if the affected person lives long enough (43, 204). The most common type of skin tumor resulting from radiation exposure is squamous-cell carcinoma, although basal-cell carcinomas occur frequently and appear to occur more frequently in the less severe and more superficial type of radiodermatitis. The time between the first evidence of skin changes and appearance of the cancer can average 7 years, with a variance of 1 to 25 years. Tumors developing in irradiated skin appear to possess a relatively low degree of malignancy with infrequent metastases. Rates of ultimate mortality reported from radiation cancer of the skin, however, have ranged from 5 to 25 percent (145, 204).

Clinical experience has dealt largely with small exposure areas. No observations are available on delayed consequences of whole-body skin exposure. Data may become available with time as a result of total-body electron exposure in the treatment of extensive skin diseases. Whole-body exposures of primates to >900 rads of 32 mev protons, has lead, as early as 1 year, to severe fibrosis and contracture of the skin with immobilization and starvation as the cause of death (178, 195). In these primates, no tumors have been seen after 2 1/2 years.

Depending on total dose and dose protraction, late radiation changes in skin vary from minor telangiectasia of cosmetic interest only to development of the most serious sequela, metastatic carcinoma. However, quantitative dose-response relationships for the various manifestations of chronic radiodermatitis do not exist. There seems to be a correlation, however, between production of an early desquamation reaction and manifestations of minimal late effects (telangiectasias, mottled pigmentation) in that clinically-evident permanent changes are observed regularly after early desquamation. Based on this premise, the single-exposure dose-response relationship for production of minimal late changes in the skin would be parallel to and approximately the same as that for early moist desquamation shown in Figure 3-46. Indirectly at least, there are observations that support the above contention (145, 266). Back extrapolation from protracted dose schedules suggest a single-dose equivalent of ~2,800 rads (small exposure fields, ~10 cm^2) for production of 50 percent probability of late necrosis (82, 145, 273).

As with early desquamation, the total dose required to produce chronic radiodermatitis should show parallel dependence on dose protraction and fractionation resembling that for early responses. This is seen in the extrapolations from tenuous observations in Figure 3-59 (82, 145, 266, 273). The late effects appear less sensitive to protraction than early effects. It should be emphasized, however, that, with sufficient protraction, enough dose can accumulate to produce severe radiodermatitis, including cancer, without ever producing any early response.

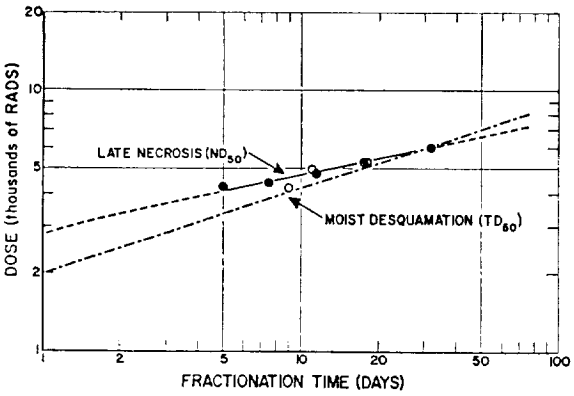


Figure 3-59

Comparison of the Time-Dose Curves for Late Radiation Necrosis and Early Moist Desquamation.

(After Langham (ed.)-NAS-NRC[145] from the data of Sulzberger et al[266], Traenkle and Mulay[273], and von Essen[82])

Table 3-60 probabilistically summarizes the suggested absorbed doses for production of late skin necrosis. As with ocular-lens changes, the time-dose response curve for skin necrosis suggests a decreasing effect with increasing dose protraction, such that the ratio of protracted dose (approximately equally distributed in daily increments over ~7 weeks or longer) to single dose (1 day) required to produce the same probability of response is ~2.3.

Table 3-60

Suggested Absorbed Doses[a] of Reference Radiation for Production of Late Skin Necrosis.

(After Langham (ed.)-NAS-NRC[145])

PROBABILITY OF RESPONSE (percent)	HIGH-INTENSITY SINGLE (1-DAY) DOSE (rads)	FRACTIONATED OR PROTRACTED DOSE[b] (rads)
10	2,000	4,600
50	2,800	6,400
90	3,600	8,200

a. Point of interest for dose estimation, 0.1 mm depth; area exposed <150 cm^2.

b. Dose protracted over 7 weeks or longer.

The late skin effects are very sensitive to the LET factor. Fast neutrons are at least 3 to 5 times as effective as 200-kVp x-rays in producing late radiation sequelae and even more effective in producing late responses than early ones (263). It is suggested that the QF in Table 3-2 be used for late skin effects and that an area effectiveness factor (F_a) of 1.25 for areas >150 cm^2 be used in risk evaluations. The recommended dose protraction

factor F_{pr} is 1 for protraction times of 7 weeks and longer and the F_{pr} linearly increases to 2.3 with decreasing time to 1 day. Equation (11) may be used to calculate the RES for late effects with these QF and F_{pr} factors. As an example, if the space-radiation dose (D) to an area of > 150 cm^2 accumulated (in approximately daily increments) during a mission of 7 weeks or longer (F_{pr} = 1) is 1,000 rads of radiation with an average QF_L of 3, the RES would be 3,750 reu. Comparison of this value with the fractionated dose-response relationship indicated in Table 3-60 (on the basis of 1 reu = 1 rad of reference radiation) suggests about a 3 percent probability of necrosis or chronic radiodermatitis. If fractionated more or less equally over 25 days (F_{pr} = 1.65), the probability of necrosis would be of the order of 50 percent (RES = 6,200 reu). In this case, however, the appearance of early skin responses would definitely have been mission-limiting. In evaluating risk from late skin effects, it should be kept in mind that from 5 to 25 percent of cases of chronic radiodermatitis (in small areas) progress to the malignant stage, and that the area damaged may have a considerable influence on the probability of malignancy since the number of potential cells exposed to malignant conversion is proportional to area. It is not clear if there is a specific body site which shows predilection to chronic necrosis or malignant change (204, 273).

General Life-Shortening and Carcinogenesis

Life-shortening

A convincing body of data indicates that a statistical sample of an animal population exposed to radiation has a shorter median life expectancy than does an unirradiated sample of the same population. The data further show that the degree of life-shortening is a function of accumulated dose. If a group of animals receives a dose of radiation insufficient to cause early lethality, the animals appear to recover completely. Blood counts return to normal, gastrointestinal symptoms disappear, and weight returns practically to normal. Organ function tests, such as liver or kidney, are within normal limits. Nevertheless, on a statistical basis, these animals die sooner than their unirradiated controls (64, 121, 133, 137, 145, 152, 196, 261). Generally, no new or different disease syndromes have been observed as late effects in irradiated animals, and all causes of death so far studied, with only minor exceptions, are accelerated by radiation. Symptoms of delayed radiation effects appear to be so much like those of aging that the syndrome has often been termed "radiation-induced aging," and typical signs of aging confirm this impression.

There is little reason to doubt that radiation exposure would have the same qualitative effect on life expectancy and general well-being of man. With regard to space crews, selection of dose limits for early effects automatically establishes certain probabilities of general life-shortening and other late responses, and forces consideration of placing limits on the associated actuarial risks as they would apply to long-duration missions and space-flight careers. No definitive data exist on the radiation dose-response relationship for general life-shortening in man, but useful data may eventually result from the Atomic Bomb Casualty Commission's studies of the Japanese atomic-bomb survivors. At present, however, it is necessary

to rely almost entirely on animal observations, tenuous extrapolation from experimental animals to man, and limited data on the mortality rates of several age-cohorts of American radiologists.

The degree of life-shortening caused by a single radiation dose delivered to young adult rats or mice is nearly a straight-line function of dose. There is a decrease in mean survival time of about 0.04%/rad with a range of 0.01%/rad to 0.10%/rad. If it is assumed that the percentage of life-span decrement per rad is the same in all species, then a single high-intensity dose of 1 rad of x or gamma radiation would have a statistical life-shortening effect in man of ~10 days (45). Alternatively, if it is assumed that the single-exposure LD_{50} dose for man is 300 rads and that equal fractions of the LD_{50} for the different species produce the same percentage life-span loss, a high-intensity absorbed dose of 1 rad at the midline would have a life-shortening effect in man of ~ 20 days (65). It may be assumed also that flight crews (in the 30- to 40-year age interval) at the time of exposure will have only about one half their life expectancy at risk. On this basis, the life-shortening effect per rad may be modified downward from the above values. A controversy exists over the methods of extrapolation from animals to man and over whether life-shortening from single high-intensity exposure increases linearly or nonlinearly with increasing dose (152, 229). It can probably be assumed arbitrarily for space-flight applications that the actuarial risk from high-intensity exposure to radiations with an LET of 3.5 keV/μ and below is of the order of 10 days/rad (145). About four times more gamma radiation is required at low dose rate than at high dose rate to produce comparable statistical life-shortening in mice. Application of a factor of 4 to the assumed life-shortening value of ~ 10 days/rad for high-intensity exposure in man gives a statistical decrement of ~ 2.5 days/rad for low-dose-rate exposure. This value is in general agreement with other estimates which have been made for man of 1 day/rad for continuous exposure at dose rates not in excess of ~0.5 rad/day (86). If the dose rate is increased above 0.5 to 1 rad/day, its effectiveness on life-shortening may be expected to increase, finally reaching a figure about four time the low-dose-rate value when the dose is received promptly. The dividing line between acute and chronic exposure is not known, but there is almost certainly a gradual transition from one to the other. Present knowledge of rate-dependence of life-shortening effects would hardly seem to justify attempts to refine the quantitative influence of dose protraction and fractionation beyond that given above. Table 3-61 and Figure 3-62 show the suggestion of the NAS-NRC Panel on dose protraction factors in life-shortening (145).

Comparison of fission neutron vs. gamma ray or x-ray effects suggest that the life-shortening effectiveness of low-LET radiation is more highly dose-rate dependent than high-LET radiation (145). The RBE of low-LET neutrons increases from 2 to 10 as protraction is prolonged (145). For the typical space proton dose and dose rate behind nominal shielding at the probable site of the life-shortening effect, the bone marrow, a QF = 1 can probably be assumed (3, 105, 106, 145). Table 3-63 represents a current estimate of quality factor for high-LET radiation of cosmic ray type (243). Applying upper limit estimates to total body dose for man of 40 m rad/day and 120 m rems/day for 2π incidence of the full galactic flux at solar minimum, it can be calculated that the life of a crewman will be shortened by about 1/4 a day for every day he spends in space under these conditions (238, 245).

Table 3-61

Suggested Reference Radiation Dose-Response Relationships for General Life-Shortening and Increased Incidence of Leukemia

(After Langham (ed.)-NAS-NRC[145])

RESPONSE	HIGH-INTENSITY EXPOSURE [a]	LOW-INTENSITY EXPOSURE [b]
Life Shortening [c]	~ 10 days/rad	~3 days/rad
Leukemia [c]	2-4 per 10^6 man-yr/rad	1-2 per 10^6 man-yr/rad

a. Assumed to be 50 rads/day and greater.
b. Assumed to be 1 rad/day and less.
c. Site of interest for dose estimation, 5 cm depth; whole-body exposure.

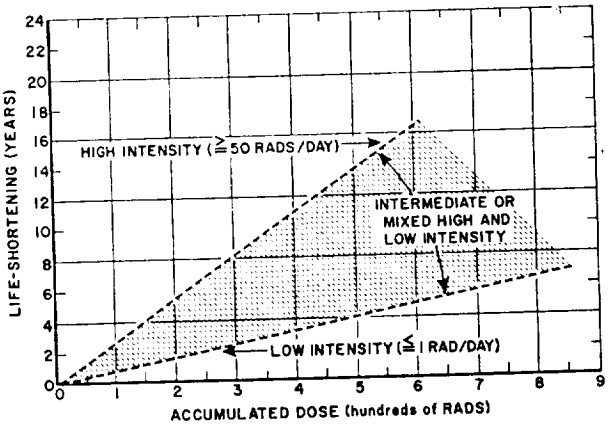


Figure 3-62

Relationship of Accumulated Dose and Intensity of Reference-Quality Whole-Body Radiation to Life-Shortening Probability

(After Langham (ed.)-NAS-NRC[145])

Table 3-63

Estimates for Life-Shortening of Man from Exposure to High-LET Ionizing Radiation

(After Schaefer[243])

Type of Radiation	Life Shortening, days/rad	
	Acute Exposure	Chronic Exposure
Low LET (Electrons, x- or gamma rays)	12	3
High LET (Low-E protons or neutrons, medium and high-E heavy nuclei)	24	24
Extremely high LET ("Microbeams" of heavy nuclei enders)	?	?

The induction of leukemia and other neoplastic diseases contributes specifically to the statistical life-shortening effect of cumulative radiation exposure (121). Although this problem has been under study since the early 1900's there are still very few quantitative dose-response data available for man. This is not due entirely to lack of information, but rather, to the complex statistical and actuarial aspects of the problem. Since no new or unique types of cancer are produced by irradiation, one must seek differences in either the age of appearance, the frequency of occurrence, or both. Since most causes of death are increasing exponentially with age beyond young adulthood, it becomes difficult to be certain without careful statistical analysis when a change in frequency may be attributed to some factor other than sheer chance. In addition, most human data involve fairly small groups-at-risk, and, therefore, the probability of detecting a significant age-specific death rate for a given malignancy is very small. The present Atomic Bomb Casualty Commission (ABCC) studies exemplify many of these statistical difficulties.

Leukemia is one neoplastic disease that characteristically occurs significantly earlier in life in irradiated populations than in nonirradiated populations and, depending upon dose, at a significantly higher percentage (121, 277). Several studies have substantiated the higher-than-average number of deaths from leukemia in irradiated human populations. The most significant data are those obtained from the survivors of the Hiroshima and Nagasaki bombings (12, 40). Exposure in both cities involved prompt weapon-quality gamma radiation with some fission neutrons, the neutron component being higher in Hiroshima. The predicted excess death rate lies between one and two deaths per million exposed per rad per year (1 to 2 per 10^6 man-years/rad). According to present indications, this rate is likely to be maintained for at least 15 to 20 years after exposure.

All other epidemiologic studies of irradiated adult humans also give strong evidence of man's sensitivity to the leukemogenic effect of ionizing radiation (277). These studies include a group of adult British males given therapeutic irradiation of the spine for ankylosing spondylitis (58), the study of professional radiologists, and the evaluation of Danish cancer registry data (84). Virtually all induced leukemias are of either the acute granulocytic, acute lymphocytic, or chronic granulocytic forms. The incidence of chronic lymphocytic leukemia is not detectably increased by radiation. Recent suggestions that the incidence of multiple myeloma, lymphosarcoma, and Hodgkin's disease is higher among the proximally irradiated Japanese (within 1,400 m of the hypocenter) should be noted with caution, since only between one and four such cases have been observed (5). However, a survey of leukemia deaths among U.S. radiologists, does offer evidence in support of a significantly increased mortality ratio from multiple myeloma (151). The latent periods for acute forms of leukemia and for chronic leukemia have been fairly well defined as 1 to 5 years and 1 to 10 years, respectively (85). The duration of an elevated death rate from leukemia is not absolutely defined and may or may not exceed 15 years (31, 58).

There is evidence from several sources that radiation to the thyroid and pituitary gland may increase incidence of thyroid adenomas and carcinoma (7, 8, 9, 155, 212, 257, 272). The dose-response relationship is not clear.

Although risk estimates for thyroid cancer have been made for the case of children exposed to single doses, no valid estimate is available for the irradiated adult. The estimate for children is similar to the leukemia risk (about 1 per 10^6 man-years/rad), but the negative data for adults suggest that the risk may be somewhat lower for that group (145).

The evidence for skin cancer has been covered above. Osteogenic sarcoma also appears to follow radiation. The present consensus is that, if no complicating bone pathology is present, at least 3,000 to 4,000 rads are required to induce bone cancer (33, 132). No risk factor can be derived from the data since they are based on case-history reports and not on epidemiologic surveys. For the same reason, the minimum sarcoma-inducing dose cannot be considered definite. Many other unfrequent tumors have been reported in following radiation of man and animals (145). For example, the ratio of mortality from all cancers among U. S. radiologists is 40 percent higher than among a non-exposed medical-specialty group (252). Unfortunately dose-response relationships are not clear.

In the case of neutron irradiation of high LET, there appears to be much less recovery of chromosome damage and a concomitant higher RBE for tumor formation (64). High LET radiations have not unequivocally demonstrated a higher tumor induction rate than that normally associated with their acute or chronic lethal effectiveness. Soviet studies suggest that protons may have a higher carcinogenic effect in small rodents than equivalent doses of x-ray or gamma radiation (270). Until more specific knowledge is available, it is recommended that the QF-LET relationship proposed by the NCRP (Table 3-2) be used to estimate the risk of malignant disease following exposure to space radiations. The dose rate sensitivity of carcinogenesis appears to be greater than that for life-shortening (40, 121, 252). However, the exact protraction factors are far from clear. Table 3-61 and Figure 3-64 present the proposed relationship for space radiations. Since none of the human data are sufficiently accurate or extensive to favor any one of the dose response models over the other, the simplest linear relationship has been accepted as adequately descriptive of existing data, although it should be appreciated that the present evidence for man and animals does not generally support simple linearity (46, 47, 151). In view of these uncertainties in dose-response curves, any errors in risk estimate will tend to be in the conservative direction (that is, to overestimate response), particularly at doses below 100 rads (145).

The organ specificity, region, area, and volume factors for carcinogenesis are far from clear. Organs in children appear generally more sensitive than those in adults. Astronaut age of predominantly 30 to 40 will certainly reduce the probability of thyroid neoplasia, may reduce the possibility of cancer involving the skeletal, connective, and integumentary tissues, but is not likely to modify the probability of leukemia.

Since the relationships of Table 3-61 and Figures 3-62, 3-63, and 3-64 may be modified by such factors as radiation quality, dose distribution, and dose rate, any attempt to refine risk evaluations by adjusting the space-radiation dose to give reference-equivalent space exposure (RES) is probably an unjustified refinement in view of the large uncertainties in the dose-response

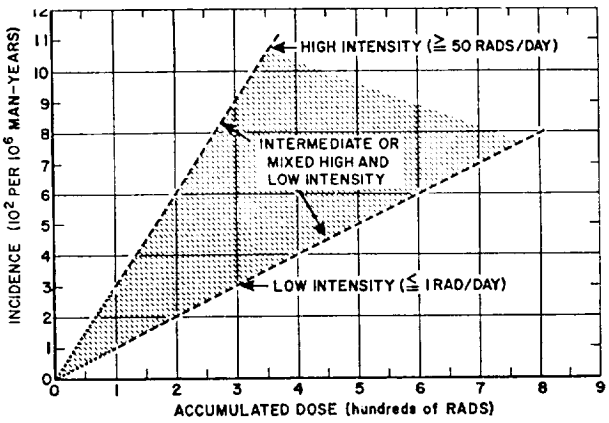


Figure 3-64

Relationship of Accumulated Dose and Intensity of Reference Quality Whole-Body Radiation to Increased Probability of Leukemia.

(After Langham (ed.)-NAS-NRC[145])

relationships. It is possible, however, to make such adjustments of RES and to record chronologically the life-shortening and leukemia-risk status of a flight crew in a manner analogous to that suggested for progressive bone marrow injury, using Equation (10) and Figure 3-53. If it is assumed that the effective tissue depth for generalized late effects is 5 cm, QF_L of the major space radiations (inside current spacecraft shielding) for production of life-shortening and leukemia will be approximately unity. QF_L, however, may be estimated from the calculated local LET using Equation (1). A dose-rate accumulation-effectiveness factor (RF_A) for each daily-dose increment may be taken from Figure 3-53. To evaluate the probabilities of life-shortening and increased leukemia incidence, it is necessary only to multiply RES (reu) for each daily increment by the respective probability values for low-intensity reference-radiation exposure shown in Table 3-61 and sum the incremental probabilities.

Genetic Manifestations

The reports of the National Research Council Committee on Genetic Effects of Atomic Radiation present a clear review of general knowledge of the genetic effects of radiation exposure and stress the responsibility of all public and private agencies to keep the radiation dose to the population below their recommended value of 10 rads/generation, or about 0.3 rads/year (145, 180). Subsequent recommendations by the International Commission on Radiological Protection suggest a dose of 5 rads in 30 years, or about 0.17 rad/year (121). The potential radiation exposure of astronaut crews should not make a significant contribution to this average per capita figure (145). The individual astronaut will undoubtedly be subject to gonad doses well above those normally allowed persons operating under the exposure limits established for routine occupational radiation hazards. The concept of permissible dose as that which entails a negligible risk of severe somatic or genetic injury has to be set aside when considering the space-radiation problem (145, 227). Even so, knowledge of the radiation dose received by the gonads permits an estimate of the probability of mutation in the spermatogonial cells and of its expression in the offspring. Such a calculation assumes

reasonable knowledge of the following parameters (the values given in parentheses are based largely on animal data):

1. The mutation rate per rad/gene ($\mu = 5 \times 10^{-8}$)
2. The number of genes per haploid set ($n = 10^4$)
3. The probability of expression of the new mutation in the first generation heterozygote ($s = 5 \times 10^{-2}$)
4. The gonad dose (D)

For example, assuming a dose of 100 rads,

$$(5 \times 10^{-8})\,(10^4)\,(5 \times 10^{-2})\,(10^2) = 2.5 \times 10^{-3}$$

would be the probability of a new mutation in the immediate offspring. Such probability statements must be accepted with considerable reserve, but they do serve to indicate that the genetic risk to the individual astronaut is not unacceptably high (145). Since the mutation rate in the advanced germinal cell stages of the mouse is twice or three times that observed in the spermatogonia, it can be recommended that conception of offspring be avoided during the postflight period in which irradiated postgonial cell stages are still present (227).

Dose rate and fractionation effects are not yet clear (225, 227). Low dose-rate exposures are not as effective as high-intensity exposures in inducing mutations. In mice, the difference may be a factor of 4 to 10 and apparently is operative at dose rates of less than 0.1 to 1.0 rad/min. However, this dose-rate dependence is observed only in the immature germ cells, not in spermatozoa. This finding is consistent with the concept that metabolic activity is a prerequisite to repair and that low dose-rate exposure, as with somatic injury, permits concurrent repair in conjunction with less damage to repair mechanisms (172). Present data on the effects of fractionation are limited to studies on mice using high dose rates and exposure intervals where high mutagenic sensitivity is known to exist. This sensitivity itself is of some concern, however, since existing data suggest that a fractionation interval of about 1 day causes the response to a second exposure to be greater than the normal expectation for that dose (224). Widely fractionated, low dose-rate exposures (comparable to most in-flight possibilities) would probably have approximately the same mutagenic effects as continuous exposures to low dose rates, but these rate and interval parameters have not yet been tested. There is a significant increase in quality factor with increasing LET, especially in protracted exposures, but the exact relationship is not clear (226, 251).

As a general operating principle for the next few decades, the NAS-NRC Panel believes that the probability of such late responses as general life-shortening and increased likelihood of malignancy may be considered of secondary importance in evaluating the risks of manned space flight (145). This attitude contrasts sharply with the manner in which occupational risks are evaluated, where late effects are of primary importance. The relative sizes of the astronaut and occupational groups provide the major reason for attaching less importance to late effects of radiation. The astronaut population

may be about 30 to 50; occupational groups may comprise hundreds of thousands of humans. Such late radiation injury is measured in statistical terms - an increase in age-specific death rate, a reduction of after-expectation of life, an increase in incidence of malignancy. Nevertheless, an awareness must be maintained in respect to the astronaut population of the late somatic and genetic manifestations of radiation injury. As noted earlier, selection of acceptable RES values for short-term responses of the gastrointestinal and hematopoietic systems will automatically entrain certain probabilities for occurrence of leukemia, generalized life-shortening, and other late manifestations.

Establishment of a career dose appears premature. If establishment of such a dose is a necessity then some value or set of organ-specific values must be established as acceptable integrated annual dose increments. Previous attempts along this line have been made, but have been thought by others to provide unrealistically low values that have no meaning or relationship to the biological effects they are designed to protect against (104, 147, 210). There is no obvious interim approach to the problem of developing radiation guides for evaluating long-term risk without also establishing career-exposure limits. Since a lack of both radiobiological knowledge and operating experience precludes the establishment of such dose limits at the present time, an alternate suggestion is offered as an approach to the question of general life-shortening. The long-term radiation risk may be compared with the accepted risks associated with piloting high-performance aircraft. It has been estimated that the latter occupation is characterized by a life-shortening probability of approximately 10 years. If this risk is assumed to be additive with the radiation hazards, the question then becomes how much additional probability of life-shortening may be acceptable. This is covered in the section on dose limits, below.

Soviet approaches to radiation safety in space flight have been reviewed (192).

SECONDARY FACTORS IN RADIATION INJURY

Environmental Factors

A number of environment factors in space flight having potential interaction with radiation differ markedly from normal terrestrial conditions. These include weightlessness, a pure-oxygen environment, periods of vibration and thermal load, and brief periods of excessive g loads. Unfortunately, there are few data on these interactions in humans. Animal studies suggest the interactions noted in Table 3-65. In this table, additive means simple summation of effects; synergistic means an interaction that produces a greater than additive effect; antagonistic means an interaction that produces a less than additive effect; and neutral means no detectable effect of the nonradiation stress alone or in combination. Synergistic potentials predominate and suggest that the conservative end of any proposed radiation dose-effect range should probably be used for first approximations in the hazards analysis.

Though animal data supporting these conclusions are available, it should be stated that this Table represents a tentative appraisal of the problem and will require revision as new data are made available (145, 294).

A synergistic factor frequently raised in considering the design of radiation shielding is the effect of space-cabin atmosphere (222). A parallel has been drawn between the mechanism of tissue damage seen in oxidation syndromes that follow radiation (221). High pressure of oxygen has been

Table 3-65

Summary of Stress Combinations and Types of Interactions

(After Langham (ed.)-NAS-NRC[145])

COMBINED STRESSES	INTERACTION
Radiation-noise	Neutral to synergistic
Radiation-hypothermia	Neutral to synergistic
Radiation-hyperthermia	Neutral to synergistic
Radiation-hypoxia	Synergistic (antagonistic for only brief periods during exposure)
Radiation-hyperoxia	Neutral to additive
Radiation-physiological factors	Synergistic
Radiation-emotional factors	Indeterminate
Radiation-vibration	Neutral to additive to synergistic
Radiation-acceleration	Antagonistic to neutral to synergistic
Radiation-weightlessness	Additive to synergistic
Radiation-vestibular factors	Neutral to additive
Radiation-emergency situations	Neutral to synergistic
Three-stress interactions	Indeterminate

used to sensitize tissue to radiation during x-ray therapy for cancer (107). Protective effects of antiradiation drugs against oxygen toxicity have also been shown. At present there appears to be no requirement for the alteration of shielding calculations due to the presence of 5 psia - 100% oxygen in future space-cabin atmospheres. However, several studies suggest that some synergism may be present. It has been shown that mice exposed to 750 r of gamma radiation from cobalt 60 given at 90 r/min have a survival about 10% lower in 5 psia - 100% oxygen than in air (23). At 900 r given at a rate of 38 r/min, the synergism was much less. On the other hand, mice exposed to 800 r of 250 kVp x-rays at only 14 r/min have shown no synergism with 5 psia - 100% oxygen (136). The type, total dose, and the dose rate of radiation may be significant variables in these studies of synergism. In tissue culture, at least, there is a steady reduction of the oxygen effect with increase stopping power until its apparent abolition by radiation $\gtrsim$ than 3000 MeV/cm^2/g (271). The exact stopping power at which the O_2 effect becomes undetectable in intact animals or isolated cells has not been established. Further research along this line is needed.

Alteration of the vestibular apparatus after human exposure to radiation has received recent study (176). Such alteration may play a significant role in degrading astronaut performance in zero g or during reentry and certainly requires further study.

Anti-radiation Therapy

The potential use of anti-radiation compounds in space operations has been recently reviewed (13, 16, 48, 145). The amino-thiols have received the most study and appear to be quite promising (13). However, the doses calculated as required for human protection are well within the human toxic range and can provide little protection against chronic or continuous radiation (13, 149, 193, 233, 265, 269, 276, 279). Much more data are required on toxicity mechanisms in humans. Maximum dose reduction for lethality is about 1.7 or 1.8, but this factor is much lower for other end points.

Other drugs such as PAPP and serotonin appear to be less effective than the amino-thiols, especially against radiation of high LET. Both MEA and PAPP protect mice against 440 MeV protons (203). Combination of different drug types has been tried with slightly more success than with individual drugs (165, 194, 269, 279, 283, 291). Combinations tend to alleviate the toxicity problem but certainly do not solve it.

Post-irradiation therapy consists of antinauseant and vomiting drugs (130, 143), intravenous fluids, anti-microbial agents, and supportive care. Leukocytes from leukemia patients (94) and bone marrow (6, 36, 59, 164) have been used with variable success in humans. The case for using autologous marrow in space operations has also been reviewed (48). Problems in obtaining sufficient amounts from each astronaut and the training of crews for administration of the marrow are superimposed on the basic storage difficulties. Cryogenic storage of blood and marrow in space is under consideration (173, 219).

Treatment of the irradiated skin with topical and oral cortisone has been tried with little success in relief of discomfort in early or late phases. In view of its penetrability and anti-radiation properties, the drug DMSO may offer some promise as a topical prophylactic against skin reactions (48).

In view of the lag times in arrival of solar flare particles after early electromagnetic warning signals and knowledge of the power-flux-time spectra of flares, it may be possible to predict the magnitude of integrated exposure expected during a flare from measurements made early in that same flare (93). In the future, prophylaxis with improved protective agents may well be tailored on a flare-by-flare basis. At the present state-of-the-art, however, prophylactic agents should not be entered into the basic hazard analysis even for acute flare exposures.

Performance After Radiation

The literature on the effects of radiation on the nervous system and behavior is large and complex (97, 111, 112, 145, 217, 282). The Soviet literature indicates many reflex changes in animals following radiation but the significance of parallel changes in humans is not clear (156, 287). Damage to the nervous system may even be the primary cause of death at very high dose levels (See Figure 3-37). The quantification of specific behavioral effects is confused by the prodromal syndrome which, by itself, can have profound

effects on behavior and yet may not involve irradiation of the central nervous system (see Table 3-36 and Figures 3-38 to 3-42). Whole body exposures of 100 to 130 rads of reference quality radiation can give fatigue, apathy, dizziness, headache, and depression which seriously alter behavior (150, 174, 207). Low doses of x-ray down to 10 rads can be detected by behavioral responses in some animals (98).

Observation of trained primates suggests that limiting mechanisms in each behavioral test may be key factors in interpretation of radiation effects. For example, protracted irradiation may tend to increase attentiveness to the immediate field of concern by depressing attention to peripheral information. Animals can continue to learn and can continue to express previously learned behavior (i.e., retained their learning) during and after considerable radiation to the head up to many hundreds or thousands of rads in short exposures. It should be remembered, however, that although the tests used involve fairly complex procedures they do not measure total performance under a wide range of circumstances. Some neurological deficits which affect behavior have followed exposure of the head to the higher doses just mentioned - often with concomitant morphologic changes (145).

Short of prolonged interplanetary flight, the microbeams of galactic radiation will probably not produce much of risk factor as far as brain, and eye damage are concerned (145, 299, 300). It is assumed the same holds true for the audiovestibular mechanism, though this is far from clear (176). Similar ignorance exists with respect to the retina where radiations of high LET may cause irreversible damage.

RADIATION DOSE LIMITS IN SPACE OPERATIONS

According to the NAS-NRC Panel, the rationale for an independent review of radiation protection in specific space flights should be reflected in the following points (145):

1. Radiation is only one of many recognized and accepted potential risks that may jeopardize the success of any flight mission.
2. Individual astronauts are carefully selected for their special skills and motivation. The application of existing standards of radiation safety established for large, occupationally exposed groups would unduly limit the ability of this small group of specialists to achieve their objectives.
3. The parameters of some space-radiation risks cannot be precisely predicted; therefore, optimal protective measures will not always be available or even feasible. Since any radiation shielding will add to the weight of a spacecraft, the reduction in risk to be achieved by the shielding must be balanced against the other uses to which this weight might have been put.

4. Since flight missions may vary in both duration and radiation exposure, the probability and importance of the radiation risk compared with those of other risks must be taken into account for each specific mission. A risk-versus-gain philosophy is most appropriate for this comparison, and the philosophy is particularly useful for evaluation of radiation risk. The latter is generally a cumulative one that should not require an urgent all-or-nothing type of decision as had been previously proposed (29, 101).

Risk Analysis

In view of this proposed risk versus gain philosophy and judgment, it is expected that the results of this judgment will probably vary for each mission. It has been suggested that the space radiation hazards and risks be evaluated in the following terms (145):

1. Immediate or early performance decrement (early responses) occurring within a few hours to one month following a major exposure.
2. Progressively increasing performance decrement or serious loss of performance over long periods of flight as a result of an accumulating exposure (progressive injury to the blood-forming system).
3. Probability of delayed or chronic radiation response that may require interrupting a planned series of flights and which may limit an astronaut's career.

Within each of these categories, the significant clinical symptomatology or responses must be defined on the basis of importance to crew safety and mission success. The relative significance of responses will be mission-dependent. The following suggestions may assist in identification of the important responses and in evaluation of their significance for each specific mission.

1. Any amount of radiation exposure should be considered as potentially detrimental and, therefore, the exposure should be kept at a minimum consistent with the risk versus gain philosophy.
2. Radiation guides should be set below the level that might result in an unacceptable probability of in-flight response capable of jeopardizing crew safety.
3. Elapsed time between recurrent or repeat use of an individual or crew should take into consideration the nature and extent of previous exposure, the predicted exposure risk of the contemplated mission, and the degree to which mission success may depend on individual or crew experience.

4. The dose or doses established for early effects automatically entails acceptance of certain probabilities of occurrence of generalized life shortening, leukemia, and other late manifestations.

5. The radiation responses may be subdivided into "in-flight" and "post-flight" categories. Although this subdivision is somewhat arbitrary, it is time-dependent and may be important under special circumstances, for which certain higher risks may be acceptable if it is clear that the latent period for expression of injury will automatically cause the response to occur post-flight.

In view of the above operational requirements and hazards analysis, it has been suggested that any radiation exposure that might exceed the dose limits set for the mission may be permitted if the concomitant risk incurred by action to avoid the radiation fields or to protect oneself against the potential injury is determined to be greater than the hazard associated with the excess radiation dose. There is no immediately obvious way of approaching the problem of setting limits for the long-term effects that does not also imply certain career-dose limits and the lack of operating experience precludes establishing a firm committment here.

As a first order approach to permit test of the trajectories for the AAP program, the radiation exposure levels have been suggested according to the following provisional operational criteria (208):

Planning Operational Dose (POD): The dose which should not be exceeded without requiring a mission modification of some degree. The degree of modification will be a function of the magnitude of the excess dose and will be formulated by mission rules. This dose will be used for mission planning purposes to determine if proposed trajectories and time lines are acceptable.

Maximum Operational Dose (MOD): The dose which should not be exceeded without specific modification of the mission to prevent further radiation exposure. Such an exposure would be considered to result in a potentially harmful in-flight response in terms of crew safety and post-flight response in terms of delayed radiation injury.

In establishing the POD and MOD limits it has been suggested that each response and its effect on the mission and crew are considered independently, and no adjustments are made for known or suspected uncertainties in radiobiological data, shielding calculations, or environmental data. As an example of this approach, the preliminary radiation limits for Apollo Applications Program are presented. Current recommendations for radiation exposure guidelines for missions of 30-60 days only are listed. It should be emphasized that the radiation units shown are for use in early planning of the AAP Program and will be updated as better knowledge becomes available.

The doses are expressed in rads of a reference radiation taken to be equivalent to 250 kVp x-rays whose mean LET is ~3.5 kev/micron of track length in wet tissue. No radiation QF or RBE has been applied.

The following doses found in Table 3-66 have been established on the assumption that the crew will be exposed to small increments of dose on each orbit and no allowance is made for pulses of radiation received at higher intensities. (See Section on Progressive Performance Decrement).

Table 3-66

Provisional Radiation Dose Limits Suggested for Preliminary Evaluation of a 30-60 Day Mission

(After Grahn[208])

Tissue	Depth	POD	MOD
Skin	0.1 mm	2.5 rads/day	5 rads/day
Eye	3.0 mm	1.25 rads/day	2.5 rads/day
Bone Marrow	5.0 cm	0.6 rads/day	1.0 rad/day

Dosimetry for Characterization of Space Radiation Exposure (145)

From a physical point of view, the type of radiation, flux density, and energy spectrum completely define the radiation field which produces a biological change. From a biological point of view, however, it is the energy transferred by this field to the biological entity under consideration which is most important. When the physics of interactions between tissues and incident radiations are known, then the physical specification suffices, although in many instances computer programs are required to apply such physical knowledge. The most logical choice for space radiation monitoring, in view of the above difficulty, appears to be a tissue-equivalent system (49, 83, 119, 127, 175).

The problems associated with radiation monitoring of a manned space flight must, in other words, be clearly distinguished from the acquisition of geophysical data or of information aimed primarily at computation of shielding requirements. The chief requirement is for an instrument which will yield a direct indication of absorbed dose in tissue in real time. Such a requirement rules out instruments which are only capable of measuring flux or even flux plus energy distribution because of the complexity of using such data to provide dosimetric information. The dependence on geometrical variation, angular incidence, self-shielding, lack of accurate physical data on interaction properties between the radiation and tissue, plus the degree of complexity of the computations themselves, tends to rule out this method. It seems reasonable to conclude that knowledge of the physical characteristics of the radiation environment, although it may provide data for other scientific or engineering purposes, is not immediately applicable to radiation monitoring. Such information, therefore, should be gathered and treated separately from the problem of crew safety. For the latter problem, a practical approach to a detector whose atomic composition is as close as possible to that of tissue and whose response is proportional to energy absorbed, rather than to

flux density, appears to be the best course to follow, even within the limitations and compromises necessary (15).

The NAS-NRC Panel has suggested that the development of such a system should take two lines of approach (145). First, a system to supply both rate ard total absorbed dose in rads as a function of mission time should be engineered for space vehicle application. Absorbed dose should be determined in such a manner as to supply values at superficial points and at critical depths in the body. Second, a tissue-equivalent system for determining the energy absorbed per event in a representative tissue volume centered at the points where absorbed dose has been measured should be developed on a simplified basis so that energy deposited per event can be classified into several broad LET groups. Total absorbed dose and dose rate should be displayed and weighted in accordance with LET distribution if the situation warrants. Absorbed dose, dose rate in rads, and LET groups should be recorded for future reference.

Dose should obviously then be defined in terms of rads, and preferably it should be measured in a tissue-equivalent system at at least three levels, including 0, 5 and possibly 10 cm in equivalent tissue depth. If possible, doses should be measured at the 0.1 mm and 3 cm levels noted in Table 3-66. Accuracy of these determinations should be no less than ±15 percent. If compromise is required, it would then seem that at least two measurements should be made: one at the equivalent level of the skin and a second at about 5 cm, assuming it to be the mean depth of the bone marrow. The spanning measurements suggested above are statistically better but probably would invoke a greater weight penalty and instrument sophistication. A practical approach to the solution of this problem is under development (83).

A very important question that has been debated repeatedly concerns the alternative whether the radiation field inside the vehicle should be probed with stationary sensors distributed throughout the ship or whether micro-sensors on the bodies of the crew members should be given preference. It could be argued in favor of the first alternative that stationary sensors would free the crewman from additional gear in his space suit. Furthermore, such sensors could be of greater weight and bulk, thereby allowing a more elaborate analysis of the local radiation level. In favor of the second alternative, sensors on the body would indicate the radiation level exactly at the location where it counts. It is even conceivable that the differential reading of a pair of sensors on the chest and back would provide a crude measure of depth-dose. However, the validity of the data beyond a measure of local surface doses is open to question. Multiple sensors do allow estimation of the general homogeneity of the dose and could be used to estimate the degree of partial body exposure for risk analyses (270). The main argument in favor of sensors on the body naturally rests in the advantage that it would cover all contingencies of each individual's activity. In terms of the Lunar Mission, surface dosimeters require no changes or adjustments whether the individual is in the heavily shielded Apollo vehicle or in the extremely light Lunar Module, or is engaged in extravehicular activity. To be sure, for the last named condition, it should be recognized that there are very high fluxes of low-energy electrons and protons at many locations in the space environment. These should be detected and measured on the outside of the vehicle prior to

any outboard excursion, since they can potentially produce a very high surface tissue dose.

A final question concerns the need of LET sensors as a component of dosimetric instrumentation in space. In discussing the problem, measurement of heavy nuclei is excluded. In its conventional interpretation, LET defines the inhomogeneity in the distribution of the ionization events at the microscopic level. The diameter of the ionization columns which heavy nuclei produce in tissue exceeds the dimensions of a single cell, creating a peculiar exposure pattern with a few cells exposed to very high doses and the surrounding bulk of the cell population remaining entirely unaffected. It is not generally agreed upon that the biological significance of this type of exposure cannot be dealt with adequately in terms of the conventional LET concept (236, 243). Some feel that the LET concept is still useful in this context, and with the appropriate study, could be applied to the primary galactic cosmic radiation (270). As has been indicated above, the dose of microbeam radiation will probably not play a significant role in gross brain or eye damage in most space missions of less than 1 year duration (145). However, one must keep in mind the irreversible nature of the lesions in such areas as the gonads, lens and retina from very high-LET radiation (270).

If heavy nuclei are excluded, the LET problem appears only of limited importance for the remaining types of ionizing agents represented in the galactic radiation beam (243). The two main components of the primary galactic beam (i.e., protons and alpha particles) produce LET values exceeding those of standard x-rays only at energies from a few MeV down to the Bragg peak. These energies are not represented at all in the incident beam. Low-energy protons and alpha particles originate only locally in nuclear disintegrations in absorbing material. It should be remembered that the spectacular multipronged disintegration stars which cosmic-ray primaries release in collisions with silver and bromine nuclei of nuclear emulsions are absent in materials made up of low Z components such as living tissue. The number of prongs per star in these substances is small and the star frequency low; hence, terminating protons and alpha particles contribute only insignificantly to total ionization dose. Since the remaining dose of the galactic beam is produced mainly by protons of very high energies, the question could be raised whether a QF considerably smaller than 1 would not be applicable to the total absorbed dose from the galactic beam. (See Table 3-16 and Figure 3-17).

The situation is different for the proton and alpha fluxes of solar particle beams. Protons and alpha particles reaching the end of their ionization ranges (so-called "enders") contribute noticeably to the total ionization dose in systems of medium-light shielding (1.5 g/cm^2) and become the predominant contributors to the total dose in the skin and subcutaneous tissue behind low shielding. Under the latter conditions, calculation suggests a QF of 4 to 5 for late effects from combined proton and alpha particle dose to a depth of a few mm. On the other hand, it is not possible to say unequivocally whether these very special conditions would justify the great complications which a separate determination of LET would introduce in dosimetric instrumentation. Since the conditions in question occur only under conditions of low shielding and are always accompanied by a very steep drop in absorbed dose in the first

mm, it is possible that measurement of dose in rads with application of a suitable QF factor to the skin dose would suffice. (See Figure 3-25).

Dose Limits for Ground Personnel

Maximum permissible radiation doses in adult radiation workers and ground personnel as recommended by the Federal Radiation Council (88) are:

Type of exposure	Condition	Dose (rem)
Radiation worker:		
(a) Whole body, head and trunk, active blood-forming organs, gonads, or lens of eye.	Accumulated dose	5 times number of years beyond age 18
	13 weeks	3
(b) Skin of whole body and thyroid	Year	30
	13 weeks	10
(c) Hands and forearms, feet and ankles.	Year	75
	13 weeks	25
(d) Other organs	Year	15

If it is not feasible to govern exposures to internal emitters by applying airborne radioactivity concentration standards, the following radiation protection standards can apply: (281)

Type of exposure	Condition	Dose (rem)
Whole body, active blood-forming organs, gonads.	Year	5
	13 weeks	3
Thyroid	Year	30
	13 weeks	10
Bone		Body burden of 0.1 microgram of radium-226 or its biological equivalent *
Other organs	Year	15
	13 weeks	5

* Exposure must be governed so that the individual's body burden does not exceed this value 1) when averaged over any period of 12 consecutive months and 2) after 50 years of occupational exposure.

The quality factor for calculating rem values applicable to low dose exposure and risk of late effects in occupational exposure are given in Table 3-2 (41).

A Radiological Safety Handbook for the John F. Kennedy Space Center is available (186). Data are available on the hazards of radioisotope thermoelectric generators for remote stations around the world (187).

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