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Relating Space Radiation Environments to Risk Estimates

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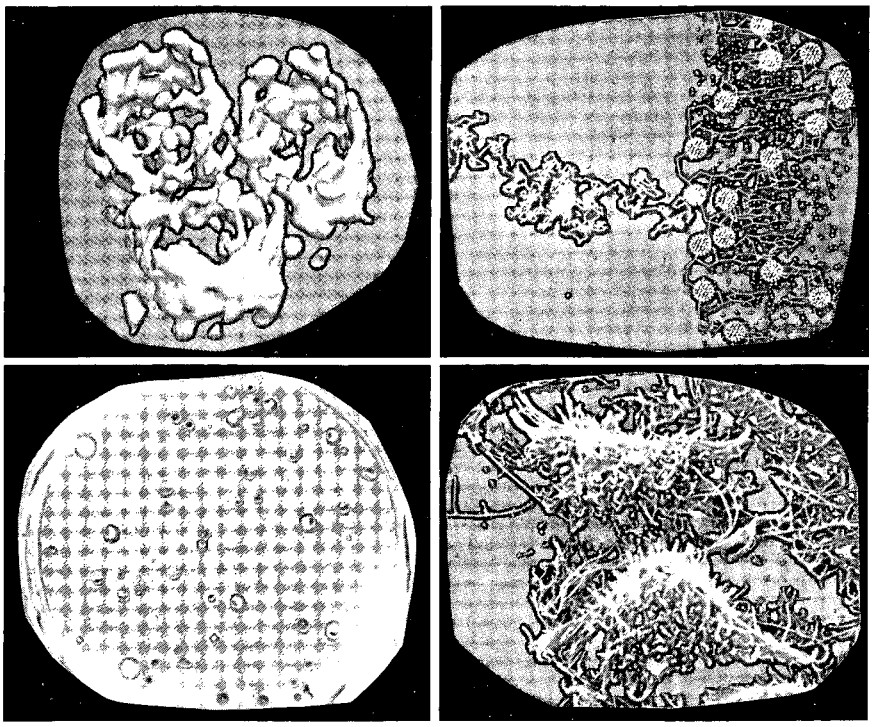

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INTRODUCTION

A number of considerations must go into the process of determining the risk of deleterious effects of space radiation to travelers on long missions. Among them are (1) determination of the components of the radiation environment (particle species, fluxes and energy spectra) which the travelers will encounter, (2) determination of the effects of shielding provided by the spacecraft and the bodies of the travelers which modify the incident particle spectra and mix of particles, and (3) determination of relevant biological effects of the radiation in the organs of interest. The latter can then lead to an estimation of risk from a given space scenario. Clearly, the process spans many scientific disciplines from solar and cosmic ray physics to radiation transport theory to the multistage problem of the induction by radiation of initial lesions in living material and their evolution via physical, chemical, and biological processes at the molecular, cellular, and tissue levels to produce the end point of importance.

This lecture will provide a bridge from the physical energy or LET spectra as might be calculated in an organ to the risk of carcinogenesis, a particular concern for extended missions to the moon or beyond to Mars. Topics covered will include (1) LET spectra expected from galactic cosmic rays, (2) probabilities that individual cell nuclei in the body will be hit by heavy galactic cosmic ray particles, (3) the conventional methods of calculating risks from a mixed environment of high and low LET radiation, (4) an alternate method which provides certain advantages using fluence-related risk coefficients (risk cross sections), and (5) directions for future research and development of these ideas.

RISKS OF RADIATION IN SPACE

We first consider the established and potential radiation risks to be expected on extended travel in space. It is accepted that, given adequate shielding against giant solar particle events, the most important radiation effects outside the earth's magnetic field are late effects: cancer, cataracts, and genetic effects caused by radiation produced both from solar particle events and from the galactic cosmic rays. The National Council on Radiation Protection and Measurements (NCRP) has published a report discussing various aspects of the space radiation risk assessment problem (Report #98, NCRP,

1989). It was concluded that the most important late effect was cancer. Recently, new assessments of cancer risk have been made from the Atomic Bomb survivor data base (Shimizu et al, 1990), and the BEIR V Committee (1990). The International Commission on Radiological Protection (ICRP) has recently published revised estimates of the risk of cancer mortality per unit of exposure. The value of 4% per Sv was given for the excess risk of cancer mortality for radiation received at low dose rate by adult members of the population (ICRP, 1991). This new value is roughly a factor of two higher than previously thought to apply. Based on these new estimates, the NCRP career Exposure Limits for near-earth orbit, suggested in Report #98, are presently under review.

Other potential late effects include cataractogenesis and possibly irreparable damage to the central nervous system accumulating from the low level of the penetrating highly ionizing component of the galactic cosmic rays (sometimes called HZE particles (Grahn, 1973)). It is not known whether the doses will be below the threshold dose for cataractogenesis and there is only speculation at present on the effects to the human central nervous system. Genetic effects to subsequent generations may become important if and when space travelers begin procreation after having been on an extended mission.

IMPORTANCE OF SPECIFIC ORGAN RISK ASSESSMENT

In the radiation exposures from large solar particle events that will sometimes occur in space flight, each organ of the body will receive a different amount of radiation due to the different amounts of shielding provided by the rest of the body. In addition, it has been determined that every organ or tissue has a different radiosensitivity for tumor induction. This is shown in Table I taken from ICRP Report #60 (1991). We note that some organs are over an order of magnitude more sensitive to cancer induction than others. Thus, in determining the overall cancer risk to the body from solar particle events, it is important to determine the exposure to each organ separately, estimate the average dose equivalent to the organ and add the contributions of risk to each organ to obtain the total risk of cancer induction in the body. In order for this to be a meaningful calculation, the external shielding (i.e., spacecraft, lunar habitat, etc.) must be very well defined, and the extent of modulation of the radiation field must be well understood.

LET SPECTRA FROM GALACTIC COSMIC RAYS

Another important concern is the risk from the galactic cosmic radiation. A good review of our knowledge in cosmic ray abundances, their energy spectra, and their modulation through the eleven and twenty-two year solar cycles has been presented in other papers in this course. The question arises: what is the relative "importance" of the different species in the cosmic rays in determining the carcinogenic risk? A feeling for this can be obtained by examining the LET spectrum of the radiation in question. The *fluence-LET* spectrum is the distribution of LET's of the particles found in the radiation environment. Mathematically, the fluence-LET spectrum, f(L), is defined such that the total fluence (number of particles per unit area), F, can be written as the integral over all LET:

$$F = \int f(L)\ dL \qquad (1)$$

where L is the LET_{∞} or dE/dx (i.e., the stopping power) of the particles and f(L) is the fluence of particles having LET between L and L + dL.

The absorbed dose, **D**, can be written in terms of the fluence-LET spectrum as follows:

$$D = k \int f(L)\ L\ dL \qquad (2)$$

where k is a constant linking fluence and dose units. We note that the integrand can also be defined as the distribution in LET of absorbed dose, D(L), the *dose-LET spectrum,* so that

$$D(L) = k\ f(L)\ L. \qquad (3)$$

A representation of the dose-LET spectrum for the galactic cosmic rays at solar minimum is shown in Figure 1. The distribution D(L) has been multiplied by L so that equal areas under the curve have equal weights since the distribution is plotted on a logarithmic scale, i.e., linearly in the log of the LET. Several of the peaks in this distribution have been identified on the curve to show the relative importance of protons, helium, silicon, and iron ions in producing absorbed dose. The peak of low energy iron is a reflection of the fact that in free space the low energy iron ions having quite large LET values are still present. It should also be noted that the actual heights of the peaks as shown on the graph are of no significance since the curve should in theory go to infinity where the LET *vs.* energy relationship for each particle type reaches a maximum or minimum.

There is a modification of this spectrum as the particles traverse matter. Coulomb effects (i.e., ionization of the atoms through which charged particles pass) will slow the particles down, thus changing their LET's and the absorbed dose they produce. Nuclear interactions will occur causing secondary particles which, in turn, will slow down and cause other nuclear interactions. The nuclear processes are very complex for all the species and energies involved in the galactic cosmic ray spectra. Computer codes have been developed to calculate the transport of the galactic spectra through matter, and a description of several of them has been presented at this course (see papers by J. W. Wilson). One output of such codes is the integral dose-LET distribution, i.e., the dose produced at the depth in question by particles above a given LET plotted as a function of LET. This distribution can be differentiated to give the differential dose-LET distribution at the depth of interest and plotted in the same manner as in Figure 1. Such a plot for 10 g/cm^2 of aluminum shielding is shown in Figure 2. The same peaks for the different ions are still seen even at this depth and the proton peak has remained constant, while the higher LET peaks are somewhat less important and the low energy, very high LET iron peak has totally disappeared. It should be noted that multiple production of nuclear secondaries has been accounted for in the calculation, but multiple coulomb and nuclear scattering and as well as straggling have been neglected, i.e., the "straight-ahead approximation" has been assumed. Note that the "valleys" at low LET have been filled up to some extent by the secondary nuclear interactions of the higher Z particles.

CONVENTIONAL METHOD OF DETERMINING THE RISK FROM MIXED RADIATION ENVIRONMENTS

Given an LET spectrum like the one shown in Figure 2, it is possible to calculate the dose equivalent, H. Then the risk of excess cancer mortality by the radiation is just the product of **R** and H, where R is the risk coefficient for low LET radiation expressed in risk per unit of dose equivalent (i.e., risk per Sv) as already discussed. The dose equivalent is found by multiplying the dose distribution by a weighting factor (called the Quality Factor, **Q(L)**), which is a function of LET, and then by integrating over all LET.

Mathematically, we have:

$$H = \int Q(L)\ D(L)\ dL \qquad (4)$$

The Quality Factor is intended to weight the higher LET components of the radiation, in line with the experimentally found observation that, per unit of absorbed dose, high LET radiation is more effective in causing biological damage (thus presumably in causing human cancer) than low LET radiation. The functional dependence decided upon comes from examination at various LET's of experimental data on dose response curves of various biological systems deemed relevant to carcinogenesis and extrapolated to low dose and low dose rates or protracted exposures.

The ICRP has recently published a new dependence of Quality Factor on LET (ICRP, 1991). The new and old (ICRP, 1977) functions are plotted in Figure 3. The three main differences are that the new function (1) remains at unity until the LET reaches 10 keV/μm, (2) rises to a value of 30 at 100 keV/μm, and (3) decreases as the LET rises above 100 keV/μm.

Multiplication of the incident galactic cosmic ray dose distribution (under no shielding, cf. Figure 1) by the *new* Quality Factor yields the curve shown in Figure 4. Because a minimum ionizing iron ion has an LET of about 145 keV/μm, near the maximum of the Quality Factor *vs.* LET curve, we see a very prominant peak in the distribution around this value. The curve as calculated behind 10 g/cm^2 of aluminum shielding is given in Figure 5. Here again the contribution around 100 keV/μm from the iron and other high Z components is quite large. This indicates that high LET radiation will play a considerable role in determining the ultimate biological response even under fairly heavy shielding.

HIT FREQUENCIES OF CELL NUCLEI BY GALACTIC COSMIC RADIATION

It is of some interest to estimate the frequency with which the nuclei of typical cells within the bodies of space travelers might be hit by tracks of the galactic cosmic radiation. Calculations have been made for two specific simple shielding configurations at solar minimum conditions (Curtis and Letaw, 1989). Case a is a point at the center of a spherical aluminum shell 1 g/cm^2 thick, and case b is a point at the center of a spherical aluminum shell 4 g/cm^2 thick and 5 cm inside the surface of a sphere of water 30 cm in diameter to approximate in a rough way the human body. The two shielding configurations are shown in Figure 6. An area of 100 μm^2 was chosen for the size of the "target" cell nucleus. This is a conservatively large estimation for the cross sectional area of many cell nuclei in the human body.

The calculation uses the "straight-ahead" approximation and includes nuclear fragmentation. Hit frequencies for the two configurations are shown as a function of the charge of the particle in Figure 7 for a three-year mission outside the geomagnetosphere at solar minimum. The free-space abundances are plotted as x's in the figure. It is interesting to note that the peaks and valleys reflecting the relative free space abundances are almost indistinguishable from the more lightly shielded configuration (case a) and persist even for the more heavily shielded configuration (case b). We see, for instance, that each cell nucleus in the case b configuration would receive over a three year mission, on the average, roughly 400 proton hits, 40 helium ion hits, 0.7 carbon ion hit, 0.5 oxygen ion hit, and so on. From these data and assuming Poisson statistics (i.e., a random process) for particle arrivals, the percentages of cell nuclei hit at least once or at least twice behind the two shielding configurations can be calculated. These

are shown for the more heavily shielded configuration in Table II for various charge groups. We note that 33% of cell nuclei will receive one or more hits during a three-year mission at solar minimum from particles with charge between 10 and 28. Some 80% of these will be single traversals only, that is, no other traversals of particles within that charge group. However, the probability is 0.86 that these cell nuclei will receive at least one hit from a particle in the charge 3-9 group and all will receive many hits from helium ions and protons.

Another way to present the results is in terms of the mean time between hits of ions with the same charge, i.e., the reciprocal of the mean frequencies. The frequencies can then be presented in terms of one hit per mean time between hits. This is shown for protons, helium ions, oxygen ions and iron ions in the second column of Table III. In the third column are shown for comparison hit frequencies simply scaled to the human body by multiplying by the ratio of the two presumably relevant areas (0.3 m^2 for the body/100 μm^2 for the cell nucleus). Because of the assumptions in this calculation, the latter are very rough values. We conclude that although the bodies of the space travelers will be hit by many galactic cosmic ray particles during a mission lasting a year or longer, each cell nucleus will be hit by very few heavy ions with high charge, and the majority will be hit by at most one very heavy charged particle (with charge above 10). This conclusion leads us to the realization that for travel on extended deep space missions, the effects of single heavy particle traversals of cells may play very a important role.

From the above discussion, we realize that it might be advantageous to split the dose equivalent, H, as calculated from Eq. (4), into its component parts, keeping the contributions from the different ion species separate. Thus, Eq. (4) can be rewritten:

$$H = \sum_{i=1}^{n} \int Q(L_i)\ D(L_i)\ dL_i \qquad (5)$$

where the summation is over the *n* different ion species in the cosmic ray spectrum. Remembering the relationship between the dose- and the fluence-LET spectra (Eq. 3), the risk then becomes:

$$R_c = R\ H = R \sum_{i=1}^{n} \int kQ(L_i)\ L_i\ f_i(L_i)\ dL_i \qquad (6)$$

INTRODUCTION OF AN ALTERNATIVE TO THE CONVENTIONAL APPROACH OF CALCULATING DOSE EQUIVALENT

It was pointed out some time ago that there is a way of defining risk without having first to estimate the dose equivalent. In 1966 it was suggested that a fractional cell lethality (FCL) be defined and calculated for astronauts caught in large solar particle events (Curtis et al, 1966). In this approach, fluence-LET spectra of protons, helium ions and heavier particles as found at depth within the body of a seated astronaut were multiplied by cell inactivation cross sections as a function of LET as measured with heavy ions on human kidney cells at the Berkeley Hilac (Todd, 1965), and then integrated over LET to obtain the number of inactivation hits. This yielded the survival, S, of cells at that depth, and the FCL was just 1 - S. The FCL was considered to be a measure of risk, since it was a direct measure of cells killed. Other similar ideas relating risk directly to a microdosimetric spectrum of energy deposited locally, (i.e., a y-

spectrum) and defining a "hit-size effectiveness function" (HSEF) have been discussed in the literature (Bond et al, 1985, Sondhaus et al, 1990).

We now formally introduce the concept of a fluence-based risk coefficient (Curtis et al, 1991). It is defined as the risk per unit fluence so that the product of it and the fluence of a particular particle species yields the risk, for instance, the excess relative risk of cancer mortality, caused by that fluence. Since it has the units of risk/ (particle/unit area), this gives it the units of area and it is called a *risk cross section.* Under the assumptions that only single particle traversals are important (see above) and that the risk probability is small compared to unity for each particle type, the risks from all particle species are additive. If we denote the risk cross section for the ith particle species by σ_i (L_i), we can write for the risk:

$$R_c = \sum_{i=1}^{n} \int \sigma_i(L_i)\, f_i(L_i)\, dL_i \qquad (7)$$

RELATIONSHIP BETWEEN RISK CROSS SECTION AND QUALITY FACTOR

Comparing Eqs. (6) and (7), we note that the risk cross section can be written in conventional terms as follows:

$$\sigma_i(L_i) = k\, R\, Q(L_i)\, L_i \qquad (8)$$

As seen in Fig. 3, the Quality Factor is defined as a single-valued function of the LET. This means that any particle of a given LET will have the same value of the Quality Factor. It is well known that because of the different *track structure* (spatial distribution of energy loss around the track's trajectory) resulting from particles with different charge, biological effects of different particles with the same LET can be quite different. The definition of the risk cross section allows for this possibility in its formulation; this is not the case in the conventional treatment.

AN EXAMPLE OF USING THE CONCEPT

The prevalence of radiation-induced tumors in the Harderian gland of the mouse has been used as an example of how the concept of risk cross section can be used to calculate expected tumor prevalence during a space flight at solar minimum outside the geomagnetic field. From fluence-response curves obtained at the Berkeley BEVALAC for several beams of charged particles with well-defined LET's, the initial slope (i.e., the slope of the curve of prevalence as a function of fluence at very low fluence) was determined. This slope is the risk cross section for Harderian tumor prevalence. The slopes in units of μm^2 are plotted in Figure 8 (Alpen et al, submitted). The solid line is an analytical expression developed simply to possess characteristics thought to pertain (Curtis et al, 1991): an initial linear increase with LET (corresponding to the region where the Relative Biological Effectiveness, RBE, is expected to be 1), a supralinear region (corresponding to the region where the RBE is increasing to values considerably greater than 1), and a region of constant cross section or plateau at high LET (corresponding to the region of "saturation" or "overkill" where the RBE is decreasing). We note that there is a fairly good fit to the data, but there is no evidence whatsoever that the plateau implied by the analytical expression has been reached in the experimental data.

An additional factor has been included in the final calculations. It is well known that at high energy, nuclear interactions of protons and helium ions with target molecules in tissue can contribute local high LET events that rival or even surpass the contribution of direct ionization losses from these particles. This phenomenon has been studied (Shinn et al, 1989), and we have included target fragmentation in the calculation when the galactic cosmic ray spectra were integrated. The total risk cross section becomes:

$$\sigma_i^*(L_i) = \sigma_i(L_i) + \sigma_{targ\ frag} \qquad (9)$$

The results when using galactic cosmic ray spectra at solar minimum calculated behind 1 g/cm^2 of aluminum shielding are given in Table IV. The results of tumor prevalence per year both including and excluding target fragmentation are shown. The contributions from protons (Z=1), helium ions (Z=2), and two higher Z subgroups (Z=3-9 and 10-28) are presented separately. The prediction is that a 6% prevalence of Harderian tumors is expected in a space mission of one year outside the geomagnetosphere from the galactic cosmic radiation behind 1 g/cm^2 of aluminum shielding. Some 60% of the total comes from the Z = 10-28 charge group. The integral prevalance plotted as a function of LET (both for the total and for the contribution not including target fragmentation) is shown in Figure 9. The prevalence due to particles with LET greater than a given LET is plotted against the LET. The conclusion is clear that most (~ 80%) of the effect is caused by radiation with LET above 10 keV/μm for this thickness of shielding. We emphasize that this percentage will vary with shielding thickness as well as time through the solar cycle. This mode of evaluation thus results in the same conclusion as from the more traditional one: a large percentage of the biological effect is due to particles at high LET's.

CONCLUSIONS AND FUTURE DIRECTIONS

This discussion of radiation risks in space flight has emphasized the problem of determining the risk of cancer induction from the radiation environment that travelers will find outside the shielding confines of our earth's magnetic field, on a return excursion to the Moon, for example, or an exploratory trip to Mars. The conventional calculation of LET spectra produced by the galactic cosmic radiation behind a typical shielding thickness has been described. From such calculations, in which a weighting factor such as the Quality Factor weights the differential dose distribution in LET, it is possible to learn the *relative* importance of the different LET components constituting the radiation. We have seen that even under 10 g/cm^2 aluminum shielding, a considerable portion of the estimated risk (as suggested by the calculated distribution in LET of the dose equivalent) arises from components of LET greater than 10 keV/μm. In the future, it will be important to validate and improve the codes by which these transport calculations are made.

We have next approached the problem from a slightly different point of view by calculating the hit frequencies of cell nuclei at a point inside a simulated human body (a 30-cm diameter sphere of water) from the various charged components of the galactic cosmic radiation under well-defined shielding configurations. We have noted that most cell nuclei, if hit at all, will be hit by only one heavy highly charged particle during a long term mission of one to three year's duration. This emphasizes the need to study at ground level the biological effects which are considered relevant to the carcinogenic process of *single* traversals of cells by a high energy heavy particle. Such cells will also be hit by larger numbers of particles with lower charge over very long periods of time. Interactive effects of such hits should be studied to determine if they will provide anything more than a small second order modulation.

Finally, we have shown how a new concept related to particle fluence, the *risk cross section,* can be used to estimate risk and have pointed out several advantages for using such a concept for evaluating risk from the galactic cosmic radiation. The development of this idea paves the way toward a mechanistic understanding of radiation-induced carcinogenesis from charged particle radiation in terms of particle traversals of the cells at risk. Clearly, considerably more ground-based research is necessary on identifying the important changes inside a cell nucleus at the molecular level caused by a traversal of a high energy heavy particle track. Only in this way will we ultimately be able to better estimate the risk of these very low fluence very high energy particles to the health of space travelers on long missions .

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TABLE I

NOMINAL PROBABILITY COEFFICIENTS FOR INDIVIDUAL TISSUES AND ORGANS[1] [2]

Tissue or organ	Probability of fatal cancer for working-age population (10^{-2} Sv^{-1})
Bladder	0.24
Bone marrow	0.40
Bone surface	0.04
Breast	0.16
Colon	0.68
Liver	0.12
Lung	0.68
Oesophagus	0.24
Ovary	0.08
Skin	0.02
Stomach	0.88
Thyroid	0.06
Remainder	0.40
Total	4.00

[1] From ICRP, 1991.

[2] The values relate to a population of equal numbers of both sexes and a wide range of ages.

TABLE II

PERCENTAGE OF CELL NUCLEI HIT [1]

Case b Shielding Configuration

(Area = 100 μm^2, Solar Min.)

Mission Duration	One Year		Three Years	
Charge Group	≥1 Hit	≥2 Hits	≥1 Hit	≥2Hits
3-9	49	14	86	59
10-16	10	0.51	27	4
17-25	1.6	0.001	4.8	0.12
26-28	0.99	0.005	2.9	0.04
10-28	12	0.8	33	6

[1] From Curtis and Letaw (1989).

TABLE III

ROUGH NUMBERS FOR HIT FREQUENCIES IN SPACE
(Solar min.)

Particle Species (and charge)	Hits/cell nucleus [1] (Area ~100 μm^2)	Hits/human body [2] (Area ~ 0.3 m^2)
Protons (Z=1)	~ 1 per 3 days	~ 10^4 per second
He ions (Z=2)	~ 1 per month	~ 10^3 per second
Oxygen ions (Z=8)	~ 1 per 6 years	~ 20 per second
Iron ions (Z=26)	~ 1 per 100 years	~ 1 per second

[1] Behind 4 g/cm^2 Al and 5 cm beneath surface of a 30-cm diameter water sphere, (from Curtis and Letaw, 1989).

[2] Obtained simply by scaling to the larger area.

TABLE IV

CONTRIBUTIONS TO HARDERIAN GLAND TUMOR RISK FROM VARIOUS CHARGE GROUPS [1]

(Solar Min., 1g/cm^2 Aluminum shell shielding)

Charge Group Z	Prevalence per year	
	Direct ionization only	With target fragmentation
1	0.0052	0.0092
2	0.0029	0.0039
3-9	0.0089	0.0101
10-28	0.0362	0.0367
TOTAL	0.0532	0.0599

[1] From Curtis et al, in press ,1991.

FIGURE LEGENDS

1. The differential dose-LET spectrum for galactic cosmic rays in free space at solar minimum. Several of the peaks are identified as being caused by specific components of the radiation. The distribution has been multiplied by L, the LET, in order to give equal weights to equal areas under the curve, since the LET scale is linear in the logarithm of the LET.

2. The differential dose-LET spectrum for galactic cosmic rays behind 10 g/cm^2 aluminum shielding at solar minimum. The "low-energy" iron peak at high LET is absent since that component is absorbed in the shielding. The distribution has been multiplied by the LET as Fig. 1.

3. The new (ICRP, 1991) and old (ICRP, 1977) Quality Factors as determined by the International Commission on Radiological Protection are shown as a function of LET.

4. The differential dose-equivalent distribution for galactic cosmic rays in free space at solar minimum. This curve gives an idea of the relative importance of the various components of the radiation in causing risk. The distribution has been multiplied by the LET as in Fig. 1.

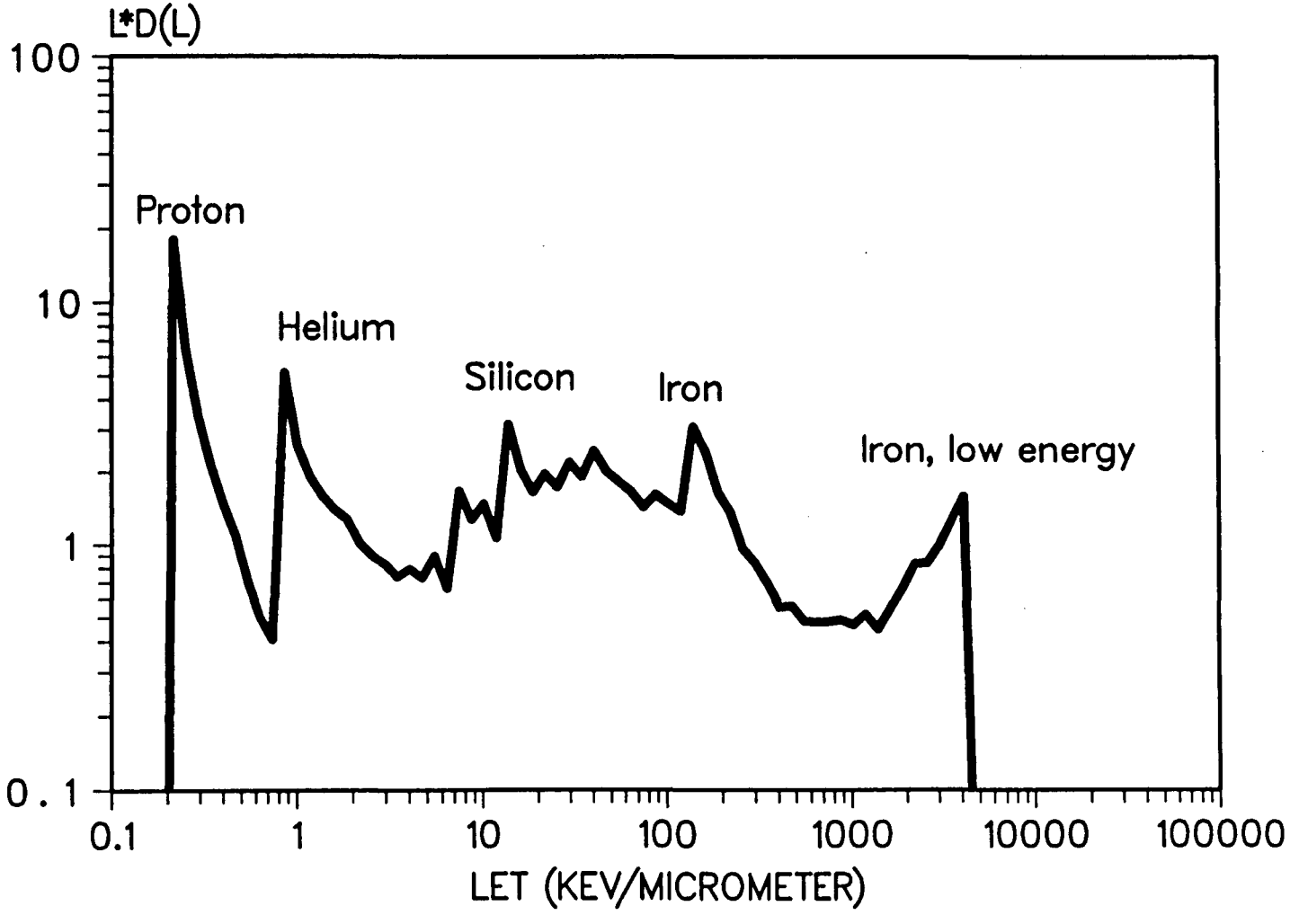
5. The differential dose-equivalent distribution for galactic cosmic rays behind 10 g/cm^2 aluminum shielding. The distribution has been multiplied by the LET as in Fig. 1.

6. Description of the shielding configurations for the hit frequency calculations. Case a: the point of interest is the center of an aluminum shell 1 g/cm^2 thick; case b: the point of interest is the center of an aluminum shell 4 g/cm^2 thick and 5 g/cm^2 beneath the surface of a sphere of water 30 g/cm^2 in diameter.

7. Frequencies of charged-particle hits caused by galactic cosmic rays in a 100-μm^2 area at the point of interest within the two shielding configurations (cases a and b defined in Fig. 6) plotted as a function of the charge of the particles for a 3-year mission outside the geomagnetosphere at solar minimum. The free-space galactic cosmic ray abundances converted to the same units are shown as **x**'s.

8. The risk cross sections for Harderian gland tumor prevalence as measured with monoenergetic beams from the Berkeley BEVALAC (Alpen et al, submitted) are shown as a function of LET. The curve is from an analytical expression as explained in the text (Curtis et al, 1991).

9. The integral probability distribution of Harderian gland tumor prevalence calculated for a year's mission at solar minimum behind 1 g/cm^2 aluminum shielding is plotted as a function of LET. It is the probability of tumor induction from LET's greater than a given LET plotted against the LET. Curves with and without inclusion of target fragmentation are shown for comparison.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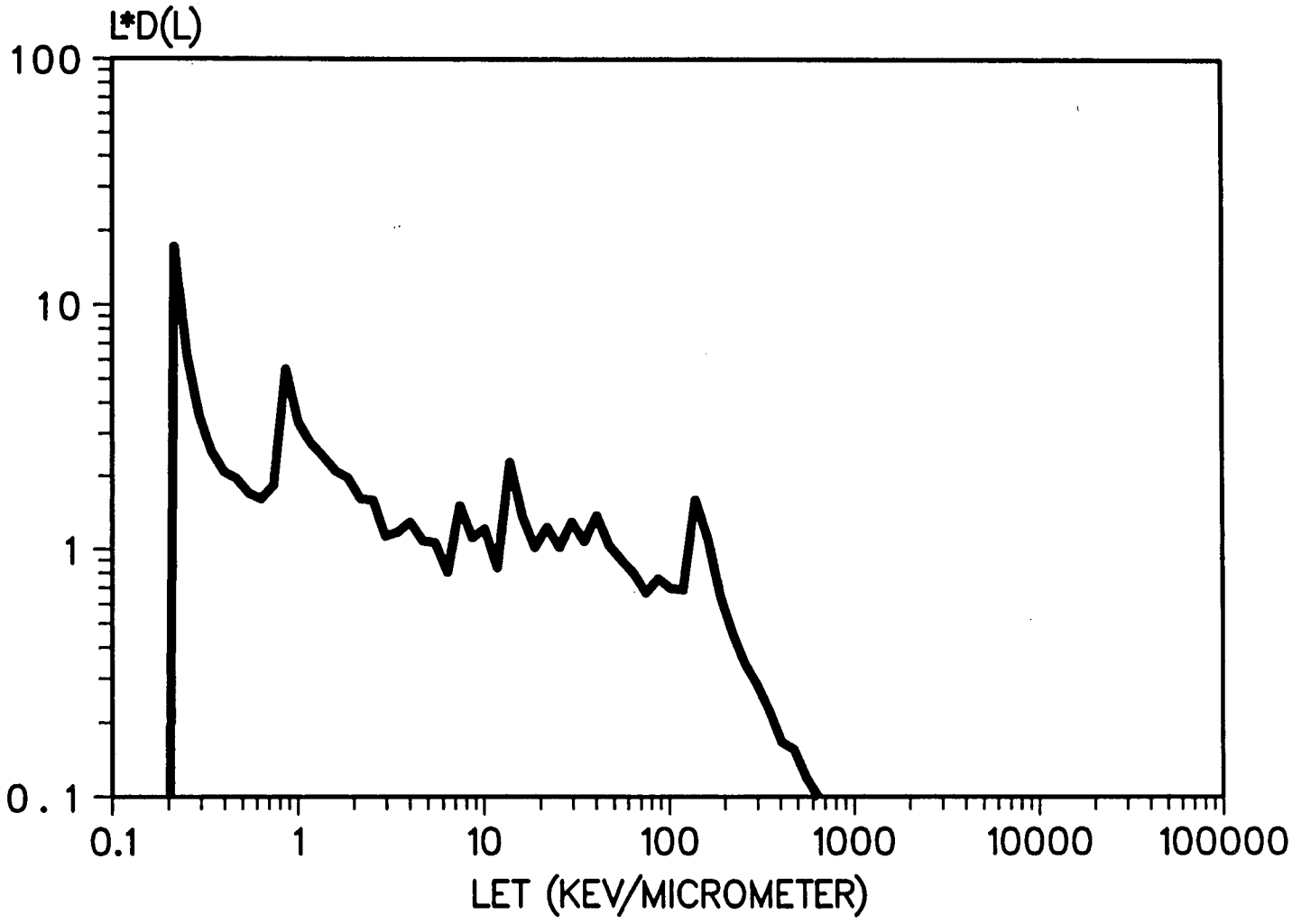
0.1
Proton
Helium
Silicon
Iron
Iron, low energy
0.1
1
10
100
1000
10000
100000
LET (KEV/MICROMETER)

Figure 1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1
10
100
1000
10000
100000
LET (KEV/MICROMETER)

Figure 2

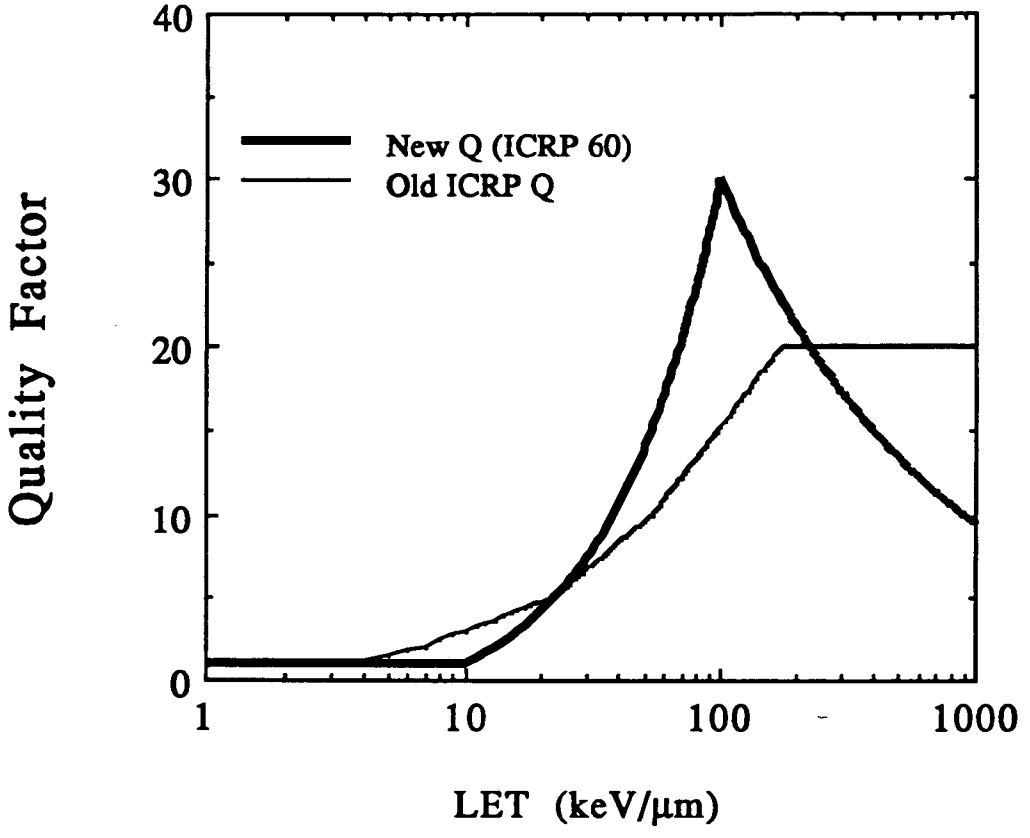

Quality Factor
40
30
20
10
0
New Q (ICRP 60)
Old ICRP Q
1
10
100
1000
LET (keV/μm)

Figure 3

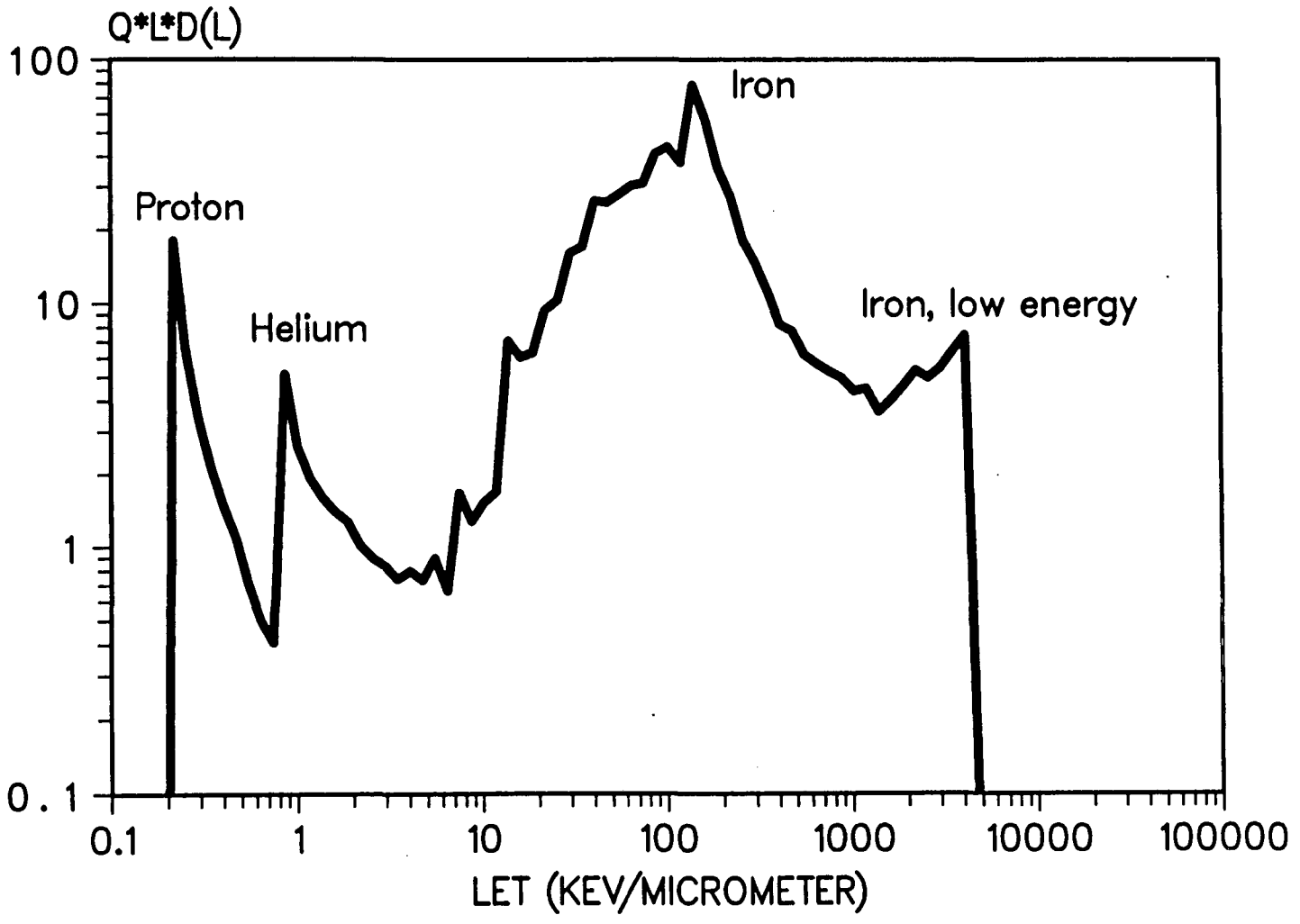

Q*L*D(L)
100
10
1
0.1
Proton
Helium
Iron
Iron, low energy
0.1
1
10
100
1000
10000
100000
LET (KEV/MICROMETER)

Figure 4

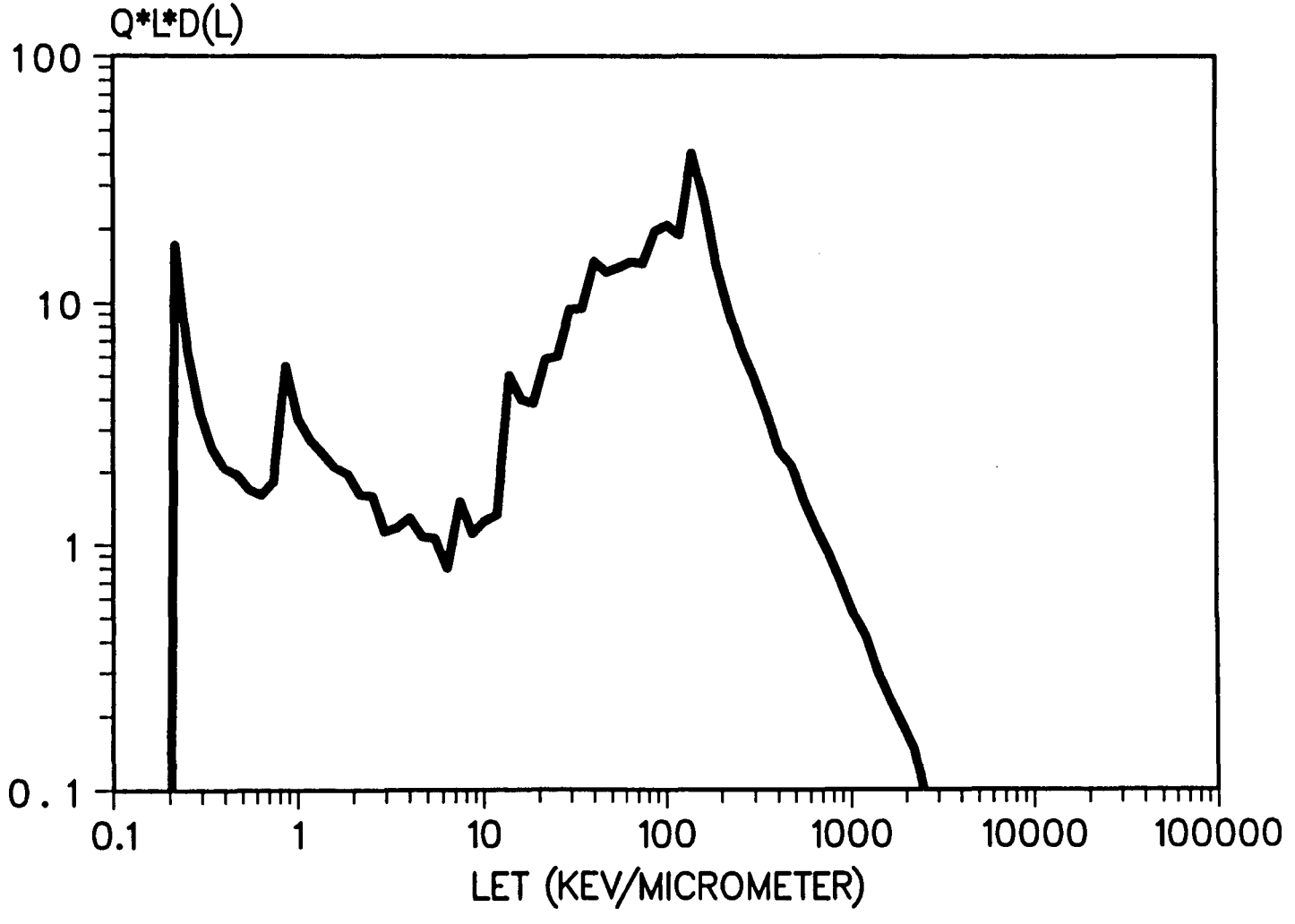
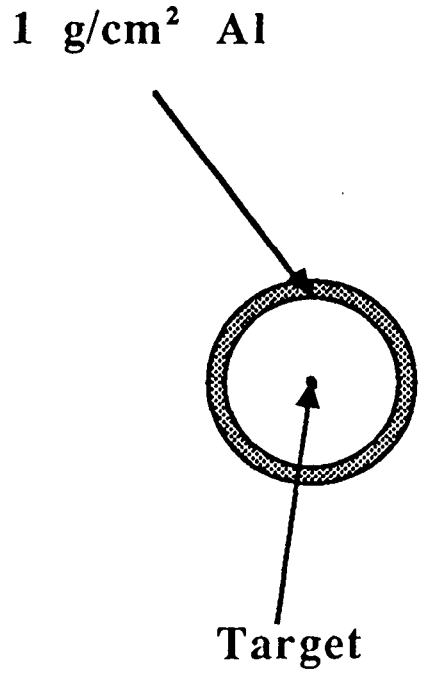

Q*L*D(L)
100
10
1
0.1
0.1
1
10
100
1000
10000
100000
LET (KEV/MICROMETER)

Figure 5

a.



b.

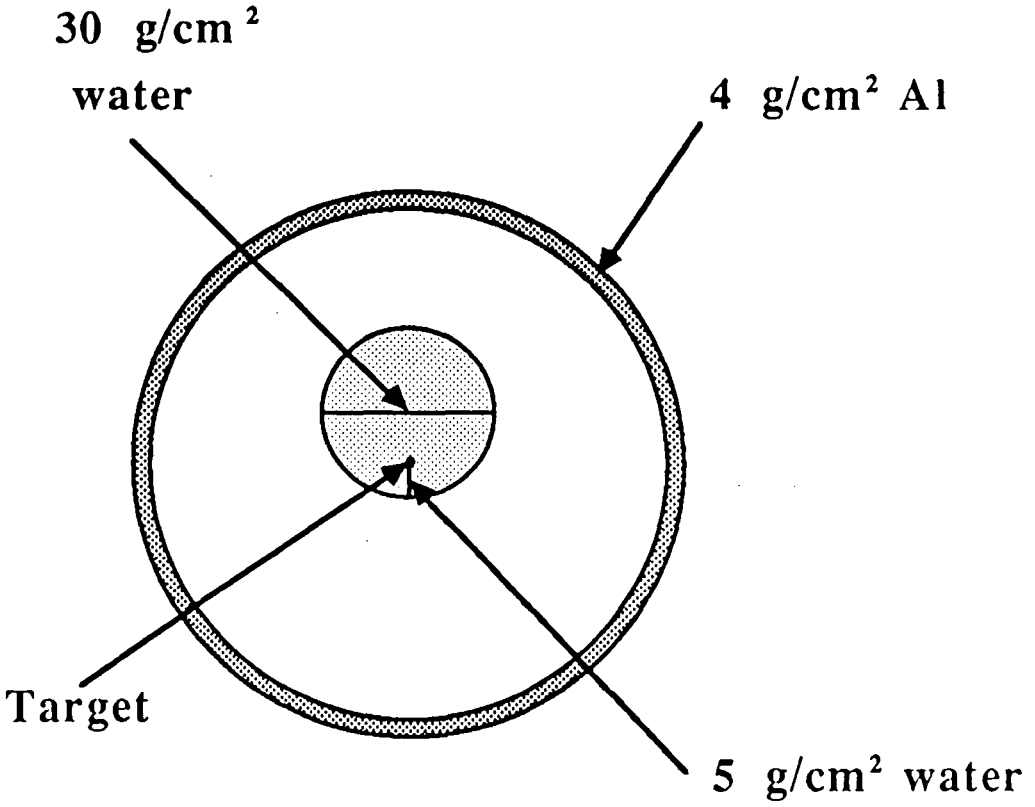


Figure 6

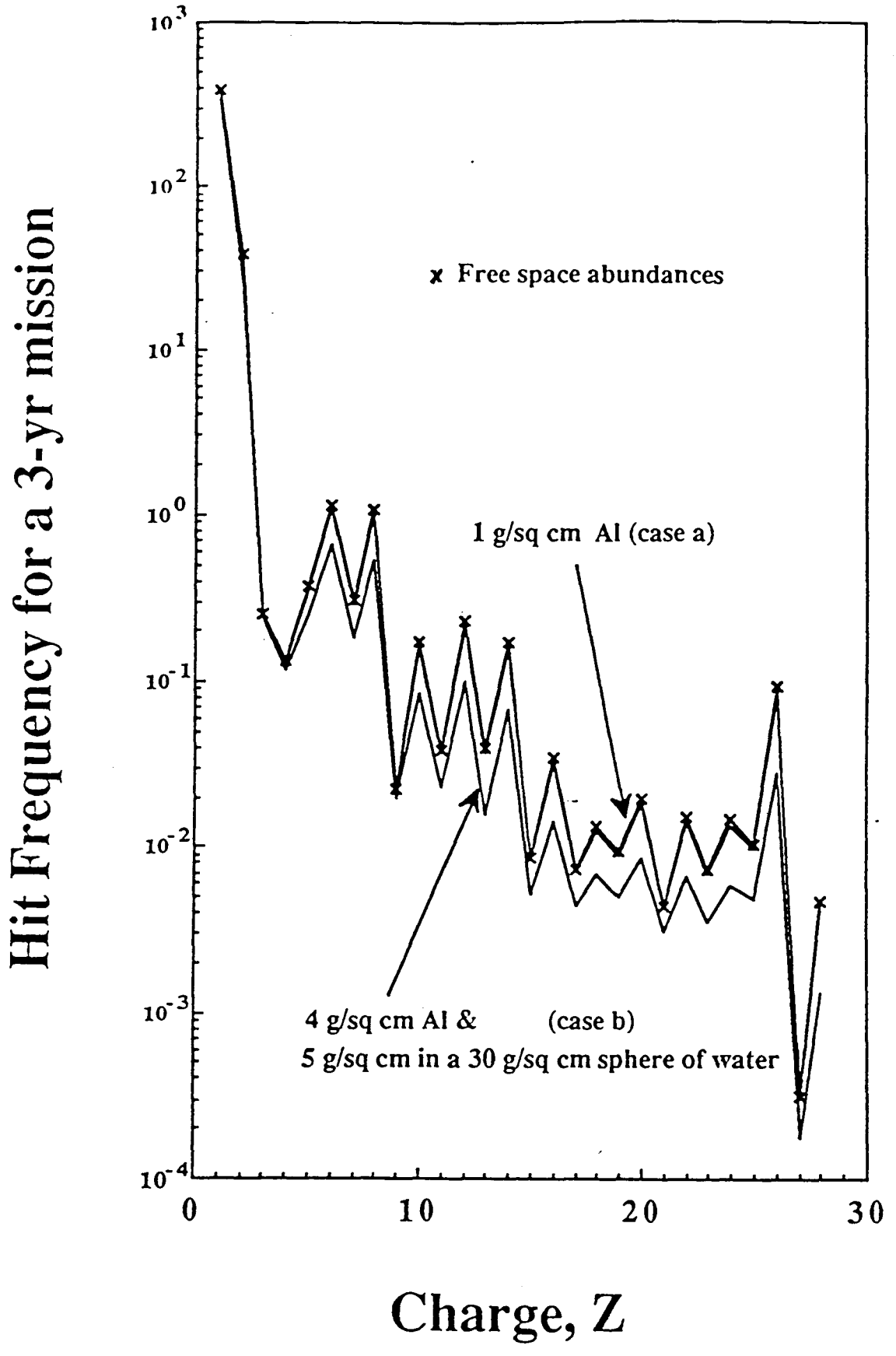

Hit Frequency for a 3-yr mission
10^3
10^2
10^1
10^0
10^-1
10^-2
10^-3
10^-4
0
10
20
30
Charge, Z
x Free space abundances
1 g/sq cm Al (case a)
4 g/sq cm Al & (case b)
5 g/sq cm in a 30 g/sq cm sphere of water

Figure 7

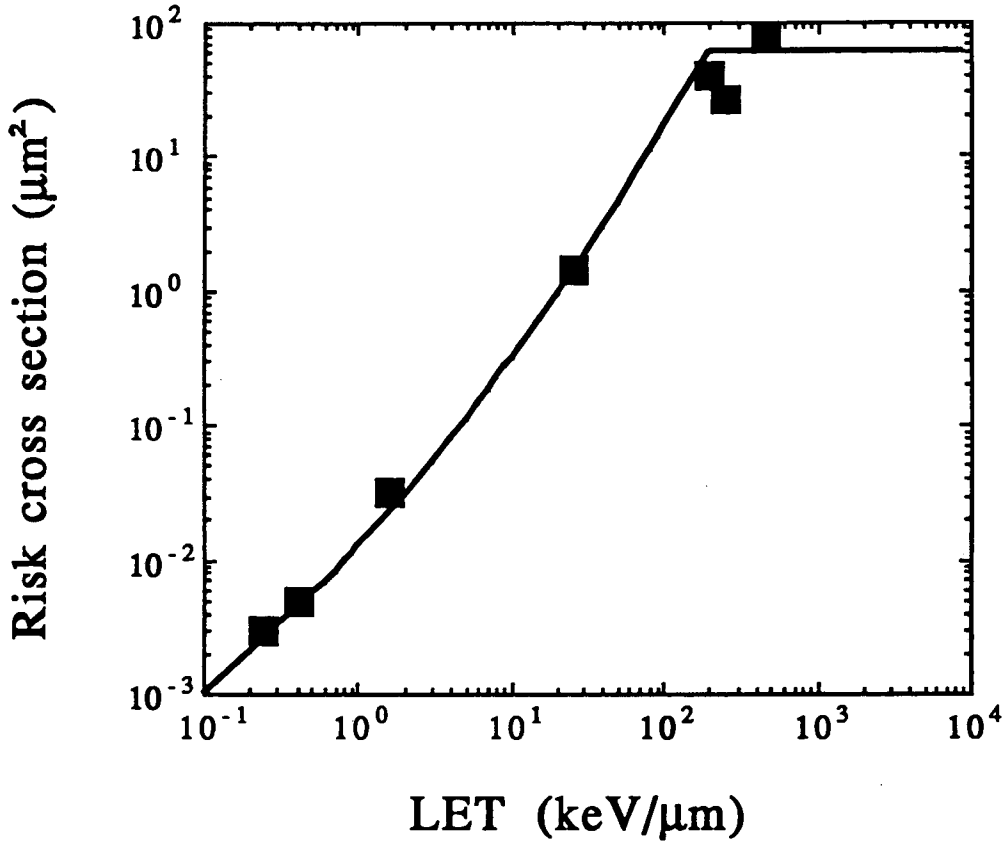

Risk cross section (μm²)
10²
10¹
10⁰
10⁻¹
10⁻²
10⁻³
10⁻¹
10⁰
10¹
10²
10³
10⁴
LET (keV/μm)

Figure 8

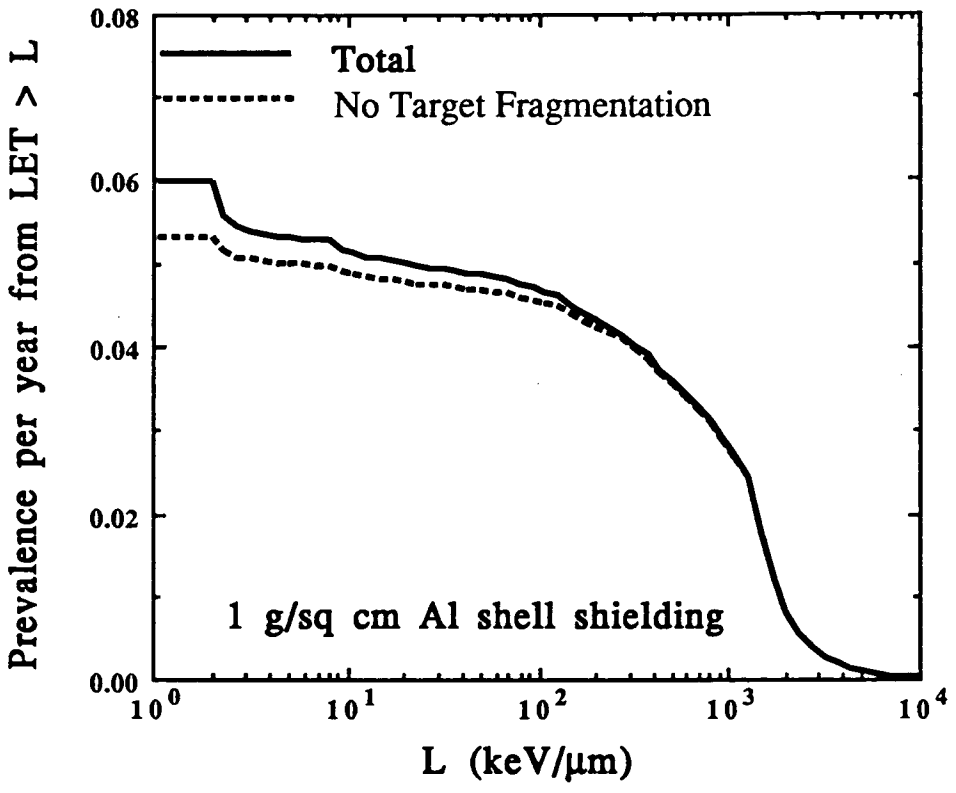

Total
No Target Fragmentation
1 g/sq cm Al shell shielding
Prevalence per year from LET > L
L (keV/μm)
0.08
0.06
0.04
0.02
0.00
10^0
10^1
10^2
10^3
10^4

Figure 9

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