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ENERGY-LOSS DISTRIBUTIONS AND FRACTIONAL CELL LETHALITY

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Stanley B. Curtis

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Donner Laboratory and Lawrence Radiation Laboratory,
University of California, Berkeley, California

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ABSTRACT

In the evaluation of the hazard from a given radiation environment, various factors other than the absorbed dose play an important role in determining the biological response. One of these is the quality of the radiation, that is, the dE/dx of the particles depositing the dose. It is convenient, especially when dealing with charged particles heavier than electrons, to display the dose at a point as a function of dE/dx. This function is called an energy-loss or dE/dx distribution. Such a representation allows an evaluation of the importance of the various dE/dx components that comprise the dose. In particular, the high dE/dx components are of interest because it has been shown that, in general, high dE/dx radiation is more effective--that is, has a higher RBE (Relative Biological Effectiveness) in producing biological damage than low dE/dx radiation. Examples are given of dE/dx distributions due to two typical solar-particle events in free space with different spectral shapes and under different shielding thicknesses. The case of a steep spectrum under thick shielding shows the proton component dominating, while the case of a flatter spectrum under thin shielding shows the helium-ion component to be slightly more important than the proton component.

A potentially fruitful way of quantifying the biological effects of a given environment is by using the inactivation cross section. This experimentally determinable quantity is equal to the probability per unit flux of a cell being inactivated, and is analogous, in this sense, to a nuclear scattering or interaction cross section. It appears to be a function of dE/dx, but does not depend on the type of heavy particle producing the dE/dx. Unfortunately, few mammalian inactivation cross sections have been experimentally determined to date. Todd has measured the inhibition of the proliferative capacity of human kidney cells in vitro and has shown that the resulting damage may be interpreted as being caused by two distinct damage mechanisms--an irreversible single-hit mechanism dominating at high dE/dx, and a reversible multi-hit mechanism dominating at low dE/dx. As an illustration, the cross sections from these experiments have been used to calculate the numbers of inactivation hits/cell for two sample radiation environments involving protons, helium ions, and heavier components: the galactic cosmic radiation in free space under 0.2 g/cm^2 water shielding and a large solar-particle event in free space. Presented in terms of the ratio of inactivation hits/cell of the heavy components to that of the protons, the results show: (1) For the galactic cosmic radiation, the very heavy components (Fe-Ni ions) cause one and a half times as much damage as protons under 0.2 g/cm^2 shielding. (2) For the solar-particle event, the helium-ion contribution is slightly less than the proton contribution, but is the same order of magnitude and remains so even at large shielding thicknesses. The heavier-component contribution is down by an order of magnitude from that of the helium ions, and drops off much more steeply with increasing shielding thickness.

The fractional number of cells inactivated or fractional cell lethality (FCL) can be calculated if the numbers of lethal hits/cell are known from both the reversible and irreversible damage mechanisms. It turns out that irreversible damage dominates for the solar events chosen for illustration. FCL values were calculated for two points inside the body at the waist of a seated astronaut for several large solar-particle events of the last solar cycle, taking into account the body self-shielding. The results show that up to 7% of the cells would have been inactivated 4 cm inside the body at the waist behind 1 g/cm^2 of vehicular shielding in the largest event.

Such calculations as this may help in the future for the evaluation of the hazard from mixed-heavy-particle radiation environments when inactivation cross sections or other suitable "malfunction" cross sections are available for more critical and perhaps irreplaceable cells in the body and when accumulated damage over a long period, such as for extended space flight, is of importance.

INTRODUCTION

The problem of evaluating the hazard from a given radiation environment can be very complex. In the first place, the differing interactions of the various types of radiation make the analysis difficult. Secondly, the shielding of the human body itself provides an added complication in the determination of the particle flux to reach a point deep within the body. Finally, the ultimate biological effect depends not only on the amount of energy deposited by the particles per unit volume (i.e., the absorbed dose) but also on such quantities as the dose rate and the ionizing power (dE/dx) of the particles. We consider here only one aspect of the problem: the dependence of the biological effect on the dE/dx of the particles depositing the dose. All material presented in this paper except that on the galactic cosmic rays has been published elsewhere.[1,2]

It has been clearly demonstrated[3,4] that the relative biological effectiveness (RBE) of radiation from charged particles in mammalian systems depends on the rate of energy loss of the particles, that is, on their dE/dx. Other parameters (such as the amount of energy deposited in a <u>finite</u> sensitive volume) may ultimately be used to describe the quality of the radiation from the biological standpoint;[5] but until more is known about the effects of microscopic distributions of energy in specific biological systems of interest, it appears reasonable to continue to use dE/dx as a rough approximation in all systems.

ENERGY-LOSS DISTRIBUTIONS

It is convenient, therefore, to express the absorbed dose at a point in terms of an energy-loss distribution or spectrum. We define this function in the following way. First, we recall the expression for the differential dose element:

$$dD = (dJ/dE')\,\epsilon'\,dE', \tag{1}$$

where dJ/dE' is the differential energy spectrum; i.e., the number of particles per unit area with energies between E' and $(E' + dE')$ at the point of dose computation, and $\epsilon' = dE'/dx$ is the rate of energy loss of a particle with energy E'.

The integral of the above expression is the absorbed dose at the point:

$$\text{dose (in rads)} = 1.6\times10^{-8}\int_0^\infty \frac{dJ}{dE'}\,\epsilon'\,dE',$$

where dJ/dE' is expressed in number of particles per cm^2-MeV, ϵ' is in MeV cm^2/g, and E' is in MeV. We assume here that the energy being lost is absorbed "locally" and so we restrict ourselves to incident charged particles heavier than electrons. In addition, we are neglecting in this first approximation the fact that, in some cases, high-energy secondary electrons or delta rays can deposit energy some distance from the track core.

We now define a function called the energy loss or dE/dx distribution function, $F(\epsilon')$, such that

$$F(\epsilon')\,d(\log \epsilon') = dD. \tag{2}$$

We use the differential of the logarithm in the definition simply for convenience, since we shall see that it is convenient to plot the distribution as a linear function of the log of ϵ'.

Equating (1) and (2) and solving for $F(\epsilon')$, we obtain

$$F(\epsilon') = 2.303(dJ/dE')\,\epsilon'^{2}/(d\epsilon'/dE') \tag{3}$$

for the energy-loss distribution function. It is seen from Eq. (3) that $F(\epsilon')$ diverges whenever $d\epsilon'/dE'$ vanishes, i.e. at the maximum and minimum of the dE/dx vs E curve. These divergences show up in the distributions as "spikes".

From the definition of $F(\epsilon')$, in units of MeV/g, we have

$$\text{dose (in rads)} = 1.6\times 10^{-8} \int_{-\infty}^{\infty} F(\epsilon')d(\log \epsilon') . \tag{4}$$

If $F(\epsilon')$ is plotted graphically as a function of ϵ' on a logarithmic scale, equal distances along the abscissas have equal weights, and the importance of different dE/dx contributions can readily be evaluated. The spikes or points of divergence give no trouble in the dose integral because the areas under them contribute a small part of the total dose in a typical exposure situation.

ENERGY-LOSS DISTRIBUTION FROM A SOLAR-PARTICLE EVENT

The proton, helium- and heavier-ion fluxes comprising a solar-particle event provide a good example of a situation where energy-loss distributions are helpful in indicating the relative importance of various dE/dx contributions. Figure 1 shows energy-loss distributions resulting from two different energy spectra behind two different shielding thicknesses. Figure 1a gives the case for thick shielding, 5 g/cm^2 water equivalent and a rather steep particle spectrum. Figure 1b gives the case for thinner shielding, 1 g/cm^2 water equivalent, and a flatter spectral shape. It was assumed that the integral particle spectra were exponential in rigidity with the form

$$J_j(\geq P) = J_{0j} \exp(-P/P_{0j}),$$

where P is the rigidity or momentum per unit charge, $J_j(\geq P)$ is the number of particles per cm^2 of the jth particle type with rigidity equal to or greater than P, and J_{0j} and P_{0j} are constants for a given event. In Fig. 1a, we have $P_{0p} = P_{0\alpha} = P_{0M} = 80$ MV, which exemplifies a rather steep spectrum, and in Figure 1b, $P_{0p} = P_{0\alpha} = P_{0M} = 180$ MV, which exemplifies a flatter spectrum. Here p, α, and M respectively stand for protons, helium ions, and ions of charge Z between 6 and 9, called M particles. In these calculations, the latter were assumed to have $Z = 8$. The J_0 values for protons and helium-ions were assumed to be sixty times that for the M particles. All these assumptions are reasonable from the limited data available on the spectra and composition of the larger solar-particle events that occurred in cycle 19.[6,7,8] In the figure, the areas under the curves have been normalized

to unity by dividing by the dose.

The difference in relative contribution to the energy-loss distributions of the proton and helium-ion components arises about equally from the change in spectral shape and the change in shielding thickness. This example illustrates the relative importance of high- and low-dE/dx particles in contributing to the dose under differing environmental conditions for two typical large solar-particle events.

BIOLOGICAL CONSIDERATIONS

In the evaluation of a hazard from a specific radiation environment, the radiosensitivity of the biological organism involved must be considered. As indicated above, one parameter upon which this sensitivity depends is the dE/dx of the particles depositing the energy. The International Commission on Radiological Protection has quantified this concept; the value of this quantity is called the quality factor (QF).[9] In addition, the following dependence of QF on dE/dx has been suggested:

$$QF(\epsilon) \doteq 0.8 + 1.6\times10^{-2}\,\epsilon$$

for $QF < 20.0$ and ϵ in MeV cm^2/g. The biologically important dose or dose equivalent in rem may be calculated as

$$\text{dose equivalent} = 1.6\times10^{-8}\int_{-\infty}^{\infty} F(\epsilon')\,QF(\epsilon')\,d(\log\epsilon')\,.$$

Here the QF acts as a weighting factor that gives more weight to the higher dE/dx portion of the distribution.

The concept of QF, however, is artificial in the sense that its dependence on ϵ has simply been agreed upon as an upper-limit extrapolation to low dose rates and low doses of RBE's from radiobiological data. It would be of interest to use a more physically meaningful quantity

whose dependence on dE/dx is perhaps similar but whose interpretation is that of the probability of a biologically significant interaction taking place. Such a quantity should be independent of dose rate, total dose, and all other physical characteristics of the radiation environment, and should have functional dependence only on the dE/dx of the radiation. This quantity could, of course, depend on biologically important parameters, e.g., on the availability of oxygen.

INACTIVATION CROSS SECTIONS

The inactivation cross section measured by Todd is an example of such a quantity.[10,11] In experiments with human kidney cells, Todd has measured the inactivation cross sections for inhibition of the cell's proliferative capacity *in vitro* as a function of dE/dx with various heavy ions at the HILAC of the Lawrence Radiation Laboratory. Evidence has been found for two types of radiation damage existing in the kidney cells studied. One type is irreparable; the other is reparable. Dependence of the inactivation cross section on dE/dx is different for the two types of damage. These cross sections are analogous to nuclear-scattering cross sections in that they are the probability per unit flux of the proliferative capacity of the cell being destroyed. The experimental cross sections are shown as a function of dE/dx in Fig. 2.[11] The cross section due to irreparable damage is labeled σ_1, and that due to reparable damage is labeled σ_2. Although both cross sections and therefore inactivation probabilities rise with increasing dE/dx, it should be noted that the reparable cross section dominates at low dE/dx, and the irreparable cross section at high dE/dx. This is consistent with the experimental fact that high dE/dx radiation in general

produces more irreparable damage, while the damage caused by low dE/dx radiation is more readily reparable.

INACTIVATION HITS PER SITE

By using inactivation cross sections and the dE/dx distribution as described above, the number of lethal or inactivating hits per site can be calculated. The expression for the number of such hits, $J(x)$, at a depth x is

$$J(x) = \int_0^\infty \frac{dJ}{dE'} \sigma(E') \, dE' , \tag{5}$$

where dJ/dE' is the differential energy spectrum at a depth x, and $\sigma(E')$ is the inactivation cross section. This may be rewritten in terms of the energy-loss spectrum $F(\epsilon')$ of Eq. (3) as

$$J(x) = \int_{-\infty}^{\infty} F(\epsilon') \frac{\sigma(\epsilon')}{\epsilon'} \, d(\log \epsilon') . \tag{6}$$

The integrand of Eq. (6) is just the dose integrand of Eq. (4) multiplied by a factor $\sigma(\epsilon')/\epsilon'$. This factor is analogous to a QF or RBE, but is independent of dose and dose rate, and depends only on the probability for inactivation, and on the dE/dx of the particle involved.

GALACTIC COSMIC-RAY HAZARD TO THE SKIN

As another example in the use of these concepts, the energy loss distribution for the galactic cosmic radiation under 0.2 gm/cm^2 of water-equivalent shielding is shown in Fig. 3. Only the most important contributions are shown here. When more than one nuclear species is present under a single designation, such as the M-particle group ($6 \leq Z \leq 9$), a representative Z and A have been chosen for that group. Recent experimental data have been used[12-16] and were extrapolated to lower energies where necessary. Above the distribution on the graph, the quantity $\sigma_1(\epsilon')/\epsilon'$ is plotted on an arbitrary scale. This indicates the weighting factor chosen in the computation of lethal or inactivation hits/site. The magnitudes of the lethal hits per site in this example are not meaningful, since the cross sections used were those measured for kidney cells in vitro. Unfortunately, inactivation cross sections for cells of the skin are not available at present. These would be more appropriate in a situation in space, for instance, where an astronaut in a space suit was engaging in extra-vehicular activity. Even in this situation, there would be shielding present—his own body and the nearby spacecraft—which would modify the result by decreasing the contributions from the high dE/dx particles, since they would not be able to penetrate the nearby material without fragmenting and producing secondary particles. However, the ratios, of the values of lethal hits per site by a heavy component to that by protons gives us a feeling for the relative importance of the various components in causing skin damage. All that is required for the validity of this analysis is that the shape of the inactivation cross section for skin cells be similar to that for kidney cells. Inactivation

cross sections have been found to have similar shapes in many different kinds of biological test objects even as far removed from human cells as haploid yeast cells and T1 bacteriophage.[11] Table I presents the ratios of irreparable lethal hits (i.e. from the σ_1 damage mechanism) of the various heavy components compared to those of protons. Only σ_1 damage was assumed, since σ_2 damage is reparable and presumably will be repaired at the low doses involved. It is seen that all components make roughly equal contributions, with the iron-nickel ion group making the largest contribution at these small depths. Deeper within the body, secondary production becomes important, and the calculation is not as straightforward. In principle, however, the calculation can be made for any position where the differential energy spectra of the various particles are known or can be calculated and the probability or cross section for a specific kind of biological damage or functional degradation is known as a function of dE/dx.

THE FRACTIONAL CELL LETHALITY CONCEPT

We now define the fractional cell lethality (FCL) as the fractional number of cells or sites killed or inactivated by the radiation.[17] If we define $\phi(x)$ as the probability that at a depth, x, a site is still active, or in other words the fractional number of sites still active, then the change in ϕ in a time dt will be given by

$$-d\phi = J(x,t)\ \phi(x)\, dt,$$

where $J(x,t)$ is the number of inactivation hits per unit time at depth x. Integrating we obtain the familiar exponential dependence

$$\phi(x) = \exp[\,-J(x)\,]\ ,$$

where $J(x)$ is the time-integrated number of inactivation hits per site from Eq. (5) or (6).

For a single-hit damage mechanism, the FCL is simply

$$FCL(x) = 1 - \phi(x) = 1-\exp[-J(x)] .$$

For a combination of a single and a multi-hit mechanism as proposed by Todd, the expression becomes

$$FCL(x) = 1-\exp[-J_{\sigma_1}(x)] \left\{ 1 - [1-\exp(-J_{\sigma_2}[x])]^n \right\},$$

where $J_{\sigma_1}(x) = \sum_j J_j(x)$ for the single-hit mechanism, and $J_{\sigma_2}(x)$ is similarly defined for the multi-hit mechanism. The summation is over the different types of particles present in the spectrum. The exponent, n, may be interpreted as the number of hits necessary to inactivate the site by the multi-hit mechanism. Its value is not important at low doses, since damage from the multi-hit mechanism is negligible.

FRACTIONAL CELL LETHALITY FROM SOLAR-PARTICLE EVENTS

As a final example, we calculate the FCL to an astronaut's kidney from several of the large events that occurred in solar cycle 19. The physical parameters for the various events are given in Table II and come from the work of Webber.[18] The contributions to the lethal hits per site from the heavier components relative to that from the protons are shown in Fig. 4 as a function of equivalent water shielding for the 12 November 1960 event. Also shown are the relative rad doses from each component for comparison. It is seen that the proton contribution dominates the He-ion contribution, although both are of the same order of magnitude and remain so, even at larger shielding thicknesses. The

M particle contribution is down by an order of magnitude from the He-ion contribution and drops off more steeply with increasing thickness.

Calculations of FCL have been made at two points within the body of a seated astronaut for the three solar-particle events given in Table II. These calculations included the self-shielding provided by the body. In this case, we write the number of lethal hits per site at a body point and behind X g/cm^2 of vehicular shielding for the jth particle type as

$$J_j(X, \text{ body point}) = \sum_i f(x_i) J_j(X + x_i) ,$$

where $f(x_i)$ is the fractional solid angle seen from the body point through a body thickness, x_i. These factors, which weight the J_j according to the distribution of body shielding around the point, have been calculated for various points within a seated 75-percentile man.[19] The two points chosen here were 4 and 6 cm into the body at the waist (right side, 25 cm up from the seat level, on the mid-sagittal line). The results are shown in Fig. 5. It is seen that up to 7% of the kidney cells 4 cm inside the waist would have been inactivated in the 12 November 1960 event under 1 g/cm^2 vehicular shielding.

This calculation is just an example of how available cross-section data might be used to determine the biological damage and thus to evaluate the radiation hazard. Certainly, other cells are more critical to the body than kidney cells. It is hoped that inactivation cross sections or perhaps some other measurable quantity such as a malfunction cross section will be measured in the future for other more critical and perhaps less easily replaceable body cells. In addition, a way must be

found to relate the calculated FCL to the functional degradation of the organ being considered. When such data become available, the problem of relating biological effects and functional degradation to particle energy can be more easily handled. It is felt that the FCL concept will be of some help in providing a quantitative measure of the hazard in situations where highly ionizing radiation and, therefore, irreparable processes play an important role.

Table I. Ratio of lethal hits by heavy particles to lethal hits by protons in the galactic cosmic radiation under 0.2 g/cm^2 water shielding.

Particle	Z	Ratio
protons	1	1.00
He ions	2	0.72
M ions	6 to 9	0.78
LH ions	10 to 14	0.89
Fe-Ni ions	26 to 28	1.5

Table II. Particle spectral characteristics of three large solar-particle events of cycle 19

Date	J_{0p}	P_{0p} (MV)	$J_{0\alpha}$	$P_{0\alpha}$ (MV)	J_{0M}	P_{0M} (MV)
7/14/59	2.6×10^{10}	80	1.99×10^{10}	87	3.32×10^{8}	87
11/12/60	8.9×10^{9}	124	1.94×10^{9}	172	3.23×10^{7}	172
11/15/60	5.9×10^{9}	114	1.92×10^{9}	156	3.2×10^{7}	156

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FIGURE LEGENDS

Fig. 1. Two examples of normalized energy-loss distributions under water shielding. (a) Shielding thickness of 5 g/cm^2 for a solar-particle event with $P_{0p} = P_{0\alpha} = P_{0M} = 80$ MV; (b) shielding thickness of 1 g/cm^2 for a solar-particle event with $P_{0p} = P_{0\alpha} = P_{0M} = 180$ MV. In both cases, J_{0p} and $J_{0\alpha}$ equal 60 J_{0M}.

Fig. 2. Inactivation cross sections for irreversible (σ_1) and reversible (σ_2) damage to the proliferative capacity of human kidney cells *in vitro* as a function of dE/dx, as measured by Todd.[11]

Fig. 3. The energy-loss distribution from galactic cosmic rays under 0.2 g/cm^2 water shielding. The upper curve gives the weighting factor $\sigma_1(\epsilon')/\epsilon'$ on an arbitrary scale for the lethal hits/site calculation.

Fig. 4. Contribution of the He ions (α) and M particles (M) to the lethal hits/site and to the rad dose relative to protons (p) as a function of thickness for the solar-particle event of November 12, 1960.

Fig. 5. Fractional cell lethalities (FCL) at two body points in a seated astronaut as a function of vehicular water-equivalent shielding from three solar-particle events.

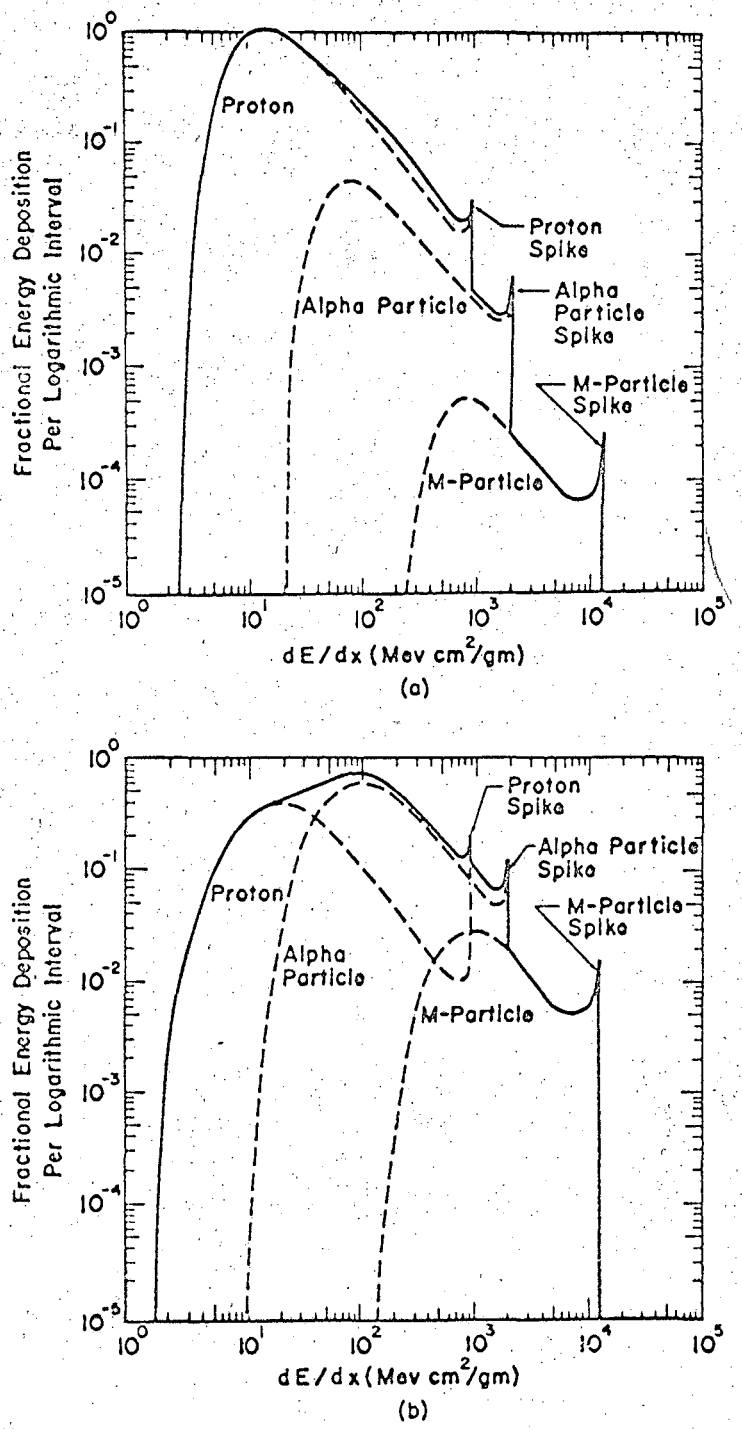

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Fig. 1

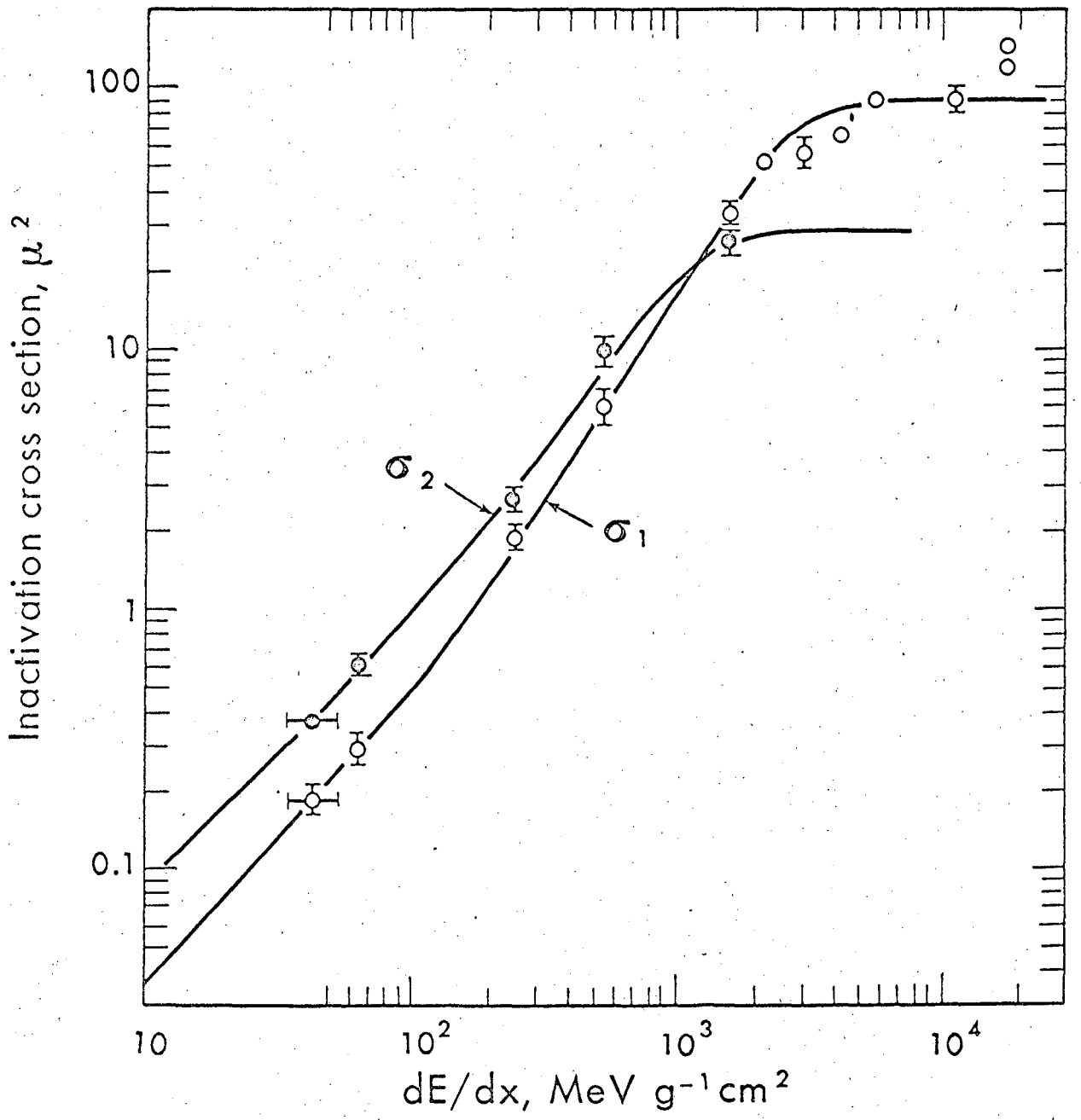

Fig. 2

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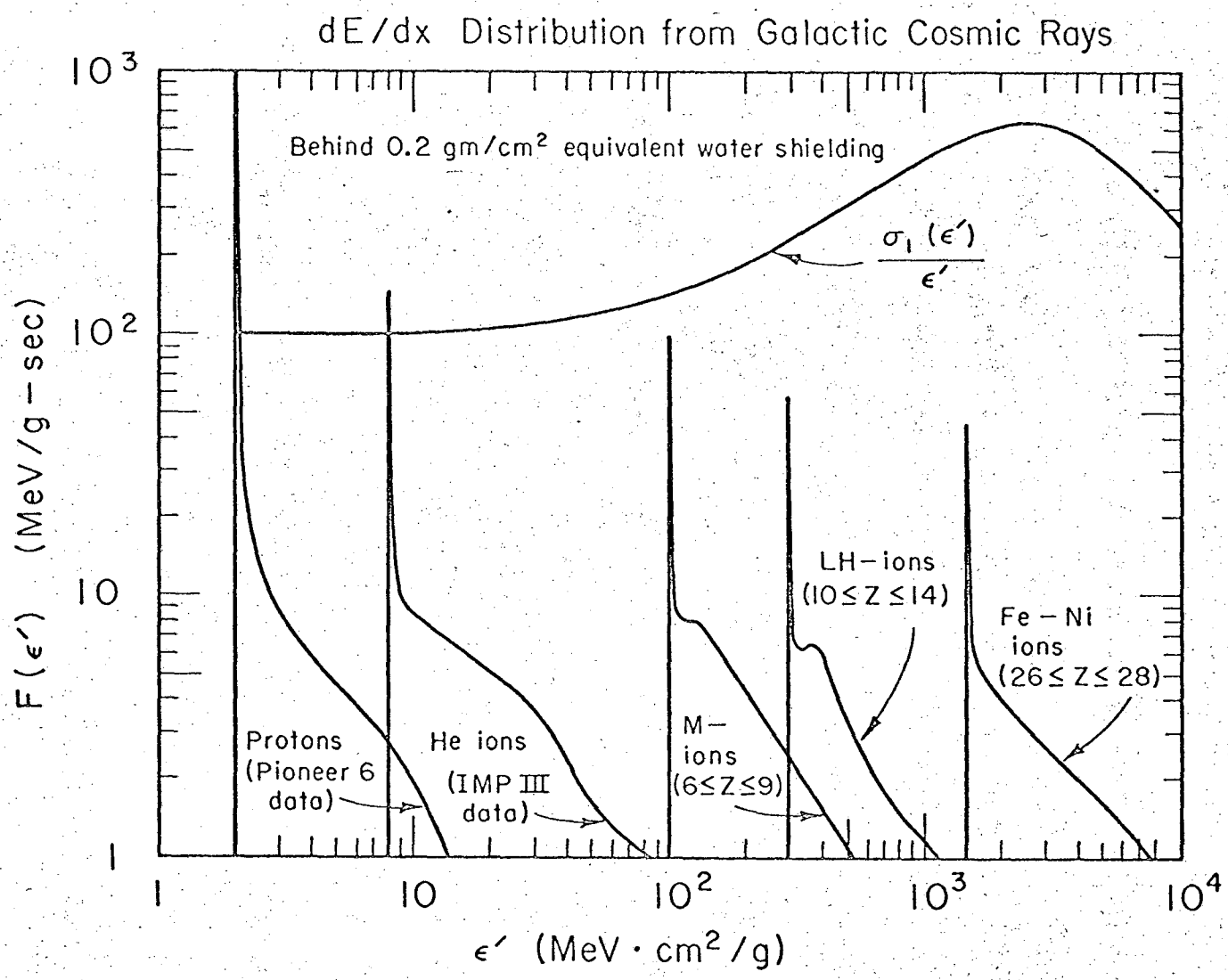

Fig. 3

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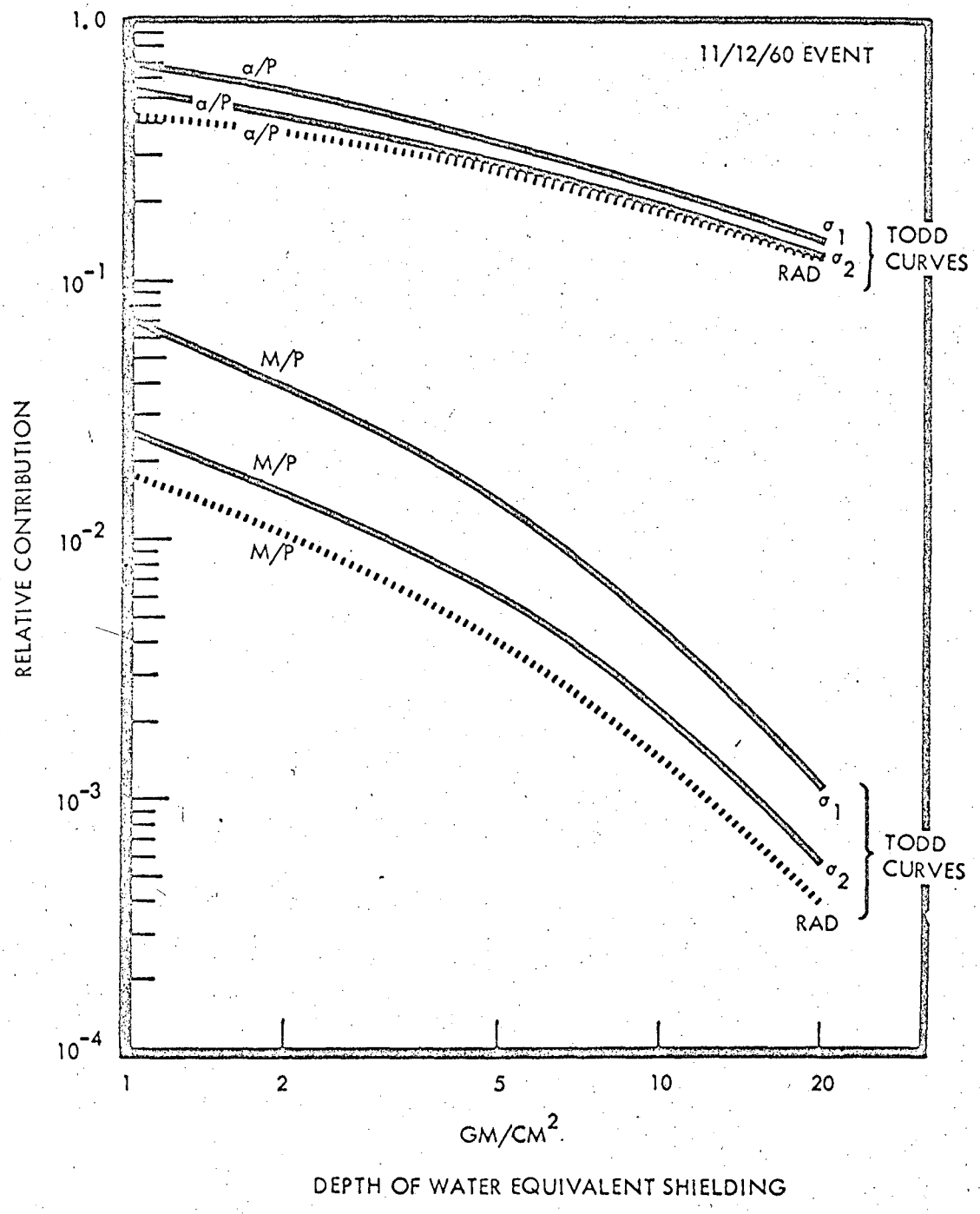

Fig. 4

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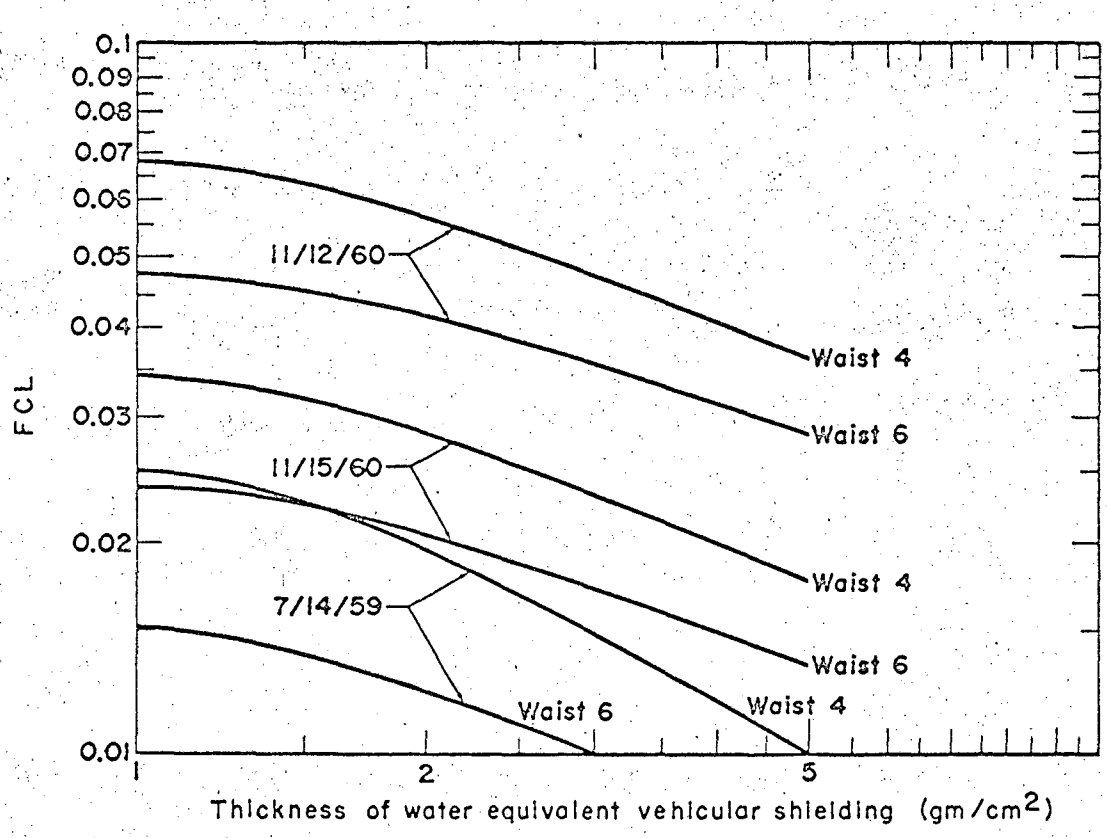

Fig. 5

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