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Radiation Risk Predictions for Space Station *Freedom* Orbits

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Abstract

Risk-assessment calculations are presented for the preliminary proposed solar minimum and solar maximum orbits for Space Station Freedom (SSF). Integral linear energy transfer (LET) fluence spectra are calculated for the trapped-proton and galactic cosmic ray (GCR) environments. Organ-dose calculations are discussed using the Computerized Anatomical Man model. The cellular track model of Katz is applied to calculate cell survival, transformation, and mutation rates for various aluminum shields. Comparisons between relative biological effectiveness (RBE) and quality factors (QF) for SSF orbits are made, and fluence-dependent effects are discussed.

Introduction

An increased concern for the possible radiation risks to astronauts is expected for Space Station Freedom (SSF) because of longer crew stays (45 to 180 days) and new information received in recent years of the harmful effects of low levels of ionizing radiation (ref. 1). The most important radiological concern for space flight continues to be late-developing effects, especially cancer (ref. 2), and our inadequate knowledge of any possible increased risk from high-energy heavy ion (HZE) particles. The reassessment of cancer risks from low levels of highly ionizing radiation is expected to lead to new recommendations on exposure limits and quality factors (QF). (See ref. 1.)

Space Station Freedom will be stationed in low Earth orbit (LEO) with the radiation fields encountered similar to those seen in recent flights of the Space Shuttle. The orbital parameters for SSF are a 28.5° circular orbit with a varying altitude to obtain constant atmospheric drag throughout the 11-year solar cycle. Preliminary plans are to utilize a maximum altitude of 270 nautical miles (n.mi.) at solar maximum and a minimum altitude of 200 n.mi. near solar minimum. The larger altitude will avoid the expanding upper atmosphere as solar activity increases. Radiation levels decrease with increasing solar activity so that only modest increases occur in dose rate at the high altitudes during solar maximum. The major radiation concerns are from the trapped protons in the South Atlantic Anomaly (SAA) and the background galactic cosmic rays (GCR). Exposures to the trapped-electron belts are expected to be small, and only an anomalously large solar proton event would contribute to exposures because of the Earth's magnetic shielding and only if there are concurrent magnetic depressions (ref. 3). The flux of trapped protons in the SAA varies within the solar cycle with a maximum dose for fixed altitude at solar minimum. Space Station Freedom will orbit the Earth with a fixed orientation, which may lead to important anisotropic

effects from trapped-proton exposures (ref. 4). The variation in GCR exposure over the solar cycle in LEO is less important because only the highest energy nuclei, assumed to receive little solar modulation, are transmitted through the Earth's magnetic field. From the Shuttle experience (ref. 5), we expect SSF exposures on the order of 50 mrad/day, varying with the amount and type of spacecraft shielding.

Traditionally, risk assessment for space flight is based on the quality factor (QF) which is solely dependent on the linear energy transfer (LET). The QF multiplies the absorbed dose (defining the dose equivalent) for the purpose of defining an equivalent scale for measuring the effectiveness of various radiation fields in producing deleterious effects in biological systems. The effectiveness of a radiation may vary from one tissue to the next, including a variation among biological responses, such that the QF represents a measure of an average effectiveness of a field. For high LET radiations, no information exists on human response, such as cancer or genetic effects, and QF values are based on comparisons with low LET radiations in radiological experiments (ref. 1). The relative biological effectiveness (RBE), defined as the ratio of an absorbed dose of a radiation in question to a reference radiation for an equivalent response, is used to make this comparison in laboratory experiments with cultured cells or animals. Experiments (refs. 6–10) find RBE values varying with ion velocity, charge, fluence level, and the biological end point such that the use of a single parameter, such as LET, is seen to represent an oversimplification for specifying the quality of a field.

For space missions, the fluence dependence found in RBE measurements for HZE particles is of particular concern. A fluence-dependent RBE is also expected for high-energy protons in the limit of small doses because of the dominance of nuclear reaction products on the proton effect in the regime (refs. 10 and 11). Radiological experiments are usually

performed at high dose and high dose rates where many ions pass through a single cell in a short period of time. In space we have protracted exposures with a relatively small accumulated dose. The expected flux rates for SSF are at about 10^6 particles/cm²/day, which is a small number if we consider that there are about 10^9 cells/cm³ of tissue (10^6 /cm² per monolayer resulting in about one hit per cell per day). Sublethal damage through intertrack effects should not contribute to SSF exposures, and intratrack effects are the major concern. Single tracks of HZE particles may have infinite RBE because of unique biological damage not possible by small doses of low LET radiation (ref. 12), and the RBE or QF may have no relevance for space missions.

Biological damage at low fluence is described by the action cross section that simply represents the probability per particle that an ion will produce the particular biological response of interest. In the absence of sublethal damage, the level of biological response is then an exponential function of the product of the fluence and the cross section. The track model of Katz (refs. 12–15), through parameterizations of the cross section in terms of ion charge and velocity, has been successful in describing the damage levels as well as the RBE in extensive comparisons to experiments with high-energy protons and HZE particles. Cellular response parameters for biological end points, obtained from track theory, combined with an accurate determination of the fluence spectra at positions of the cells at risk can then be used to assess the level of biological damage expected on space missions. Of particular concern is the formation of neoplasms and mutations that are thought to be early processes in the formation of cancers. The relationship between such early responses and cancer induction in humans would represent the most vital piece of information for risk assessment. Dose-rate effects as related to repair mechanisms and cell cycle may become important modifying factors for the low-exposure levels in space, but, unfortunately, they are not understood at this time.

In this paper we present risk-assessment calculations for SSF orbits. The differential flux of particles from the SAA and GCR sources, behind aluminum and tissue shielding, is evaluated for the SSF orbits using the charged-particle transport codes BRYNTRN (ref. 16) and HZETRN (ref. 17), respectively, where the effects of nuclear fragmentation of the GCR nuclei and target nuclei in the shielding are evaluated. Anisotropic exposures from trapped protons are assumed to be averaged out by random astronaut motion over a long mission, but they should be considered in future work. The absorbed dose and

dose equivalent are considered for the skin, eye lens, and blood-forming organs (BFO) using the Computerized Anatomical Man (CAM) model to represent astronaut's self-shielding (ref. 18). The expected risk associated with a given dose equivalent can then be determined by following NCRP Rep. No. 98 (ref. 2). We also apply the cellular track model of Katz to the proposed SSF orbits for biological end points of cell death (loss of cell reproductive capability), confluent transformation, and mutations of mammalian cell cultures. Estimates of production rates for stochastic effects will be helpful if their relationship to tumor induction is realized, and they may be more meaningful for low levels of high LET exposures where the dose-equivalent concept may not apply.

Radiation Transport and Particle Fluence Spectra

The SAA protons and GCR particles are transported through spacecraft and tissue shielding using the transport codes BRYNTRN (ref. 16) and HZETRN (ref. 17), respectively. The transport equation for high-energy nuclei is written in the straight-ahead approximation as (ref. 19)

$$\left[\frac{\partial}{\partial x} - \frac{\partial}{\partial E} \tilde{S}_j(E) + \Sigma_j(E) \right] \Phi_j(x, E) = \sum_{k>j} \Sigma_{jk}(E) \Phi_k(x, E) \quad (1)$$

where Φ_j is the flux of ions of type j with atomic mass A_j and charge Z_j at x moving along the x -axis with energy E (given in units of MeV/amu), \tilde{S}_j is the change in E per unit distance, Σ_j is the macroscopic nuclear absorption cross section, and Σ_{jk} is the macroscopic fragmentation cross section for the production of ion j from ion k . The target-fragment fields are written in terms of the production collision density as

$$\Phi_\alpha(x, E_{\alpha j} : E_j) = \frac{1}{S_\alpha(E_\alpha)} \int_{E_\alpha}^{\infty} \frac{d\Sigma_{\alpha\beta}(E')}{dE'} \times \Phi_j(x, E_j) dE' \quad (2)$$

where the subscript α labels the target-fragment type, S_α denotes the stopping power or LET, and E_α and E_j are particle energies given in units of MeV. The transport equation is solved using a characteristic transformation and a perturbation series in the number of nuclear collisions. Details on the methods of solution and nuclear interaction data bases for nucleon and GCR transport are found in references 16,

17, and 20. We note that the nuclear fragmentation cross sections are poorly known in many instances and that the accurate determination of the particle fields is seen to be highly dependent on the use of the correct cross sections (refs. 17, 21, and 22).

The radiation environments assumed are from the AP-8 model (ref. 23) for the trapped protons and from the NRL CREME solar minimum model for the GCR particles (ref. 24). The integral LET spectra from SAA primary and secondary protons are shown for various shield thicknesses in figures 1 and 2 for SSF maximum and minimum orbits, respectively. In figure 3 the GCR integral LET spectra are shown for the solar minimum environment for SSF. The variation of the GCR environment at these altitudes is small and, therefore, ignored herein. Secondary particle production through nuclear fragmentation becomes more important for increased shielding, which is seen in figure 3 where there are more particles at larger shield values.

Dose and Dose Equivalent

The absorbed dose is defined as

$$D(x) = \sum_j \int dE_j \Phi_j(x, E_j) S_j(E_j) + \sum_j \sum_\alpha \int_0^\infty dE_\alpha dE_j \Phi_\alpha(x, E_\alpha : E_j) S_\alpha(E_\alpha) \quad (3)$$

where the summation is over all particles in the radiation field. Similarly, the dose equivalent is defined as

$$H(x) = \sum_j \int dE_j \Phi_j(x, E_j) S_j(E_j) Q(S_j) + \sum_j \sum_\alpha \int dE_\alpha dE_j \Phi_\alpha(x, E_\alpha : E_j) S_\alpha(E_\alpha) Q(S_\alpha) \quad (4)$$

where the LET-dependent quality factor from ICRP-26 (ref. 25) is represented as

$$Q(S) = \begin{cases} 1 & (S \leq 4 \text{ keV}/\mu) \\ 12.5 \ln[1 + (S/43.75)] & (4 < S < 172.95 \text{ keV}/\mu) \\ 20 & (S \geq 172.95 \text{ keV}/\mu) \end{cases} \quad (5)$$

In figure 4 the variation in dose and dose equivalent with solar cycle is illustrated with calculations compared at 200 n.mi. for solar minimum and solar maximum.

The Computerized Anatomical Man (CAM) model (ref. 18) represents a 50th-percentile U.S. Air Force man who weighs 191.9 lb and is 69.1 in. in height. Shield distribution functions are calculated for several body organs using ray-tracing methods with 512 rays used for convergence. Organ doses are then evaluated through averaging over the rays representing the distribution functions. In figures 5–9, we show dose and dose-equivalent calculations for the surface dose and for several organs from combined SAA proton and GCR exposures versus aluminum shield thickness. In figure 10 the BFO dose using a 5-cm-depth approximation is shown. The 5-cm approximation is seen to overestimate the dose and dose equivalent in comparison with the BFO results of the CAM model. The spacecraft shielding for SSF is also expected to be modeled through ray-tracing techniques. Exposure limits expected for SSF crews (ref. 2) are shown in table I with values in parentheses giving average daily rates for reaching the corresponding limit, which may be compared with our results. A substantial fraction of the BFO limit can be expected for extended stays if adequate shielding is not provided. We note that for most shield values, an average QF for SSF, defined as the ratio of dose equivalent to absorbed dose, is expected to be less than 2.

Track Model Predictions for SSF

In the parametric track model of cellular damage developed by Katz (ref. 13), biological damage from fast ions is caused by delta-ray production and proceeds through two modes of response. In the ion-kill (intratrack) mode, damage occurs through the action of single ions, whereas in the gamma-kill (intertrack) mode, cells not damaged in the ion-kill mode can be sublethally damaged from a passing ion and then inactivated by the cumulative addition of sublethal damage due to delta rays from other passing ions. The response of the cell is described by four cellular response parameters, two of which (m , the number of targets per cell, and D_0 , the characteristic X-ray dose) are extracted from the response of the system to X-ray or gamma-ray irradiation. The other two (σ_0 , interpreted as the cross-sectional area of the cell nucleus, within which the sites are located, and κ , a measure of the size of the damage site) are found from survival measurements with track segment irradiations by energetic charged particles. The surviving fraction of a cellular population N_0 after irradiation by a fluence of particles F is written as (ref. 13)

$$\frac{N}{N_0} = \pi_i \times \pi_\gamma \quad (6)$$

where the ion-kill survivability is

$$\pi_i = e^{-\sigma F} \quad (7)$$

and the gamma-kill survivability is

$$\pi_\gamma = 1 - \left(1 - e^{-D_\gamma/D_0}\right)^m \quad (8)$$

The gamma-kill dose fraction is defined as

$$D_\gamma = (1 - P)D \quad (9)$$

where D is the absorbed dose and P is the fraction of cells damaged in the ion-kill mode given by

$$P = \frac{\sigma}{\sigma_0} \quad (10)$$

where σ is the single-particle inactivation cross section. For the protracted exposures to be experienced on SSF, we assume that all sublethal damage is repaired such that

$$\frac{N}{N_0} = e^{-\sigma F} \quad (11)$$

Our calculations revealed that in actuality, this assumption is not necessary since the gamma-kill contribution is indeed small at these fluence levels.

For the mixed-radiation fields seen in space, the ion-kill term is written as (ref. 26)

$$\begin{aligned} \sigma F = n \sum_j \int dE_j \Phi_j(x, E_j) \sigma_j(E_j) \\ + n \sum_\alpha \sum_j \int dE_\alpha dE_j \Phi_\alpha(x, E_\alpha : E_j) \sigma_\alpha(E_\alpha) \end{aligned} \quad (12)$$

where the subscripts j and α label the projectile and target fragments, respectively, n is the number of days in the SSF mission, and σ_j is defined as

$$\sigma_j = \sigma_0 \left(1 - e^{-Z_j^{*2}/\kappa\beta_j^2}\right)^m \quad (13)$$

where β is the particle velocity and Z^* is the effective charge number. The first term on the right-hand side of equation (12) then corresponds to the contributions from the incident space radiation, including projectile fragments; and the second term, from target fragments produced from shielding materials.

The RBE relative to X rays delivered at a high dose rate for a given survival level is defined as

$$\text{RBE} = \frac{D_X}{D_0} \quad (14)$$

where

$$D_X = -D_0 \ln \left[1 - \left(1 - \frac{N}{N_0}\right)^{1/m}\right] \quad (15)$$

is the X-ray dose at which this level is obtained. At low fluences where $D \ll D_0$ and only ion kill contributes, the RBE is approximately (refs. 26 and 27)

$$\text{RBE} \approx \frac{D_0}{\text{LET}} \sigma^{1/m} F^{-1+(1/m)} \quad (16)$$

For an exponential X-ray response, $m = 1$, and we find from equation (16) that the RBE is fluence independent. If the radiation environment is steady with time, the RBE is found to vary with mission duration as (ref. 26)

$$\text{RBE}(n_1) = \left(\frac{n_1}{n_2}\right)^{-1+(1/m)} \text{RBE}(n_2) \quad (17)$$

where n_1 and n_2 are arbitrary numbers of days in space and the low-fluence conditions mentioned are assumed to hold. Equation (17) can then be used to scale the RBE values discussed below to a mission of arbitrary duration on SSF, assuming that the radiation environment is fairly constant.

Of particular concern is the fluence dependence of the RBE found in measurements with high LET particles, indicating a rising RBE with decreasing fluence (refs. 6-10). In the track model, this effect is seen to arise because fast ions may act through single-particle action, whereas X rays or gamma rays act only through sublethal damage. In the limit of a small X-ray dose, the RBE may approach infinity (ref. 12) since no effect from X rays will be registered, whereas a nonzero response can be expected for single-ion tracks. For high-energy protons, the LET from direct ionization is small, on the order of that of gamma rays, and biological damage at low fluence proceeds predominantly through the production of nuclear fragments in tissue. The effective radiation field of the incident protons and high LET tissue secondaries can then lead to high RBE values at low dose. The effect of the tissue fragments on the proton action cross section for neoplastic transformation of C3H10T1/2 cells is shown in figure 11. The dotted line shows the contribution of primary protons and is seen to be negligible at high energies. Thus, in the

single-track regime (low fluence), target fragments dominate.

Cellular response parameters derived from radiological experiments are listed in table II. In figure 12, we show results of calculations for the fraction of neoplastic transformations of C3H10T1/2 cells in 90 days behind aluminum shielding, assuming a local host medium of tissue. In figure 13 we show the resulting values of RBE for transformations for the trapped-proton and tissue secondary contributions. The RBE is seen to increase with shielding because of the decreasing fluence of particles as indicated by equation (16). In table III we give estimates of production rates for several end points in mammalian cell cultures. For cell death, the effects of normal turnover and repopulation of cells will have to be considered in order to understand these loss rates. Fractional cell-killing values for values of T-1 human kidney cells, with the CAM model representation of kidney shielding included, from the trapped protons are shown in figure 14. For trapped exposure, the high RBE values computed are solely due to nuclear secondaries formed in tissue and would approach 1 if their effects were excluded. In figure 15, RBE values from the GCR components at 90 days are shown. The RBE values computed in all cases are many times greater than the corresponding QF values, although the damage rates predicted in table III are not excessive.

Concluding Remarks

Calculations for assessing biological risk on Space Station *Freedom* (SSF) orbits have been presented based on the quality factor (QF) which is solely dependent on the linear energy transfer (LET) and using the fluence-dependent cellular track model. Results indicate that the low fluence levels expected for SSF orbits present relative biological effectiveness (RBE) values for end points in mammalian cells more severe than quality factors in ICRP-26 and which vary rapidly with spacecraft shielding because of varying particle fluences. In both approaches, damage from the South Atlantic Anomaly (SAA) protons is expected to dominate over the background galactic cosmic ray (GCR) exposure. In the track model, the damage from SAA protons occurs predominantly through the production of nuclear secondaries in tissue. The accurate description of fragmentation in tissue is seen to be vital for applications of the track structure model to the proton-dominated exposures of SSF.

The track model is limited to end points studied in radiological experiments such that the biological cross section is described. More importantly, the

relationship between the number of transformations induced and the resulting probability of tumor formation must be known before risk assessment can be made. Nevertheless, our study indicates that if quality factors are to be used to assign risk from high LET fields, important questions related to the rapid variation of RBE with fluence at low fluence must be answered when applying such a system to exposures in space.

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April 9, 1991

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Table I. Short-Term Dose-Equivalent Exposure Limits for Protection Against Nonstochastic Effects
[Values in parentheses give average daily rates for reaching the corresponding limit]

Time period	Dose equivalent, cSV, for—					
	BFO		Eye lens		Skin	
30 days	25	(0.833/day)	100	(3.33/day)	150	(5.0/day)
Annual	50	(0.137/day)	200	(0.548/day)	300	(0.822/day)

Table II. Cellular Response Parameters

Cell-damage type	m	σ_0 , cm ²	κ	D_0 , cGy
T-1 kidney cell death	2.5	6.7×10^{-7}	1000	170
C3H10T1/2 cell death	3.0	5×10^{-7}	750	280
C3H10T1/2 cell transformation	2.0	1.15×10^{-10}	750	26 000
Chinese hamster mutation	3.5	5.75×10^{-11}	1000	5 940 000

Table III. Cell-Damage Rates Behind Aluminum Shielding for 90 Days on SSF

Environment	T-1 kidney death	C3H10T1/2 death	C3H10T1/2 transformation	Chinese hamster mutation
Al shield thickness, 1 g/cm ²				
Trapped protons at 270 n.mi. (solar max.)	1×10^{-2}	7×10^{-3}	5×10^{-6}	4×10^{-7}
Trapped protons at 200 n.mi. (solar min.)	4×10^{-3}	3×10^{-3}	2×10^{-6}	2×10^{-7}
GCR (solar min.)	3×10^{-4}	3×10^{-4}	1×10^{-7}	$< 1 \times 10^{-8}$
Al shield thickness, 10 g/cm ²				
Trapped protons at 270 n.mi. (solar max.)	4×10^{-3}	3×10^{-3}	2×10^{-6}	2×10^{-7}
Trapped protons at 200 n.mi. (solar min.)	1×10^{-3}	1×10^{-3}	8×10^{-7}	$< 1 \times 10^{-8}$
GCR (solar min.)	3×10^{-4}	2×10^{-4}	1×10^{-7}	$< 1 \times 10^{-8}$

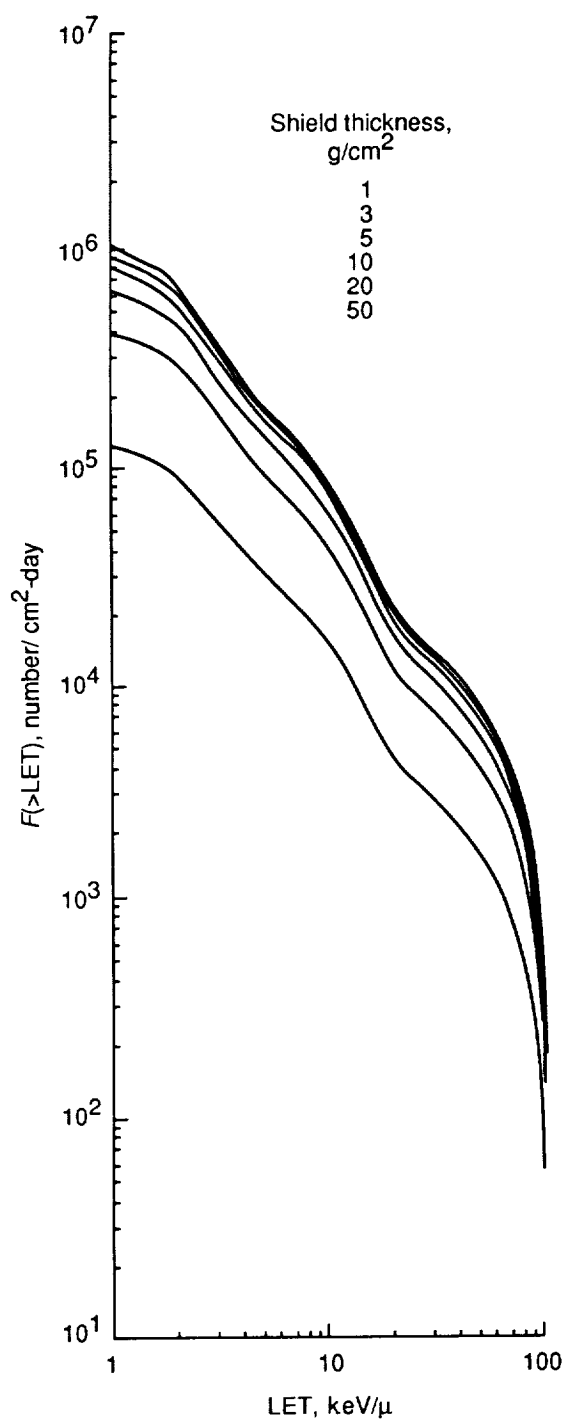


Figure 1. Integral LET spectrum for combined primary and secondary protons in 28.5° orbit at 270-n.mi. altitude (solar maximum) for various aluminum shields.

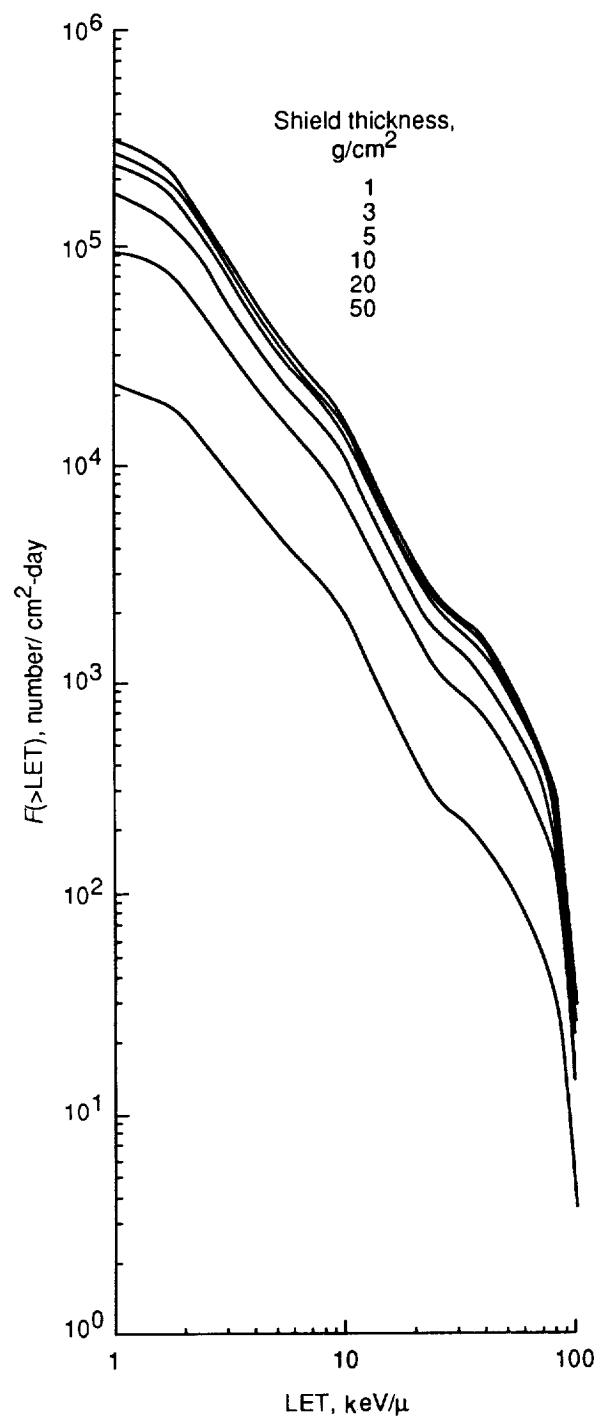


Figure 2. Integral LET spectrum for combined primary and secondary protons in 28.5° orbit at 200-n.mi. altitude (solar minimum) for various aluminum shields.

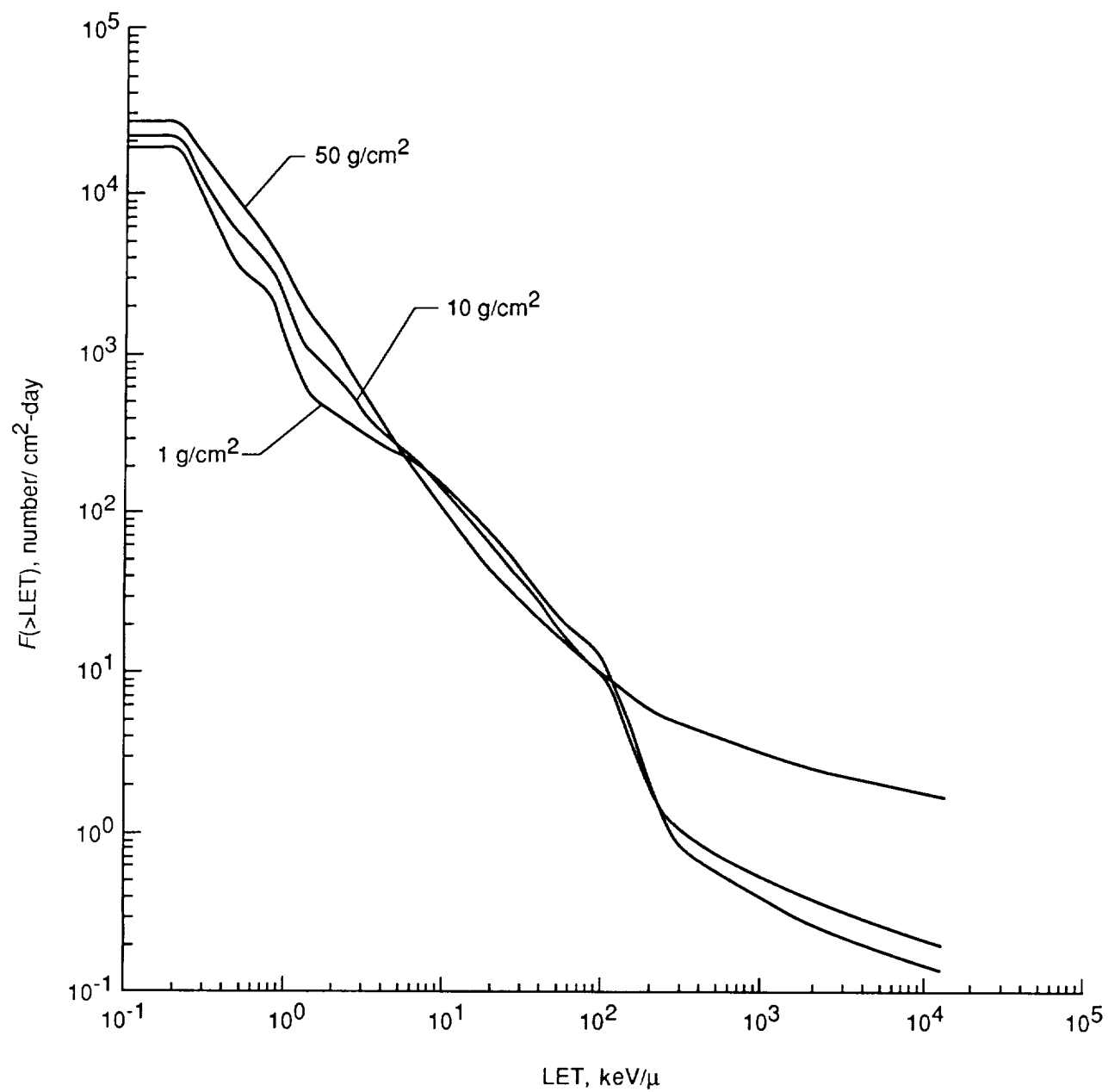
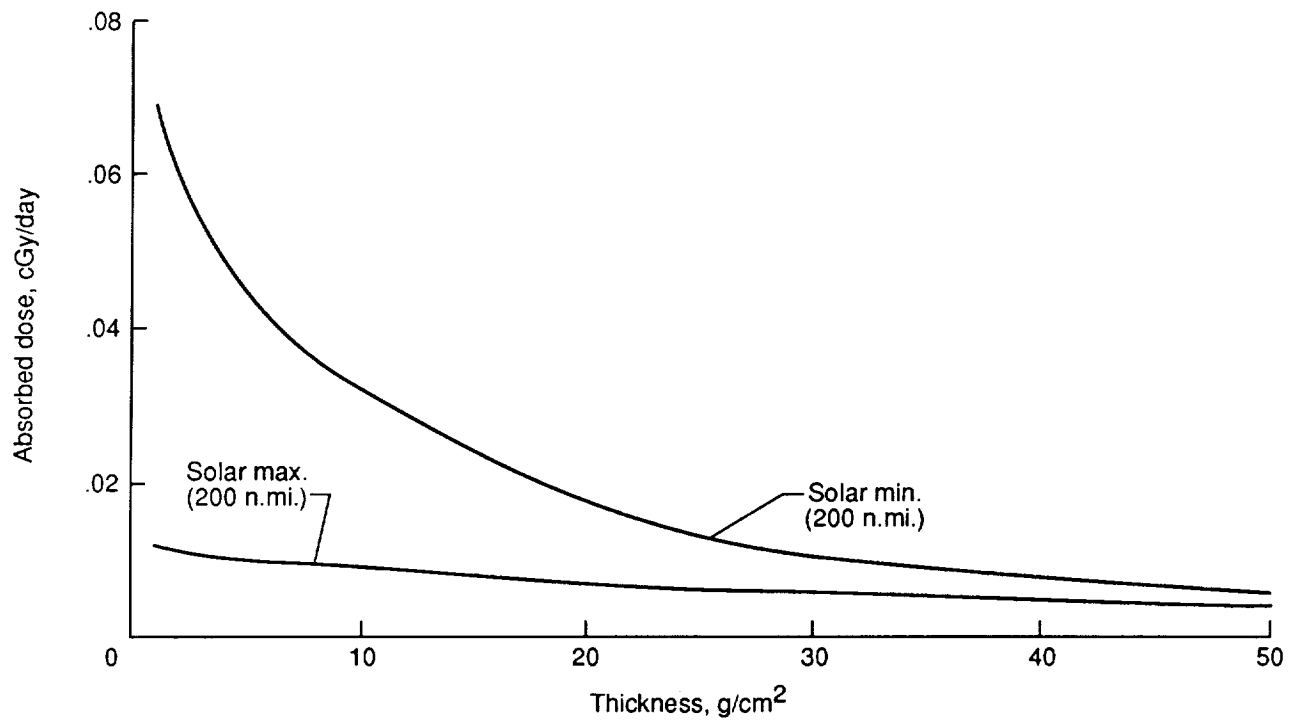
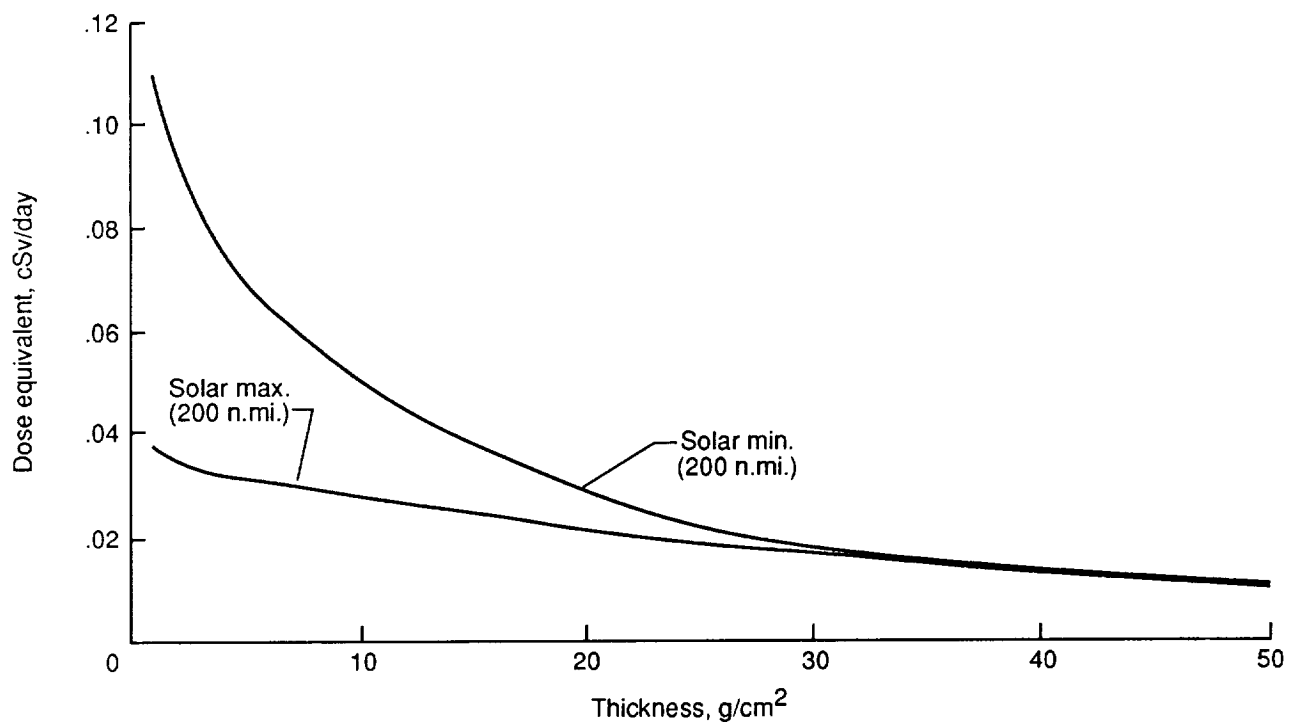


Figure 3. Integral LET spectrum for GCR particles and their secondaries (solar minimum) in SSF orbit for various aluminum shields.

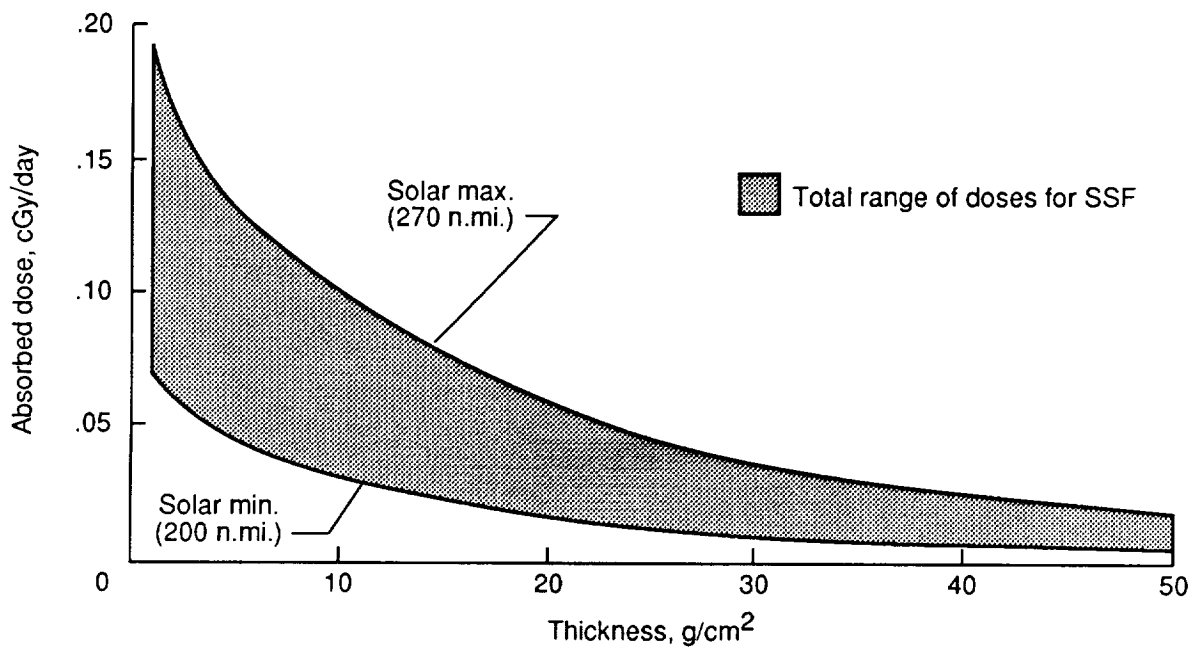


(a) Absorbed dose.

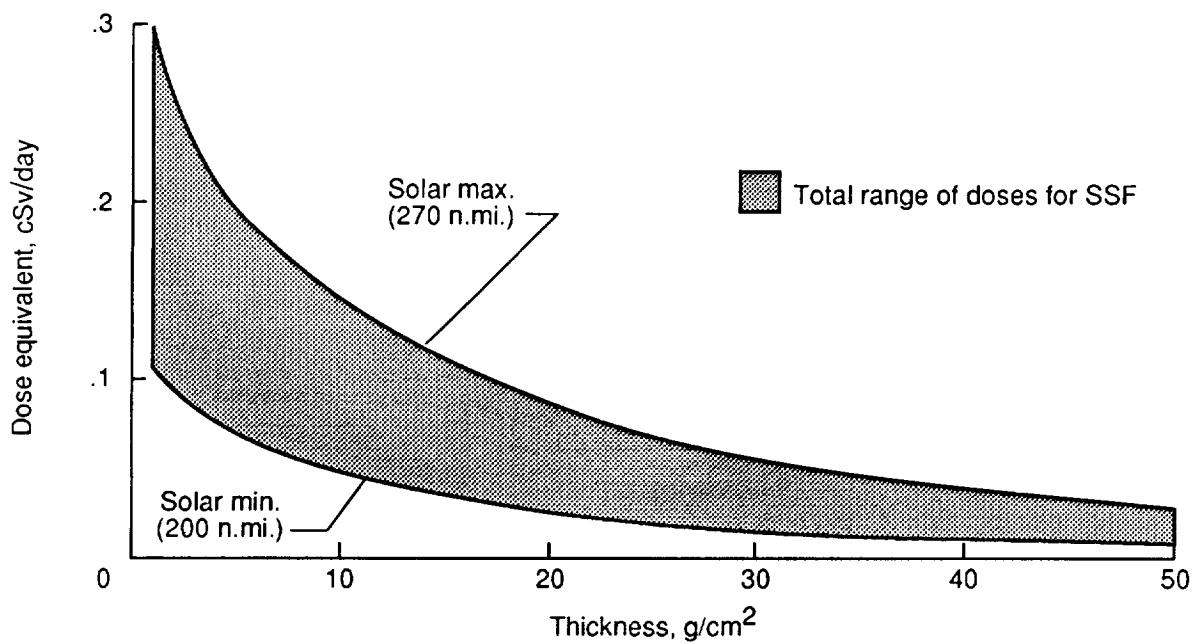


(b) Dose equivalent.

Figure 4. Exposure versus aluminum shield thickness in 28.5° orbit at 200-n.mi. altitude for solar minimum and maximum for combined trapped protons and GCR particles.

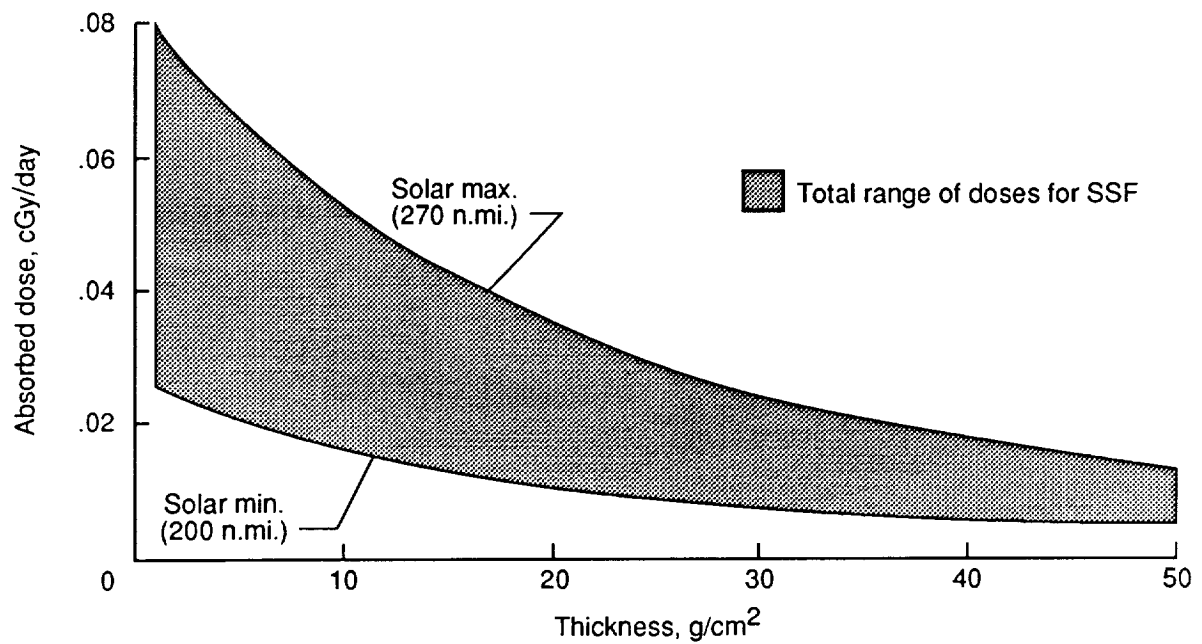


(a) Absorbed dose.

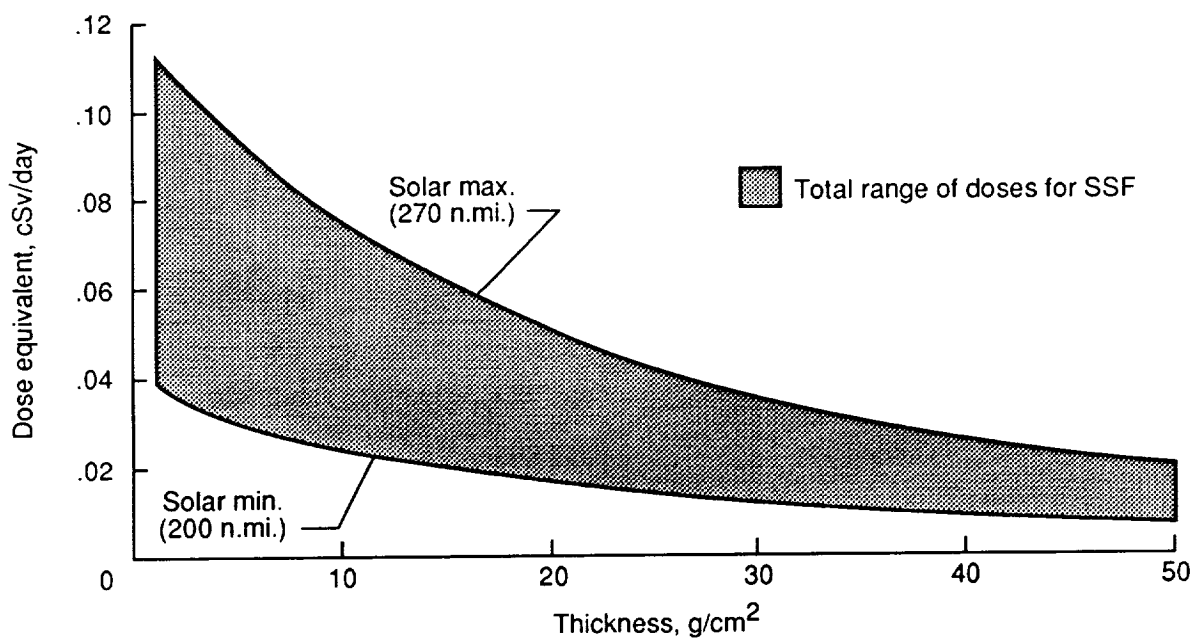


(b) Dose equivalent.

Figure 5. Exposure versus aluminum shield thickness for SSF minimum and maximum exposed orbits for combined trapped protons and GCR particles.

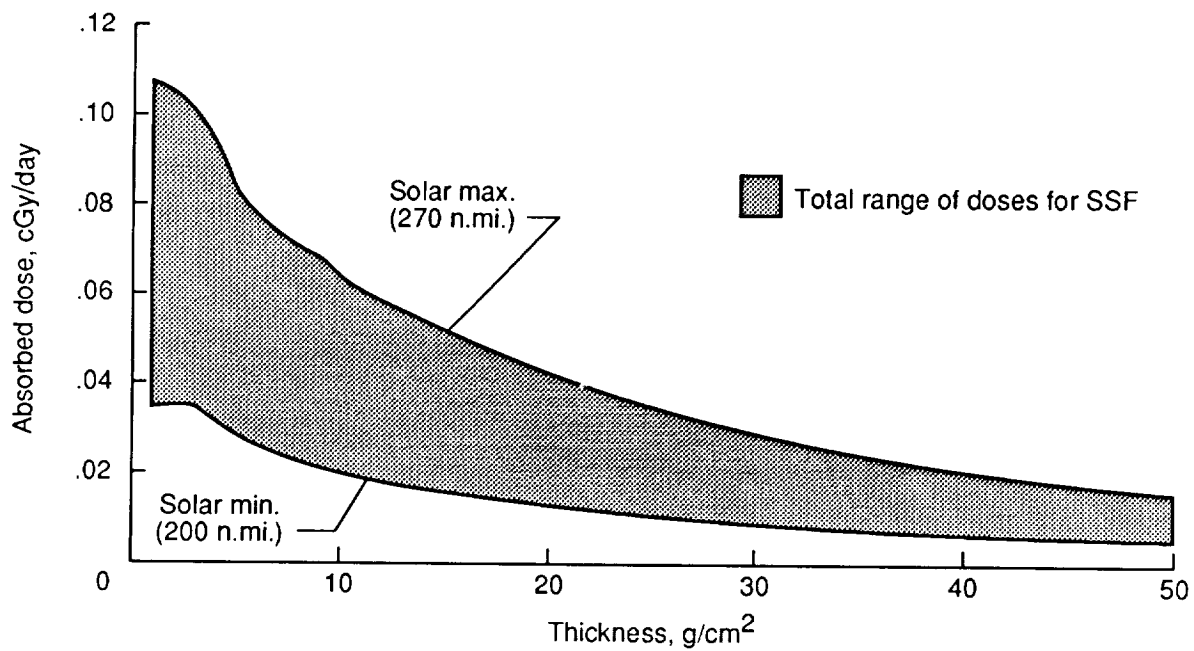


(a) Absorbed dose.

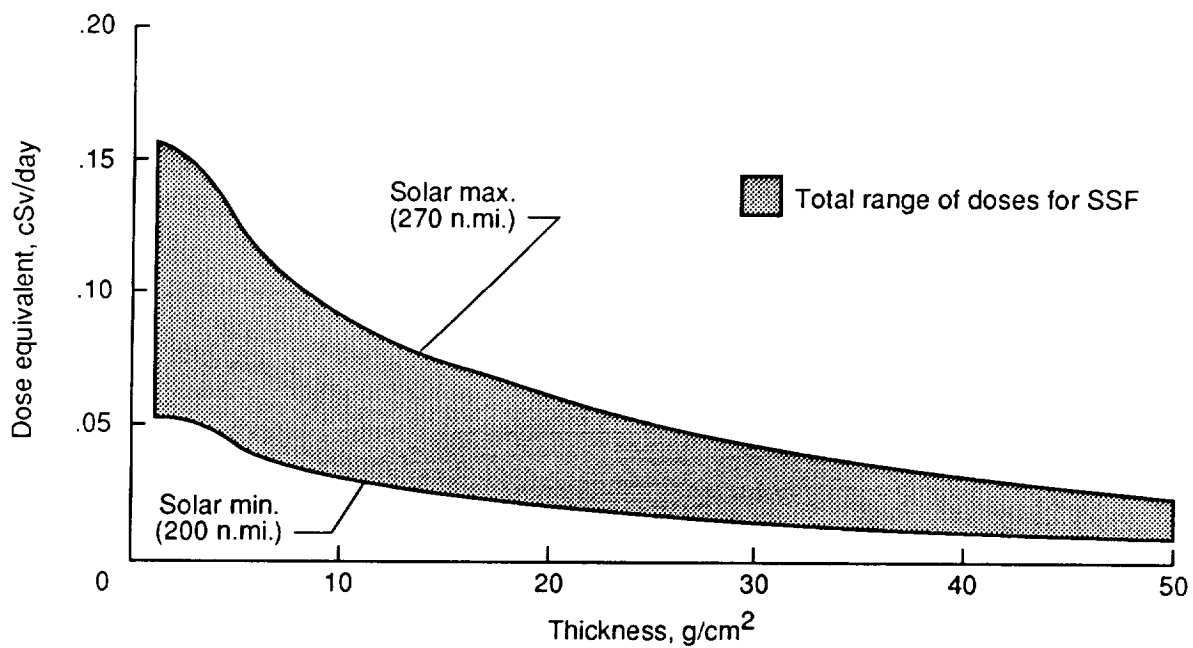


(b) Dose equivalent.

Figure 6. Exposure at the BFO using the CAM model versus aluminum shield thickness for SSF orbits from combined trapped protons and GCR particles.

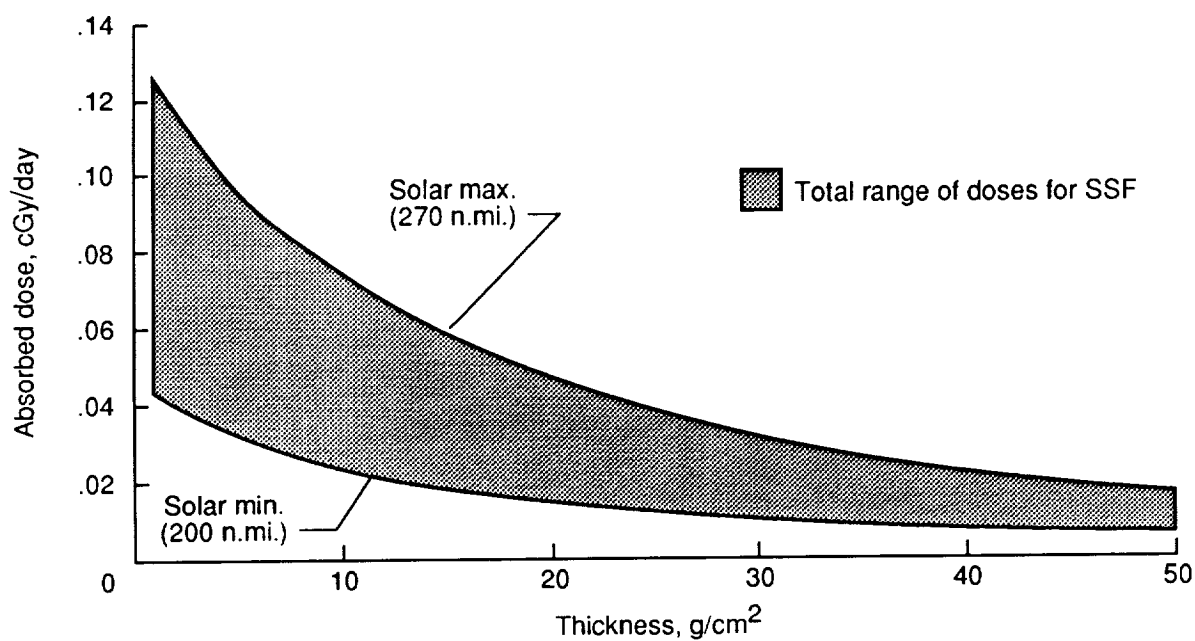


(a) Absorbed dose.

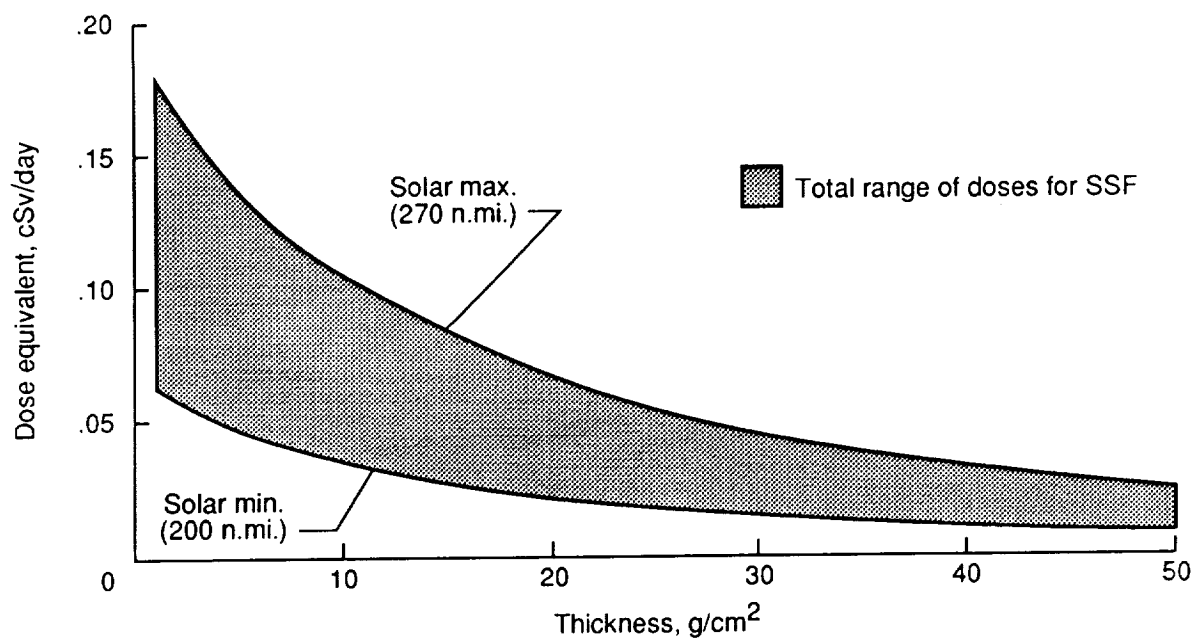


(b) Dose equivalent.

Figure 7. Exposure at average skin location using the CAM model versus aluminum shield thickness in SSF orbits.

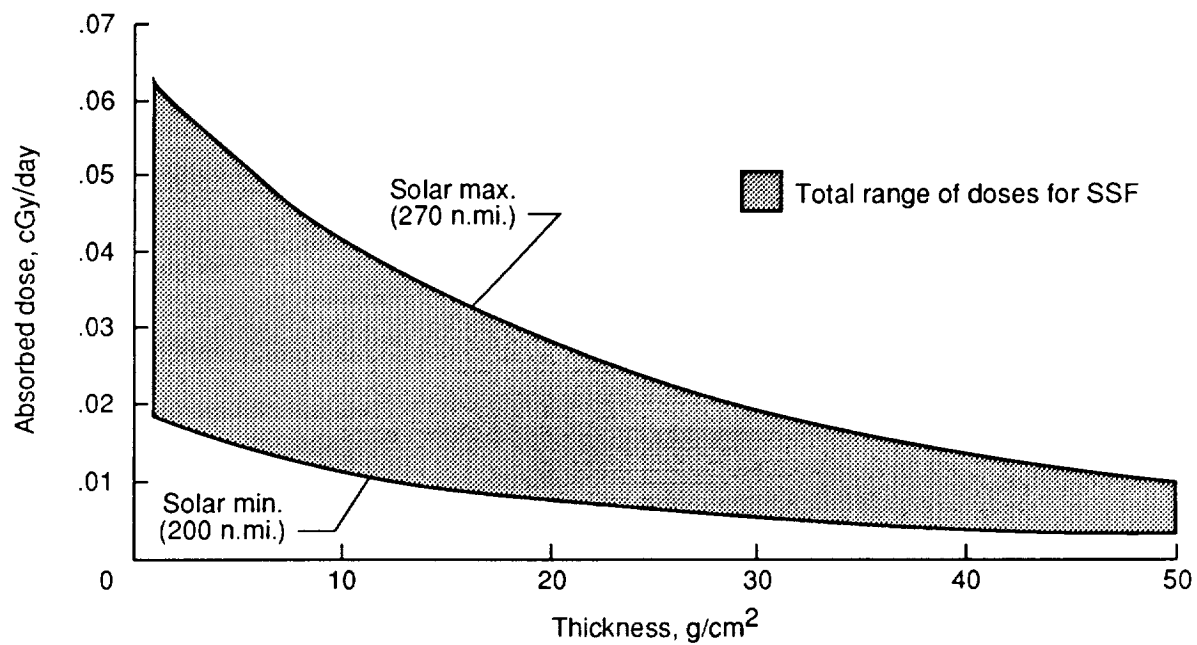


(a) Absorbed dose.

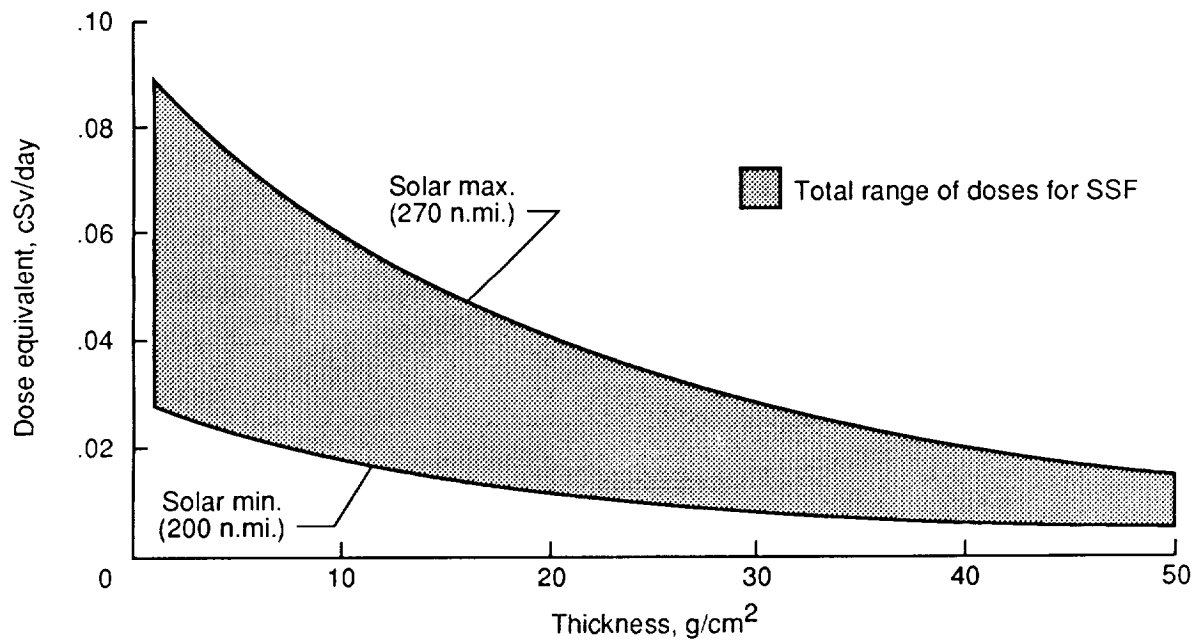


(b) Dose equivalent.

Figure 8. Exposure at the eye using the CAM model versus aluminum shield thickness in SSF orbits.

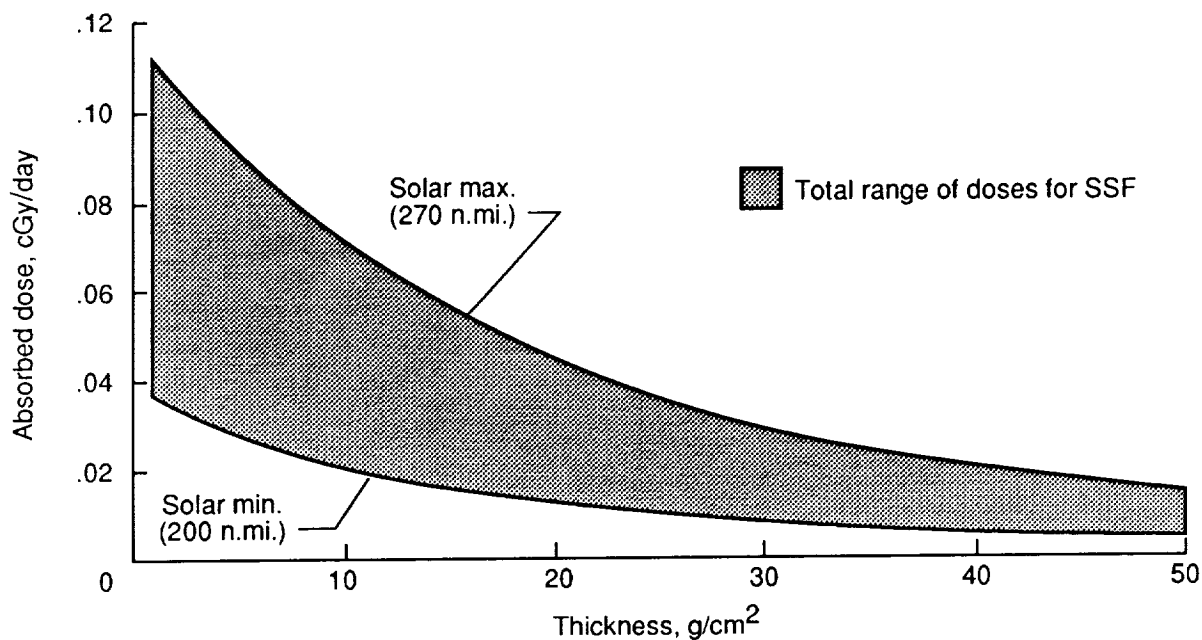


(a) Absorbed dose.

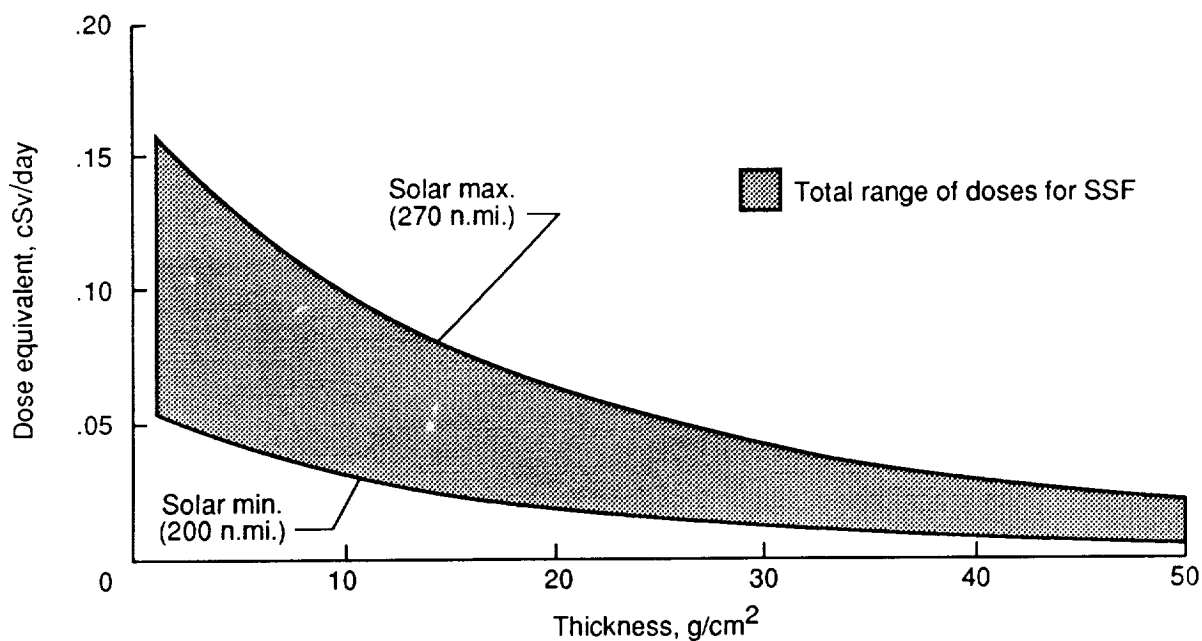


(b) Dose equivalent.

Figure 9. Exposure at the kidney using the CAM model versus aluminum shield thickness in SSF orbits.



(a) Absorbed dose.



(b) Dose equivalent.

Figure 10. Exposure at the BFO using 5-cm-depth approximation versus aluminum shield thickness in SSF orbits.

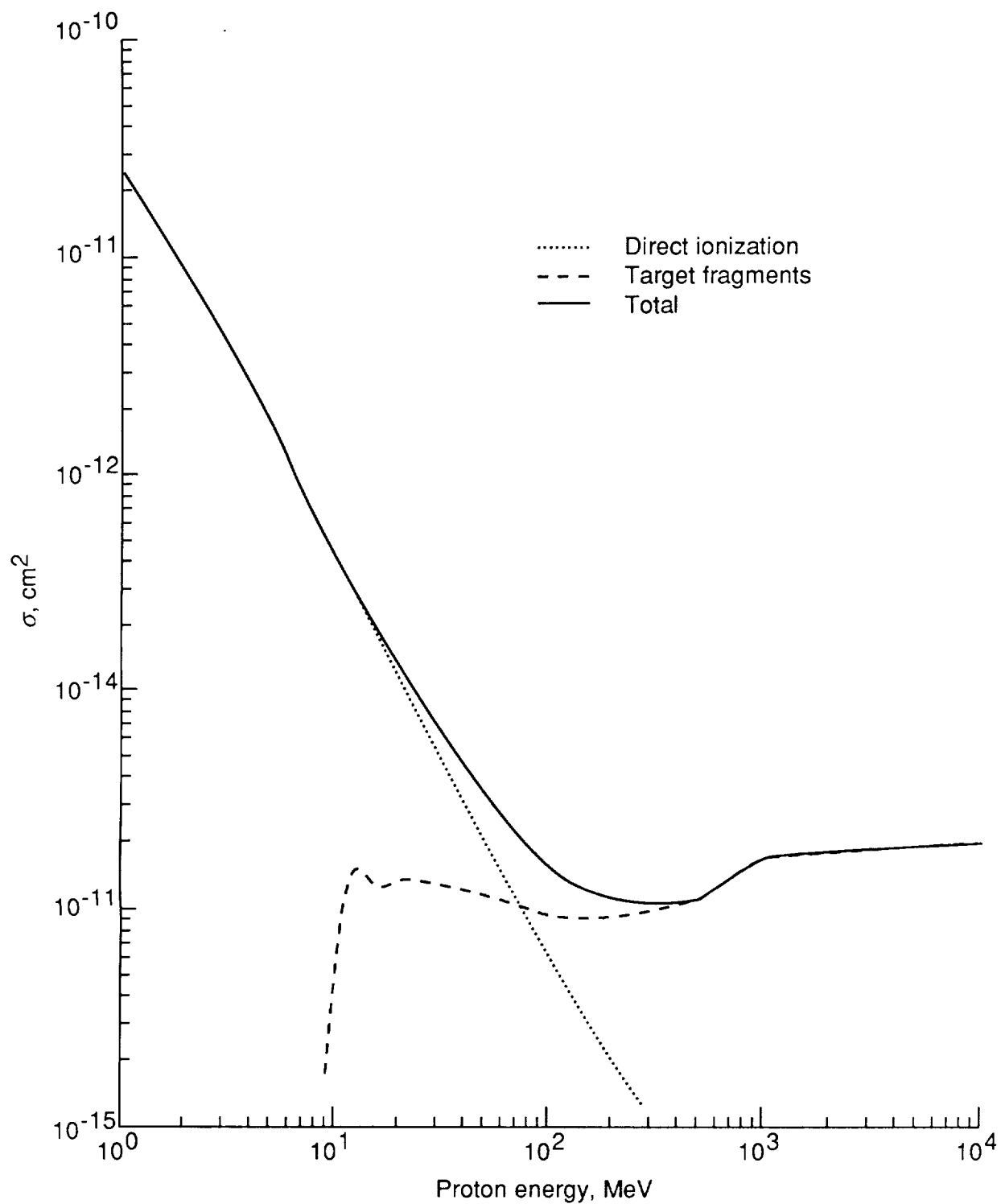


Figure 11. Cell-transformation cross sections including effects of nuclear reactions for protons in C3H10T1/2 cells according to the Katz model.

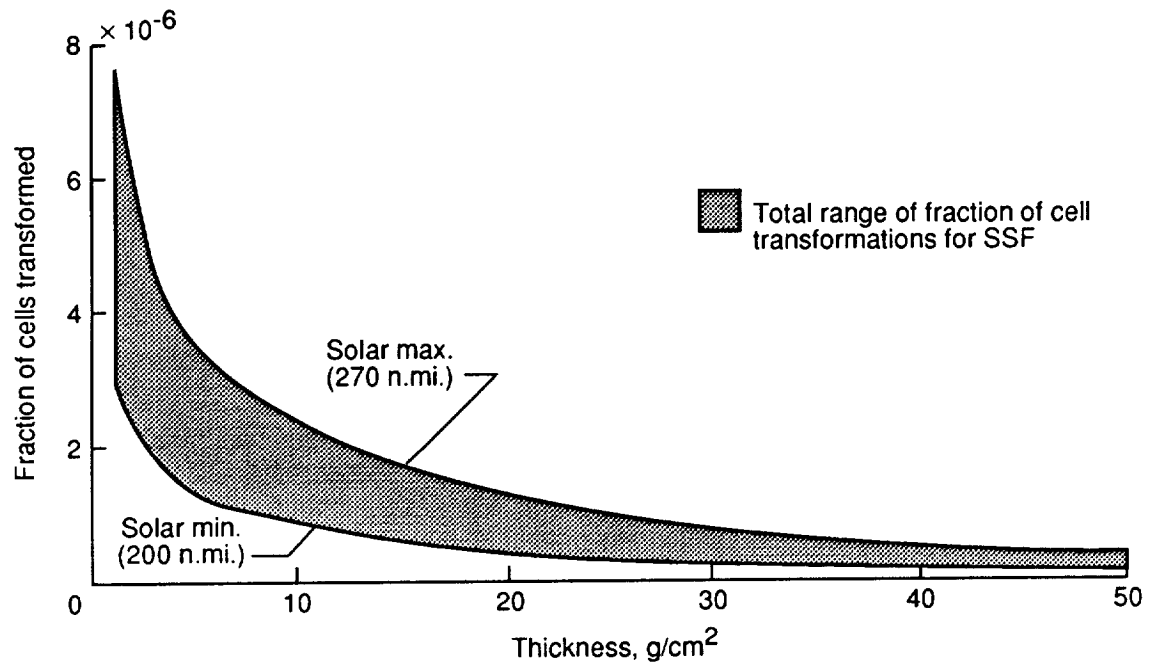


Figure 12. Fraction of C3H10T1/2 cells transformed versus aluminum shield thickness in SSF minimum and maximum exposed orbits for trapped protons.

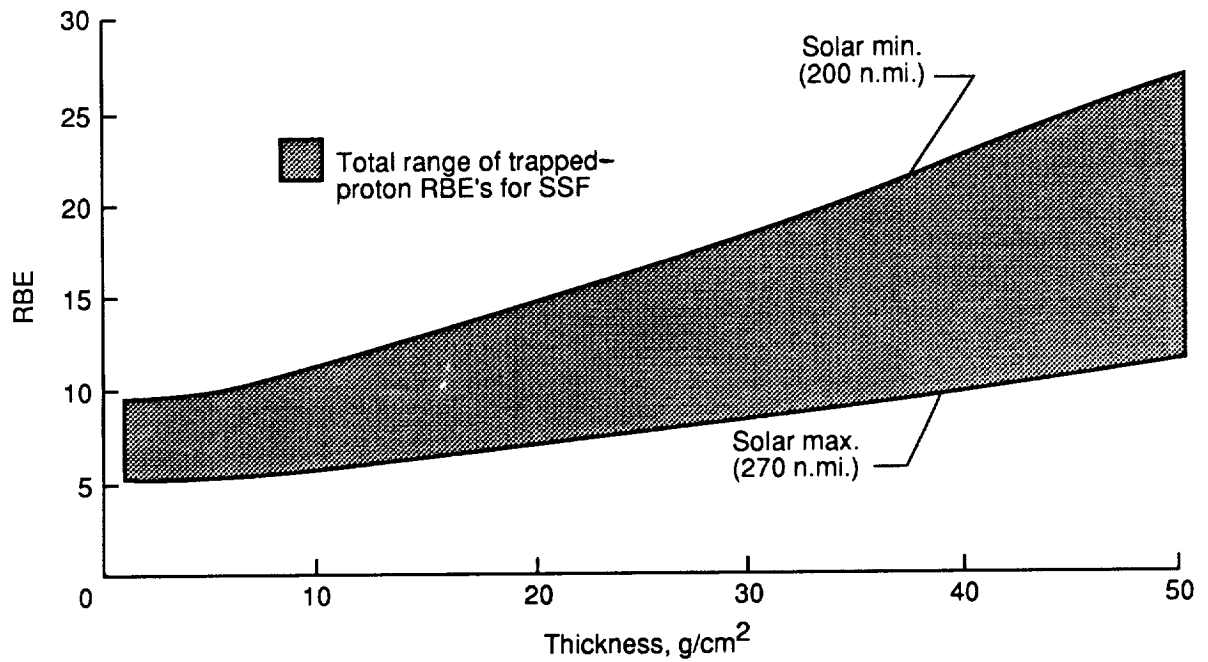


Figure 13. RBE for transformation of C3H10T1/2 cells versus aluminum shield thickness for 90 days in SSF minimum and maximum exposed orbits for trapped protons.

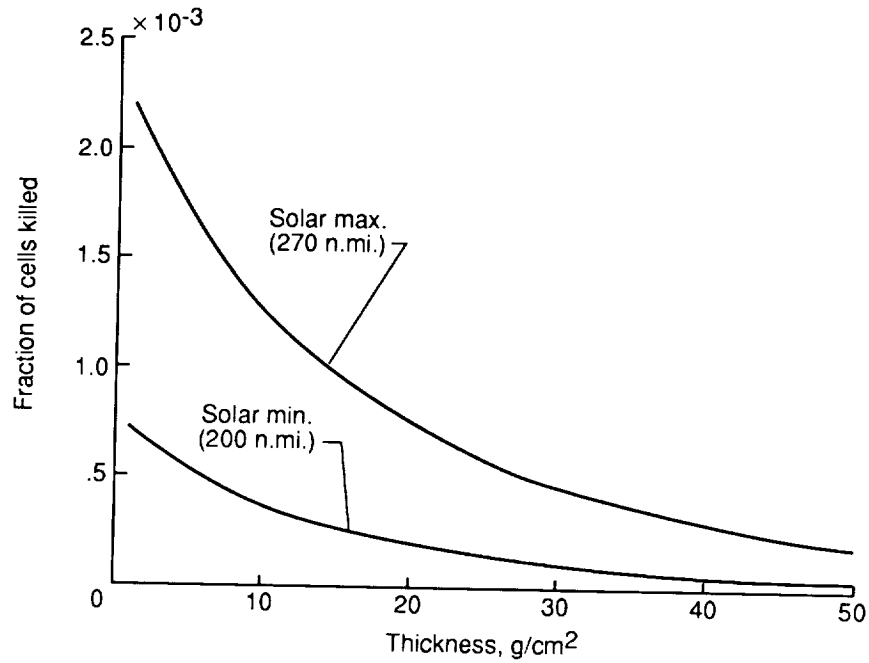


Figure 14. Fraction of T-1 kidney cells killed (loss of cell reproductive capability) behind CAM model kidney shielding versus aluminum shield thickness for 90 days in SSF minimum and maximum exposed orbits for trapped protons.

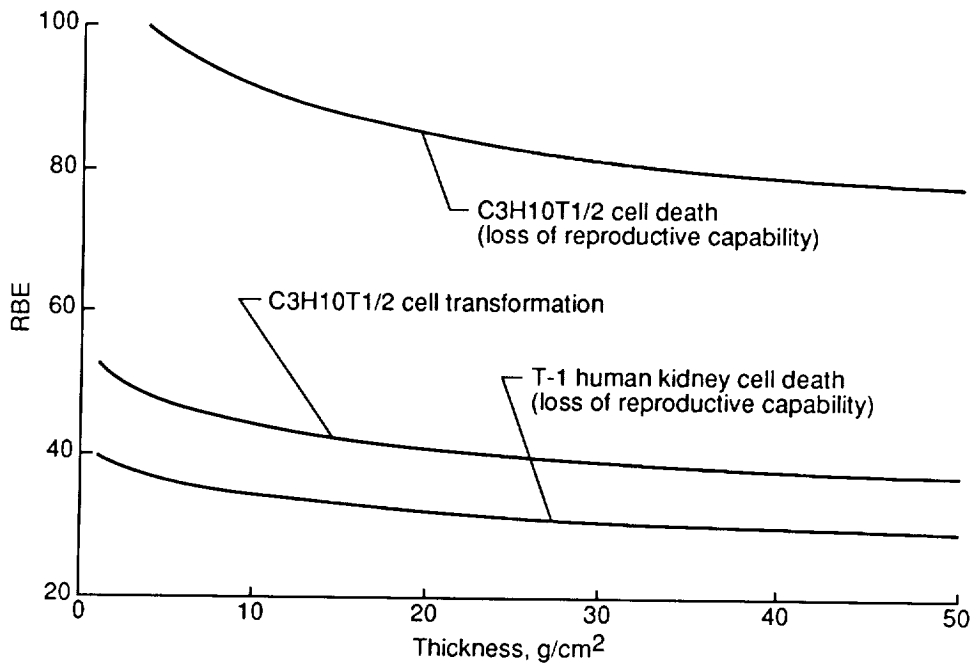


Figure 15. RBE values for several end points versus aluminum shield thickness for 90 days in SSF orbit from GCR particles.



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16. Abstract Risk-assessment calculations are presented for the preliminary proposed solar minimum and solar maximum orbits for Space Station <i>Freedom</i> (SSF). Integral linear energy transfer (LET) fluence spectra are calculated for the trapped-proton and galactic cosmic ray (GCR) environments. Organ-dose calculations are discussed using the Computerized Anatomical Man model. The cellular track model of Katz is applied to calculate cell survival, transformation, and mutation rates for various aluminum shields. Comparisons between relative biological effectiveness (RBE) and quality factors (QF) for SSF orbits are made, and fluence-dependent effects are discussed.			
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