Risks due to X-ray Flares during Astronaut Extravehicular Activity

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Abstract.

Solar hard X-ray flares can expose astronauts on lunar and deep space extravehicular activities (EVAs) to dangerous acute biological doses. We combine calculations of radiative transfer through shielding materials with subsequent transfer through tissue to show that hazardous doses, taken as $\geq 0.1$ Gy, should occur with a probability of about 10% per 100 hours of accumulated EVA inside current spacesuits. The rapid onset and short duration of X-ray flares and the lack of viable precursor events require strategies for quick retreat, in contrast to solar proton events, which usually take hours to deliver significant fluence and can often be anticipated by flares or other light-speed precursors. Our results contrast with the view that only particle radiation poses dangers for human space exploration. Heavy-element shields provide the most efficient protection from X-ray flares, since X-rays produce no significant secondary radiation. We calculate doses due to X-ray flares behind aluminum shields and estimate the required shield masses to accompany EVA rovers.

1. Introduction

The risk for space travelers due to solar radiation, as well as Galactic cosmic rays, has been studied extensively for nearly four decades, but the predictability and frequency of potentially lethal doses of high-energy solar radiation is still far from understood. A long series of studies suggest that the most energetic solar proton events (SPEs) [Reames, 1996; Shea and Smart, 1990, 2000; Miroshnichenko, 2003; Reames, 1999] can produce lethal biological doses that require careful consideration of radiation risks for manned space travel [e.g. Silberberg and Tsao, 1979; Letaw et al., 1989; Dyer et al., 1996; Schimmerling et al., 1996; Badhwar, 1997; Cucinotta et al., 2001; Miroshnichenko, 2003; Cucinotta et al., 2004; De Angelis et al., 2004a, b; Getselev et al., 2004; Wilson et al., 2004; Cougnet et al., 2004; Johnson et al., 2005; O'Brien, 2005; Kim et al., 2006].

The August 1972 SPE, often taken as the standard high-fluence event for protection studies, was not an isolated anomaly. The February 1956, November 1960, October 1989, and October-November 2003 events produced solar-particle fluxes sufficiently large that an astronaut on the moon protected by only a spacesuit would likely have perished, and many events approaching these fluences have been recorded [Shea and Smart, 2000; Miroshnichenko, 2003]. The durations of these events (many hours, see Miroshnichenko 2003) are large enough that evacuation to shelter would prevent serious exposure to an astronaut on an EVA. Observations of associated lightspeed precursors (photon flares and CME eruptions), while not always available for the strongest SPEs [Reames, 1999], could allow further time for escape.

Given the number of studies that have taken on the computational difficulties of the propagation of solar cosmic rays in the inner heliosphere and the resulting biological doses, it is surprising that the simpler study of the effects of ionizing photons, in particular X-rays from solar flares [Haisch et al., 1991; Hudson, 1991], have not been considered. It is easy to show that the fluence from the most energetic hard X-ray flares of the last 50 years would result in an amount of energy absorbed per gram of tissue larger than the lethal dose for an unprotected human at 1 AU. Acute doses for various biological endpoints are well studied, especially because of their relevance to oncological research. The frequency of very energetic flares is similar to that of the most energetic SPEs; their durations are so small that evacuation is much more problematic; and there are no early and reliable precursors that can be used to predict their onset as in the case of SPEs. Furthermore, the shielding requirements are nontrivial and somewhat different from those for solar energetic particles, preferring material of high atomic number, in contrast to the mostly polymer construction of current spacesuit designs. Bone surrounding blood-forming marrow might mitigate hematological effects, but the risk of carcinogenesis remains significant. We calculate here the expected doses from energetic solar X-ray flares and use their observed frequency-fluence relation to estimate the probability per unit time of a hazardous flare exposure.

2. The Largest Solar Flares

Solar flare photons span energies between $10^{-1}$ and $10^6$ keV [Haisch et al., 1991; Hudson, 1991; Kanbach et al., 1993; Ryan, 2000]. RHESSI observations [e.g. Battaglia et al., 2005] show that the spectrum of hard X-rays (HXR) from solar flares extends to energies as low as 10–25 keV, where "hard" refers to the power-law part of the spectrum produced by particle acceleration rather than thermal processes. The HXR spectrum is usually fit by the form $F(E) \sim E^{-p}$ [Crosby et al., 1993; Lee et al., 1993; Bromund et al., 1995; Veronig et al., 2002; Qiu et al., 2004]. The range of the estimated power-law index (log-log slope) $p$ is large for HXR flare spectra, with $2 < p < 6$ and a median around...
3.5. Additionally, a small correlation between $p$ and total X-ray output has been suggested by RHESSI [Battaglia et al., 2005].

X-class solar flares (the most energetic flares) have typical durations of about 5–30 min [Crosby et al., 1993; Veronig et al., 2002], with the more energetic flares tending to last longer. As with spectral slope, flare durations vary widely, but even with the longest of these durations the dose should be considered acute (i.e. effects have rapid onset), in contrast to low-level, extended exposure to Galactic cosmic rays. The acute lethal dose is almost always independent of dose rate [Sparrow et al., 1967], so we assume that the nonlethal acute dose depends on total fluence, not flux, and hence on the flare total energy release.

The total photon energy release $W$ in flares is difficult to estimate and varies by at least a factor of $10^3$, but a large number of studies using EUV, soft X-ray (SXR), and HXR satellite events roughly agree that the differential distribution $dN/dW$ of flare energy releases is a power law with index about $-1.6$ to $-1.8$ over at least six orders of magnitude in total energy release $W$ [Hudson, 1991; Lee et al., 1993; Crosby et al., 1993; Bromund et al., 1995; Aschwanden et al., 2000; Lin et al., 2001; Güdel et al., 2003; Qiu et al., 2004].

The largest X-ray releases observed among contemporary solar flares is $\sim 10^{42}$ erg [Hudson, 1991; Crosby et al., 1993]. Eleven X-class flares occurred during the extraordinary solar outbursts between 18 October 2003 and 5 November 2003 [Gopalswamy et al., 2005], with SXR releases in the GOES 1-8 Å (2–10 keV) band peaking at $2 \times 10^{31}$ erg for the largest bursts. Observations using the SORCE instrument’s Total Irradiance Monitor yielded a total flare energy at all wavelengths for the 28 October flare of $4.6 \times 10^{32}$ erg [Woods et al., 2004]. Radiation and charged particles from these flares compressed the Earth’s Van Allen belt to within 20,000 km of the surface [Baker et al., 2004], damaged the orbiting Mars Odyssey communication instruments, and reduced polar ozone levels significantly [Randel et al., 2005].

Although most of our calculations are for a $10^{31}$ erg flare, it is unlikely that much more energetic flares than considered here have occurred. Such flares have been serendipitously discovered in other solar-like stars with otherwise normal characteristics [Schaefer et al., 2000]. Upper limits on proton fluences inferred from cosmogenic isotopes in lunar samples [Reedy et al., 1983; Reedy, 1996], tree ring records of $^{14}$C [Lingenfelter and Hudson, 1980], and the statistics of impulsive nitrate events [McCracken et al., 2001] suggest that the frequency-fluence relation steepens for high-energy particle fluences above about $10^{10}$ cm$^{-2}$, probably due to streaming-limited fluxes associated with self-confinement by ion-wave interactions [Reames, 1999]. In contrast, no such limit, empirical or theoretical, has been established for photon flares, other than an upper limit to avoid divergence of total energy [Hudson, 1991; Aschwanden, 1999].

We adopt a mean recurrence time for $10^{31}$ erg flares of 10 yr, agreeing with the estimate of Hudson [1991] and broadly consistent with the dozen or so $10^{31}$–$10^{32}$ erg events that have been observed since GOES soft X-ray monitoring began in 1976. The average HXR frequency-energy release statistic $dN/dW$ is taken as a power law in $W$, with log-log slope $-1.7$. Thus, we take the mean time between events of energy release $W_{31}$ (in units of $10^{31}$ erg) as

$$\tau(W) = 0.2 W_{31}^{0.7}\text{ yr}. \tag{1}$$

3. Methods

For our calculations, solar flare photon number spectra are assumed to be distributed as power laws, $E^{-p}$, with $2 < p < 6$ (see above). The flare spectrum is assumed to extend from 10 keV to 511 keV. The 10 keV lower limit is taken to simulate the HXR flares, which often begin to flatten to a thermal form around this energy, while the upper limit is set high enough that a negligible number of incident photons are at higher energies, even for the shallowest spectra (lowest $p$). Henceforth, total energy release quantities refer only to the energy between 10 and 511 keV.

We transport the incident ionizing radiation using a single-scattering approximation. The primary interaction processes at X-ray energies are photoelectric absorption and Compton scattering. The photoabsorption opacity is much larger, so the radiative transfer can be computed by employing an effective opacity, in which both Compton scattering and photoabsorption are treated as absorption processes. By including the Compton cross section in the opacity, in effect assume that the material is optically thin to Compton scattering, such that at most a photon will scatter no more than once before exiting. Errors incurred by this approximation are negligible, as we have verified using Monte Carlo simulations [Smith et al., 2004]. The Compton-scattering coefficient was computed exactly using the Klein-Nishina formula, while the photoabsorption cross section was approximated using the empirical form of Setlow and Pollard [1962]:

$$\sigma_p(E) = 2.4 \times 10^{-30} (1 + 0.008Z) \left(\frac{Z}{E}\right)^3 \text{ cm}^2, \tag{2}$$

where $Z$ is the atomic number of the target nucleus and $E$ is the energy of the incident photon in units of 511 keV.

We found this form to be an excellent fit to cross section data for light elements and for energies higher than the K edge. The K edges of carbon and aluminum are at (respectively) 284 and 1560 eV [Henke et al., 1993] and are well below our spectral cutoff energy of 10 keV, so our calculation is in the energy regime that is consistent with the cross section fit. The effective opacity of the material is then

$$\kappa(E) = \sigma_p(E) + Z \sigma_c(E), \tag{3}$$

where $A$ is the mean atomic mass in amu of the target material, $Z$ is the mass of a hydrogen nucleus, $Z$ is its atomic number, and $\sigma_c$ is the Compton cross section. The single-scattering approximation results in exponential attenuation of the incident fluence. In terms of the effective opacity $\kappa$ and the areal density $\Sigma$ (g cm$^{-2}$) of the shielding material, the attenuated energy fluence $F$ is

$$F(E, \Sigma) = E \frac{dN}{dE} \exp\left[-\kappa(E)\Sigma\right]. \tag{4}$$

After transport through the shielding material, the photon fluence is converted to a biological dose by assuming that the remaining radiation is absorbed by pure water. Much of the damage by ionizing radiation is thought to be “indirect,” involving chemical reactions initiated by energy deposited in the bulk cell water or first hydration layer, rather than “direct” ionization of DNA [von Sonntag, 1987; Ward, 1999]; although this terminology is now recognized as an oversimplification [Fieledien and O’Neill, 1991]. We then estimate the dose as a function of areal density of the shielding by integrating the attenuated photon energy spectrum over the effective opacity of water $\kappa_w(E)$ (calculated using the single-scattering method above):

$$D(\Sigma) = \int_{10}^{511} \kappa_w(E) F(E, \Sigma) \, dE, \tag{5}$$

where the attenuated fluence $F$ is given above, and the limits of integration are in keV. This is essentially the skin dose.
4. Minimum Hazardous Dose

To estimate risks to humans exposed to solar flare X-rays, critical levels of acute radiation exposure for various types of health outcomes (e.g. hematologic damage, organ failure, cancer, and lethality) are needed. Particularly useful is the minimum dose at which a given enhancement in occurrence of a disease relative to the average population occurs. We use dosimetry units for which 1 gray = 100 rad = 10^(-1) erg g^-1 absorbed. For reference, a chest X-ray delivers around 0.1 mGy. Since X-ray photons have small linear energy transfer, dE/dx, their “quality factor” or “biological effectiveness” is very close to unity, independent of energy, so dose in Gy is approximately the dose in sievert. The duration of exposures to flare X-rays of large fluence will be relatively short (10–60 min in most cases), so we restrict the discussion to evidence concerning acute human radiation-syndrome data.

At doses above about 0.5 Gy, summaries of a number of sources data relevant to acute radiation syndrome in humans are available [Alpen, 1998; Turner, 1995], as well as detailed specialized studies [Dickinson and Parker, 2002; Satoh et al., 1996; Dubrova et al., 1997; Dubrova, 2003]. There is general agreement that severe damage, primarily hematologic and without assured recovery, occurs around 1 Gy. Estimates of the whole-body acute lethal dose vary from 2 to 5 Gy [UNSCEAR, 2001; Alpen, 1998; Turner, 1995]. An upper limit to the radiation risk dose in an exposed individual is the ratio of the average spontaneous rate of mutations over a large number of genes to the induced mutation rate per Gy for low-LET (linear energy transfer) irradiation, recognizing that the mutation rate is highly variable among loci; this ratio is the mutation doubling dose. An estimate using 135 human genes for spontaneous rates and 35 mouse genes for induced rates [Sankaranarayanan and Chakraborty, 2000] gives a doubling dose of 0.8 Gy. This dose is for chronic (i.e. continual) irradiation, and a dose rate reduction of a factor of three is usually assumed for acute doses, suggesting a doubling dose of 0.3 Gy for acute irradiation. However this estimate remains uncertain and subject to various definitions [Sankaranarayanan and Chakraborty, 2000; UNSCEAR, 2001].

Most work on X-ray and gamma-ray radiation risk to exposed individuals (not genetic disease endpoints) comes from studies of carcinogenesis. There is little doubt that the incidence of cancer due to radiation-induced genomic instability rises with acute dose above 0.2–0.3 Gy. Below this dose there is continued debate whether there is a “linear-no-threshold” relation between risk and ionizing-radiation dose, or whether doses below about 0.1 Gy result in “adaptive response” causing endogenous DNA damage prevention and immune stimulation [Feinendegen, 2005]. Alternatively, it is also likely that risk at low doses is larger than the linear-no-threshold extrapolation, even increasing with decreasing dose, because of bystander effects, as reviewed in Hall [2004]. The situation is further complicated by the existence of a significant fraction of humans with predisposing mutations to cancers induced by ionizing radiation (e.g. Sankaranarayanan and Chakraborty, 2000). It is certain that mutations, often at significant loci [Sparrow et al., 1972; Sankaranarayanan, 1982; Forster et al., 2002], chromosomal abnormalities in blood lymphocytes [Violot et al., 2005], and clustered DNA damage [Sutherland et al., 2000] occur at much smaller doses in the range 0.01 to 0.1 Gy, but their impact on health risk has been difficult to assess because of the evidence for adaptive response at low doses. A compelling discussion by Brenner et al. [2003] argues that there is good human epidemiological evidence for increased cancer risk at an acute X-ray or gamma-ray dose of 0.05 Gy, and reasonable evidence for enhanced risk above 0.01 Gy, although the risk enhancement is in the 1–10% range.

Acute critical doses for significantly increased risk for other disease endpoints may also be of order 0.1 Gy or less, especially for hematological diseases. For example, the summary of delayed somatic (i.e. bodily) effects due to acute doses by Hansineier [2002] indicates that gastrointestinal tract syndrome (leading to loss of digestion ability, bleeding ulcers, and diarrhea) sets in at 0.1 Gy for X-rays.

The risk of a particular genetic-disease endpoint per Gy of irradiation is more difficult to estimate, since the disease-specific induced mutation rate varies greatly and most models assume mutation-selection equilibrium. Inspection of risk estimates in humans and animal models for a number of genetic-disease classes that include 26 human disorders encompassing 135 genes [Sankaranarayanan and Chakraborty, 2000] indicates that risks for genetic-disease endpoints at 0.1 Gy due to acute X-ray doses are probably much smaller than the risks for somatic disease in an exposed individual discussed earlier. These estimates include corrections for potential recoverability and concentrate on low-LET radiation, X-rays, and γ-rays. The results are given for chronic irradiation, even though they are based on experiments with high dose-rate irradiation.

The “dose-rate reduction factor” for acute doses is believed to be roughly a factor of three, a factor which is well established in studies of specific-locus mutations. Using this factor to correct the results, it is found that risks in terms of the excess over an average population for acute doses are typically 1% per Gy. But these results were typically obtained at equivalent acute doses in the range 0.5–3 Gy, so linearity of the above risk with dose cannot be assumed.

A review of evidence for enhanced mutation rates in human populations exposed to doses as low as 0.25 Gy in the Chernobyl accident and nuclear weapons tests in Kazakhstan is given in Dubrova [2003]. But the main point is that the risks for genetic disease endpoints at 0.1 Gy might be smaller than the risk for somatic disease in an exposed individual.

Given that the linear increase of cancer risk with dose for acute doses above 0.1 Gy seems unequivocal and that the threshold for delayed somatic-disease endpoints appears to also be about 0.1 Gy, we adopt this biological dose as an upper limit for significant risk increase due to X-rays, with the

![Figure 1. Acute biological doses behind polymer shielding (representative of current spacecrafts) due to a 10^{31} erg X-ray flare as a function of flare spectral index, p, and areal density, Σ. The dose is roughly independent of p for shields with areal densities smaller than about 2 g cm^-2. For larger shielding columns, the dose becomes sensitive to the spectral shape because more of the incident spectrum is attenuated, and hence reshaped, before being absorbed by the model water column. Areal densities of polymer in excess of 2 g cm^-2 are needed to reduce the X-ray dose to below our adopted maximum acceptable acute dose of 0.1 Gy.](image-url)
understanding that enhanced risk for several disease endpoints, but especially cancer [Brenner et al., 2003], may still be significant at lower doses. An important unresolved point is that adoption of a critical dose depends on the risk enhancement that one is willing to accept; our reading of the literature suggests a 10% enhancement in potentially fatal disease endpoints at 0.1 Gy, and less than 1% enhancement for genetic disease endpoints at this dose.

5. Results

5.1. Risk Estimate

We can now compare our adopted upper dose limit of 0.1 Gy with that received behind polymer shielding, which is representative of current spacesuit design, such as the space shuttle Extravehicular Mobility Unit [Ross et al., 1997]. We use pure carbon as a proxy for the mostly polymer construction of spacesuits because only the carbon atoms in polymers significantly absorb X-rays. The results, assuming a $10^{31}$ erg solar flare, are shown in Fig. 1.

The typical areal densities of spacesuit components (~0.5–1.5 g cm$^{-2}$) provide little protection during a large solar flare. A relatively common $10^{31}$ erg flare would deliver over 0.2 Gy behind the current spacesuit—twice our adopted upper limit. A thicker spacesuit could reduce this dose to 0.1 Gy, but larger flares do occur, albeit less often. Bolstering spacesuits simply decreases the frequency of dangerous doses and does not eliminate the threat.

What is the likelihood of being exposed to doses above 0.1 Gy? If we use for simplicity a typical flare spectral index of 3.5, we find that the approximate dose behind polymer shielding of areal density $\Sigma \gtrsim 0.3$ g cm$^{-2}$ is

$$D(\Sigma, W) = 0.21 W_{31} \Sigma^{-1.4} \text{Gy},$$

where $W_{31}$ is the flare energy release in units of $10^{31}$ erg, and $\Sigma$ is the areal density of the shielding material in g cm$^{-2}$. Using the flare energy-recurrence frequency relation given in the discussion of flare properties to eliminate $W_{31}$, the mean time between flares delivering at least a given dose $D_{0.1}$ behind an polymer shield of areal density $\Sigma$ is

$$\tau(\geq D) = 0.08 D_{0.1}^{0.7} \Sigma \text{yr},$$

where $D_{0.1}$ is the dose in units of 0.1 Gy. If we then assume that the time between flare events is typically much longer than the duration of exposure and that flares occur at random, the probability of exposure to a dose of at least the critical dose is

$$P(D, \Sigma) = 1 \times 10^{-3} D_{0.1}^{0.7} \Sigma^{-1}$$

per hour of EVA.

So in just 100 hours of EVA in the current spacesuit, an astronaut would accumulate a 10% risk of a dangerous exposure to solar flare X-rays.

5.2. X-rays Compared to Solar Energetic Particles

The previous section discusses only the risk due to photons from flares. The risk due to solar energetic particle events (SPEs) is certainly not negligible, but we argue that the risk during EVAs is significantly smaller than estimated here for hard X-rays from flares. The largest SPEs, such as the 14 July 2000, Feb 1956, and Aug 1972 events, had 1 AU fluences in the range $10^{10}$–$10^{11}$ cm$^{-2}$ [Miroshnichenko, 2003], although per-particle energies were 100 times smaller than for Galactic cosmic rays. Silberberg and Tso [1979] estimated the incidence of flares that produce SPEs with 1 AU doses greater than 1 Gy as about one per decade, similar to what we estimate for $10^{31}$ erg photon flares that produce 2 Gy doses, as shown in the Fig. 1. A similar frequency for SPEs with fluences above $10^{10}$ cm$^{-2}$ can be derived from the recorded number of large events [Reedy, 1996; Shea and Smart, 1990; Miroshnichenko, 2003], nitrate ice core reconstruction covering several centuries [McCracken et al., 2001], and probability models [Feynman et al., 1993, 2002]. In contrast, $10^{31}$ erg X-ray flares that also require substantial shielding are about 50 times more frequent.

Spacesuits are the last line of defense until shelter is reached, so exposures during a flare will depend on the ratio of the time to reach shelter to the time to deliver the total fluence. Figure 2 shows that the doses behind aluminum shielding are significantly smaller than that received inside spacesuits of the same areal density because of the strong dependence of the photoabsorption cross section on atomic number of the target material. But X-ray flares leave only 10–30 min to reach shelter before the total fluence is delivered.

Hard X-ray flares are impulsive, with rise times of minutes or less. The time to withdraw to adequately shielded shelter is very small. A reliable energetic hard X-ray flare precursor signature occurring more than an hour before the flare maximum would be needed for this purpose. There are many signatures that have been proposed as flare precursors [Martin, 1980; Simnett, 1993], such as UV brightening, soft X-ray enhancements, microwave radio signatures, Hα filament disturbances, strong magnetic shear, sunspot motions, and the beginnings of chromospheric mass ejections (CMEs), whose onset is now known to slightly precede an associated flare on average. Most of these precursors are only observable for less than a few minutes before the onset of the flare, so do not give sufficient warning and would require elaborate monitoring systems. Some radio precursors, such as polarization signatures, are observed up to tens of minutes before a flare, but are not observed in the majority of flares. Similarly, for all precursor signatures there are flares seen without the precursor and observations of “precursors” that are not followed by a flare; no precursors are necessary and sufficient [Golub and Pasachoff, 1997].

Even more seriously, none of these precursors are predictors of the energy release of the flare itself, so given the large frequency of flares that pose no biological hazard, use of these precursors would likely result in a large false-alarm rate.

Most probabilistic approaches for flare prediction are based on a combination of historical rate of flaring for a given sunspot classification group and additional information such as shear, magnetic topology, and previous large-flare activity [Gallagher et al., 2002]. A more recent Bayesian method relies only on flare event statistics [Wheatland, 2005]. These methods are most suited for probabilistic prediction of quantities like the number of flares of a certain class in a given year, but not for EVA hazard warnings. For example, the Bayesian method would have predicted a 20% probability for an X-class flare on 4 Nov 2003, using data up to one day before, including a highly clustered series of strong and weak flares in the week before, yet actually the most energetic flare in several decades was about to occur [Wheatland, 2005], a flare almost an order of magnitude more energetic than the model flare used in the calculations reported here.

Such prediction algorithms might be useful for policies requiring no EVAs in windows of a week or so, when the probability of a large flare can be somewhat more accurately estimated, but this could greatly curtail manned exploration, depending on where the threshold for significance is placed and the reliability of the prediction. For example, less reliable predictions would require lower thresholds for significance (and thus higher alarm rates) to maintain acceptable risk levels.
hours of accumulated EVA. The onset and duration of X-ray flares is rapid enough and possible precursors are unreliable enough that avoidance would be difficult. The simplest solution for X-ray protection on rover-based EVAs could be the inclusion of a mobile body shield to supplement the shielding provided by the spacesuit until shelter can be reached.

Acknowledgments. DSS was supported by the NSF Graduate Student Research Fellowship and Harrington Doctoral Fellowship Programs. JMS was supported by the NASA Exobiology Program, Grant NNX04GK33G. This work was carried out as part of the research of the NASA Astrobiology Institute Virtual Planetary Laboratory Lead Team, which is supported through the NASA Astrobiology Institute.

References
Alphen, E. L. (1998), Radiation Biophysics, Academic Press, San Diego.
Aschwanden, M. J. (1999), Nonthermal flare emissions, in The Many Faces of the Sun: A Summary of the Results from NASA’s Solar Maximum Mission, edited by K. T. Strong et al., pp. 273–300, Springer, NY.
Aschwanden, M. J., T. D. Tarbell, R. W. Nightingale, C. J. Schrijver, A. Title, C. C. Kankelborg, P. Martens, and H. P. Warren (2000), Time variability of the “quiet” Sun observed with TRACE. II. Physical parameters, temperature evolution, and energetics of extreme-ultraviolet nanoflares, Astrophys. J., 555, 1047–1065.
Badhwar, G. D. (1997), Deep space radiation sources, models, and environmental uncertainty, in Shielding Strategies for Human Space Exploration, edited by J. W. Wilson et al., pp. 17–29, NASA Conf. Pub. 3360.
Baker, D. N., S. G. Kanekal, X. Li, S. P. Monk, J. Goldstein, J. L. Burch (2004), An extreme distortion of the Van Allen belt arising from the “Halloween” solar storm in 2003, Nature, 432, 878–881.
Battaglia, M., P. C. Grigis, A. O. Benz (2005), Size dependence of solar X-ray flare properties, Astron. Astrophys., 439, 737–747.
Brenner, D. J., et al. (2003), Cancer risks attributable to low doses of ionizing radiation: Assessing what we really know, Pub. Natl. Acad. Sci. U.S.A., 100, 13761–13766.
Bromund, K. R., J. M. McTiernan, and S. R. Kane (1995), Statistical studies of ISEE 3/ICE observations of impulsive hard x-ray solar flares, Astrophys. J., 455, 733–745.
Cougnet, C., et al. (2004), Radiation exposure and Mission Strategies for Interplanetary Manned Missions (REMSIM), Earth, Moon, and Planets, 94, 279–285.
Crosby, N. B., M. J. Aschwanden, and B. R. Dennis (1993), Frequency distributions and correlations of solar X-ray flare parameters, Solar Phys., 143, 275–299.
Crosby, N. B., O. H. W. Siegmund, P. W. Vedder, and J. V. Vallera (1993), Extreme Ultraviolet Explorer deep survey observations of a large flare on AU Microscopii, Astrophys. J. Lett., 414, L49–L52.
Cucinotta, F. A., W. Schimmerling, J. W. Wilson, L. E. Peterson, G. D. Badhwar, P. B. Saganti, and J. F. Dicello (2001), Space Radiation Cancer Risks and Uncertainties for Mars Missions, Radiat. Res., 156, 682–688.
Cucinotta, F. A., W. Schimmerling, J. W. Wilson, L. E. Peterson, P. B. Saganti, and J. F. Dicello (2004), Uncertainties in estimates of the risks of late effects from space radiation, Adv. Sp. Res., 34, 1383–1389.
De Angelis, G., B. M. Anderson, W. Atwell, J. E. Nealy, G. D. Qualls, and J. W. Wilson (2004a), Astronaut EVA exposure estimates from CAD model spacesuit geometry, J. Radiat. Res., 45, 1–9.
De Angelis, G., M. S. Clowdsey, R. C. Singletary, and J. W. Wilson (2004b), Mars radiation environment model with visualization, Adv. Space Res., 34, 1328–1332.
Dickinson, H. O., and L. Parker (2002), Leukemia and non-Hodgkin’s lymphoma in children of male Sellafield radiation workers, Int. J. Cancer., 99, 437–444.
Dubrova, Y. E. (2003), Long-term genetic effects of radiation exposure, Mutat. Res., 544, 433–439.
Sutherland, B. M., P. V. Bennett, O. Sidorkina, and J. Laval (2000), Clustered DNA damages induced in isolated DNA in human cells by low doses of ionizing radiation, Proc. Natl. Acad. Sci. USA, 97, 103–108.

Turner, J. E. (1995), Atoms, Radiation, and Radiation Protection, 2nd ed., Wiley, NY.

UNSCEAR Hereditary Effects of Radiation (2001), United Nations, New York.

Veronig, A., M. Temmer, A. Hanslmeier, W. Otruba, and M. Messerotti (2002), Temporal aspects and frequency distributions of solar soft X-ray flares, Astron. Astrophys., 382, 1010–1080.

Violot, D., R. M’kacher, E. Adjadj, J. Dossou, F. de Vathaire, and C. Parmentier (2005), Evidence of increased chromosomal abnormalities in French Polynesian thyroid cancer patients, Eur. J. Nucl. Med. Mol. Imaging., 32, 174–179.

von Sonntag, C. (1987), The Chemical Basis of Radiation Biology, Taylor & Francis, NY.

Ward, J. F. (1999), Ionizing radiation damage to DNA: A challenge to repair systems, in Advances in DNA damage and repair: Oxygen radical effects, cellular protection, and biological consequences, edited by Dizdaroglu, M., and A. E. Karakaya, pp. 431–439, Kluwer/Plenum, NY.

Wheatland, M. S. (2005), A statistical solar flare forecast method, Space Weather, 3, S07003.

Wilson, J. W., M. S. Clowdsley, F. A. Cucinotta, R. K. Tripathi, J. E. Nealy, and G. de Angelis (2004), Deep space environments for human exploration, Adv. Sp. Res., 34, 1281–1287.

Woods, T. N., F. G. Eparvier, J. Fontenla, J. Harder, G. Kopp, W. E. McClintock, G. Rottman, B. Smiley, and M. Snow (2004), Solar irradiance variability during the October 2003 solar storm period, Geophys. Res. Lett., 31, L10802–10806.
