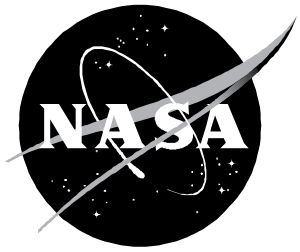
NASA Technical Paper 3643



Astronaut Protection From Solar Event of August 4, 1972

John W. Wilson and Francis A. Cucinotta
Langley Research Center • Hampton, Virginia

T. D. Jones
Oak Ridge National Laboratory • Oak Ridge, Tennessee

C. K. Chang
Christopher Newport University • Newport News, Virginia

National Aeronautics and Space Administration
Langley Research Center • Hampton, Virginia 23681-0001

April 1997

Available electronically at the following URL address: http://techreports.larc.nasa.gov/ltrs/ltrs.html

Printed copies available from the following:

NASA Center for AeroSpace Information
800 Elkridge Landing Road
Linthicum Heights, MD 21090-2934
(301) 621-0390

National Technical Information Service (NTIS)
5285 Port Royal Road
Springfield, VA 22161-2171
(703) 487-4650

Abstract

The dose rates in the blood-forming organ of a typical astronaut for four space shielding conditions are used to study the astronaut health effects of the solar particle event which began on August 4, 1972. This event was chosen as it was the most hazardous event for which detailed measurements have been made and for which dire predictions of the potential health effects have at times been suggested. The code used for health effects is the biological model developed for tactical nuclear weapons warfare survival of young adults in a 1g environment. We find the risks of early lethality to be very small especially if appropriate medical action (antibiotics and blood transfusions) is taken soon after the exposure. The primary concern would then be for the development of cancer later in life. Although leukemia could occur relatively soon after the exposure, the risk of solid tumors might be best controlled by using mature individuals for the mission, and thereby offset cancer risk by balancing life span remaining against the long latency periods associated with solid tumors. Use of genetic selection criteria could further reduce health risks during the mission. A possible space experiment to evaluate synergistic effects of the microgravity environmental stress and other space-related stress factors is discussed.

Introduction

From the earliest discussions of space exploration, the problem of protection against space radiations has been a concern (ref. 1); the possibility of adverse health effects from a large solar particle event was reinforced by the occurrence of a very large ground level event indicating arrival of high-energy particles on February 26, 1956 (ref. 2). The hazard posed by a possible large solar particle event was addressed in the Apollo missions, and the allowable exposures for this high-risk mission of great national importance were quite large (200 rem for blood forming organ and ocular lens, 700 rem for skin, and 980 rem for hands and feet where the older unit rem is equal to 1 cSv) and provided some balance against the other risks associated with the mission (ref. 3). For these short 2-week missions, these limits would allow the return of the astronauts for proper medical treatment. Although radiation health workers today find these numbers shocking, it was recognized at that time that the added mechanical complexity associated with providing added radiation protection from such events would dramatically increase the mechanical risks associated with the mission. Indeed, some deaths occurred from design complexity, but no known deaths from radiological exposure.

The radiations received during the Apollo missions were mainly from the galactic cosmic ray background, but on August 4, 1972, between the Apollo 16 and 17 missions, began the most significant solar particle event (mainly protons with small percentage of other ions) known even to the present time (refs. 4 and 5). This event has been studied in the past and many predictions of the possible health effects have been suggested. Most of these judgments were based on examination of human early radiation response data for single exposures and often based on responses to exposures during radiation therapy sessions with seriously sick individuals (ref. 6). Clearly, the interpretation of the event could be quite different if repair/recovery effects and specific space-related factors are taken into account.

As a result of the needs for tactical nuclear warfare planning, extensive databases of radiation response have been made for many animals, and models of the cellular and tissue level dynamics were derived by fitting the databases. These models are used herein to extrapolate the available human data to protracted exposures. We reexamine the August 4, 1972, event by using these recently published biological models. Clearly, the use of such models will enhance our understanding of large solar particle events and allow the development of practical methods for managing the associated risks.

Event of August 4, 1972

The first indication of an important solar event was the observation of an optical flare at 0621 UT on August 4, 1972. The associated coronal mass ejection (ref. 7) resulted in a significant accumulation of particle fluence by 0700 UT. The particle flux remained high for the next 11 hours over which the major accumulation of fluence of energetic particles occurred (fig. 1). Low-energy particles continued to arrive through 1000 UT on August 5, 1972. The doses behind a water equivalent shield of 0.4, 1, 5, and 10 cm were evaluated by Wilson and Denn (ref. 4) and are shown in figure 2. The shield thicknesses used in figures 2(a), (b), (c), and (d) correspond approximately to a space suit, pressure vessel, work area, and storm shelter, respectively. Although the exposures are known to have large dose gradients within

the body, we assume for evaluation of the risk that the exposures are uniformly distributed within the indicated organs. The actual risk estimates may be somewhat lower as a result of the nonuniform distribution within the organ. In estimating the dose to specific organs, self-shielding factors are included according to the approximations of Wilson and Denn (ref. 4).

In the past, risk was managed for missions following the Apollo program by setting dose limits to control both the early response and the cancer risk of the astronaut. The risk limits used from the Skylab mission through Shuttle operations saw little change (ref. 8), and maximum allowable exposure limits for career, yearly, and 30-day intervals were as shown in table 1. The 30-day limitation was specifically to control the early effects of radiation, since exposure recovery is in about 4 weeks. For example, the 30-day limitation is the controlling factor in shielding for short-duration space exploration to near objects like the moon wherein the protection of the astronaut requires a "storm shelter" of approximately 10 cm of polyethylene (fig. 2(d)). Meeting these limitations assures the control of adverse health risks of the astronaut but gives us no insight into the health risks of accidental exposure during future manned space exploration nor the development of a philosophy to appropriately deal with such an accident. These two items are discussed in the following sections.

Biological Response Models

Exposures at which significant health effects occur have been summarized from various sources by the National Council on Radiation Protection and Measurements (NCRP) and are shown in table 2. The dose associated with 50 percent mortality (LD_{50} value) is affected by the degree of medical support; intensive medical care can greatly increase the chances of survival (ref. 8). Recent practical experience was gained as a result of the Chernobyl accident where most exposures were characterized as a relative uniform whole-body exposure due to gamma rays and surface exposure an order of magnitude larger from beta emitters (ref. 9), which is somewhat similar to space exposure distributions (ref. 4). There were no deaths among those whose whole-body exposure was less than 2 Gy. All patients of exposures to the marrow system of doses greater than 2 Gy were given supportive care including isolation, antibiotics, and in extreme cases, transfusions and transplants (ref. 9). Only one death occurred for exposures between 2 and 4 Gy with intense supportive care.

The diagnostics of the Chernobyl accident relied on biological and physical dosimetry. The blood elements within exposed individuals were monitored within 12 hours of the accident and taken as an indication of the level of exposure. To understand this methodology, we show the kinetics of the marrow system in figure 3. The stromal cells reside on the bone surface and provide the substrate on which long-term stem cell populations normally reside. The stromal cells consist of those populations associated with the yellow marrow. The stromal cells provide growth factors which are responsible for the rate of cell propagation among the various stem populations. The long-term repopulating stem cells differentiate into lymphoid and myeloid stem populations. Humoral factors added by the stromal cells control the rate of progression of these differentiated stem populations. All other blood elements are produced by further differentiation among these two stem populations. Radiation injury to these stem and stromal populations will have its ultimate consequences in the peripheral blood. The time course of these peripheral blood elements (specifically the lymphocytes, neutrophils, and platelets) were used to estimate the level of exposure (ref. 9). Kinetic models of the stem and stromal populations based on animal studies are used in the present report to develop the understanding of the anticipated response of the astronaut to solar particle event exposure.

An analysis of experimental animal survival data demonstrated that very few animals ever died at 70 percent of their LD_{50} (lethal dose for 50 percent mortality) and very few ever survived at 130 percent of their LD_{50} (ref. 10). Further studies (ref. 11) used a much larger database and found the ratio of dose at 90 percent mortality to dose at 10 percent mortality (LD_{90}/LD_{10}) to be on the order of 2.0 or somewhat less for most species. (See fig. 4.) Thus, the range of exposure from survival to early death is very narrow.

The prompt-dose LD_{50} of each animal experiment depends on a host of conditions including strain, sex, age, diet, food supplements, cage care, bacterial environment, cage mates. In addition, each investigator-laboratory combination produces a distinct LD_{50} value even if all players attempt to replicate exactly the same experiment. To predict doses that will not kill any animals and doses that will have no survivors would be easy. However, poor accuracy would result from attempts to predict graded mortality over the short-dose range between the LD_{90} and the LD_{10} in one species of mouse from an LD_{50} and standard deviation (σ) from another species of mouse. A very small change in biological and physical factors can easily change a condition of total survival to total mortality. The specific conditions of the space environment is expected to play an important role in addition to the specific genetic makeup.

The animal database has been used to estimate a prompt-dose LD_{50} and probit slope (σ) within each

experiment (ref. 12). Mortality for protracted exposures within the same animal colony can then be analyzed or predicted on this common scale defined by stem and stromal cell kinetics provided that the only variables are related to the physical radiation environment, such as dose, dose rate, radiation quality, dose fractionation. Cell kinetics models should transfer reasonably well within a species, but the particular LD_{50} and probit slope estimates may vary twofold within different healthy strains of inbred animals—especially when studied by different investigators at different laboratories. Some mouse strains have an LD_{50} of 8 Gy and other strains treated the same way may have an LD_{50} of about 4 Gy (ref. 13). Similar factors are expected to be active in human exposures: the space environment and related space factors.

The human LD_{50} to marrow seems to be about 3 Gy for the atomic bomb survivors (ref. 14). But the LD_{50} for man can be increased with antibiotics, blood transfusions, and cytokine therapy to about 6 Gy. Intensive medical care including bone marrow or blood stem cell transplant could increase survivability to high levels as shown in table 2, but such medical procedures carry additional attendant risks (ref. 9) that may be modified by preconditioning to the space environments. Conversely Morris and Jones (ref. 11) have modeled 13 species of test animals and predicted the LD_{50} for man to be only about 1.8 Gy if confined in a cage under nonsterile conditions similar to that used for test animals. Such shifts may in fact be typical for space exposure and would be an important determinate of astronaut health. The genetic selection of astronauts and their conditioning may increase their radioresistance but space environmental factors, such as stress of close confinement, stress from microgravity, cabin atmosphere, may decrease their radioresistance. Food and water should give a survival advantage beyond that of the atomic bomb survivors but microgravity stress, confinement, and so forth may lead to greater risk. To itemize the various factors and to explore what the published experimental data say about the factors taken individually would be useful.

Cancer incidence from radiation exposure in inbred animals is even more variable than acute radiation injury. Strains where almost all animals get cancer (without any exposure) and low-cancer strains exist. Analyzing animal cancer data in terms of cell kinetics has not been attempted but would be of great value in understanding the solar particle exposure effects. Cancer risk estimation for mice should be predictable in terms of cell kinetics and a risk coefficient valid for the exposed animals at risk. Such an analysis could indicate the approximate degree of risk to man and assess the general degree of validity to be expected from the model within a mouse's lifespan of about 2 years.

Dose Protraction Effects in Risk Management

The primary early radiation syndrome depends on the exposure level (ref. 6). The expected exposures of deep organs is a few greys in space exploration, and the corresponding early biological effects would be dominated by the loss of stem cell populations within the bone marrow and the associated stroma cells. The initial radiation injury occurs in individual irradiated cells but ultimately shows through the loss of cells from the tissue system for which intercellular communication (cytokines or growth control factors, fig. 3) demands increased replacements to the tissue system. In this respect the injury to the stromal cells plays an important role as a primary source of cytokine production for the proliferation of stem cells, their progeny, and control of the amplifying divisions and differentiation processes in maturing cells that are progeny of the hematopoietic stem cells. The ability to replace lost cells depends on the sustained damage to the stem and stroma population. The repair rates at the cellular level are typically minutes or hours, whereas the tissue recovery period is typically days to weeks. For the August 1972 event, the exposure time is long compared with cellular repair processes but short compared with the time required for organ recovery. Because the exposure time is long compared with repair rates, the cellular repair processes are anticipated to greatly reduce the injury of the exposure anticipated for this event.

Risk of early lethality to the astronaut is related largely to depression of the immunological system and other blood elements through cell killing of various stem cell or the stromal cell populations (fig. 3). Because bone marrow doses are not expected above a few greys, the primary treatment is to supplement the immunological system with antibiotics. Clearly, the efficacy of this action for space-stressed astronauts needs to be understood. In developing such an understanding, we use the models developed for application to military operations to study the effects of protracting the dose over a period of many hours.

We use the model for early lethality as adapted by Young, Jones, and Morris (ref. 15) to examine the repair/recovery effects in humans due to rather large exposures. Figure 5 shows the mortality for a 2-Gy dose to the bone marrow by 250 kVp X rays (the 250 kVp refers to the peak voltage of alternating current X-ray machine) delivered as multiple equal fractions 1 hour apart. Each fraction was given in a 15-min exposure. Mortality can be quite large when received in a single high dose rate expose. (Note that Jones estimates that the bone marrow LD_{50} of 250 kVp X rays is 2.15 Gy, whereas that of ^{60}Co gamma rays is 2.95 Gy.) Supportive medical treatment is expected to allow survival as shown in the figure. As the

number of fractions is increased, the mortality drops dramatically to less than 10 percent (even without medical treatment) beyond 15 fractions (or equivalently 15 hours). The stem and stroma cell survival at the end of each fractionated exposure is shown in figure 6. Stem cell survival for the single 2-Gy bone marrow dose is very low (much less than 10 percent). As the number of fractions is increased, the stem cell survival shows a dramatic increase approaching 40 percent. Likewise, similar changes in the stromal cell population and repopulation reduces the mortality for the 20 fractions at 2 Gy to 10 percent. Clearly, cellular repair and repopulation are effective in reducing the risk when the exposure is highly fractionated with adequate time between fractions for repair. The stem and stroma cell populations during and after exposure to a 2-Gy bone marrow dose given as 20 fractions are shown in figure 7. The recovery period in this case is about 2 to 4 weeks.

The equivalent dose for the solar event is uncertain and we have assumed the equivalence to 250 kVp X rays which may be somewhat conservative. Figure 8 shows the estimated stem and stroma populations within the astronaut for the August 4, 1972, exposures with various shield configurations. Although substantial decreases in the stem and stroma populations occur for the space suit, the effects are clearly reduced within the pressure vessel and further reduced to a small perturbation when shielded within an equipment area. The value of having a shelter to further protect the astronaut is not clear from the present results on early lethality. Figure 9 shows mortality for a 2-Gy exposure as a function of dose rate corresponding to exposure periods of 15 min to 20 hours. Also shown in figure 9 are the results of the exposures based on the August 4, 1972, event within a space suit and pressure vessel at the appropriate average dose rate. If the astronaut remained in the work area or went to the storm shelter, then no real threat to the survival of the astronaut would exist as would be true for the space suit or pressure vessel if appropriate medical treatment was given. The corresponding stem cell survival is shown in figure 10 as a function of the exposure period along with similar results for exposures at 1 Gy. The corresponding mortality for the astronaut for a 1-Gy exposure protracted over several hours or more is very large.

The estimated total cancer risk for young adult males for the August 4, 1972, event in the case of accidental exposure shows strong protraction effects as seen in figure 11. The fatal cancer risk would be about a factor of 2 smaller. The much smaller leukemia risk is shown in figure 12. Leukemia would appear in 4 to 10 years after exposure and would be the most meaningful risk for a middle-aged adult population. Of lesser importance to a middle-aged population are the solid tumors which do not appear for 20 to 30 years.

Operations on the lunar surface would reduce the accidental exposure levels by at least a factor of 2 because of shielding below the local horizon. Added protection could be found by using a cliff or crater wall. The probability of early death is probably negligible in these cases. Furthermore, protection would be gained from the Martian atmosphere for Mars surface operations.

The present calculations were meant to apply to healthy adult individuals on future NASA exploration missions. Because the models are developed for 1*g* ground-based data, the specific stress associated with space travel is not accounted for in the model. It would seem desirable to compare the model and its associated database with exposures in space of small mammals as a means of testing the effects of microgravity and other space stress factors.

Small Mammal Experiment

Many factors affect the LD_{50} of human and animal populations. Some of the factors (genetic, nutrition, general health, bacterial environment) have been tested in laboratory experiments, but specific space-related stress factors have not been tested. Such a test could be made with relative ease because large health risks are involved and good statistics could be obtained with relatively small populations. In addition to the relative small number of mice, the duration of the experiment would be only a few weeks during the recovery period. Follow-up observations for cancer induction could be made but may be of little meaning with such a small population.

Concluding Remarks

On the basis of a biological response model for use in estimating biological effects in young adult males and the estimated flux spectra of the August 4, 1972, solar event, repair and recovery play an important role in determining the early response in solar particle event exposures. In the present analysis, such events are unlikely to result in early death in the case of accidental exposure if adequate supportive medical procedures are provided. Although the August 4, 1972, event is the largest event for which detailed observations are available, some uncertainty still exists of the size of the event the astronaut may ultimately encounter. Indeed, a reevaluation of the flux spectra of the August 4, 1972, event has been suggested because the satellite data may exhibit some saturation and the spectral shape above 60 MeV was not measured. Even if a larger event is indicated, the reduction of dose on the lunar surface is likely not to change these conclusions for lunar-based operations.

The marrow cell kinetics models that have been developed are based on uniform marrow doses and

conventional sources of X/gamma photons and fission/fusion neutrons. Hematopoietic stem cells circulate freely within the body and are usually treated as having random distribution within hematopoietic marrow. In contrast, marrow stroma is typically described as the cytokine-producing yellow marrow that serves as a necessary and sufficient substrate upon which the hematopoietic stem cells can proliferate. Highly nonuniform exposures to marrow of the body would seem to ensure that sparred stromal tissues could serve as a favorable microenvironment upon which circulating stem cells could attach and proliferate. Because several studies have indicated that restoration of normal myelopoiesis/hematopoiesis can be monoclonal, it seems that nonuniform marrow irradiations could, in principle, greatly amplify the probability of survival beyond that expected from molecular repair and compensatory repopulation associated with protracted irradiations. Published experimental data on nonuniform marrow irradiation should be analyzed in terms of cell kinetics. The result should serve to either realistically model nonuniform exposures or to design a minimum set of simple mouse experiments that will elucidate the process adequately.

Another source of uncertainty has been the use of existing model coefficients evaluated from conventional therapeutic and environmental sources for applications to space radiation environments. Because the rate constants in the cell kinetics models have been specified, evaluated, and validated in terms of target size and hit effectiveness considerations such as DNA per cell and electron track density associated with radiation tissue interactions, it is desirable to use published biophysical data to develop more realistic cellular rate constants for cell injury. These two refinements may change current conclusions in a consistent direction or they may tend to offset each other, depending upon the way that existing calculations are applied to space missions. Clearly, the importance of space missions, the value placed on human life, the visibility of space endeavors, and the need to optimize protection, shielding, and survival all mandate that continued emphasis should be placed on development of more realistic models of injury and response.

A further uncertainty in the present model is the effects of space-related stress factors, especially microgravity. Changes in the biological response due to shifts in LD_{50} value are noted in animal experiments from various factors such as strain, sex, age, diet, food supplements, general care, social factors. The microgravity environment is known to cause perturbations in the blood system that would undoubtedly result in increased radiosensitivity but the effects of such pertinent factors are not yet quantified by space experiments. A small mammal experiment with limited numbers of animals could provide valuable information on the space-related stress factors.

The outcome of an improved understanding of the early response to solar event exposures may reduce the corresponding shielding requirements because repair and recovery of the blood-forming organ appear unlikely to result in any serious early response for this organ. The primary protection concern may then be cancer induction. Such an outcome could have an impact on the shielding requirements in future deep-space missions and potentially reduce the mission cost.

NASA Langley Research Center
Hampton, VA 23681-0001
December 10, 1996

References

1. Armstrong, Harry; Haber, Heinz; and Strughold, Hubertus: Aero Medical Problems of Space Travel—Panel Meeting, School of Aviation Medicine. *J. Aviation Med*., vol. 20, no. 6, Dec. 1949, pp. 383–417.

2. Schaefer, Hermann J.: Cosmic Ray Dosage During the Giant Solar Flare of February 26, 1956. *J. Aviation Med*., vol. 28, no. 4, Aug. 1957, pp. 387–396.

3. Billingham, John; Robbins, Donald E.; Modisette, Jerry L.; and Higgins, Peter W.: Status Report on the Space Radiation Effects on the Apollo Mission. *Second Symposium on Protection Against Radiations in Space*, Arthur Reetz, Jr., ed., NASA SP-71, 1965, pp. 139–156.

4. Wilson, John W.; and Denn, Fred M.: *Preliminary Analysis of the Implications of Natural Radiations on Geostationary Operations*. NASA TN D-8290, 1976.

5. Nealy, J. E.; Simonsen, L. C.; and Striepe, S. A.: Natural Radiation Environment Fluence and Dose Predictions for Missions to the Moon and Mars. *Proceedings of the Topical Meeting on New Horizons in Radiation Protection and Shielding*, American Nuclear Soc., Inc., 1992, pp. 181–187.

6. Langham, Wright H., ed.: *Radiobiological Factors in Manned Space Flight.* National Academy of Sciences, National Research Council, 1967.

7. Kahler, S. W.; Sheeley, N. R., Jr.; Howard, R. A.; Michels, D. J.; Koomen, M. J.; McGuire, R. E.; Von Rosenvinge, T. T.; and Reames, D. V.: Associations Between Coronal Mass Ejections and Solar Energetic Proton Events. *J. Geophys. Res*., vol. 89, Nov. 1, 1984, pp. 9683–9693.

8. National Council on Radiation Protection and Measurements: *Guidance on Radiation Received in Space Activities*. NCRP Rep. No. 98, July 31, 1989.

9. Guskova, A. K.; Barabanova, A. V.; Baranov, A. Y.; Gruszdev, G. P.; Pyatkin, Y. K.; Nadezhina, N. M.; Metlyaeva, N. A.; Selidovkin, G. D.; Moiseev, A. A.; Gusev, I. A.; Dorofeeva, E. M.; and Zykova, I. E.: Appendix—Acute Radiation Effects in Victims of the Chernobyl Nuclear Power Plant Accident. *Sources, Effects and Risks of Ionizing Radiation—United Nations Scientific Committee on the Effects of Atomic Radiation: 1988 Report to the General Assembly, With Annexes*, United Nations, 1988, pp. 613–647.

10. Jones, T. D.: Hematologic Syndrome in Man Modeled From Mammalian Lethality. *Health Phys*., vol. 41, July 1981, pp. 83–103.

11. Morris, M. D.; and Jones, T. D.: Hematopoietic Death of Unprotected Man From Photon Irradiations—Statistical Modeling From Animal-Experiments. *Int. J. Radiat. Biol.*, vol. 55, no. 3, 1989, pp. 445–461.

12. Morris, Max D.; Jones, Troyce D.; and Young, Robert W.: A Cell Kinetics Model of Radiation-Induced Myelopoiesis—Rate Coefficient Estimates for Mouse, Rat, Sheep, Swine, Dog and Burro Irradiated by Photons. *Radiat. Res.*, vol. 135, no. 3, Sept. 1, 1993, pp. 320–331.

13. Morris, M. D.; and Jones, T. D.: A Comparison of Dose-Response Models for Death From Hematological Depression in Different Species. *Int. J. Radiat. Biol.*, vol. 53, no. 3, 1988, pp. 439–456.

14. Levin, S. G.; Young, R. W.; and Stohler, R. L.: Estimation of Median Human Lethal Radiation-Dose Computed From Data on Occupants of Reinforced-Concrete Structures in Nagasaki, Japan. *Health Phys*., vol. 63, no. 5, 1992, pp. 522–531.

15. Young, Robert W.; Jones, Troyce D.; and Morris, Max D.: Dose-Rate RBE Factors for Photons—Hematopoietic Syndrome in Humans vs. Stromal Cell Cytopenia. *Health Phys*., vol. 67, no. 5, Nov. 1, 1994, pp. 495–508.

Table 1. Ionizing Radiation Exposure Limits

[From NCRP 98 (ref. 8)]

Exposure interval	Dose equivalent, Sv, for—		
	Skin	Ocular lens	Bone marrow
30 days	1.5	1	0.25
Annual	3	2	0.50
Career	6	4	[a]1–4

[a]Varies with gender and age at initial exposure.

Table 2. Exposure Levels for Single High-Dose Rate Exposure at Which Health Effects Appear in Healthy Adults

[From NCRP 98 (ref. 8)]

Health effect	Dose, X or gamma radiation, Gy
Blood count changes in population	0.15–0.25
Blood count changes in individual	0.5
Vomiting, effective threshold	1.0
Mortality, effective threshold	1.5
LD_{50} with minimal supportive care	3.2–3.6
$LD_{50}$with supportive medical treatment	4.8–5.4
LD_{50} with autologous bone marrow or blood stem cell transplant	11.0

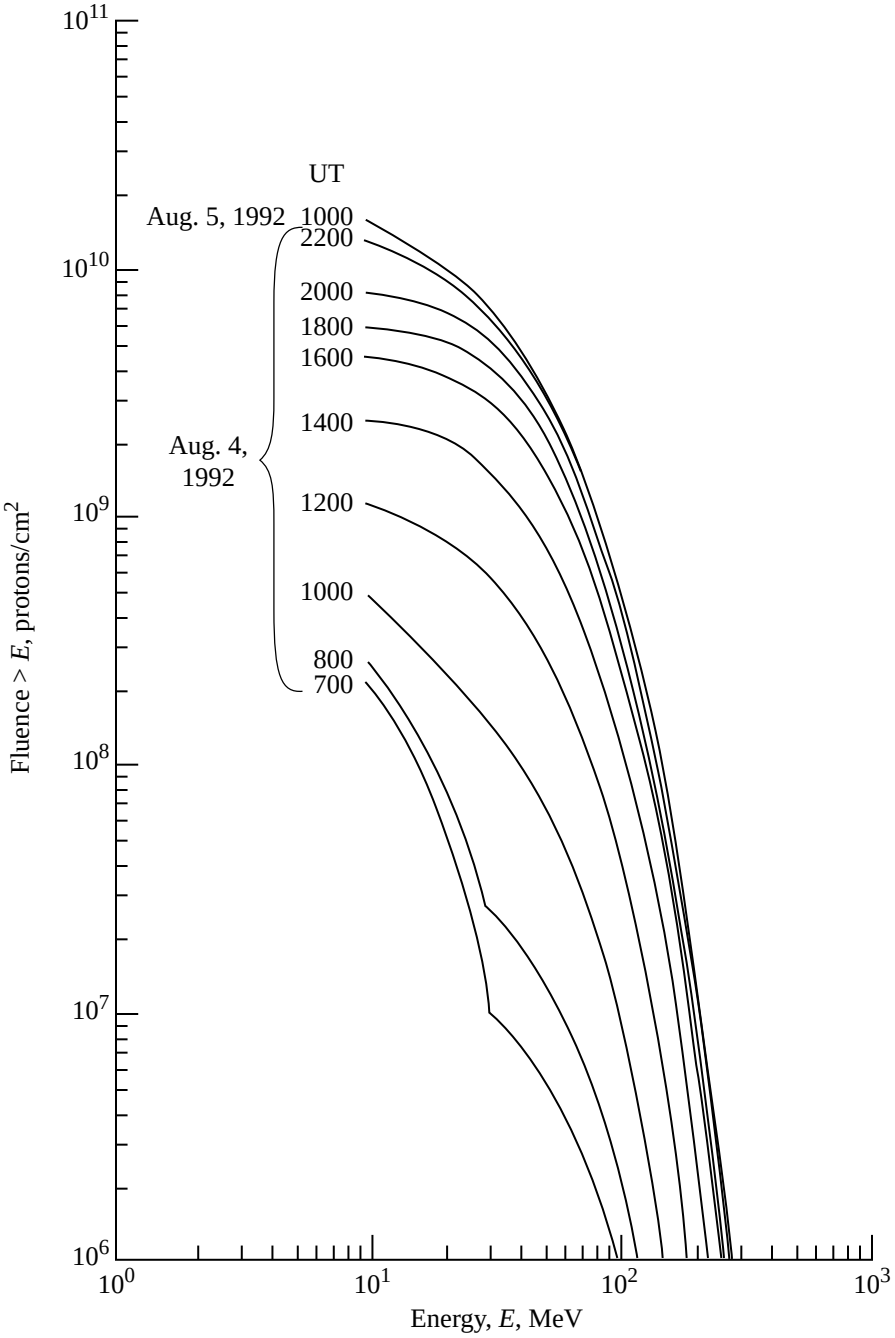
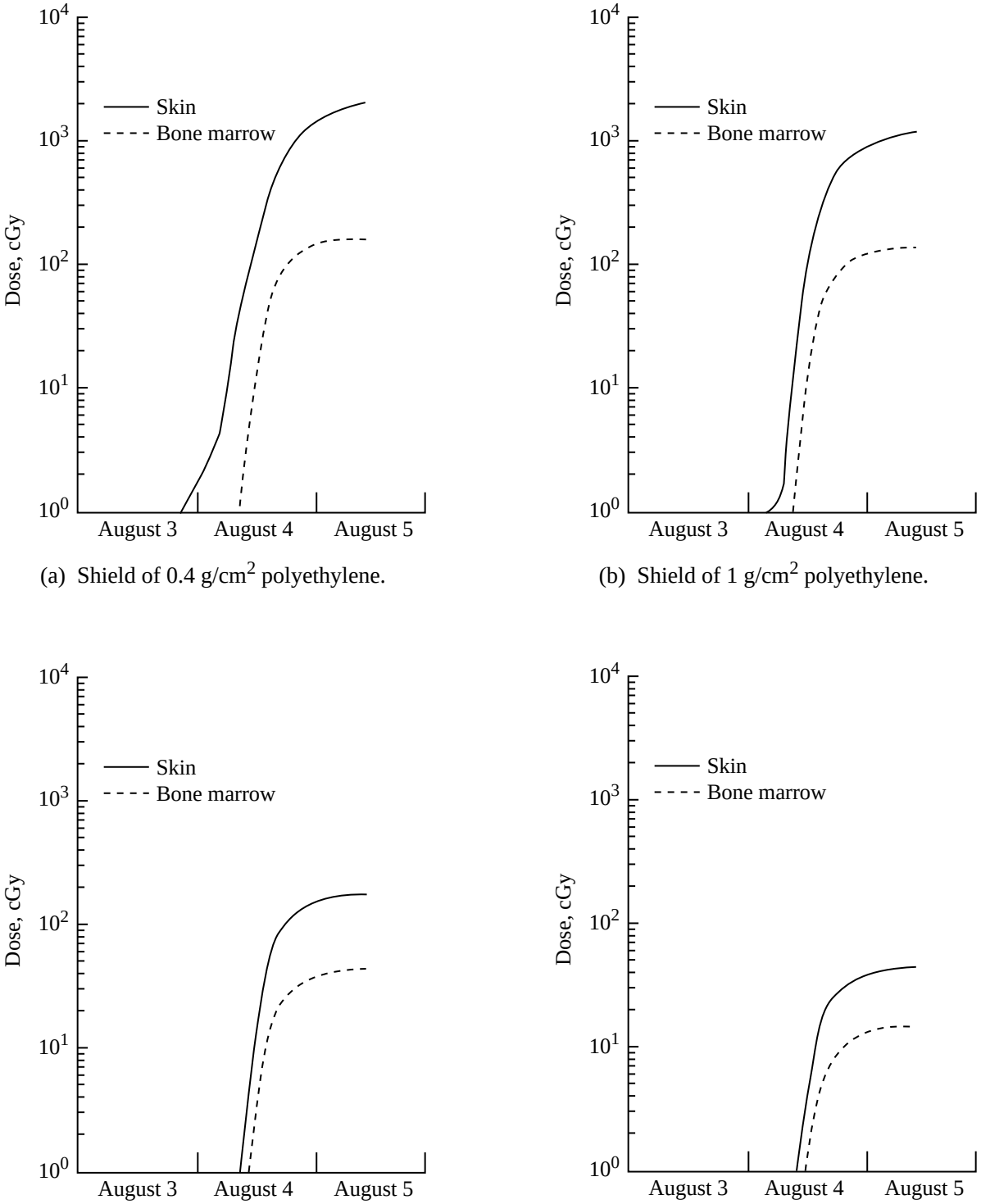


Figure 1. Fluence from August 1972 solar event as function of time and energy.



(a) Shield of 0.4 g/cm^2 polyethylene.

(b) Shield of 1 g/cm^2 polyethylene.

(c) Shield of 5 g/cm^2 polyethylene.

(d) Shield of 10 g/cm^2 polyethylene.

Figure 2. Dose to shielded astronaut for August 1972 event.

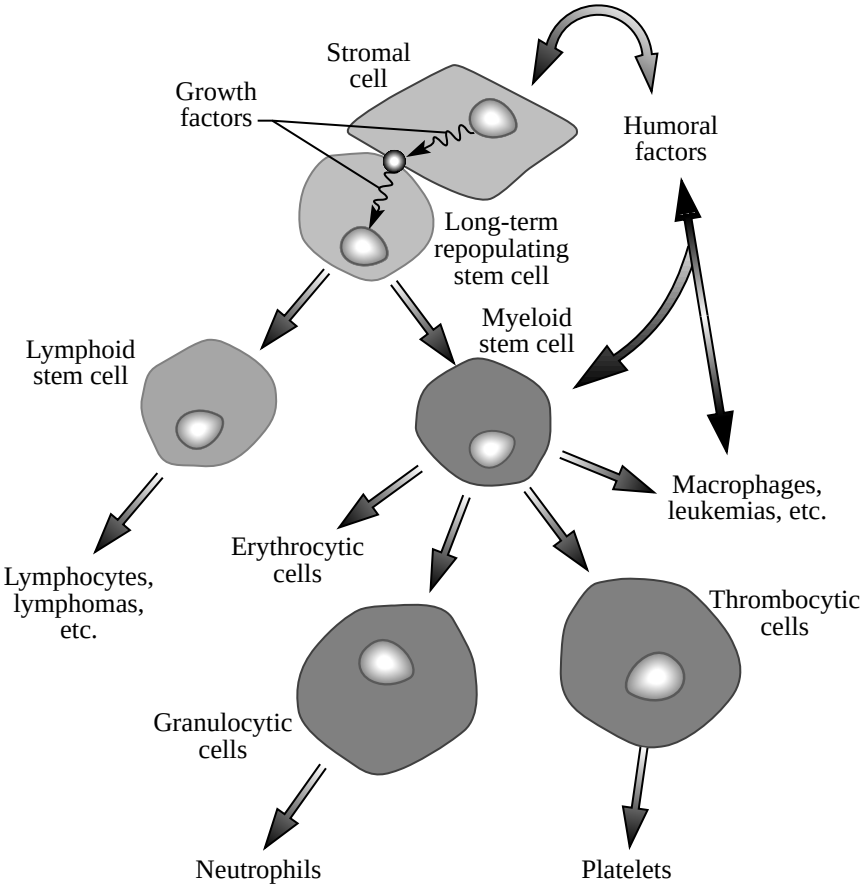


Figure 3. Cell populations and humoral factors controlling peripheral blood elements.

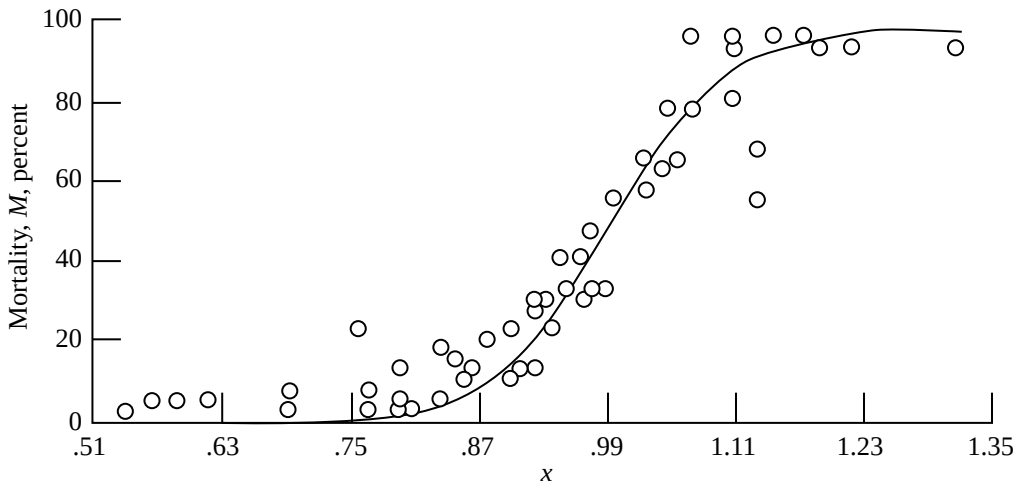


Figure 4. Mortality as function of bone marrow dose for various test animals (ref. 10). $M = \frac{A}{1 + B\,e^{-cx}}$ where ($A = 98.62$, $B = 5.09 \times 10^7$, and $C = 17.93$); $x = \frac{D}{\mathrm{LD}_{50}}$.

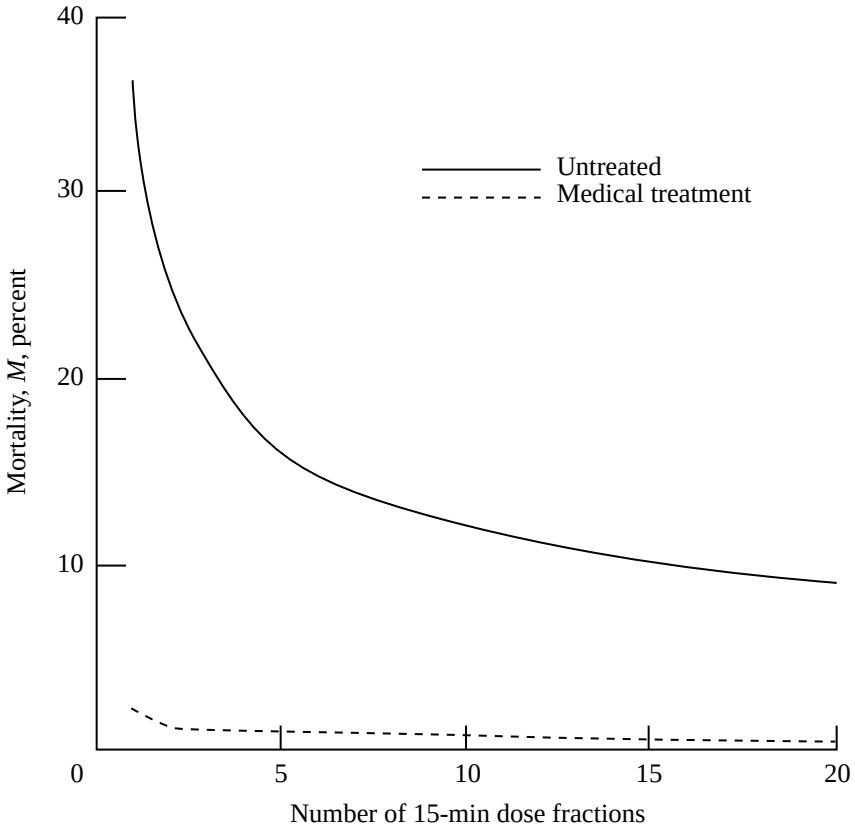


Figure 5. Mortality for hourly fractionated 2-Gy bone marrow dose as function of number of fractions.

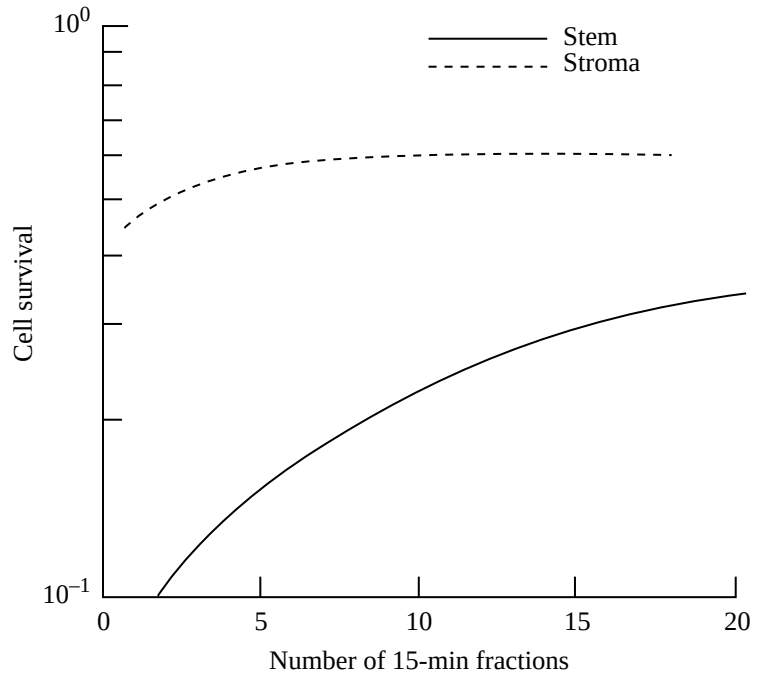


Figure 6. Stem and stroma cell survival at end of exposure period for a fractionated 2-Gy total bone marrow dose.

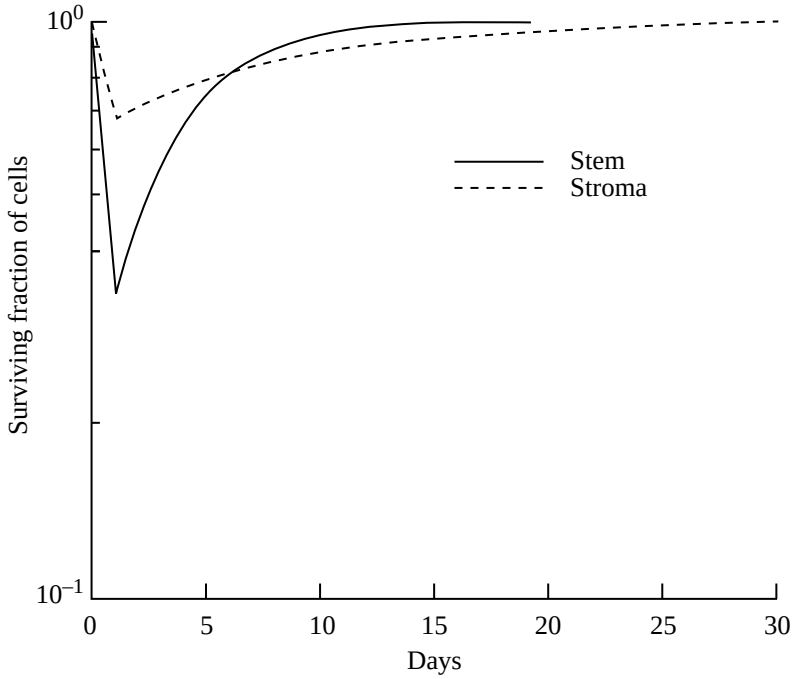


Figure 7. Surviving fraction of stem cell population for 20 hourly fractions of 2-Gy total bone marrow dose showing recovery period.

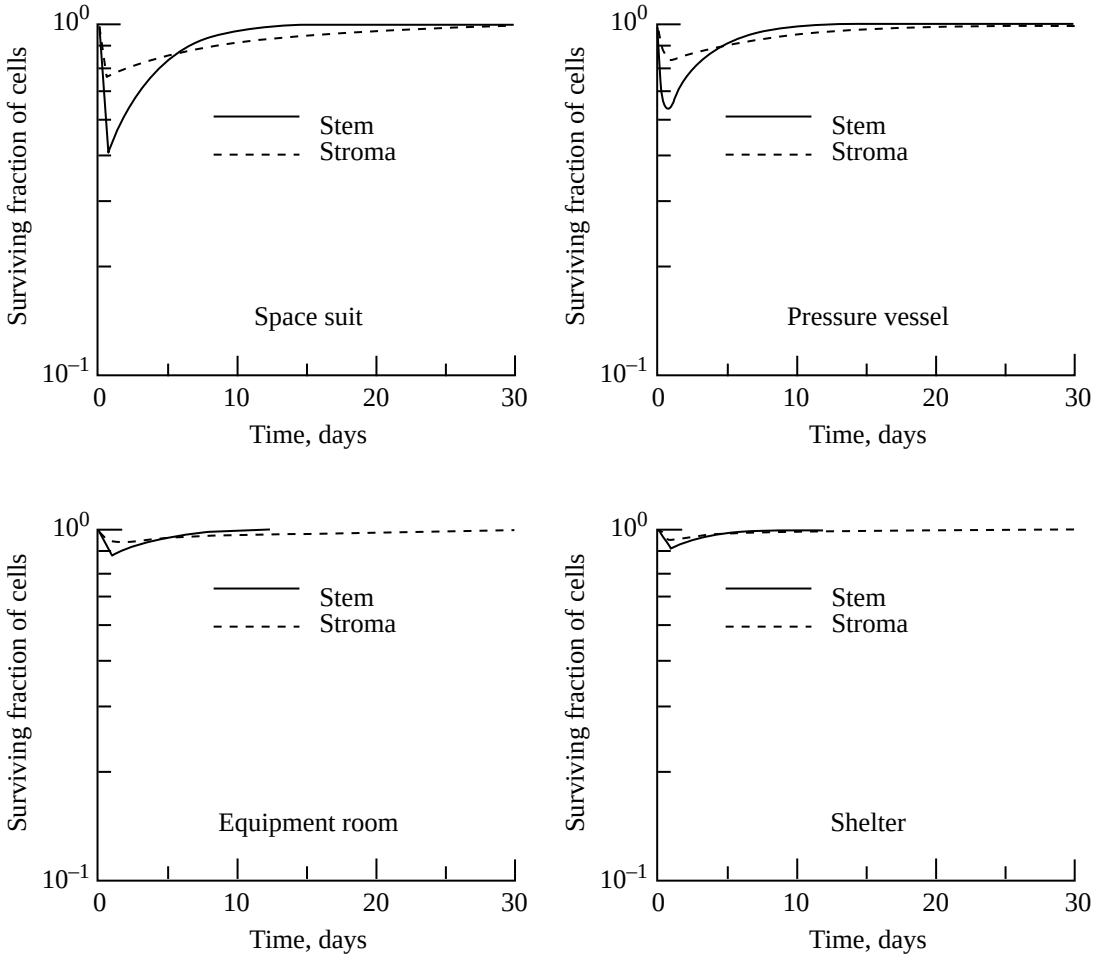


Figure 8. Bone marrow cell populations for August 4, 1972, exposures.

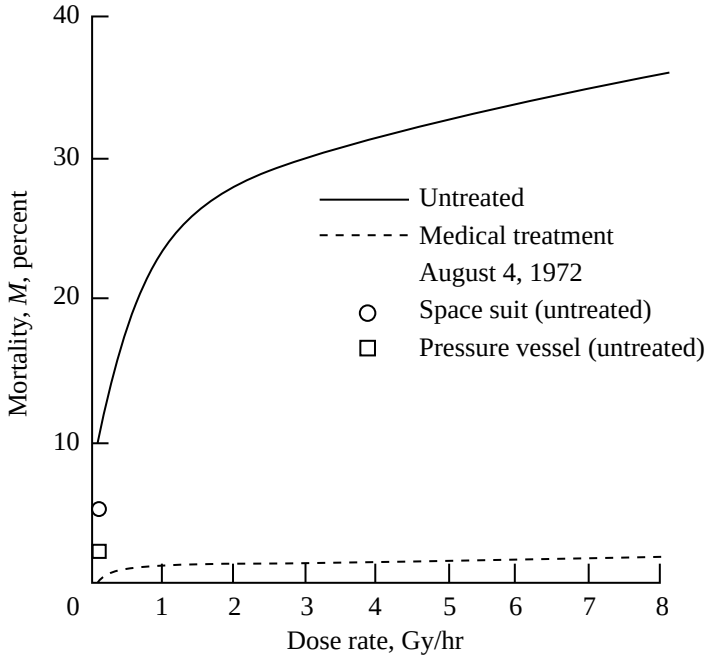


Figure 9. Mortality following 2-Gy bone marrow dose as function of dose rate. Exposures to August 4, 1972, event in space suit and pressure vessel at event average dose rate also shown.

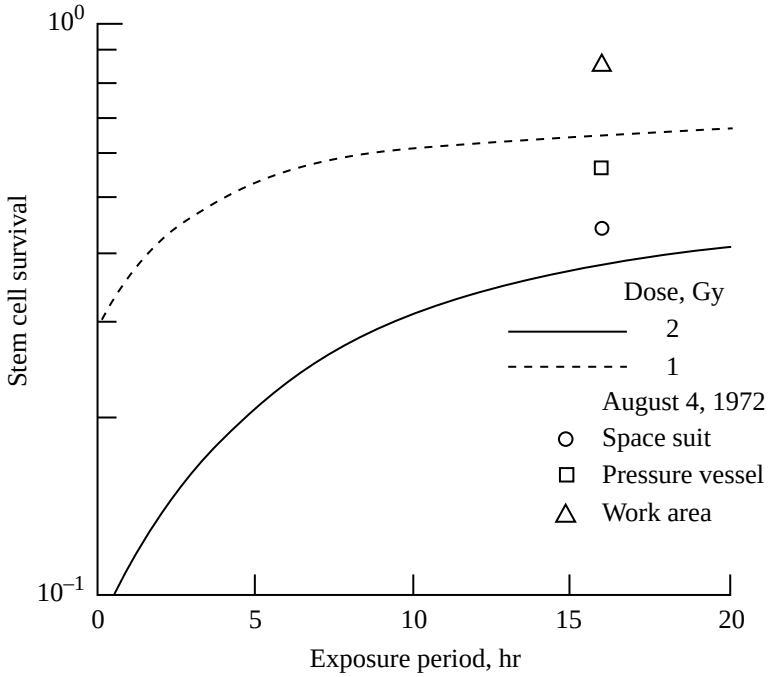


Figure 10. Stem cell surviving fraction for 1- and 2-Gy bone marrow doses as function of exposure period. Results for August 4, 1972, event for several shield configurations also shown.

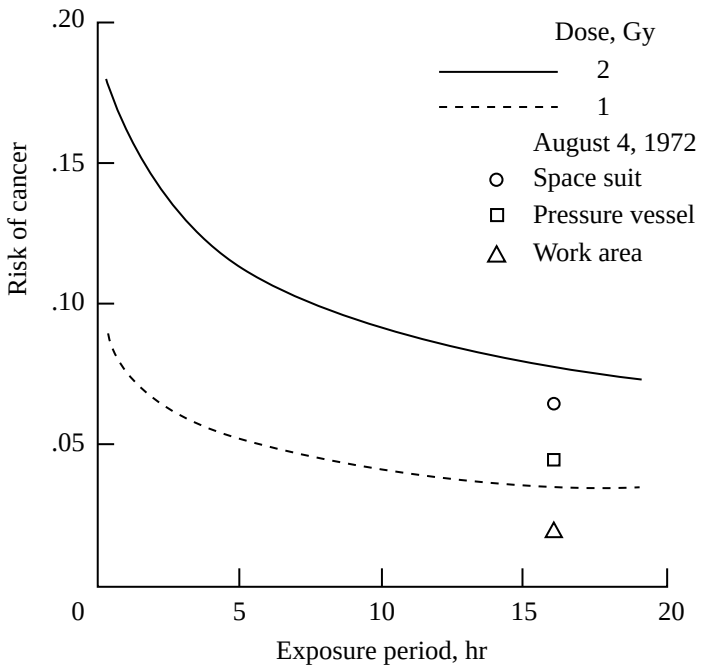


Figure 11. Cancer risk for 1- and 2-Gy bone marrow doses as function of exposure period. Risk from August 4, 1972, event for various shield configurations also shown.

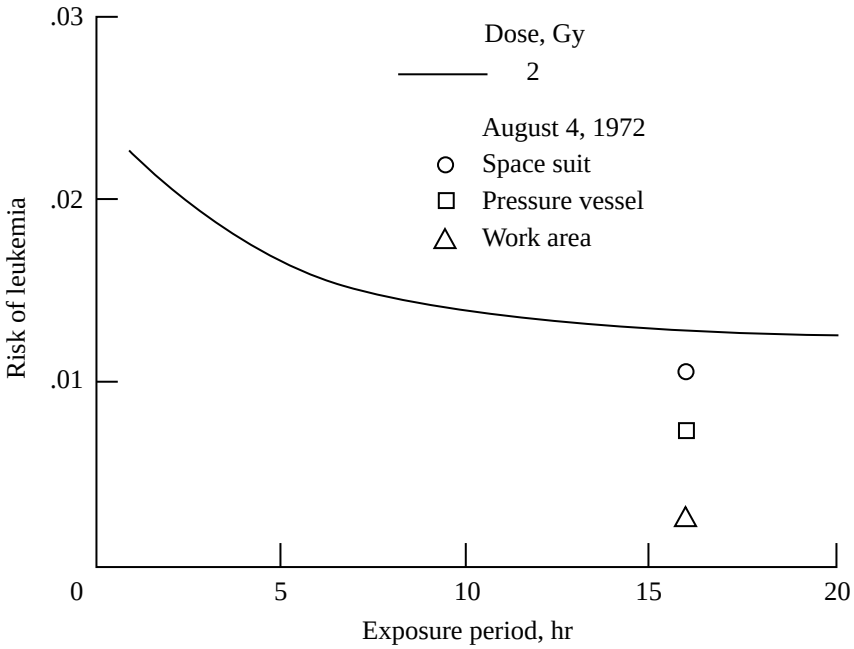


Figure 12. Risk for leukemia from 2-Gy bone marrow dose as function of exposure period. Risk for August 4, 1972, event for several shield configuration also shown.

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. AGENCY USE ONLY *(Leave blank)*	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	April 1997	Technical Paper

4. TITLE AND SUBTITLE
Astronaut Protection From Solar Event of August 4, 1972

5. FUNDING NUMBERS
WU 199-45-16-11

6. AUTHOR(S)
John W. Wilson, Francis A. Cucinotta, T. D. Jones, and C. K. Chang

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
NASA Langley Research Center
Hampton, VA 23681-0001

8. PERFORMING ORGANIZATION REPORT NUMBER
L-17590

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)
National Aeronautics and Space Administration
Washington, DC 20546-0001

10. SPONSORING/MONITORING AGENCY REPORT NUMBER
NASA TP-3643

11. SUPPLEMENTARY NOTES
Wilson and Cucinotta: Langley Research Center, Hampton, VA; Jones: Oak Ridge National Laboratory, Oak Ridge, TN; Chang: Christopher Newport University, Newport News, VA.

12a. DISTRIBUTION/AVAILABILITY STATEMENT
Unclassified–Unlimited
Subject Category 52
Availability: NASA CASI (301) 621-0390

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*
The dose rates in the blood-forming organ of a typical astronaut for four space shielding conditions are used to study the astronaut health effects of the solar particle event which began on August 4, 1972. This event was chosen as it was the most hazardous event for which detailed measurements have been made and for which dire predictions of the potential health effects have at times been suggested. The code used for health effects is the biological model developed for tactical nuclear weapons warfare survival of young adults in a 1*g* environment. We find the risks of early lethality to be very small especially if appropriate medical action (antibiotics and blood transfusions) is taken soon after the exposure. The primary concern would then be for the development of cancer later in life. Although leukemia could occur relatively soon after the exposure, the risk of solid tumors might be best controlled by using mature individuals for the mission, and thereby offset cancer risk by balancing life span remaining against the long latency periods associated with solid tumors. Use of genetic selection criteria could further reduce health risks during the mission. A possible space experiment to evaluate synergistic effects of the microgravity environmental stress and other space-related stress factors is discussed.

14. SUBJECT TERMS
Solar-particle event; Dose rates; Biological response models; Shielding

15. NUMBER OF PAGES
16

16. PRICE CODE
A03

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
Unclassified	Unclassified	Unclassified	