

Mortality from Late Central Nervous System Effects Among US Astronauts 1959-2022

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Article

Keywords: neurology, spaceflight, radiation

Posted Date: September 29th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10438220/v1>

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Additional Declarations: No competing interests reported.

Abstract

Objective

Late Central Nervous System (CNS) effects are currently under surveillance by NASA as a possible long-term health consequence of space radiation. Despite decades of follow-up, the small size of the astronaut cohort and long latency of these conditions have made conventional risk estimation difficult, leaving a gap in evidence to support deep-space missions to the Moon and Mars, where radiation exposures will exceed those of low Earth orbit.

Methods

We use data on NASA astronauts from the Astronaut and Spaceflight Participant Archive database linked to mortality information from the National Death Index to calculate standardized mortality ratios from late CNS effects from 1959 to 2022. We introduce a Bayesian surveillance system that determines how many deaths in the future are required to obtain various levels of probability for reductions or increases in mortality risk.

Results

Standardized mortality ratio (SMR) point estimates are below parity for both men and women. For males, $SMR = 40$ (95% CI = 5–145). For females $SMR = 0$ (95% CI = 0–4611) with only 0.08 expected. The surveillance system shows that, over 10 years, 17 total deaths for males and 3 for females would be required to achieve a 97.5% probability that $SMR > 100$.

Conclusions

The confidence intervals for both SMRs convey considerable uncertainty in the estimates. The Bayesian framework transforms this uncertainty into prospective surveillance thresholds, quantifying how many future deaths would signal excess or reduced risk and providing a transparent, updatable tool for monitoring rare outcomes in small cohorts.

1. INTRODUCTION

Human spaceflight exposes astronauts to several ubiquitous hazards, including the deep space radiation environment. Galactic cosmic rays (GCR) and solar particle events (SPEs) present a risk of both acute and late effects. Acute effects include sensorimotor disruption, possible motor neuron effects, and potential sleep disturbances¹.

The most widely recognized late effects include cancer and cardiovascular disease, but space radiation also poses a risk to the central nervous system (CNS). The National Aeronautics and Space Administration (NASA) has identified late neurological disorders as long-term health concerns for astronauts, including Alzheimer's disease, dementia, premature aging, and white matter disease^{2,3}. These concerns arise from protracted low-dose-rate exposures to protons, HZE particles, and neutrons at doses estimated for design reference missions (approximately 0.5-1 Gy)².

Evidence for these risks comes from both experimental animal and observational human sources: experimental studies showing neurocognitive and behavioral effects in rodents exposed to lower doses of HZE ions and detrimental CNS changes observed in patients treated with high-dose gamma-ray or proton beam radiation³. Observational studies have also documented white matter changes in astronauts post-flight via diffusion magnetic resonance imaging⁴ and an increased white matter hyperintensity burden on fluid-attenuated inversion recovery sequences⁵.

While imaging studies reveal structural changes shortly after spaceflight, mortality outcomes provide insight into the long-term consequences that may occur decades later. Alzheimer's disease and related dementias are progressive and, in some cases, ultimately fatal², making death records a useful endpoint for assessing lifetime risk. The microvascular pathology suspected in radiation-induced white matter disease—endothelial dysfunction, small vessel ischemia, blood-brain barrier disruption—may increase risk of cerebrovascular disease and subsequent dementia⁶, conditions that lead to substantial mortality in the general population⁷. Mortality surveillance offers pragmatic advantages over incidence surveillance alone, since mortality can be viewed as an ultimate consequence of chronic conditions, and its timing is easier to objectively assess.

Even with a clear-cut outcome such as death, astronaut cohorts still present a fundamental surveillance challenge: small sample sizes, heterogeneous exposure profiles, and competing mortality risks all constrain statistical precision. Traditional standardized mortality ratio (SMR) analyses, which compare observed deaths to the expected deaths in the general population, can quantify relative risk. However, with sparse data, they yield wide confidence intervals and correspondingly low confidence.

This study presents conventional SMR analysis and proposes a Bayesian surveillance framework for mortality from late CNS effects—dementia and motor neuron disease—among NASA astronauts. Using astronaut biographical data from the Astronaut and Spaceflight Participant Archive paired with mortality information from the NDI, we calculate cause-specific (neurological) SMRs for male and female astronauts compared to US general population rates. We then implement a prospective Bayesian system that projects mortality boundaries forward ten years, explicitly defining thresholds for protective, indeterminate, and excess mortality zones as evidence accumulates. The goal is not to declare definitive answers with insufficient data, but to establish a transparent surveillance infrastructure that maximizes the utility of available evidence, along with a picture of future probable trajectories and probability intervals, which can be updated as the cohort ages into higher-risk decades and new data are collected.

2. METHODS

2.1 Subjects

Astronaut data were obtained from the Astronaut and Spaceflight Participant Archive, an open-source database containing demographic and vital data on international astronauts and commercial spaceflight participants⁸. For this analysis, we included all NASA astronauts selected from 1959 through 2021 (Classes 1 to 23). Follow-up was calculated from the date of selection until either the date of death or 31 December 2022, whichever came first.

Cause of death information was obtained from the National Death Index, which provided International Classification of Diseases (ICD) codes (8th, 9th and 10th editions) for underlying causes of death matched to astronauts known to be deceased as of 31 December 2022. We defined neurological mortality as death attributed to late CNS effects coded as one of a set of ICD codes representing conditions hypothesized to be associated with cumulative space radiation exposure (Table I). The protocol for obtaining National Death Index data and conducting astronaut mortality studies was reviewed by Solutions IRB and verified as Exempt research according to 45CFR46.104(d).

General population reference mortality rates were obtained from the CDC WONDER database for the same ICD codes (Table I), stratified by calendar year and 10-year age groups (35–44, 45–54, 55–64, 65–74, 75–84, 85 + yr)^{9,10,11}. Rates were available for the years 1968–1978 (with ICD-8 codes), 1979–1998 (with ICD-9 codes), and 1999–2016 (with ICD-10 codes). For the period from 1959–1967, we applied the 1968 rates; for the period from 2016–2022, we applied the 2016 rates. Given the young age of astronauts during the pre-1968 period and the low baseline rates of late CNS effects at these ages, we anticipate that the impact on the expected numbers of deaths of the extrapolation of 1968 rates to earlier years will be negligible.

All analyses were conducted in R (R Foundation, Vienna, Austria). Claude Opus 4.5 (Anthropic, 2025) was used for copy-editing assistance and language refinement. All conceptual content, structure, and final text are the author's own.

2.2 Statistical Analysis

To index the relative risk of death from neurodegenerative causes among astronauts compared to that of the US general population, we computed standardized mortality ratios as the quotient of the observed neurodegenerative deaths and the expected number, under mortality rates in the US general population. Expected deaths were computed based on sex, age, and calendar year. Traditional 95% confidence intervals around the SMRs were calculated by assuming the observed count of deaths follows a Poisson distribution.

As a forward-looking methodology, we propose a surveillance tool allowing for probabilistic inferences regarding the comparative rate of late CNS mortality. The motivating question is: How many deaths from

late CNS effects would be required to obtain a given level of probability that $SMR \neq 100$? A Bayesian framework is ideal, providing a method for updating the SMR with incremental data while permitting direct probabilistic statements about the posterior distribution.

Under a Bayesian framework, we model observed deaths O as Poisson with unknown rate λ , where $\lambda = SMR \times E$, E being the expected rate in the age-, sex-, and calendar-year-matched general population. A Gamma distribution with shape parameter α and rate parameter β (i.e., $\text{Gamma}(\alpha, \beta)$) provides a conjugate prior for λ , and thus for the SMR, meaning the posterior remains in the Gamma family, where α encodes cumulative observed deaths and β encodes cumulative expected deaths. Given new data, the posterior distribution on the SMR is $\text{Gamma}(\alpha + j, \beta + E_i)$, where j is the cumulative number of deaths from late CNS effects and E_i is the expected deaths in projection year i . Note that in our surveillance framework, j represents hypothetical cumulative deaths over the ten-year projection, evaluated across a range (0 to 20) to establish prospective surveillance thresholds—not observed deaths from actual follow-up.

At the start of the projection, we initialize α as the observed deaths from late CNS effects at the end of follow-up and β_1 as the expected deaths at the end of follow-up. For each simulated future year i ($i = 1, \dots, 10$), we (1) project the astronaut age distribution forward by shifting 10% of each 10-year age group to the next older category, assuming uniform distribution of ages within groups; (2) recalculate the age-weighted general population mortality rate ($gp.rate.w$) using the projected age distribution and 2016 ICD-10 reference rates (the most recent available from CDC WONDER); (3) calculate the expected deaths for that year (E_i) as $n \times gp.rate.w$; (4) compute the posterior distribution $\text{Gamma}(\alpha + j, \beta_i + E_i)$ for each hypothetical cumulative death count j from 0 to 20, and calculate credible intervals and $P(SMR > 100)$; and (5) update $\beta_{i+1} = \beta_i + E_i$ for the next projection year.

From these posteriors we identify upper, lower, and intermediate surveillance boundaries. The lower boundary is the protective effect threshold, defined as the maximum j where the upper 97.5% CrI for $SMR < 100$. The upper boundary is the excess mortality threshold, defined as the minimum j where the lower 2.5% CrI for $SMR > 100$. We also define intermediate boundaries of j where $P(SMR > 100)$ crosses 25%, 50%, and 75%, representing heuristic transitions between zones of evidence strength.

The simulation accounts for both accumulating person-time and the changing age structure of the cohort over the 10-year projection period. Under this system, newly selected astronauts can be integrated into the simulation by increasing the counts in the appropriate age pools, though we have not done so here. Similarly, for longer simulations, astronauts could be removed from the oldest age bin at a designated attrition rate (for example, based on observed all-cause mortality rates for the astronauts). The comparison need not be to the general population—any sensible comparison group can be used. Finally, while this method simplifies the cohort aging process, future person-years could be projected using the exact age structure of the starting cohort, though this is unlikely to affect the threshold estimates.

3. RESULTS

Table II displays the actuarial data for the SMR analysis. Between 1959 and 2022, 299 male astronauts were selected, contributing a combined 9604 person-years of observation, with a mean follow-up period of 32.1 year and a mean age of 53.0 for male person-years. There were 61 female astronauts in the cohort, selected between 1978 and 2022. They contributed 1422.6 person-years to the analysis, with mean follow-up time 23.3 year and mean age of a person-year of 46.9 year.

Among male astronauts, 2 deaths had late CNS effects as the underlying cause, while 4.99 deaths were expected, equating to an SMR of 40 (95% CI = 5–145). The point estimate suggests that male astronauts are at less than half the risk of the US general population; however, the wide confidence interval, inclusive of 100, means that we cannot rule out the possibility that male astronauts may actually be at increased risk.

Only 0.08 deaths from late CNS effects were expected among female astronauts, and no such deaths were observed. The SMR for female astronauts was therefore 0.0 (95% CI = 0–4611). The confidence interval here reflects the comparatively small amount of follow-up contributed by female astronauts. With an upper bound of 4611, the confidence interval is completely uninformative.

Combining the data for men and women yielded an observed count of 2 deaths and an expected count of 5.07, for an SMR of 39 (95% CI = 5–142). Just as in the individual estimates, the pooled confidence interval is uninformative.

Figures 1 and 2 display Bayesian surveillance boundaries for male and female astronaut late CNS effects mortality over a 10-year projection period. Color zones represent strength of evidence: dark blue (strong protective), light blue (moderate protective), light grey (indeterminate), coral (moderate excess), dark red (strong excess).

For male astronauts (Fig. 1), the boundaries span a moderate range over the 10 years, reflecting the amount of accumulated evidence (9,604 person-years, 2 deaths). In projection Year 1, observing 2 or fewer cumulative deaths would indicate a protective effect with 75% probability, while 9 or more deaths would indicate excess mortality with 97.5% probability. Deaths from 3 to 5 fall in the indeterminate zone, and 6–8 deaths define the moderate increase zone (25% probability). By Year 10, as person-time accumulates and the cohort ages, these thresholds increase to 8 (protective, 25% probability) and 17 (excess, 97.5% probability), reflecting higher expected mortality that occurs due to additional follow-up in an aging population. In Year 1, even 0 observed deaths cannot establish a protective effect with 97.5% certainty; by Year 2, sufficient expected deaths have accumulated to cross this threshold, and the strong protective boundary remains at 0 deaths for the remainder of the simulation.

For female astronauts (Fig. 2), boundaries are substantially narrower, reflecting sparse data (1,423 person-years, 0 deaths). The pervasively narrow zones illustrate limited inferential power. Most plausible death counts (up to 1 death in the first half of the simulation and up to 2 deaths in the second half)

remain only minimally informative. The blue protective zones remain constant such that only the total absence of mortality from late CNS effects would constitute evidence of reduced risk in the 10-year simulation window. By Year 10, observing 3 or more deaths would indicate excess mortality with 97.5% probability.

4. DISCUSSION

The results of the conventional SMR analysis suggest that US astronauts have not been at increased risk of death from late CNS effects between 1959 and 2022. However, this result is based on only 2 deaths across 11,026 total person-years and cannot be confirmed with statistical significance under the frequentist SMR framework. While six decades of follow-up time may seem substantial, the resulting SMR confidence interval (5–142) reflects considerable uncertainty—we cannot rule out mortality rates either well below or moderately above the general population reference. In fact, with the data used here, an SMR of 220 (11 observed deaths against 4.99 expected) would be required among males before a result would register as meaningful at $p < 0.05$. Among females, the opposite applies: the expected count is so small (0.08) that as few as 2 observed deaths would yield a "significant" SMR of 2,500 — a result that is statistically significant but epistemologically meaningless. These power limitations are not unique to a general-population comparator — they reflect the small size of the astronaut cohort itself, and would persist, and in many cases would be exacerbated, under any reference population. For astronauts embarking on unprecedented deep-space missions to the Moon or Mars, definitive answers may not emerge for 30 + years or more, given disease latency periods and the small crews involved. As the most recent comparable cohort with extensive follow-up, current and former LEO astronauts will themselves serve as the most defensible counterfactual against which deep-space mission outcomes can eventually be assessed — making sustained surveillance of the existing corps a prerequisite for evaluating the long-term health consequences of exploration-class missions. What is needed is a method for maximizing the utilization of available data and a transparent framework for ongoing surveillance as evidence accumulates.

Stratification by cumulative spaceflight duration or mission type was not feasible given the sparse outcome counts. Subdividing 2 deaths across exposure categories would only reduce person-years within strata, shrink expected counts, and widen already-uninformative confidence intervals. As evidence accumulates over decades, future analyses may permit exposure-response evaluation.

The Bayesian surveillance system presented here provides such a framework. Figures 1 and 2 demonstrate an intuitive approach: establishing prospective boundaries that define when accumulated deaths constitute evidence of increased risk, decreased risk, or remain consistent with general population rates. This forward-looking approach is more informative than repeatedly calculating SMRs with wide, overlapping confidence intervals. By explicitly quantifying the number of deaths over a specific timeframe that would constitute an excess risk, the system transforms uncertainty into actionable surveillance thresholds.

The surveillance boundaries themselves tell a story about data adequacy. Male astronauts, with 9,604 person-years and two deaths, yield boundaries that rise steadily and in tandem as evidence accumulates. Female astronauts, with only 1,423 person-years and zero deaths, yield essentially uninformative boundaries—the lower threshold remains at zero across all projection years, and the upper boundaries only increase by 1 in a step-like fashion. This contrast highlights a fundamental point: sometimes precision isn't achievable, and acknowledging inferential limits is more valuable than relying on false confidence.

A critical feature of this surveillance system is the ability to account for cohort aging. The median age of the male astronaut cohort at the end of follow-up is approximately 68 year, with 49% already in the 65–84 age range. Over 10 year of projection, expected late CNS effects mortality nearly doubles as the cohort ages into higher-risk categories. Our approach dynamically adjusts boundaries to reflect this reality. While simulations with even higher fidelity could be constructed (e.g., accounting for changes in person-year distribution at the individual level), the gain in precision would be negligible, especially when weighed against the increase in complexity.

Our approach offers a key advantage over traditional retrospective SMR calculations. Rather than repeatedly reporting statistically non-significant findings as data slowly accumulate, it projects forward to quantify precisely what future observations would constitute actionable evidence. The framework answers 'how many deaths would we need to see to conclude excess risk?' rather than perpetually stating 'current data are inconclusive.'

Another advantage is that, although the surveillance framework establishes binary thresholds for operational clarity, the Bayesian approach enables a more nuanced interpretation. For any observed death count, one can calculate $P(\text{SMR} > 100)$ directly, providing continuous estimates of the strength of the evidence rather than binary zones.

The general idea of a surveillance framework presented here is limited neither to mortality outcomes from late CNS effects nor to any particular comparator. The approach applies to any rate-based outcome where accumulating evidence must be continuously evaluated against a reference population, and any (or multiple) defensible reference rates can be substituted into the expected-events calculation. A surveillance suite running multiple comparators in parallel — for example, the general population, pilot cohorts, and military aviators — would allow signals to be triangulated across reference frames, with concordant trips across multiple comparators constituting stronger evidence than a single-comparator signal. Similarly, such a suite could track multiple endpoints, such as incidence rates for cancer subtypes, cardiovascular events, or cerebrovascular disease, by substituting incidence for mortality and disease-free person-time for total person-time in expected events calculations. The framework is similarly applicable beyond astronaut populations, such as small occupational cohorts with unique exposures (such as nuclear workers, pilots, and offshore workers). The core principle remains constant: explicitly define what evidence would constitute alarm or reassurance, account for changing cohort characteristics over time, and acknowledge when data are insufficient to distinguish signal from noise.

The gradient probability zones in Figs. 1 and 2 make this inferential uncertainty visually transparent, a feature particularly valuable when communicating surveillance results to non-statistical audiences such as mission planners or regulatory bodies. We advocate for the adoption of this prospective Bayesian surveillance framework as a new standard for monitoring long-term health outcomes in small, high-exposure cohorts, ensuring that limited data are used to their maximum inferential potential.

One limitation is that ICD code revisions across eras (ICD-8, ICD-9, ICD-10) introduce potential inconsistencies, though we selected conditions with relatively stable clinical definitions (Parkinson's disease and Alzheimer's disease). Similarly, we applied 1968 rates to observations made prior to 1968 and 2016 rates to observations made after 2016. Both of these potential sources of error should have minimal impact on the male astronaut SMR, as the average age of a person-year before 1969 is 35 year, where the incidence and mortality from late CNS effects are negligible, and such data represent only 2% of the overall observation time. Observations made after 2016 wield a larger influence as they are 14% of the total observed person-years in men and 22% among women. Nevertheless, we believe any bias in the SMR introduced by this rate substitution would be minimal, as mortality rates from late CNS effects have likely been stable in the last 10 years.

This analysis demonstrates the feasibility of implementing prospective Bayesian surveillance for rare outcomes in small, aging cohorts. The framework is generalizable to other astronaut health outcomes of concern (cardiovascular disease, cancer subtypes, cerebrovascular disease) and could inform surveillance protocols for commercial spaceflight participants as that population expands. Transparency about inferential limits, as illustrated by the less informative boundaries for female astronauts, is preferable to perpetual null findings that offer little more than a reminder of insufficient statistical power.

Declarations

ETHICS DECLARATION

The protocol for this research was reviewed by Solutions IRB, LLC. It was ruled Exempt according to 45CFR46.104(d) - secondary use of identifiable private information or identifiable biospecimens collected for purposes other than this research study. No contact with the study subjects was made or attempted.

FUNDING DECLARATION

This research had no funding.

COMPETING INTERSTS

The authors have no competing interests to declare.

ETHICS STATEMENT

The protocol under which this study was performed was reviewed by Solutions IRB, a fully-accredited independent Institutional Review Board. The protocol was verified as Exempt research according to 45CFR46.104(d).

Author Contribution

RR conceived of the study, compiled the dataset, and designed and performed the analysis. DD and MR provided subject matter expertise on neurology and radiation exposure. SD contributed to the methods and statistical analysis plan. All authors contributed to the manuscript.

ACKNOWLEDGEMENTS

The authors wish to thank the astronauts of the NASA Astronaut Corps for their continued bravery in exploring outer space.

Data Availability

Astronaut demographic and vital data were obtained from the Astronaut and Spaceflight Participant Archive (ASPA) via the Organization for Space Medicine, Engineering, and Design website (osmed.org). The database may be downloaded at <https://github.com/osmed-admin/aspa> . The cause of death data were obtained separately from an application made by the research team to the US National Death Index (NDI) at the National Center for Health Statistics (NCHS). While the ASPA is publicly available, the NDI death data cannot be shared in accordance with NCHS/NDI rules. The protocol under which this study was performed was reviewed by Solutions IRB, a fully-accredited independent Institutional Review Board. The protocol was verified as Exempt research according to 45CFR46.104(d).

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Tables

Table I. ICD codes for late CNS effects under study.			
Condition	ICD-10	ICD-9	ICD-8
Vascular dementia	F01	290.4	–
Parkinson disease	G20	332	342
Other degenerative diseases of the nervous system	G30-G31	331.0, 331.2, 331.9	290.1
Cerebral ischemia	I67.8, I67.9	437.0, 437.1, 437.9	437.x

Table II. Actuarial data and SMR for late CNS effects among US astronauts, 1959-2022.			
Variable	Males	Females	Total
Period	1959-2022	1978-2022	1959-2022
Astronauts	299	61	360
Person-years	9604.0	1422.6	11026.6
Mean follow-up, yr	32.1	23.3	30.6
Mean age of person-years, yr	53.0	46.9	52.2
Obs. Deaths	2	0	2
Exp. Deaths	4.99	0.08	5.07
SMR, % (95% CI)	40 (5, 145)	0 (0, 4611)	39 (5, 142)

Figures

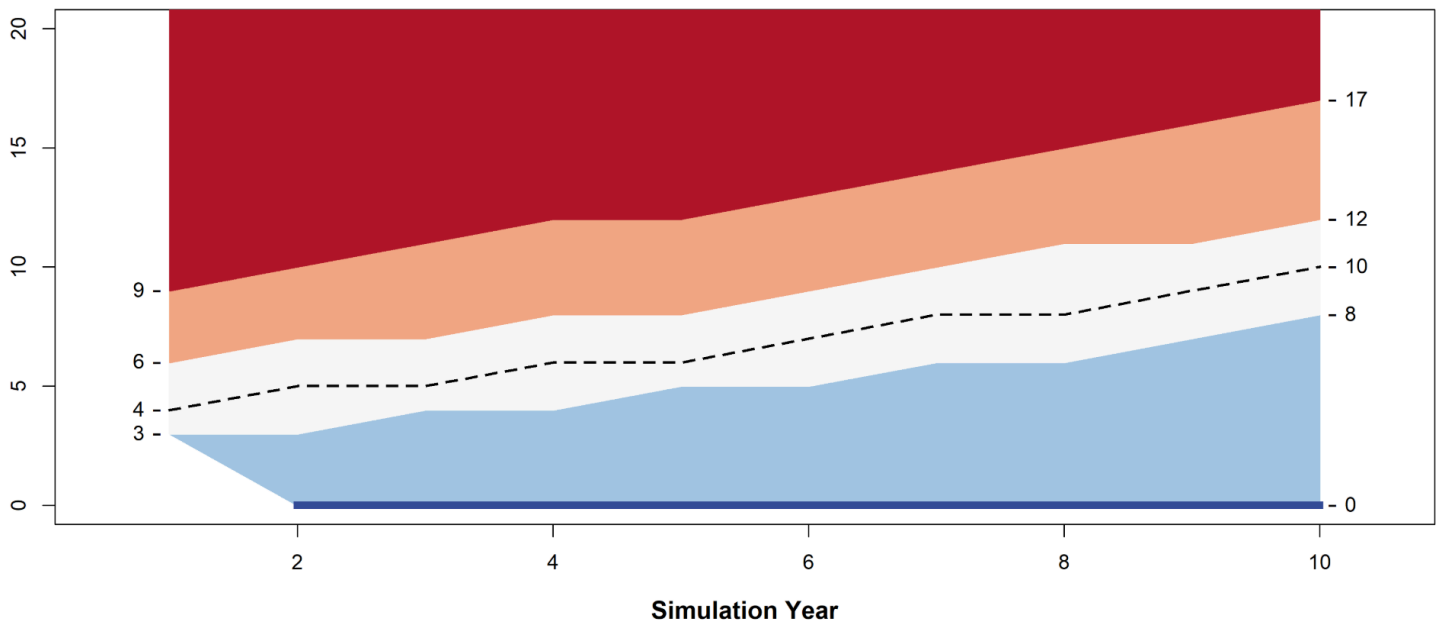


Figure 1

Bayesian surveillance boundaries for late CNS effects mortality among male astronauts over a 10-year projection. Color zones represent posterior probability that SMR>100: dark red (>97.5%, strong excess),

coral (75-97.5%, moderate excess), light grey (25-75%, indeterminate), light blue (2.5-25%, moderate protective), dark blue (<2.5%, strong protective). Black dashed line indicates equipoise ($P(SMR>100)=50\%$). Values on the left margin show death count thresholds at projection year 1, while numbers in the right margin show death count thresholds at projection year 10.

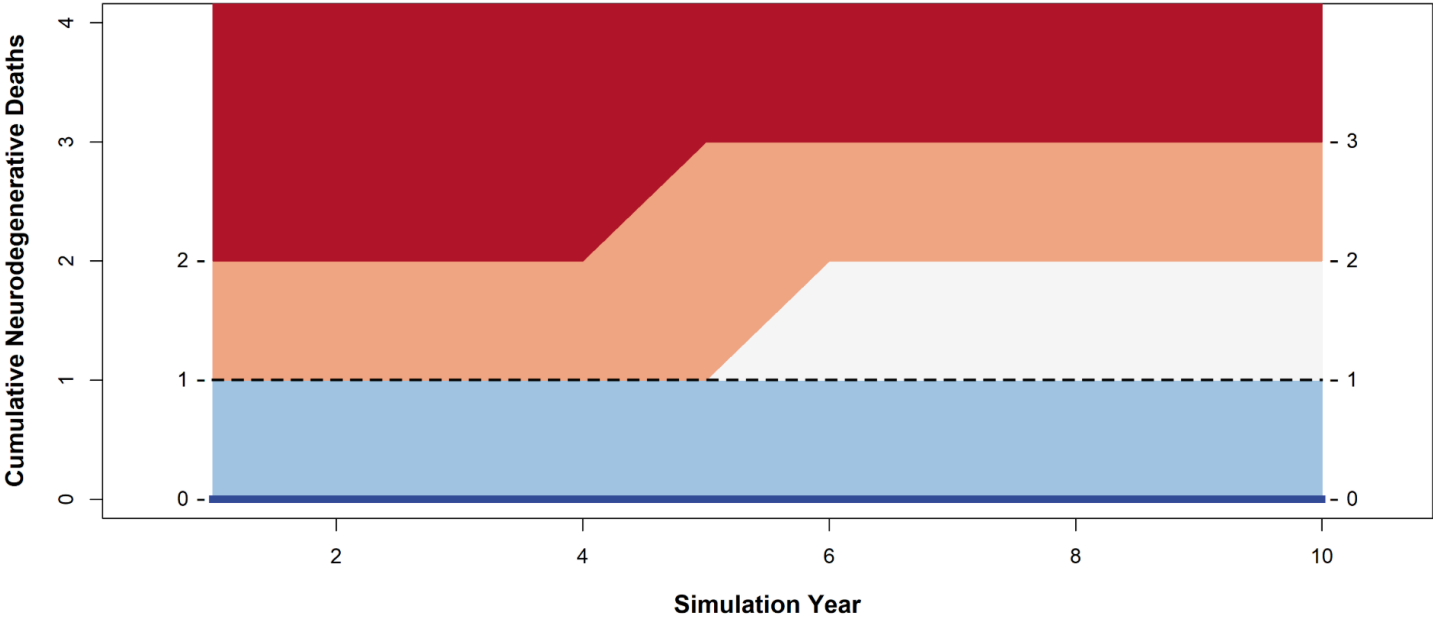


Figure 2

Bayesian surveillance boundaries for late CNS effects mortality among female astronauts over a 10-year projection. Color zones represent the posterior probability that $SMR>100$, as in Figure 1. Black dashed line indicates equipoise ($P(SMR>100)=50\%$). Values on the left show death count thresholds at projection year 1, while numbers on the right show death count thresholds at projection year 10. The low thresholds reflect sparse accumulated observation time (1,423 person-years), zero baseline deaths, and low expected counts into the future. This demonstrates limited inferential power to distinguish between protective and harmful effects.