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Illustrative Bayesian Reanalysis of Published Micronucleus Frequencies: A Retrospective Methodological Comparison of Classical and Jeffreys-Rule Estimates in Selected Radioprotection Groups

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Featured Application

This work illustrates how the choice between classical background subtraction and a Jeffreys-rule Bayesian transformation can materially change the apparent magnitude of protection derived from published cytokinesis-block micronucleus (CBMN) frequencies when observed values approach the spontaneous background. It is intended as a methodological caution for the retrospective interpretation of micronucleus frequencies reported on a normalised MN/500 binucleated-cell scale, as well as an argument for reporting raw micronucleus counts together with their exact cell denominators in future CBMN studies. It is not a validated tool for dose assessment, radiation protection practice, or clinical decision-making.

Abstract

Published cytokinesis-block micronucleus (CBMN) frequencies, normalised to 500 binucleated cells (MN/500 BC), were used to illustrate how the choice of statistical parametrisation affects background-subtracted radioprotection metrics. An illustrative Jeffreys-rule transformation was applied to published normalised frequencies from eight representative experimental groups, drawn from two previously published human-lymphocyte CBMN datasets generated by a single laboratory, within the broader retrospective context of four source studies (2000–2026): an irradiated control, four pre-irradiation radioprotective treatments, one post-irradiation treatment, one radiosensitiser, and an iodine-131 biodosimetry cohort. It was not used as a statistically valid Bayesian inference based on the original experimental counts. Because the original integer micronucleus counts and the exact numbers of binucleated cells scored per condition could not be recovered, the analysis is presented as a descriptive methodological comparison. On the normalised scale, the transformation increased the net MN value when observed frequencies were close to background, with a maximum correction of 2.38 MN/500 BC, and approached the analytically expected asymptotic offset of 0.5 MN/500 BC under the illustrative working parametrisation, as the observed values substantially exceeded the background. Consequently, within the selected groups analysed, the apparent magnitude of protection calculated from the transformed values was lower than the classical estimates for the selected pre-irradiation treatments,

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showing the largest classical MP values—for example, 79.5% versus 93.8% for rosmarinic acid. The ordering of the selected radioprotective-treatment groups by MP was unchanged. These results illustrate the sensitivity of background-subtracted MN metrics to statistical parametrisation under low-signal conditions. They must not be interpreted as valid experimental uncertainty estimates, as dose estimates, or as clinical decision criteria.

Keywords: ionising radiation; CBMN assay; normalised micronucleus frequencies; Jeffreys prior; illustrative Bayesian calculation; retrospective methodological reanalysis; radiation protection

1. Introduction

The cytokinesis-block micronucleus (CBMN) assay in human peripheral blood lymphocytes has established itself as the most widely used cytogenetic method for the quantification of radiation-induced chromosomal damage, both for biodosimetry and for the evaluation of genotoxic and radioprotective agents. Described in its modern form by Fenech and Morley [1], the assay detects micronuclei (MN)—cytoplasmic bodies containing acentric chromosome fragments or whole chromosomes excluded from the main nucleus during cell division—in binucleated cells produced by the addition of cytochalasin B. Scored as MN per 500 binucleated cells (MN/500 BC), the assay is recommended by the International Atomic Energy Agency (IAEA) [2,3], the OECD (Test Guideline 487) [4], and the ISO (17099:2014) as a validated tool for both routine biomonitoring and emergency biodosimetry.

Our research group has made systematic use of the CBMN assay over more than two decades to evaluate the radioprotective activity of a broad panel of natural compounds, including procyanidins from grape seeds, phenolic compounds from Olea europaea, and rosemary phenolics, as well as other reference substances, such as amifostine, vitamins C and E, and quercetin [5–8]. These studies comprise both in vivo experiments in mouse bone marrow and in vitro CBMN experiments in human peripheral blood lymphocytes; only the latter were used as data sources in the present work. These experimental findings are in line with recent clinical and mechanistic reviews highlighting the potential of natural polyphenols as radioprotective agents in thoracic cancer patients and other settings [9,10].

Despite the biological validity and widespread adoption of the CBMN assay, the statistical treatment of MN frequency data has remained, for the most part, anchored to a classical frequentist framework: the net MN count is estimated as the arithmetic difference between the total count in the treated group and the background frequency ($n - b_0$), and the dose is then estimated by the inversion of the calibration curve. This approach is reliable and well-validated when net counts are substantially higher than the background. However, it presents a fundamental and well-documented limitation in the scenario that is arguably the most important for radioprotection research; when the radioprotective agent is highly effective, the MN frequency in the treated group is reduced toward the background level, producing net counts that approach zero, may become negative (which is physically inadmissible), and are associated with very large relative uncertainties. This limitation has been documented by Vinnikov et al. [11] and Ainsbury et al. [12], and has motivated the development of dedicated Bayesian software tools [13]. Beyond Jeffreys-rule inference for Poisson counts, several Bayesian approaches have been developed for cytogenetic biodosimetry, including negative binomial models for overdispersed chromosome aberration data and Bayesian estimation of net counting rates [14,15].

The Bayesian framework for Poisson count data provides a natural and mathematically rigorous solution to this problem. By treating the unknown net signal as a random

variable with a probability distribution rather than a fixed quantity, Bayesian inference yields an estimate—the posterior expected value E[s]—that is always positive, respects the physical constraint $s \geq 0$, and provides a full posterior distribution that quantifies measurement uncertainty in a coherent and interpretable way. In 2008, our group [16] applied this framework to biological dosimetry in thyroid cancer patients treated with ^{131}I, using the Jeffreys non-informative prior for the Poisson rate parameter. The Jeffreys-rule Bayesian estimator for the Poisson mean has since been recommended and extended by multiple independent groups, including applications to partial body and overdispersed cytogenetic data [17,18], and dedicated software (CytoBayesJ) has been developed for its routine implementation [13]. However, the Bayesian framework has rarely been used to retrospectively re-examine how it would modify the radioprotection metrics already reported in published CBMN studies with natural antioxidants. We emphasise that the Jeffreys-rule Bayesian estimator itself is not novel, as it has been established and applied in cytogenetic biodosimetry since 2008 [16–18]. The principal contribution of the present study is a transparent retrospective methodological illustration of how this estimator modifies net-count and radioprotection metrics in eight representative CBMN groups, of which seven are derived from Alcaraz et al. [5] and one from Serna et al. [16], within a broader retrospective context of four previously published studies. The selected groups span the principal low-count and higher-count conditions relevant to radioprotection and biodosimetry, allowing direct comparison of classical and Jeffreys-rule values of the net MN frequency, magnitude of protection (MP), and dose reduction factor (DRF). Because only frequencies normalised to 500 binucleated cells are available for these historical groups, this comparison is descriptive and does not constitute a formal count-based Bayesian inference.

These earlier findings are methodologically relevant to the present reanalysis because they exemplify the near-background regime in which post-exposure MN frequencies remain close to baseline values, making classical net-count estimation particularly fragile and the apparent magnitude of protection strongly dependent on how the background is subtracted.

Here, we apply a single Jeffreys-prior transformation to eight representative CBMN groups selected from previously published studies, using the normalised MN/500 BC scale on which these data were reported. The analysis illustrates how the resulting correction is largest when observed MN frequencies are close to the background, and how it modifies standard radioprotective metrics under near-background conditions.

The aim of this study is to apply the Jeffreys-rule transformation illustratively to eight representative experimental groups selected from previously published CBMN studies; to quantify, for each group, the resulting correction $\Delta = E[s] - (n - b_0)$ on the normalised MN/500 BC scale; to compare classical and transformed values of the standard radioprotection parameters, magnitude of protection (MP), and dose reduction factor (DRF); and to derive cautious methodological recommendations for the reporting and interpretation of near-background CBMN data. Because the original integer MN counts and the exact numbers of binucleated cells scored are not recoverable from the source publications, no experimental uncertainty estimates, credible intervals, or formal tests of Poisson conformity have been reported.

2. Materials and Methods

2.1. Study Design and Overview

A retrospective methodological analysis was conducted using previously published CBMN datasets from the Department of Radiology and Physical Medicine, University of Murcia. Four prior studies provided the broader historical context for the present work. For transparent and reproducible numerical presentation, the detailed Bayesian analysis was restricted to eight representative experimental groups selected to span the main

experimental categories and the range of observed background-to-signal conditions. These eight groups included an irradiated control, four pre-irradiation radioprotective treatments, one post-irradiation treatment, one radiosensitiser, and one ^{131}I patient cohort. The broader set of previously published groups is cited as retrospective context but is not presented here as a fully reconstructed or independently reproducible group-by-group reanalysis. In this manuscript, 'a priori' means that the eight groups and the three selection criteria were fixed before E[s], Δ, MP_Bayes, or any other transformed quantity was calculated; it does not mean that selection was independent of the published n and b_0 values, because coverage of the observed n/b_0 range was one of the prespecified descriptive criteria. The selection was based on three criteria: availability of a matched reported background frequency, coverage of the observed-to-background ratio across the near-background and higher-signal range, and inclusion of the irradiated control and quercetin as contrasting reference conditions. No group was selected or discarded on the basis of the magnitude of the resulting correction. Nevertheless, because not all eligible published groups were included, the possibility of selection bias cannot be excluded, and the values reported here should not be read as a representative average of the source studies.

2.2. Source Studies and Inclusion Criteria

Studies were eligible if they employed the CBMN assay in human peripheral blood lymphocytes following international guidelines (IAEA EPR-Biodosimetry 2011, OECD TG 487, ISO 17099); reported MN frequencies in binucleated cells for irradiated controls and at least one radioprotective or reference substance; and provided sufficient information to recover, for each experimental group, the reported MN frequency and corresponding background frequency (b_0). Four previously published studies from our laboratory met these criteria and constitute the retrospective context of the present work: rosemary phenolics and other antioxidants in γ-irradiated human lymphocytes [8]; chemical genoprotection with phenolic compounds in X-irradiated human lymphocytes [6]; the comparative antioxidant CBMN study of Alcaraz et al. [5]; and the Bayesian biodosimetry study of chromosomal damage in iodine-131-treated thyroid cancer patients [16]. To avoid any ambiguity about the provenance of data, we state explicitly that the eight representative groups analysed in detail here were taken from two of these datasets: the irradiated control, the four pre-irradiation treatments (rosmarinic acid, carnosic acid, vitamin E, and amifostine), the post-irradiation rosmarinic acid group, and the quercetin radiosensitiser group are those reported by Alcaraz et al. [5], whereas the iodine-131 patient cohort is that reported by Serna et al. [16]. Our earlier in vivo experiments with grape-seed procyanidins [6] and Olea europaea leaf phenolics [7] are cited only as a biological background, as they were performed in mouse bone marrow and reported micronucleated polychromatic erythrocytes on a different biological scale; they were therefore not used as data sources.

We deliberately restricted the present reanalysis to these four studies, all conducted in our own laboratory over more than two decades (2000–2026) using a consistent CBMN protocol, cytochalasin-B concentration, and scoring criteria, in order to minimise inter-laboratory methodological heterogeneity (differences in donor radiosensitivity, culture conditions, scorer criteria, or irradiation source/dose rate) as a confound on the Bayesian correction Δ. Consequently, the present work should be read as a retrospective methodological reanalysis of our own previously published biodosimetry data, and the derived metrics (E[s], Δ, MP, DRF) should not be treated as universally generalisable across external laboratories without further validation.

The present manuscript does not provide a complete reconstructed dataset for every historical experimental group reported in these studies, and it does not provide experimental uncertainty estimates for any group. It provides the published normalised frequencies

and the corresponding illustrative Jeffreys-rule values for the eight representative groups selected for methodological illustration. Consequently, the quantitative statements of the present work are restricted to these representative, descriptive comparisons.

2.3. Cytokinesis-Block Micronucleus Assay (Summary)

All source studies employed the standard CBMN assay based on the method used by Fenech and Morley [1], which was slightly simplified by IAEA [2,3]. Heparinised whole blood (1–2 mL) was cultured at 37 °C for 72 h in RPMI-1640 or F-10 medium with foetal bovine serum, phytohaemagglutinin, and antibiotics. Cytochalasin B (3 μg/mL) was added at 44 h to block cytokinesis. Cells were subjected to hypotonic treatment (0.075 M KCl), fixed in methanol–acetic acid, and then stained with May–Grünwald–Giemsa. At least 3000–5000 binucleated cells (BCs) were scored for each experimental condition, in accordance with OECD Test Guideline 487 and IAEA recommendations [4]. The results were expressed as MN per 500 binucleated cells (MN/500 BC), which is a normalised reporting unit rather than the number of cells actually scored. For each experimental condition, trained observers scored at least 3000–5000 binucleated cells by light microscopy under double-blind conditions [1–3]. Because the results were published as MN/500 BC, the group-specific integer MN counts and the exact numbers of binucleated cells actually scored in each condition cannot be recovered from the published tables. Throughout the present work, MN/500 BC is therefore treated as a published summary measure on a normalised scale and not as a Poisson event count with a known exposure denominator.

2.4. Irradiation and Calibration Curves

In the radioprotection studies, blood samples were irradiated in vitro with doses ranging from 0.1 to 16 Gy of X-rays Andrex SMART 200E machine (YXLON International, Hamburg, Germany) or with γ-rays from a ^{137}Cs blood irradiator (IBL 437C, CIS Paris, France). Calibration curves were established by irradiating control samples at multiple doses; a linear–quadratic model $Y(D) = aD + bD^2$ was fitted for the ^{131}I study, and a linear model $D = \alpha + \beta \cdot MN$ was used for the antioxidant study [5]. Original calibration parameters were used without refitting. In these expressions, Y(D) is the observed MN yield (MN/500 BC) at absorbed dose D (Gy); a and b are the linear and quadratic dose–response coefficients, respectively, obtained by least-squares fitting of the irradiated control calibration data; D is the estimated absorbed dose (Gy) inferred by inverting the calibration curve; and α and β are the intercept and slope of the linear regression of the dose on MN yield, respectively, used for the antioxidant study calibration.

2.5. Extraction of MN Count Data

For each experimental group, the following quantities were extracted from the original publications: n = MN frequency normalised to 500 binucleated cells (MN/500 BC); b_0 = background MN frequency in non-irradiated controls (MN/500 BC); and the calibration-curve parameters. The published datasets provided MN frequencies normalised to 500 binucleated cells rather than original integer MN counts linked to exact denominators for every experimental condition. Although 3000–5000 binucleated cells were reported as the typical scoring range in the source studies (Section 2.3), exact group-specific denominators and raw MN counts were not consistently recoverable. The normalised MN/500 BC values were therefore used only as published summary measures for an illustrative methodological comparison and were not treated as original Poisson event counts for the purpose of formal uncertainty estimation.

2.6. Illustrative Bayesian Transformation of Normalised MN Frequencies

We let n denote the published MN frequency normalised to 500 binucleated cells and b_0 the corresponding published background frequency, with both treated as fixed known quantities. For illustrative purposes only, the Jeffreys-rule transformation was applied to these normalised values as if n were a Poisson observation with mean $s + b_0$, where $s \geq 0$ denotes the net quantity of interest, and the Jeffreys non-informative prior for the Poisson rate parameter (proportional to the inverse square root of the rate) was adopted. This is a working parametrisation on the MN/500 BC scale and not a formal Poisson likelihood for the original experimental counts, because those counts and their exposure denominators were not available. Application of Bayes' theorem yields the posterior distribution as

$$p(s \mid n, b_0) \propto (s + b_0)^{n-\frac{1}{2}} \exp[-(s + b_0)], \quad s \geq 0, \tag{1}$$

which was normalised using the incomplete gamma function [16]. The Bayesian point estimate was taken as the posterior mean E[s], which converges asymptotically to the classical estimate $(n - b_0)$ for large count numbers.

Because this transformation is applied to normalised frequencies rather than to integer counts with known denominators, every quantity derived from it—E[s], the correction Δ, and the metrics computed from them—is descriptive. Such quantities must not be interpreted as statistically valid posterior variances, credible intervals, or estimates of experimental uncertainty, and no quantity of that kind is reported in this revision. A valid count-based treatment would require the original integer MN counts together with the number of binucleated cells scored per condition as an exposure offset, and preferably a joint or hierarchical model for the treated and background counts rather than a fixed b_0. Posterior means are nevertheless retained solely as deterministic point summaries of the specified illustrative transformation, because they permit a transparent like-for-like comparison with classical background subtraction. Their retention does not restore the missing sampling information and does not support posterior dispersion, credible intervals, hypothesis testing, or experimental uncertainty claims.

2.7. Dose and Radioprotection Metrics

Throughout this section the following notations are used: n = published MN frequency per 500 binucleated cells; b_0 = background MN frequency in non-irradiated, untreated control cells (MN/500 BC); s = net MN quantity, defined as $s = n - b_0$ under the classical framework and as s = E[s] (posterior mean of the illustrative transformation defined in Section 2.6) under Bayesian parametrisation; s(ctrl) = net value for the irradiated, untreated control group; and s(trt) = net value for the irradiated group treated with the test substance. No standard deviations are defined for these quantities, as the original counts and cell denominators required to characterise their sampling variability are not available (Section 2.5).

Classical and Bayesian net counts. For each irradiated group, the classical net micronucleus count is defined as

$$s_{\text{class}} = n - b_0. \tag{2}$$

Under the Bayesian framework, the Jeffreys-rule posterior for the Poisson mean yields a posterior mean $E[s]$, which we take as the Bayesian net MN count,

$$s_{\text{Bayes}} = E[s]. \tag{3}$$

Bayesian correction is then defined as

$$\Delta = s_{\text{Bayes}} - s_{\text{class}}, \tag{4}$$

and quantifies how much the classical net count is adjusted once the Jeffreys-rule posterior is taken into account.

Magnitude of protection (MP). For radioprotective agents, the magnitude of protection is expressed as the percentage reduction in the net MN yield in the treated-and-irradiated group relative to the irradiated control. Using classical net counts, this is defined as

$$\text{MP}_{\text{class}}(\%) = 100 \times \left(1 - \frac{s_{\text{trt,class}}}{s_{\text{ctrl,class}}}\right), \tag{5}$$

where $s_{\text{ctrl,class}}$ and $s_{\text{trt,class}}$ are the classical net counts for the irradiated control and the treated-and-irradiated groups, respectively. Analogously, the Bayesian magnitude of protection is obtained by replacing s_{class} with the corresponding posterior means:

$$\text{MP}_{\text{Bayes}}(\%) = 100 \times \left(1 - \frac{E[s_{\text{trt}}]}{E[s_{\text{ctrl}}]}\right). \tag{6}$$

Both MP values are therefore expressed directly as percentages. They are deterministic functions of the published normalised frequencies; in this revision, no standard deviation or credible interval is attached to them, because none can be validly derived without the original counts and cell denominators.

Dose reduction factor (DRF). In studies where calibration curves are available, the dose reduction factor compares the equivalent dose inferred from the irradiated control with that inferred from the treated-and-irradiated group. Formally, this is expressed as

$$\text{DRF} = \frac{D(\text{MN/control})}{D(\text{MN(treated)})}, \tag{7}$$

where $D(\text{MN/control})$ denotes the equivalent dose that would be inferred, from the calibration curve, using the micronucleus yield in the irradiated control group; and $D(\text{MN (treated)})$ is the equivalent dose inferred in the same way from the micronucleus yield in the treated-and-irradiated group. In other words, DRF quantifies by what factor the apparent dose would have to be reduced to reproduce, without protection, the level of chromosomal damage observed in the presence of the radioprotective agent. In this work, DRF values reported in the main tables were calculated using classical net counts and the original calibration curves from the source studies for direct comparability with the published results. Bayesian DRF values are not reported, because a coherent Bayesian recalculation would require the raw count-level dose–response data used to construct each calibration curve, together with the exact binucleated-cell denominator and exposure information at every calibration dose. These inputs are unavailable for the historical datasets; re-inverting published curves after transforming only their normalised summary frequencies would therefore mix incompatible statistical scales. Every DRF value quoted in this work is the classical value reported in the source studies.

3. Results

3.1. Dataset

Eight representative experimental groups were analysed in detail. They were selected, before any calculation was performed, from the broader retrospective set of previously published experiments so as to include an irradiated control, four pre-irradiation radioprotective treatments, one post-irradiation treatment, one radiosensitiser, and one iodine-131

patient cohort, as well as to span the observed-to-background ratios from n close to b_0 up to $n \approx 3b_0$. The conformity of the underlying data to Poisson distribution could not be assessed, as the donor-level or culture-level MN counts and their corresponding cell denominators are not available in the source publications. No dispersion test is therefore reported, and no claims are made regarding the presence or absence of overdispersion.

Table 1, Figure 1, and Supplementary Material, Table S1 report the eight representative groups analysed in detail. These groups were selected from the broader historical literature to cover the observed range of background-to-signal conditions and the principal experimental categories. They are intended as illustrative examples for the present methodological comparison and do not constitute a newly defined reduced biological dataset. The Supplementary Materials provide the numerical values and the illustrative Python code (Python 3.12.14 with SciPy 1.13.1) supporting these eight calculations. All values reported are transformations of published normalised frequencies and carry no associated estimates of experimental uncertainty.

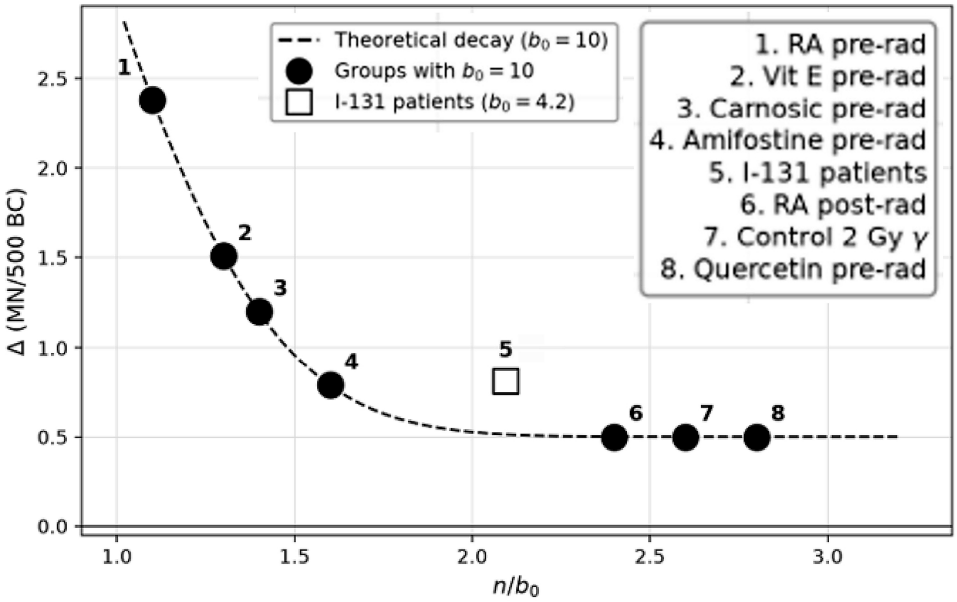


Figure 1. Correction $\Delta = E[s] - (n - b_0)$ in MN/500 BC, as a function of the ratio n/b_0 for the eight representative experimental groups (circles—seven groups with $b_0 = 10$; open square—the iodine-131 biodosimetry cohort, $b_0 = 4.2$). Numbered labels identify each group (1—rosmarinic acid pre-irradiation; 2—vitamin E pre-irradiation; 3—carnosic acid pre-irradiation; 4—amifostine pre-irradiation; 5—iodine-131 patients; 6—rosmarinic acid post-irradiation; 7—2 Gy γ control; 8—quercetin pre-irradiation). The dashed curve illustrates the analytical behaviour of Δ for $b_0 = 10$, as derived in Section 3.4. It is not a fitted universal function of n/b_0: outside the asymptotic regime, Δ depends on n and b_0 separately. As n becomes sufficiently larger than b_0, the correction approaches 0.5 MN/500 BC because the truncation at s = 0 becomes negligible.

Table 1. The published normalised MN frequencies, classical and Jeffreys-rule net values, correction Δ, published gross magnitude of protection, background-subtracted magnitudes of protection, and classical dose reduction factor for the eight representative experimental groups. Classical, Bayesian, and originally published quantities are reported in separate columns and must not be compared across definitions.

Substance/Condition	n	b_0	s_class	E[s]	Δ	MP_gross (%)	MP_class (%)	MP_Bayes (%)	DRF
Control irradiated (2 Gy γ)	26	10	16.0	16.5	0.5	—	—	—	—
Rosmarinic acid, pre-irradiation	11	10	1.0	3.38	2.38	58	93.8	79.5	7.2
Carnosic acid, pre-irradiation	14	10	4.0	5.20	1.20	46	75.0	68.5	4.8

Table 1. *Cont.*

Substance/Condition	n	b_0	s_class	E[s]	Δ	MP_gross (%)	MP_class (%)	MP_Bayes (%)	DRF
Vitamin E, pre-irradiation	13	10	3.0	4.51	1.51	50	81.2	72.7	4.9
Amifostine, pre-irradiation	16	10	6.0	6.79	0.79	38	62.5	58.9	3.3
Quercetin, pre-irradiation (radiosensitiser)	28	10	18.0	18.5	0.5	<0	−12.5	−12.1	—
Rosmarinic acid, post-irradiation	24	10	14.0	14.5	0.5	8	12.5	12.1	1.8
Iodine-131 patients (patient-averaged) [16]	8.8	4.2	4.6	5.41	0.81	—	—	—	—

n = published MN frequency (MN/500 BC); b_0 = background MN frequency (MN/500 BC); s_class = classical net value = $n - b_0$; E[s] = posterior mean of the illustrative Jeffreys-rule transformation; Δ = E[s] − s_class; MP = magnitude of protection; DRF = dose reduction factor. Three distinct families of quantities are reported side-by-side and are not interchangeable: (i) MP_gross is the originally published non-background-subtracted value taken from reference [5], and is reproduced here only for comparison with the source publication; (ii) MP_class and DRF are the classical background-subtracted values, with DRF being computed with the original calibration curves of the source studies; and (iii) MP_Bayes is obtained by substituting E[s] for s_class in the same MP definition. No Bayesian DRF is reported (Section 2.7). For the irradiated control, s_class = 16.0 and E[s] = 16.5 MN/500 BC. No standard deviations or credible intervals are given for any entry, as the original integer MN counts and the exact numbers of binucleated cells scored are not available (Section 2.5). Values are from Alcaraz et al. [5] and Serna et al. [16]. For the iodine-131 cohort, no classical DRF value was available from the source publication; therefore, no DRF value is reported in this table.

3.2. Behaviour of the Correction at Low and High Normalised Frequencies

Across the representative groups, the correction Δ was largest when the observed MN frequency was close to the background and approached 0.5 MN/500 BC as the observed value became substantially larger than the background. This behaviour follows from the truncation of the Jeffreys-rule posterior at s = 0. At low values, Δ depends on both n and b_0 individually; therefore, n/b_0 should be regarded as a useful descriptive indicator rather than as the sole determinant of the correction. The classical and transformed net MN values, the corresponding corrections Δ, and the derived radioprotective metrics are summarised in Table 1 and Figure 1. Because Δ is a deterministic function of the published values of n and b_0 on the MN/500 BC scale, it should be read as a property of the chosen parametrisation and not as a biological quantity.

3.3. Impact on Radioprotective Efficacy Metrics

Among the representative groups, the largest corrections occurred in the groups with the strongest reported radioprotective effects. For rosmarinic acid (RA) administered before 2 Gy γ-irradiation, the classical net MN value was 1 MN/500 BC, whereas E[s] = 3.38 MN/500 BC (Δ = 2.38). No posterior standard deviation is quoted for this or any other group, since it cannot be validly obtained from normalised frequencies. A comparable classical net value was reported in two studies conducted approximately 15 years apart, which illustrates the behaviour of the transformation under similar near-background conditions. This pattern is shown in Figure 2, which compares classical and Jeffreys-rule net MN values for the eight representative groups: the largest discrepancies between the two parametrisations appear at low net values, whereas irradiated controls and the radiosensitiser cluster near the identity line.

When the transformed net values were used to recalculate the magnitude of protection, MP (%) = 100 × (1 − s_trt/s_ctrl), the ordering of the five selected radioprotective-treatment groups by MP was unchanged across the groups analysed. However, the size of the difference between the classical and the transformed values depended on how close the observed frequency was to background. Figure 3 summarises this behaviour by plotting the

Bayesian MP against the classical MP for the five representative radioprotective-treatment groups: the selected pre-irradiation treatments showing the largest classical MP values lie below the identity line, whereas less effective agents and the post-irradiation treatment fall close to it.

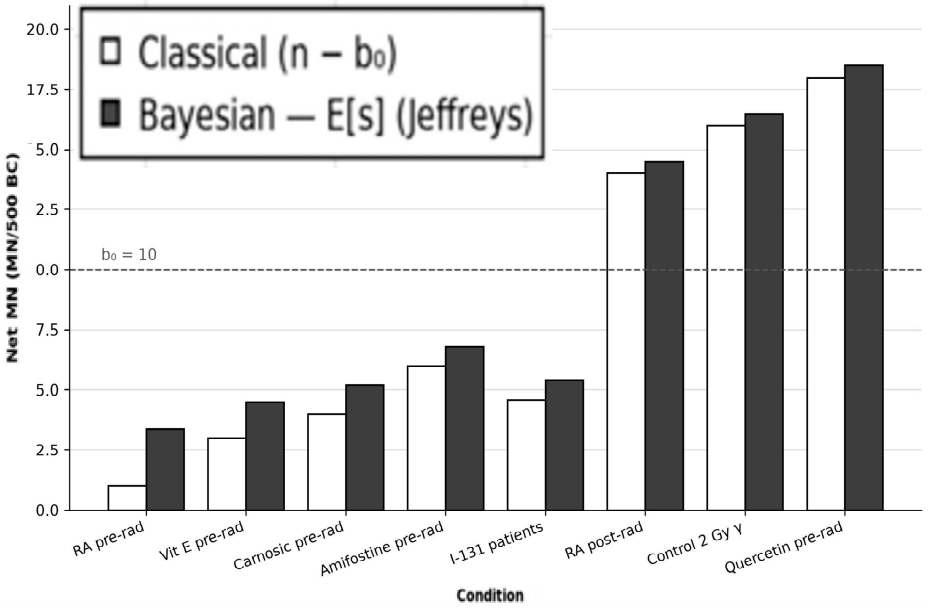


Figure 2. Comparison of classical (white/open bars) and Jeffreys-rule (dark-grey bars) net MN values for the eight representative experimental groups, sorted by increasing classical net value. The largest discrepancies between the two parametrisations are visible at low values (left side of the figure). The horizontal dashed line marks the reported background value b_0 = 10 MN/500 BC for the seven Alcaraz et al. [5] groups and is shown only as a reference. No error bars are shown because the original integer MN counts and exact cell denominators were not available; therefore, no statistically valid experimental uncertainty can be derived from the published normalised frequencies.

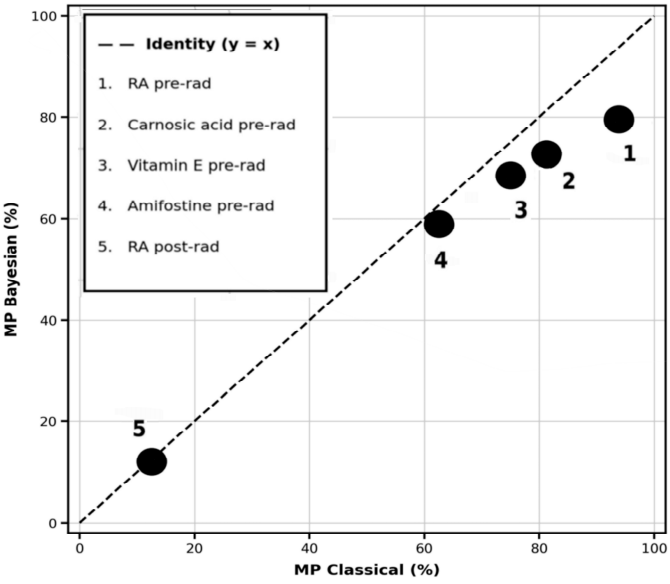


Figure 3. Magnitude of protection (MP, %), calculated with classical (*x*-axis) and Jeffreys-rule (*y*-axis) net MN values. Each point corresponds to one substance. The diagonal dashed line is the identity line (no difference between parametrisations). Points below the diagonal indicate lower Bayesian MP values, observed for the substances administered before irradiation.

Under a single net-value definition—MP = (s_ctrl − s_trt)/s_ctrl × 100, applied identically to classical and transformed values—all five groups yielded Bayesian MP values

below the corresponding classical values (Figure 3). The difference was largest for the groups with the strongest reported protection administered before irradiation: rosmarinic acid pre-irradiation (MP_class = 93.8%, MP_Bayes = 79.5%, difference −14.3 percentage points), vitamin E pre-irradiation (81.2% versus 72.7%, −8.5), carnosic acid pre-irradiation (75.0% versus 68.5%, −6.5), and amifostine pre-irradiation (62.5% versus 58.9%, −3.6). In contrast, rosmarinic acid administered after irradiation gave almost identical values under both parametrisations (12.5% versus 12.1%, −0.4 percentage points), consistent with its small correction (Δ = 0.5, Table 1). These differences are arithmetic consequences of the parametrisation and are not accompanied by any statistical tests.

This downward shift in the Bayesian MP relative to the classical MP follows directly from the truncation of the Jeffreys-rule posterior at s = 0; for groups in which n is close to b_0, the posterior mean E[s_trt] exceeds the classical net value $n - b_0$, which reduces the numerator of the MP ratio and yields a lower apparent efficacy. E[s] is non-negative by construction, which avoids the physically inadmissible negative net values produced by classical subtraction. This property does not, however, provide any information about the uncertainty of the derived MP ratio. Because the original counts and cell denominators are unavailable, no credible interval, posterior standard deviation, or Monte Carlo propagation is reported for MP in this study. MP is a nonlinear ratio of two uncertain quantities, so its uncertainty would, in any case, require the original count data to be characterised properly.

Taken together, these representative comparisons show that the classical and Jeffreys-rule parametrisations can yield materially different apparent radioprotective efficacies when observed MN frequencies are close to the background. In the groups examined here, the transformed values were lower for the strongest pre-irradiation radioprotective groups.

3.4. Analytical Behaviour of the Correction: Derivation of $\Delta \to 0.5$

Under the Jeffreys-rule prior used throughout this study, the posterior distribution of the total intensity $\lambda = s + b_0$ given an observed total count n (summed Poisson counts, conjugate Gamma–Poisson update) is Gamma(n + 0.5, 1), which has mean n + 0.5 rather than n. The posterior expected net count is therefore $E[s] = E[\lambda] - b_0 = n + 0.5 - b_0$ whenever the truncation at $s \geq 0$ has a negligible effect on the posterior mean, which occurs once n is sufficiently larger than b_0. Writing the classical (non-Bayesian) net-count estimate as sclass $= n - b_0$, this gives $\Delta = E[s] - \text{sclass} \to 0.5$ MN/500 BC as n/b_0 increases. This 0.5 offset is an analytical asymptotic consequence of the Jeffreys-rule working parametrization. Table 2 illustrates the numerical approach to this asymptote for $b_0 = 10$ by direct evaluation of the posterior mean, matching the dashed curve shown in Figure 1.

Table 2. Numerically evaluated $\Delta(n/b_0)$ for $b_0 = 10$, illustrating its approach to the analytically derived asymptotic value $\Delta = 0.5$ MN/500 BC as n/b_0 increases. The values were obtained by direct posterior mean evaluation and not by simulation; $n/b_0 \gtrsim 2$ is descriptive for this example and is not a universal threshold.

n (MN/500 BC)	n/b_0	Δ (MN/500 BC)
11	1.10	2.380
13	1.30	1.510
14	1.40	1.196
16	1.60	0.787
20	2.00	0.527
24	2.40	0.501
26	2.60	0.500
30	3.00	0.500
40	4.00	0.500

In practice, this means that for groups in which the observed count is substantially larger than background (illustratively, $n/b_0 \gtrsim 2$, for $b_0 = 10$ in Table 2), e.g., the control, RA post-radiation, and quercetin in this dataset, the Bayesian and classical net-count estimates differ by an essentially constant 0.5 MN/500 BC offset that carries no biological information; it is only in the low-count regime (n/b_0 close to 1, e.g., the pre-radiation antioxidant groups) that the Bayesian correction departs substantially from the classical subtraction and provides its principal added value, as discussed in Section 3.3.

4. Discussion

The present retrospective methodological analysis of eight representative CBMN groups shows that the Jeffreys-rule transformation produces a constrained, non-negative net MN value when observed frequencies are close to the background, and that the resulting correction is largest exactly in that regime, approaching the analytically derived asymptotic offset of 0.5 MN/500 BC at higher observed values under the illustrative working parametrisation. The central limitation of this exercise, however, must be stated plainly. The published data are normalised frequencies; a reported value of, for example, 11 MN/500 BC obtained after scoring some 5000 binucleated cells corresponds to roughly 110 observed micronuclei, and the statistical behaviour of 110 events is very different from that of 11. Applying a count-based posterior directly to the normalised value therefore does not reproduce the sampling variability of the original experiment. For this reason, all quantities reported here are treated as descriptive transformations of published summary values, and no experimental uncertainty estimate is derived from them. What the analysis does demonstrate is that background-subtracted radioprotection metrics are highly sensitive to the chosen parametrisation whenever the signal approaches the background [12].

These findings relate to our earlier CBMN-based radioprotection work with natural polyphenols. Grape-seed procyanidins and phenolics from Olea europaea leaves were shown to protect against X-ray- and γ-ray-induced chromosomal damage in mouse bone marrow, as well as rosemary phenolics in human lymphocytes [6–8]. The in vivo mouse experiments are cited here only as a biological background, because micronucleated polychromatic erythrocytes are scored on a different biological scale and were not used as data sources. Within the present framework, the human-lymphocyte observations exemplify the near-background regime in which classical net-value estimates become fragile and in which the apparent effect size depends strongly on the statistical treatment of the background.

The limitations of standard frequentist statistical approaches for cytogenetic biodosimetry have been documented in the extensive body of literature. Beyond dose estimation for homogeneous whole-body exposures, Bayesian models have also been successfully applied to partial body irradiation scenarios and zero- inflated aberration counts [17–19], further underscoring the flexibility of the Bayesian framework for complex biodosimetric problems. The development of CytoBayesJ [13] and other related software tools reflects this community consensus. Słonecka et al. [20] likewise showed, in a peer-reviewed application of a simplified Bayesian method to cytogenetic biological dosimetry, that Bayesian estimators offer a self-consistent treatment of low-count cytogenetic data, provided that the analysis is performed on genuine counts. Our results are fully consistent with these observations.

Among the substances reanalysed, rosmarinic acid (RA) presents the most instructive case. In both studies by Alcaraz et al. (2011) [21] and Alcaraz et al. (2026) [5], the same experimental design yielded the same classical net value for RA administered before irradiation: $n - b_0 = 1$ MN/500 BC, which is virtually indistinguishable from background variability under classical criteria. The transformation returns E[s] = 3.38 MN/500 BC ($\Delta = 2.38$), and the apparent magnitude of protection falls from 93.8% to 79.5%. No posterior

standard deviation is attached to this value. The recurrence of the same reported classical net value in two studies conducted about 15 years apart illustrates how the correction behaves under comparable near-background conditions, but it should be read as illustrative rather than as an independent validation of reproducibility. In contrast, when RA was administered after irradiation, n = 24 MN/500 BC and $\Delta \approx 0.5$, the asymptotic minimum of the correction (Section 3.4), so both parametrisations were in agreement. The practical implication is that studies relying exclusively on classical background subtraction may report apparent magnitudes of protection for prophylactically effective substances that are strongly dependent on the subtraction rule employed.

The present findings are also related to our earlier clinical study of patients undergoing diagnostic nuclear medicine scintigraphic examinations, in which no significant increase in CBMN frequency was detected after the doses administered in routine practice: lymphocyte MN yields measured before and after the procedures remained within the spontaneous background variability of the assay [19]. Such data correspond to conditions in which post-exposure MN frequencies remain very close to individual baseline values ($n \approx b_0$). In this regime, any positive net signal is necessarily small, and the value obtained depends markedly on how the background is handled. It is important to state that a frequency compatible with background does not demonstrate the absence of an effect, and equally that the present illustrative calculations provide no basis for the opposite claim. A properly conducted count-based Bayesian reanalysis of individual patient data, using raw MN counts and the exact numbers of cells scored, would be required before any statement about residual chromosomal damage in such cohorts could be made. Nothing in the present work should be used for clinical decision-making, for patient-level risk communication or for dose assessment in nuclear medicine.

Similarly, because the iodine-131 cohort and the scintigraphic examination dataset were not reanalysed from individual raw counts in the present study, no patient-specific estimates are reported here and the clinical examples serve only as conceptual illustrations of the near-background regime.

Several limitations of the retrospective reanalysis design must be acknowledged. First, and most importantly, the analysis is performed on MN frequencies normalised to 500 binucleated cells rather than on the original integer MN counts with their exact cell denominators; a normalised frequency is not a Poisson observation, so all Bayesian quantities reported here are descriptive transformations and not statistically valid inferences. Second, for this reason no posterior standard deviation, credible interval, background-sensitivity analysis or Monte Carlo propagation is reported. Third, the conformity of the data to a Poisson distribution could not be tested, since donor-level counts and replicate numbers are unavailable; no claim is therefore made about overdispersion, and the illustrative Papworth analysis based on an assumed number of replicates has not been included. Fourth, the background rate b_0 was treated as a fixed known quantity taken from the source publications; propagating background uncertainty would require a joint or hierarchical model fitted to the original counts of both the treated and the control cultures [14,15]. Fifth, the detailed presentation is restricted to eight groups selected a priori from the broader historical context, so selection bias cannot be excluded and the values shown are not a representative average of the source studies. Sixth, the data were extracted from published tables as group means rather than individual-donor values, and in vivo mouse bone marrow data were excluded because they are scored on a different biological scale. Finally, the calibration curves were derived under specific irradiation conditions, so the classical DRF values quoted from the source studies should not be extrapolated to other radiation qualities.

Compared with our previous Bayesian study in iodine-131-treated thyroid cancer patients, where Jeffreys-rule inference was applied to individual patient data to stabilise

dose estimates in a single clinical dataset [16], the present work is a purely methodological comparison performed on published normalised frequencies from eight representative groups. It shows how the correction changes as observed MN frequencies approach background and how this affects the point values of MP, but it produces no new dose estimates and no uncertainty estimates, and it should not be read as a reanalysis of any of the underlying cohorts.

Our work is complementary to previous contributions by Ainsbury and coworkers, who developed and implemented a broad family of Bayesian and Bayesian-like tools for calibration-curve fitting and dose estimation, culminating in the CytoBayesJ software package [11–13]. Those tools operate on genuine count data. In contrast, the present study examines what happens when a single Jeffreys-rule transformation is applied to already normalised published frequencies, and its main practical message concerns the reporting of primary data rather than the estimation of doses.

Our findings support several concrete methodological recommendations for CBMN studies. First, primary data should be reported as raw micronucleus counts together with the exact number of binucleated cells scored per condition and, wherever possible, per donor or culture; without these quantities neither a valid count-based Bayesian analysis nor a proper assessment of Poisson behaviour is possible retrospectively, as the present work demonstrates. Second, individual-donor background frequencies (b_0) should be reported so that the background can be modelled jointly with the treated counts instead of being fixed. Third, when raw counts are available and observed values are close to background, a Jeffreys-rule or hierarchical Bayesian analysis is a useful complement to classical subtraction, because it enforces a non-negative net signal and represents uncertainty explicitly. Fourth, in the present dataset, a ratio n/b_0 below two or thereabouts identified the groups in which background subtraction was least stable; this is a dataset-specific descriptive observation and not a universal decision threshold. Finally, the analysis code and numerical inputs should be made openly available so that other laboratories can reproduce the workflow.

Natural extensions of this work require primary data rather than published summaries. They include prospective applications to ongoing experiments, in which donor- or culture-level raw MN counts, exact condition-specific numbers of binucleated cells scored, and exposure information are recorded; the development of joint or hierarchical Poisson models, in which treated and background counts are modelled together, and the exact cell denominator for each observation is incorporated as an exposure offset—for example log(number of binucleated cells scored); and extension to complementary genotoxicity endpoints, such as the comet assay and γ-H2AX foci counting, where the same problem of a small signal close to the background arises. Only with such data can the differences illustrated here be translated into validated uncertainty estimates for radioprotection metrics.

The present reanalysis was restricted to micronucleus (MN) frequencies, the endpoint reported by all four source studies. In principle, the same Jeffreys-rule Poisson framework applies equally to any other CBMN cytome endpoint that is scored as a count per fixed number of cells and is expected to follow or exceed Poisson statistics at lower frequencies, including nucleoplasmic bridges and nuclear buds, as defined in the full human lymphocyte cytome assay of Fenech et al. [22,23], since the same truncated Gamma–Poisson posterior for the net rate $s = \lambda - b_0$ would apply, with n and b_0 redefined as the observed and background counts of the anomaly of interest. We did not apply the method to nucleoplasmic bridges or nuclear buds here because none of the four source studies reported these endpoints alongside MN counts for the same cultures, and we did not want to introduce new results that were not derivable from the original datasets. Extending the present framework to a full cytokinesis-block micronucleus cytome analysis, jointly modelling MN, nucleoplasmic

bridges, and nuclear buds (e.g., via a multivariate or hierarchical Poisson model with a shared background structure), is a natural direction for future work.

5. Conclusions

This retrospective methodological analysis of eight representative CBMN groups illustrates, within the selected groups analysed, that the apparent efficacy of a radioprotective treatment, as measured by background-subtracted micronucleus metrics, can depend materially on the statistical parametrisation chosen when observed frequencies approach the spontaneous background. Applying a Jeffreys-rule transformation to the published normalised frequencies yields non-negative net values; a correction that is largest near the background and approaches the analytically derived asymptotic offset of 0.5 MN/500 BC at higher values under the illustrative working parametrisation; and magnitudes of protection that are lower than the classical values for the selected pre-irradiation treatments showing the largest classical MP values, all while leaving the ordering of the selected radioprotective-treatment groups by MP unchanged. Because the original integer MN counts and the exact numbers of binucleated cells scored could not be recovered, these results are illustrative and descriptive; they are not valid experimental uncertainty estimates, they provide no dose estimates, and they must not be used for radiation protection practice or clinical decision-making. The principal practical conclusion is a reporting recommendation: CBMN studies should publish raw micronucleus counts together with the exact cell denominators and individual-donor background frequencies, which would allow properly specified count-based Bayesian analyses to be performed, both prospectively and retrospectively.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/app16178593/s1, Supplementary Material provides the illustrative Python code and numerical values for the eight representative groups analysed in detail.

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micronucleus counts and the exact numbers of binucleated cells scored per condition were not available for the historical groups analysed and could not be recovered from the source publications; this is the reason why no experimental uncertainty estimates are reported. No fully reconstructed group-by-group dataset was provided for the historical experiments cited as retrospective context. Further information on the original published studies may be requested from the corresponding author, subject to the availability of the original records.

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