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Evaluating Human Health Risks from Exposure to Agricultural Soil Contaminants using One- and Two-Dimensional Monte Carlo Simulations

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Abstract

The health and well-being of Indigenous Peoples are closely connected to the state of their lands. While natural soils are important for food security initiatives within these communities, they may also expose people to harmful contaminants. Consequently, this study – guided by Indigenous community members and leaders – evaluates the human health risks associated with contaminants in soils intended for agricultural purposes on Indigenous Peoples' lands in regions of Australia and Canada. Soil samples were collected from 47 sites in seven locations and analyzed for metals, metalloids, and organochlorine pesticides. Non-carcinogenic and carcinogenic risks were assessed for children, youths, and adults using one- and two-dimensional Monte Carlo simulations. The results indicate that there is a non-carcinogenic risk of exposure to lead (Pb) for children (HQ = 1.83) in Australia and an oral ingestion risk due to inorganic arsenic (As) for children (HQ = 1.05) in Newfoundland. Carcinogenic risks from As exposure were also identified for children ($R = 1.68 \times 10^{-5}$) and adults ($R = 1.20 \times 10^{-5}$) in Newfoundland from oral ingestion. However, no non-carcinogenic or carcinogenic risk from dermal exposure was found for all tested contaminants. The results indicate a potential need for targeted interventions, such as soil remediation, when and where possible, or community education, to reduce exposure risks.

1. Introduction

The health and well-being of Indigenous Peoples are directly tied to the health and well-being of their lands (Tsuji et al. 2023), where activities are carried out for sustenance and traditional cultural practice (Ahmed et al. 2022, 2023; Tsuji et al. 2022). The practice of agriculture has a long-standing history for Indigenous communities worldwide (Emmanuel et al., 2023), and some Indigenous Peoples have reintroduced or adopted agricultural activities, such as crop cultivation, to enhance healthy eating and increase the local availability of fruits and vegetables within their communities (Brimblecombe et al., 2017; Richmond et al. 2021; Wilton et al. 2023). However, the nature of crop cultivation, particularly in a garden setting, involves direct contact with soil and may increase exposure to toxicants, especially when individuals are unaware of their presence in the soil (Moriarity et al. 2024). Consequently, the presence of contaminants in soils used for agricultural activities may pose health risks, particularly in communities that rely on the land for sustenance or as part of traditional (Reyes et al. 2015) or non-traditional lifestyles. Thus, soil contamination with toxicants, such as metals and organic compounds, is a significant environmental and public health concern (Hoover et al. 2012; Kumar 2019; Raimi et al. 2022; Tsuji et al. 2019), particularly in Indigenous communities where agricultural activities are becoming more common, especially in the context of improving food security.

Contaminated soils may expose individuals to toxicants directly from the soil via dermal, inhalation, or ingestion of the soil itself or on unwashed crops (Food and Agriculture Organization [FAO] 2021; Ramires et al. 2024). Additionally, there is also a potential for indirect exposure through the accumulation in crops, and depending on the nature of the toxicant, the exposure may pose non-carcinogenic or carcinogenic health risks (Chen et al. 2024). Metals

like cadmium (Cd), mercury (Hg), and chromium (Cr) pose risks to the nervous and renal systems and are sometimes carcinogenic, depending on the valence state (Clarkson and Magos, 2006; Godt et al. 2006; Zhitkovich 2011); while metals and metalloids, such as, arsenic (As) and lead (Pb) from anthropogenic or natural sources can cause acute or chronic health impacts as well. For example, low-levels of Pb are known to cause neurological impairments and cognitive delays (Lanphear et al. 2016; Santa Maria et al. 2018; Ramirez-Ortega et al. 2021), while inorganic As is a classified Group A carcinogen associated with specific types of cancers and other health issues (Chatterjee et al. 2017; Chen et al. 2023; Martinez and Lam 2021). Organohalogens, including PCBs, dioxins and certain pesticides, are also persistent in the environment, are bioaccumulative and have been linked to immune suppression, cancer and other health impacts (Eskenzai et al. 2009; Ruder et al. 2014; World Health Organization [WHO], 2023a)

Given the potential risk of exposure to soil toxicants – and the increasing number of Indigenous communities, worldwide utilizing community and home gardens for various health and well-being programming (Brimblecombe et al. 2017; Emmanuel et al. 2023; Tsuji et al. 2019) – there is a need for a comprehensive health risk assessment that accounts for variability and uncertainty of the potential exposure risks. Therefore, the purpose of this study was to evaluate the human health risks associated with exposure to soil toxicants in different regions of Australia and Canada, and assess the risk of exposure to soil contaminants for Indigenous community members in these regions. Following the collection of soil samples from multiple locations on Indigenous Peoples' homelands, samples were analyzed for metals, metalloids (hereafter metal(loid)s), and organochlorine pesticides. The non-carcinogenic and carcinogenic exposure risk of these

toxicants in soil were examined using one-dimensional (1D) and two-dimensional (2D) Monte Carlo (MC) simulations, with 2DMC providing a more comprehensive assessment by capturing both the variability in exposure levels and uncertainties in input parameters. This approach is essential for accurately understanding potential health impacts. The goal of the study was to provide peoples of these regions with health risk information that empowers them to make more informed health decisions.

2. Methods

2.1 Soil sampling sites

In Australia, samples of potential soils to be used in agricultural activities were collected from eight sites in Queensland (**Figure 1A**) in 2023, including lands used for agriculture, residential areas, and a remediated gas station. Nine sites on Indigenous lands in New South Wales (**Figure 1A**) were also sampled in 2016, covering different terrains like floodplains and grassy areas, and differing land uses, agricultural and traditional cultural areas.

In Newfoundland, Canada (**Figure 1B**), 15 sites were selected as potential areas for agricultural activities, including forested areas and developed zones (e.g., sites with histories of agricultural use and near a school) and sampled between 2017 and 2020. Lastly, in the western James Bay of subarctic Ontario, Canada, 24 sites (**Figure 1B**) were selected across two remote Indigenous communities, situated on community lands, and one site near an abandoned Mid-Canada Radar Line station; these sites were sampled between 2015 and 2018. This remote region is unique compared to the others of the present study, as there is no all-season road in and out of the communities, apart from seasonal ice roads which typically open from mid-January to early-

April (Hori et al. 2016, 2018). **Figures 1A and 1B** shows the relative locations of the sampling sites; however, the specific dates and locations of the soil sampling have been anonymized to safeguard the privacy of the community partners, in accordance with the requests of community representatives. Samples were collected between 2010 and 2023.

2.2 Soil Sampling and Analysis

Composite samples, each comprising at least 10 individual soil samples ($n \geq 10$) per site, were collected using soil probes (AMS, American Falls, Idaho, USA): a 14-inch probe in Australia and a 33-inch reinforced probe in Canada. Sampling depths varied regionally, with 0-15 cm in Australia, 0-10 cm in Newfoundland due to shallow bedrock, and 0-20 cm in western James Bay for deep-rooted crop considerations. Soil from mixed piles was collected with a spade and shovel and stored in 20 L food-grade plastic buckets, with debris removed. Australian samples were sieved, mixed, bagged, and kept cool before analysis at NSW Department of Primary Industries (Wollongbar, NSW) for metals and metalloids, and Symbio Laboratory (Brisbane, QLD) for organohalogens; while Canadian samples were frozen until analyzed at Queen's University's Analytical Service Unit for all contaminants. A total of 13 toxic contaminants were assessed for non-carcinogenic and carcinogenic human health risks from the soil samples, including five metal(loid)s and nine organochlorine pesticides (**Table 1**).

Metals, metalloids and organohalogens were measured in soil samples by inductively coupled plasma mass spectrometry (ICP-MS) and gas chromatography with mass spectrometry (GC/MS). Samples were dried, ground, and digested by acid digestion in a solution of nitric and hydrochloric acids. The digestion process was performed using DigiPrepTM technology to fully

break down the samples. After digestion, samples were filtered, diluted, and analyzed with an Agilent 7700x ICP-MS in the same manner as USEPA Method 200.7, but with a modification to account for the soil matrix. Organohalogen analysis consisted of drying and homogenizing the samples which were spiked with DCBP and extracted using Soxhlet extraction with dichloromethane. Gel permeation chromatography (GPC) cleanup of the Florisil column was carried out after extraction, and the extracts were then analysed using an Agilent GC 6890 with a 5975 mass selective detector.

Arsenic speciation was carried out by Liquid Chromatography-Inductively Coupled Plasma Mass Spectrometry (LC-ICP-MS) and performed at ALS Laboratories using an Agilent 1200 series HPLC instrument paired with an Agilent 7700 ICP-MS. Quality control standards for arsenic analysis included the NIST 1568B Rice Flour reference material certified for DMA and monomethylarsonic acid (MMA), blanks, laboratory control standards, and matrix spikes.

When testing extractable micronutrients (Cu, Zn, Mn, Fe, etc.), air-dried soil samples were mixed with a buffered extractant solution of diethylenetriaminepentaacetic acid (DTPA), triethanolamine and $CaCl_2$. Extracts were centrifuged, filtered, diluted, and analysed on an Agilent 5110 ICP-OES following USEPA Method 6010D. The recoverable element amounts were calculated using a modified USEPA Method 200.2, hot dilute mineral acid digestion. Ingredients were purified and measured by ICP-OES using reagent blanks, spiked replicates, internal standards, and certified reference materials. The instrument was calibrated and tested with matrix spikes, replicates, and repeated calibration checks.

Furthermore, soil sample preparation was performed with a modified QuEChERS extraction protocol to maximize extraction yields for PCB and pesticide analysis using a Thermo-Scientific Triple Quadrupole GC-MS for PCB analysis and a High-Resolution LC-MS Orbitrap for pesticide testing. The samples were homogenised, spiked with the appropriate solvents and salts, centrifuged to remove organic content, and put through an additional clean-up step to eliminate contaminants. Analyte concentrations were measured by calibration curves and a quality control check was carried out at 20 samples to verify instrument and protocol performance, preserving spike recoveries in the 70-130% range. The limit of detection was 1.0 ng/g for DDT and its metabolites, 2 ng/g for Aldrin and Dieldrin, and ranged from 0.5 µg/g (Cobalt) to 5 µg/kg (Lead) for metals.

2.3 Probabilistic Health Risk Assessment

This study focuses on assessing human health risks from exposure to soil contaminants. Complementing this study, Moriarity et al. (2024) provides an ecological assessment using pollution indices, demonstrating elevated Pb contamination in Australia, significant organochlorine pesticide levels in Western James Bay, and As concentrations in Newfoundland soils that exceed safety guidelines. The ecological risk assessment findings directly inform this human health risk assessment study.

Health risk assessments that use a 1D Monte Carlo (1DMC) simulation consider either variability or uncertainty in the calculation. Specifically, input parameters are varied one-at-a-time so that the fewest number of iterations are required to produce a single distribution of the risk outcome. Examples include the variability of human exposure to pesticides that contaminate

water or soil, or the uncertainty in the toxicity of a particular chemical found in food. While a 1DMC may be appropriate to evaluate how uncertainty in data or variability in a population affects the estimate of risk, it falls short in its ability to differentiate between variability and uncertainty in a population. This separation can be accomplished with a 2D Monte Carlo (2DMC) simulation that uses nested loops to assess risk. Using a 2DMC simulation enables a more complete risk illustration, which would not be possible with a traditional 1DMC analysis. A 2DMC is appropriate when uncertainties in model inputs and/or natural variability in a population impact the risk estimate. The former is a result of the underlying model design, while the latter is an inherent characteristic of the studied population.

A two-stage probabilistic risk assessment (PRA) was carried out to assess the risk of exposure to metal(loid)s in soils. A one-dimensional (1DMC) PRA was first conducted to evaluate the risk of exposure by varying the intake parameters through an iterative process. Additionally, a two-dimensional (2DMC) PRA was carried out for results passing the defined non-carcinogenic or carcinogenic risk threshold from the simulated 1DMC estimates to further investigate variability, but also uncertainty in the simulations. The processes of the 1D and 2DMC are shown in **Figure 2A and 2B**.

2.3.1 1DMC

To quantify the risk of exposure to toxic soil contaminants in soils intended for agricultural activities, variables derived from the literature or generated for the current study (**Table 2**) were used in an intake calculation based on the oral (I_O) or dermal (I_D) ingestion (eqn. 1 and 2) rates

and those respective values in the non-carcinogenic (eqn. 10) or carcinogenic risk of exposure equations (**Figure 2A**).

$$I_O\left(\frac{mg}{kg\ d}\right) = \frac{IR\ \left(\frac{mg}{d}\right) \times C\ \left(\frac{mg}{kg}\right) \times EF\ (\frac{d}{y}) \times ED\ (y)}{BW\ (kg) \times AT\ (d)} \times 10^{-6} \left(\frac{kg}{mg}\right) \quad (1)$$

$$I_D\left(\frac{mg}{kg\ d}\right) = \frac{SA\ (cm^2) \times AF\ \left(\frac{mg}{cm^2 d}\right) \times C\ \left(\frac{mg}{kg}\right) \times EF\ (\frac{d}{y}) \times ED\ (y) \times ABS}{BW\ (kg) \times AT\ (d)} \times 10^{-6} \left(\frac{kg}{mg}\right) \quad (2)$$

To estimate the intake rate (IR) for youths and adults, mean soil ingestion rates from two studies about Indigenous Peoples' soil intake from agricultural and cultural activities were modeled using given parameters. The first study IR parameters (Doyle et al. 2012) were as follows in mg/day: mean (μ): 74; standard deviation (σ): 91; P_{50}: 60, and P_{90}: 193. The second study IR parameters (Irvine et al. 2014), also in mg/day, were: μ: 32; σ: 88; P_{50}:18; and P_{90}: 152. The parameters for each of these distributions indicated an approximate log-normal distribution and was a reasonable assumption for the IR distribution. Aggregating these two distributions resulted in a combined approximated log-normal distribution with parameters (mg/day) μ: 53; σ: 90; P_{50}: 27; and P_{90}: 120 that were used to model the IR for adults (**Table 2**). Additionally, for the IR for children, we hypothesized a scenario where children consumed three times the amount typically ingested by youths and adults, as they have more hand-to-mouth behaviors (Özkaynak et al. 2022), by scaling the IR for youths and adults μ and σ by a factor of three (**Table 2**).

The approximated EF values were derived from first-hand observations by a team researcher (M.W.) over the years working in the study regions and a detailed overview can be found in **Table S1**. The Canadian exposure frequency (EF_{CAN}) was a randomized triangular distribution,

with a mode of 60 days per year, a maximum of 75 days, and a minimum of 45 days, derived from agricultural activities from mid-May to mid-October. The Australian exposure frequency (EF_{AUS}) differed slightly due to climate, approximating 108 days per year on a random triangular distribution (**Table 2**), consolidated for both Northern Queensland (NQLD) and New South Wales (NSW), as the individual community EFs only differed by about one day (**Table S1**).

For I_D, the absorption factor (AF) for soil was modeled using a random triangular distribution based on EPA guidelines (EPA 2023), with different parameters for children, youths, and adults to account for varying hand-to-mouth contact frequencies and consistent with a subsistence lifestyle (**Table 2**). Specifically, children had a higher maximum value, whereas the maximum value for youths was adjusted to match that of adults, creating an AF category between children and adults. Adults had a lower AF mode due to reduced hand-to-mouth contact. Surface area (SA) was normally distributed to align with body weight (BW), per Health Canada's [HC] (1995) soil exposure guidelines. The averaging time (AT) was uniformly based on the exposure duration (ED) multiplied by 365 days for non-cancer risk and 25,550 for cancer risk estimates (**Table 2**). BW followed a normal distribution established for the region (**Table 2**), and contaminant concentration (*C*) was log-normal for each contaminant. All remaining variables used in the equations 1 and 2 and their distribution types are provided in **Table 2**.

For equations 3 and 4, the reference dose (RfD) is in mg/kg•d, and the slope factor (SF) is in $(mg/kg•d)^{-1}$.

$$HQ = \frac{I_{O,D}}{RfD} \quad (3)$$

$$R = I_{O,D} \times SF \quad (4)$$

The reference doses and slope factors for each contaminant are presented in **Table S2**. Non-carcinogenic risk calculations were carried out for all metal(loid)s (**Table S3**) and pesticides (**Table S4**), and carcinogenic risk calculations were carried out for inorganic As – a Group A human carcinogen (Integrated Risk Information System [IRIS] 2017) – and all detectable pesticides, with all risk calculations conducted only when $n \geq 5$ (**Tables S3 and S4**). We note that although Cr(VI) is a Group A human carcinogen, Cr(III) is the predominant form in soil (Agency for Toxic Substances and Disease Registry [ATSDR] 2012) and is therefore not considered for this risk assessment; we do not consider the other metal(loid)s to be a carcinogenic risk from oral or dermal exposure in soils.

Following the input of these variables into the R environment (R Core Team 2024), Monte Carlo simulations were then carried out by randomly sampling 10^5 independent iterations from all non-deterministic distributions for each contaminant. We then simulated the 95th-percentile (P_{95}) HQ or R (risk), where the focus was on results greater than 1.00 for the HQ, and 1.00×10^{-5} or 1.00×10^{-6} for R depending on location, respectively. The Incremental Lifetime Cancer Risk [ILCR] (1.00×10^{-5}) was used to assess the carcinogenic risk for Newfoundland and the *de minimis* risk (1.00×10^{-6}) was used for Western James Bay, as these are the respective risk thresholds for each province (HC, 2012). In the absence of an equivalent risk threshold for Australia, we applied the *de minimis* risk for that region as well. We also assessed the total hazard (THQ) as the sum of the HQ for each exposure pathway, the Hazard Index (HI) as the sum of the HQ for each contaminant, the total HI (THI) as the sum of the HI, the total risk (TR) as the sum of the R for each exposure pathway, the incremental cancer risk (ICR) as the sum of the R for each contaminant, and total incremental cancer risk (TICR) as the sum of the ICR. Sensitivity

286 analyses were carried out using the Spearman-rank correlation coefficient using 1.00×10^5
287 iterations and $p < 0.05$ for simulated estimates of the HQ > 1.00 or R > 1.00×10^{-5} or 1.00×10^{-6}.
288 Furthermore, to estimate the uncertainty of the HQ or R, the μ and σ of the bootstrap P_{95}
289 estimates were computed from 1.00×10^4 bootstrapped samples, and a 95% confidence interval
290 (BCI) was derived from the 2.5th- and 97.5th-percentiles.
291
292 **2.3.2 2DMC**
293 For the simulated 1DMC estimates that surpassed the non-carcinogenic or carcinogenic risk
294 threshold, a 2DMC was carried out to further assess the uncertainty of the modeled risk of
295 exposure to metal(loid)s or organohalogens in soil. As shown in **Figure 2B**, the approach
296 considered variability, in addition to uncertainty, among measurement methods (e.g., individual
297 variations in consumption patterns and population differences) using an inner loop (variability)
298 nested in an outer loop (uncertainty) of iterations. The same probability distributions as the
299 1DMC (**Table 2**) were assigned to intake parameters (i.e., AT (for non-carcinogenic only), BW,
300 ED, and EF) to reflect variability in populations, with *C* of metal(loids) in soil considered as an
301 uncertainty variable because of potential measurement or sample collection differences over time
302 and location. The IR was treated as a hybrid variable, incorporating variability and uncertainty
303 (**Figure 2B**). This was carried out this way because the IR can vary across individuals (e.g.,
304 behaviour, physiology) and there is uncertainty in the true population IR distribution as it was
305 modeled off of two studies, albeit for Indigenous Peoples', with small sample sizes. Therefore,
306 the IR was modeled to be variable within the inner loop using a log-normal distribution and
307 uncertain in the outer loop using a normalized mean and standard deviation of the IR. For every
308 outer loop iteration, an inner loop iteration was carried out, with 1.00×10^3 outer iterations and

1.00×10^4 inner iterations, resulting in a total of 1.00×10^7 simulations overall. The 2DMC uncertainty was assessed at the median of the P_5 and the P_{95} of the of the variability of non-carcinogenic or carcinogenic risk. A sensitivity analysis was also performed using the same method as the 1DMC.

2.4 Statistical Methods

All statistical analyses were performed in R version 4.4.0 (R Core Team 2024), with a significance level set at $p < 0.05$. The following R environment packages were used for the 1D, 2DMC and sensitivity analysis: stats (R Core Team 2024), EnvStats (Millard, 2023), and HMisc (Harrell, 2023); and visualization: ggplot2 (Wickham, 2016), ggsci (Xiao, 2018), ggthemes (Arnold, 2019), reshape2 (Wickham, 2007), and gridExtra (Auguie, 2017).

3. Results

3.1 Soil contamination

Tables S3 and S4 show the descriptive statistics of metal(loid) and organohalogen contaminants in soil by location. Australia showed the highest mean soil contamination concentrations of metals, particularly with Pb (mean = 58.25 mg Pb/kg), whereas Western James Bay had the lowest even though it had the most detectable samples (n = 92), with Cr (mean = 20.63 mg Cr/kg) as the primary contaminant. In Newfoundland, the mean As concentrations (mean = 31.29 mg As/kg) were the highest detectable metal(loid) in soil (**Table S3**). Pesticides were absent in Australia (**Table S4**), while Newfoundland had nine detections and Western James Bay had the most, with 155 detections, making it the region with the highest levels of pesticide contamination (**Table S4**). The detected pesticides in Western James Bay included DDT (mean = 0.50 mg

DDT/kg) and its metabolites, along with aldrin, dieldrin, and endosulfan sulfate, among others. Newfoundland also showed detections of DDT metabolites. The predominant pesticides or metabolites, in descending order of concentration, were *p,p'*-DDT, *p,p'*-DDE, and *o,p'*-DDT.

3.2. Non-carcinogenic risk of exposure to contaminants from soils intended for agricultural activities (1DMC)

3.2.1 1DMC

There is a non-carcinogenic oral risk of exposure for children (HQ = 1.83 (BCI: 1.79, 1.90); **Figure 3A**) to Pb in tested soils in Australia and to As (HQ = 1.05 (1.02, 1.09); **Figure 3B**) in tested soils in Newfoundland, respectively, (**Table 3**), driven by the *C* of the metal(loid) according to the sensitivity analyses (**Figure 3A and 3B; Table S5**). The mean HQ for children's exposure to Pb is 0.24, with a 6.18% probability of exceeding an HQ of 1.00. For As, the mean HQ is 0.13, with a 2.73% likelihood of reaching or surpassing this threshold (**Figure 3A and 3B**). The HI, and therefore the THQ and THI for children exceeds 1.00, but is primarily from oral exposure to Pb (**Table 3**); the same measures are exceeded for oral exposure to As in Newfoundland for children as well (**Table 3**). No other metals or pesticides (**Table S6**) HQ exceed a value of 1.00, nor do any HQ values for dermal exposure for metal(loid)s or pesticides (**Tables S6**).

3.2.2 2DMC

At the P_{95} of the variability of non-carcinogenic risk for the oral exposure to Pb for children in Australia (**Figure 5A**), the median uncertainty was 2.05 (interquartile range: 1.48, 2.86). The sensitivity analysis showed that the IR (rho = 0.56) was the most influential exposure factor,

surpassing the *C* from the 1DMC (**Figure 3A; Table S5**). For the oral exposure to As for children in Newfoundland, the median uncertainty was 1.22 (0.87, 1.82) at the P_{95} of the variability of non-carcinogenic risk (**Figure 5B**). The sensitivity analysis showed that the *C* (rho = 0.57) was the most influential exposure factor (**Table S5**).

3.3 Carcinogenic risk of exposure to contaminants from soils intended for agricultural activities

3.3.1 1DMC

There is an oral carcinogenic risk of exposure to As for children ($R = 1.68 \times 10^{-5}$ (1.64×10^{-5}, 1.72×10^{-5}); **Figure 4A**) and adults ($R = 1.18 \times 10^{-5}$ (1.16×10^{-5}, 1.23×10^{-5}); **Figure 4B**) from an ingestion exposure to the tested soils in Newfoundland (**Table 4**). The mean R for children's exposure to As is 2.53×10^{-6}, with a 6.57% probability of R exceeding of the ILCR threshold of 1.00×10^{-5}. For adults, the mean R is 1.79×10^{-6}, with a 3.79% likelihood of surpassing this threshold (**Figure 4A and 4B**). The sensitivity analyses showed that the *C* of As in the tested soil held the strongest influence for oral exposure (**Figure 4A and 4B; Table S5**) for both demographics in Newfoundland. The ICR exceeds the ILCR risk for these demographics (**Table 4**) in Newfoundland and is driven by the oral exposure to As. Lastly, although no individual pesticide R values exceed the *de minimis* risk in Western James Bay (**Table S7**), the exposure to all pesticides (TICR) does exceed the *de minimis* risk for adults in this region (**Table 4**). There is no dermal carcinogenic risk from exposure to any metal(loid)s or organochlorine pesticides (**Table S7**).

3.3.2 2DMC

In Newfoundland, the median uncertainty at the P_{95} of the variability of carcinogenic risk of exposure to As for children was 2.03×10^{-5} (1.35×10^{-5}, 2.60×10^{-5}) (**Figure 5C**). For adults, the median uncertainty at the P_{95} of the variability of carcinogenic risk was 1.46×10^{-5} (9.61×10^{-6}, 2.13×10^{-5}) (**Figure 5D**). The sensitivity analysis (**Table S5**) showed that the *C* of As in soil was the main exposure factor for children and adults (rho = 0.63).

4. Discussion

This study shows there are potential risks from exposure to metal(loid)s in soils intended for agricultural activities on Indigenous lands. In the Australian 1DMC simulations, children face a non-carcinogenic risk from oral exposure to Pb (HQ = 1.83) (**Figure 5A**) and in Newfoundland, children have a non-carcinogenic risk from exposure to As (HQ = 1.05) (**Figure 5B**), with soil contaminant concentration playing a significant role in both cases. Furthermore, our analyses indicate carcinogenic risks from As exposure affecting child ($R = 1.68 \times 10^{-5}$) and adult demographics ($R = 1.20 \times 10^{-5}$) in Newfoundland (**Figure 5C and 5D**). Despite these risks, the non-carcinogenic and carcinogenic risk values generally remain below risk thresholds for other metal(loid)s, indicating that under current exposure conditions, there is little to no risk from the remaining metals or any pesticides. Further, the 1DMC model, while simpler, provided a clear indication of the primary drivers of risk – namely, the *C* of contaminants in the soil. However, the 1DMC results are limited by their inability to separate the effects of variability and uncertainty, potentially underestimating or overestimating the risk by treating them as interchangeable.

The 2DMC builds on the 1DMC by going a step further to incorporate uncertainty (e.g., variations in *C*) as well as variability within the population (e.g., differences in IR). For example, while the 1DMC simulations identified a non-carcinogenic risk of exposure to Pb in Australia and As in Newfoundland for children, respectively, the 2DMC simulations revealed that the potential risk varied more significantly based on the same intake parameters (**Figure 5A and 5B**). The median uncertainty at the P_{95} of variability of non-carcinogenic risk for Pb exposure in Australian children was greater (HQ = 2.05) than the results of the 1DMC, suggesting that the risk could be higher than the risk identified in that model. The uncertainty was influenced by not only the *C* in this simulation, but the IR as well. In fact, the latter parameter was weak in the 1DMC sensitivity analysis (rho = -1.00×10^{-3}), but the strongest intake parameter in the 2DMC (rho = 0.56), thus emphasizing the role of the uncertainty in the IR driving the variability of Pb exposure risk. Similarly, for children's As exposure in Newfoundland, the 2DMC model suggested a higher potential non-carcinogenic risk (HQ = 1.22) than the 1DMC model; a similar increase in the IR was also seen in the sensitivity analysis, but the *C* remained the most influential risk parameter in the 2DMC simulations, only slightly however (**Table S5**).

For the carcinogenic risk of exposure to As for children ($R = 2.03 \times 10^{-5}$) and adults ($R = 1.46 \times 10^{-5}$) in Newfoundland, the R values derived from the 2DMC simulations are also consistently higher than those from the 1DMC simulations (**Figure 5C and 5D**), reflecting the 2DMC model's ability to capture a broader range of potential risk outcomes. Additionally, for the carcinogenic risk of exposure to As, for both children and adults in this same region, the sensitivity analysis showed slight increases in the *C* and IR (**Table S5**), suggesting a potential relationship between these parameters and the resulting risk. In other words, the *C* of As in the

423 soil and the amount of soil ingested (i.e., IR) are important determinants for assessing the
424 carcinogenic risk, not just the *C* as shown in the 1DMC sensitivity analysis.
425
426 The different results between the 1DMC and 2DMC simulations demonstrate the strengths and
427 weaknesses of each method. The 1DMC model provides a baseline risk estimate that is easier to
428 interpret but potentially less comprehensive in accounting for all sources of uncertainty. The
429 2DMC model, on the other hand, offers a more detailed risk assessment by considering the
430 relationship between variability and uncertainty. However, this increased complexity could
431 potentially lead to an overestimation of risk, especially when the input parameters were already
432 conservatively modeled. Therefore, we urge caution when interpreting the results of these
433 simulations independently. Instead, we recommend an integrated approach, viewing the risks as
434 informed by two distinct yet complementary methods.
435
436 **4.1 Non-carcinogenic risk of exposure to As in soils intended for agricultural purposes**
437 According to the CCME (2001), children have twice the percentage of daily exposure to As from
438 oral ingestion via soil than adults, and therefore, this warrants further discussion as the results
439 indicate that there is a non-carcinogenic risk of exposure to As from agricultural soils in
440 Newfoundland. For children, oral exposure – especially from hand-to-mouth activity – to
441 inorganic As in soils may be neurotoxic and can lead to early onset cognitive delays and
442 neurological deficits, and several studies (e.g., Wasserman et al. 2004; 2011; 2015; Wang et al.
443 2007a; von Ehrenstein et al. 2007) have demonstrated that IQ scores are negatively correlated to
444 oral As exposure, but mainly from drinking unfiltered well-water.
445

Historically, it was thought that exposures to only large doses of As were associated with negative health outcomes, like lowered IQ, but studies have demonstrated an association of lowered IQ scores with exposure to low concentrations of As as well (Tollins et al. 2014; Vaidya et al. 2023), which suggests the need for monitoring exposure to any concentration of inorganic As in soils. There is also an association with exposure to As in childhood and reduced lung function in later life (Dauphiné et al. 2011) which suggests that there is a critical exposure window in childhood and a need for intervention to avoid long-term health effects. It is noted, however, that these studies provide mixed evidence regarding the age of exposure to inorganic As, the age of onset of deficits due to previous exposure, and the specific dose that elicits a negative health outcome (Tsuji et al. 2015). Therefore, further study is required to fully elucidate the relationship between exposure to inorganic As and negative health outcomes. Additionally, exposure to inorganic As has been linked to type 2 diabetes (Kim et al. 2013; Navas-Acien et al. 2008) and cerebrovascular and cardiovascular diseases (Chen et al. 2011; Lee et al. 2002; Moon et al. 2012) such as hypertension and stroke.

The non-carcinogenic risk for As exposure in agricultural soils in this study is consistent with findings from other studies, which have reported comparable risk levels for children in regions with elevated As contamination in soils (Alsafran et al. 2021; Kharazi et al. 2021). While it is difficult to draw direct comparisons across regions due to different environmental and exposure conditions, the results suggests a need for further research to understand impact of As on children in different agricultural contexts. Additionally, the non-carcinogenic risk for As in our study is approximately 7% lower than the aforementioned studies, stressing both the importance

of localized assessments and the existing gaps in the literature regarding cross-regional risk evaluations.

4.2 Carcinogenic risk of exposure to As in soils intended for agricultural purposes

The International Agency for Research on Cancer (IARC) classifies As as a known human carcinogen (IARC, 2004), and the risk of cancer is higher from oral ingestion than dermal ingestion (Demissie et al. 2024). Of the 16 contaminants assessed in this study, As is the only contaminant with a carcinogenic risk to children and adults, but not youth. In particular, children have an oral carcinogenic risk of exposure to As; the ICR for children exceeds the ILCR and is also driven by the oral exposure to As (**Table 4**). There is also an oral carcinogenic risk of exposure to As for adults (**Table 5**). The results of this study demonstrate an increased carcinogenic risk from As exposure in soil, approximately 4% higher than a similar study (Agyemen et al. 2021), but lower than Mishra et al. (2024), which reported a carcinogenic risk nearly two orders of magnitude greater. For adults, however, the carcinogenic risk is generally comparable across these studies with little differences noted. What does this mean?

Following the exposure to As, for children, there is a potential association with acute lymphoblastic leukemia (ALL) and possibly epigenetic changes that may lead to leukemia (Infante-Rivard et al. 2007; Metayer et al. 2013). However, the association of As exposure with childhood leukemia is not fully elucidated and research is ongoing. Comparable studies on soil contamination have shown that children face higher As-related cancer risks from consuming crops grown in soils with elevated As concentrations, highlighting the vulnerability of this age group to soil-based contaminants in agricultural settings (Mishra et al. 2024). Furthermore, for

adults, there are several studies (e.g., Ahsan and Chen 2006; Ferreccio et al. 2000; López-Carrillo et al. 2014; Smith et al. 2006; Steinmaus et al. 2003) that show an association of pre-cancerous (i.e., skin lesions) or cancerous outcomes (i.e., cancer of the bladder, lung) following exposure to As from different media. Steinmaus et al. (2003), for example, found that exposure to even low concentrations of As are linked to an increased risk of bladder cancer, similar to findings from studies in contaminated agricultural regions, where adults faced elevated cancer risks from As in As-contaminated soil (Agyeman et al. 2021).

4.2.1 As in tested soils from Newfoundland

A spatial study of cancer outcomes in Newfoundland indicates that there may be added burden from environmental pollutants, like As, potentially leading to an additional lifetime risk of cancers (Rahman et al. 2020). We note however, as in most studies, the risk is estimated from the consumption of well-water, not soil, and that the geology of Newfoundland is highly varied, so we extrapolate the results of these studies to compare to our own with a cautionary approach. Though, this leads us to an important point: there are few studies on the risk of exposure to As from soils intended for agricultural activities, and more work is needed to understand if the risk is significant or not – if it poses the same risks as other routes of exposure, such as, ingestion from water or inhalation, and if health outcomes are strictly related to the species of As. For example, in our study, the prominent form of As in tested soils in Newfoundland was As(V) (Moriarity et al. 2024), which has different toxic properties than As(III) but can still pose a risk to health if ingested (Genchi et al. 2022). Lastly, to date, there is no evidence in the literature indicating increased cancers or other health outcomes as a result of exposure to As exposure from soil in our study region. Nevertheless, we present our results from this risk assessment that

a possibility of an oral risk of exposure to As from soils intended for agricultural activities may exist.

4.3 Non-carcinogenic risk to children from the exposure to Pb in agricultural soils

Studies on soil Pb contamination reveal similar non-carcinogenic risks for children, largely due to ingestion pathways. For example, Li et al. (2022) found that children exposed to Pb-contaminated agricultural soils had associated elevated blood Pb levels, with children under the age of three showing concentrations that exceeded acceptable thresholds. In regions with significant Pb contamination, non-carcinogenic risks may surpass thresholds, particularly for younger children who are more prone to frequent hand-to-mouth behaviors (Agyeman et al. 2021), which expands on ingestion rates as a route of exposure, however, findings from other similar assessments do not support the results of this study (Sarvestani et al. 2024; Yuan et al. 2021). These conflicting results likely reflect differences in soil Pb, variations in assessment methods and/or assumptions about ingestion rates and the use of different risk thresholds. However, these findings highlight the importance of continued and targeted investigations in contaminated agricultural areas and potential interventions to reduce health risks associated with soil ingestion.

Children are vulnerable to ingesting Pb-contaminated soil which can lead to increased blood Pb concentrations and impact development of the nervous system (EPA 2020; Roberts et al. 2022; WHO 2023b). The non-carcinogenic risk for children in Australia could indicate a need for intervention if the tested soils were to be used for agricultural purposes, to mitigate Pb exposure from ingested soils, particularly among young children, who are susceptible due to their frequent

hand-to-mouth activities and lower body weights, thus increasing the body burden of Pb (Chisolm 2008; Ramirez-Ortega et al. 2021). Chronic exposure to Pb, even at low levels, can lead to significant health issues (Lanphear et al. 2016), including cognitive impairment, developmental delays, and exacerbation of existing health problems (Lasley 2018; Ruebner et al. 2019; Schultheiss et al. 2020). However, it is important to consider that specific soil Pb concentrations found in this study and their implications for children's health need to incorporate more detailed factors like the bioaccessibility of Pb in the soil, and the exposure scenarios relevant to children in particular regions, globally.

4.4 Recommendations

The findings suggest a potential need to mitigate exposure to specific toxicants (i.e., Pb, As), in specific regions. Recommended actions include soil replacement where feasible, issuing public health advisories, and implementing community education programs to minimize direct contact with contaminated soil, particularly among children. Since remediation is not always possible for naturally occurring contaminants such as As, implementing community-led awareness programs to help educate parents and caregivers about the risks of exposure could be helpful in reducing risk (e.g., ensuring children's hands are washed frequently). Regular screening of biomarkers (e.g., blood, urine) for Indigenous Peoples living in these regions could also be part of a public health response to monitor the ongoing risk to health, but it is invasive. Lastly, ongoing monitoring of As and Pb concentrations in soils and additional research are necessary to fully understand the spatial distribution of contamination and its potential health impacts over time.

4.5 Limitations

This study has limitations that should be discussed. The soil collection period was over the course of 10 years and non-consecutive, and there may have been slight differences in collection methods across the different sites; this extended period could introduce variability or uncertainty due to environmental or land use changes. Importantly, the human health risk assessment models rely on estimated and randomized modeled variables, which may not capture the complex dynamics of exposure to the measured contaminants. Additionally, some intake parameters, although from other similar studies, may not represent specific communities included in the present study. A disadvantage of the 2DMC is that it is highly sensitive to errors in the input data, which could result in potential overestimation of the risk simulations. An additional concern is that the EF's are based on observations for adults, therefore, we had to make the assumption that children were on the land when their parents were, but this assumption might not fully capture the actual EF's for children. Despite these potential limitations, we are confident that our findings provide a global assessment of the human health risks associated with exposure to agricultural soil contamination on Indigenous lands.

5. Conclusion

This study assessed the risk from exposure to contaminants in soils intended for agricultural activities using 1DMC and 2DMC probabilistic risk simulations. Findings indicate that Pb was a primary contaminant of concern in Australian soils, and As in Newfoundland soils posed the greatest health risks. For children in Australia, exposure to Pb presents a potential non-carcinogenic risk, while in Newfoundland, As exposure poses both non-carcinogenic and carcinogenic risks for children. Adults also face a carcinogenic risk of exposure to As in Newfoundland, however, the risk from other metal(loid)s and organic pesticides remain below

risk thresholds for all demographics. The 2DMC approach revealed more variability in the PRA by incorporating uncertainty and demonstrated factors such as *C* and IR were both important intake parameters for the non-carcinogenic risk of exposure from Pb and As for children. These findings stress the need for targeted interventions, such as monitoring and mitigation strategies, in regions where agricultural activities occur on contaminated soils. We recommend follow-up and monitoring in these regions to mitigate exposure and to ensure safe agricultural activities.

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Figure Captions

Figure 1. Relative location map of participating communities in (A) Australia and (B) Canada where soils were sampled.

Figure 2. (A) The first figure shows a one-dimensional Monte Carlo (1DMC) simulation for assessing oral soil ingestion. Parameters (C, IR, ED, EF, AT, BW) are set with probability density functions (PDFs) and iterated (i = 1 to 10^5). Intake (I) is calculated and used to evaluate non-carcinogenic risk using the RfD or carcinogenic risk using the SF. The histograms show the risk probability distributions, with key thresholds marked. (B) The second figure shows a two-dimensional (2DMC) Monte Carlo simulation process, combining both variability and uncertainty iterations. Uncertainty iterations (i = 1 to 10^3) encompass nested variability iterations (i = 1 to 10^4) for all parameters. The C parameter is included separately as uncertainty. The hybrid aspect of the intake IR is highlighted, as it is subject to both uncertainty and variability. The resulting intake values are then used to evaluate non-carcinogenic or carcinogenic risk. Box plots at the bottom display the distribution of non-carcinogenic and carcinogenic risks across different percentiles. (*): indicates that AT is only considered in iterations for non-carcinogenic risk as the AT for carcinogenic risk is a fixed factor, not modeled from a PDF.

Figure 3. (A) Probability distribution of non-carcinogenic risk for children exposed to lead (Pb) in Australia and (B) for children exposed to arsenic (As) in Newfoundland, with the hazard quotient (HQ) threshold set at 1.00 (orange dashed line) and the 95th-percentile (P_{95}) in red. Below each histogram, bar plots show the sensitivity analysis of risk parameters. The bars represent the correlation coefficients (ρ) of each parameter (AT: Averaging Time, BW: Body Weight, C: Concentration, ED: Exposure Duration, EF: Exposure Frequency, IR: Intake Rate) with non-carcinogenic risk.

Figure 4. Probability distribution of carcinogenic risk for children (A) and for adults (B) exposed to arsenic (As) in Newfoundland, with the incremental lifetime cancer risk (ILCR) threshold set at 1.00 x 10^{-5} (orange dashed line) and the P_{95}. Below each histogram, bar plots show the sensitivity analysis of risk parameters. The bars represent the correlation coefficients (ρ) of each parameter (BW: Body Weight, C: Concentration, ED: Exposure Duration, EF: Exposure Frequency, IR: Intake Rate) with carcinogenic risk.

Figure 5. Uncertainty boxplots illustrate the distribution of non-carcinogenic (A and B) and carcinogenic (C and D) variability of risk at 5th, 25th, 50th, 75th, and 95th-percentiles from the 2DMC models. Panel (A) shows non-carcinogenic risk for children exposed to lead (Pb) in Australia. Panel (B) shows non-carcinogenic risk for children exposed to arsenic (As) in Newfoundland. Panel (C) shows carcinogenic risk for children exposed to arsenic (As) and panel (D) shows carcinogenic risk for adults exposed to arsenic (As), both in Newfoundland. The orange lines connect the 1DMC model values at each percentile, providing a reference for comparison. The dashed red lines indicate threshold values for acceptable risk levels: a hazard quotient (HQ) of 1.00 for non-carcinogenic risk and an incremental lifetime cancer risk (ILCR) of 1.00 x 10^{-5} for carcinogenic risk. Each boxplot shows the median (black line), interquartile range (IQR), and outliers of the risk distributions.

Table 1. Contaminants assessed for human health risk in soils intended for agricultural purposes.

Contaminant	
Carcinogenic risk (n=8)	**Non-carcinogenic risk** (n=5)
(As) Arsenic	(As) Arsenic
Dieldrin	(Co) Cobalt
o,p'-DDD (2,4-Dichlorodiphenyl-dichloroethane)	(Cr) Chromium
o,p'-DDE (2,4-Dichlorodiphenyl-dichloroethylene)	(Ni) Nickel
o,p'-DDT (2,4-Dichlorodiphenyl-trichloroethane)	(Pb) Lead
p,p'-DDD (4,4-Dichlorodiphenyl-dichloroethane)	
p,p'-DDE (4,4-Dichlorodiphenyl-dichloroethylene)	
p,p'-DDT (4,4-Dichlorodiphenyl-trichloroethane)	

Table 2. Variables used to calculate the intake of contaminants from soils intended for agricultural purposes.

Intake variable	Children	Youths	Adults	Unit	Distribution	References
$ABS_{Co,Cr,Ni}$		0.010		-	Deterministic	Environmental Protection Agency [EPA] (2023)
ABS_{As}		0.032		-	Deterministic	EPA (2023)
ABS_{Pb}		0.006		-	Deterministic	Health Canada (2021b)
$ABS_{Pesticides}$		0.100		-	Deterministic	EPA (2023)
AF	0.20(0, 3.30)	0.20(0, 0.30)	0.07(0, 0.30)	mg/cm^2 d	Triangular	International Atomic Energy Agency (1995); Men (2021), EPA (2023)
$AT_{Co,Cr,Ni}$	*U*(0, 4015)	*U*(0, 7300)	*U*(0, 25550)	d	Uniform	Based on the ED · 365 in this study
$AT_{As,\ Pesticides}$		25550		d	Deterministic	AT for carcinogenic risk
BW	*N*(30, 7.5)	*N*(60, 15)	*N*(90, 22.5)	kg	Normal	CCHS 2.2 data in Health Canada (2021a)
C		-		mg/kg	Lognormal	Current study
EF_{AUS}		108(87, 130)		d/y	Triangular	Current study
EF_{CAN}		60(45, 75)				
ED	*U*(0, 11)	*U*(0, 17)	*U*(0, 70)	y	Uniform	Canadian Community Health Survey (CCHS) 2.2 data in Health Canada (2021a)
IR	μ: 159; σ: 270; P_{50}: 81; P_{90}: 360	μ: 53 σ: 90; P_{50}: 27; P_{90}: 120		mg/d	Lognormal	Adapted from Doyle et al. (2012) and Irvine et al. (2014)
SA	*N*(1020, 255)	*N*(1590, 398)	*N*(2080, 520)	cm^2	Normal	Adapted from Health Canada (1995)

Key: *N*(M, SD): normal distribution, mean and standard deviation; IR values: μ: mean; σ: standard deviation; P_{50}: 50^{th}-percentile (median); P_{90}: 90^{th}-percentile; AF and EF values: mode(min, max); *U(*min, max): uniform distribution, minimum and maximum values.

Table 3. Non-carcinogenic risk of exposure to contaminants from soils intended for agricultural activities.

Location	Contaminant		Demographic								
			Children			Youth			Adult		
			HQ_D	**HQ_O**	**HI**	**HQ_D**	**HQ_O**	**HI**	**HQ_D**	**HQ_O**	**HI**
Australia	Metals	Co	1.88E-03	2.32E-02	2.51E-02	1.84E-04	3.39E-03	3.57E-03	1.37E-04	2.65E-03	2.79E-03
		Cr	5.61E-05	6.92E-04	7.48E-04	5.19E-06	9.66E-05	1.02E-04	4.07E-06	7.83E-05	8.24E-05
		Ni	1.75E-03	2.23E-02	2.41E-02	1.67E-04	3.14E-03	3.31E-03	1.31E-04	2.48E-03	2.61E-03
		Pb	1.21E-02	**1.83E+00**	**1.84E+00**	1.18E-03	2.56E-01	2.58E-01	9.10E-04	2.02E-01	2.02E-01
		THQ	1.58E-02	**1.88E+00**		1.54E-03	2.63E-01		1.18E-03	2.07E-01	
				THI	**1.89E+00**		**THI**	2.65E-01		**THI**	2.08E-01
Western James Bay		As	1.43E-02	5.95E-02	7.38E-02	1.35E-03	8.41E-03	9.76E-03	1.06E-03	6.59E-03	7.65E-03
		Co	2.11E-04	2.80E-03	3.01E-03	1.99E-05	3.94E-04	4.13E-04	1.55E-05	3.10E-04	3.26E-04
		Cr	9.51E-06	1.26E-04	1.36E-04	8.98E-07	1.80E-05	1.88E-05	7.06E-07	1.41E-05	1.48E-05
		Ni	4.06E-04	5.40E-03	5.81E-03	3.83E-05	7.69E-04	8.07E-04	3.01E-05	5.96E-04	6.26E-04
		Pb	7.70E-04	1.23E-01	1.24E-01	7.23E-05	1.75E-02	1.75E-02	5.69E-05	1.35E-02	1.36E-02
		THQ	1.57E-02	1.91E-01		1.48E-03	2.71E-02		1.16E-03	2.11E-02	
				THI	2.06E-01		**THI**	2.85E-02		**THI**	2.22E-02
Newfoundland		As	2.76E-01	**1.05E+00**	**1.32E+00**	2.65E-02	1.46E-01	1.73E-01	2.05E-02	1.17E-01	1.38E-01
		Co	3.39E-04	4.40E-03	4.74E-03	3.24E-05	6.21E-04	6.53E-04	2.46E-05	4.93E-04	5.17E-04
		Cr	1.18E-05	1.56E-04	1.68E-04	1.16E-06	2.25E-05	2.37E-05	8.92E-07	1.76E-05	1.85E-05
		Ni	7.94E-04	1.00E-02	1.08E-02	7.61E-05	1.45E-03	1.53E-03	5.88E-05	1.14E-03	1.20E-03
		Pb	1.57E-03	2.48E-01	2.49E-01	1.49E-04	3.54E-02	3.56E-02	1.16E-04	2.77E-02	2.78E-02
		THQ	2.79E-01	**1.31E+00**		2.68E-02	1.84E-01		2.07E-02	1.47E-01	
				THI	**1.59E+00**		**THI**	2.11E-01		**THI**	1.6E-01
Western James Bay	Pesticides	Dieldrin	7.28E-03	8.55E-03	1.58E-02	7.15E-04	1.21E-03	1.93E-03	5.29E-04	9.38E-04	1.47E-03
		Endosulfan sulfate	1.02E-05	1.26E-05	2.28E-05	9.59E-07	1.76E-05	1.86E-05	7.41E-07	1.39E-05	1.46E-05
		p,p'-DDE	4.21E-03	4.62E-03	8.83E-03	4.13E-03	6.71E-04	4.80E-03	3.11E-04	5.08E-04	8.19E-04

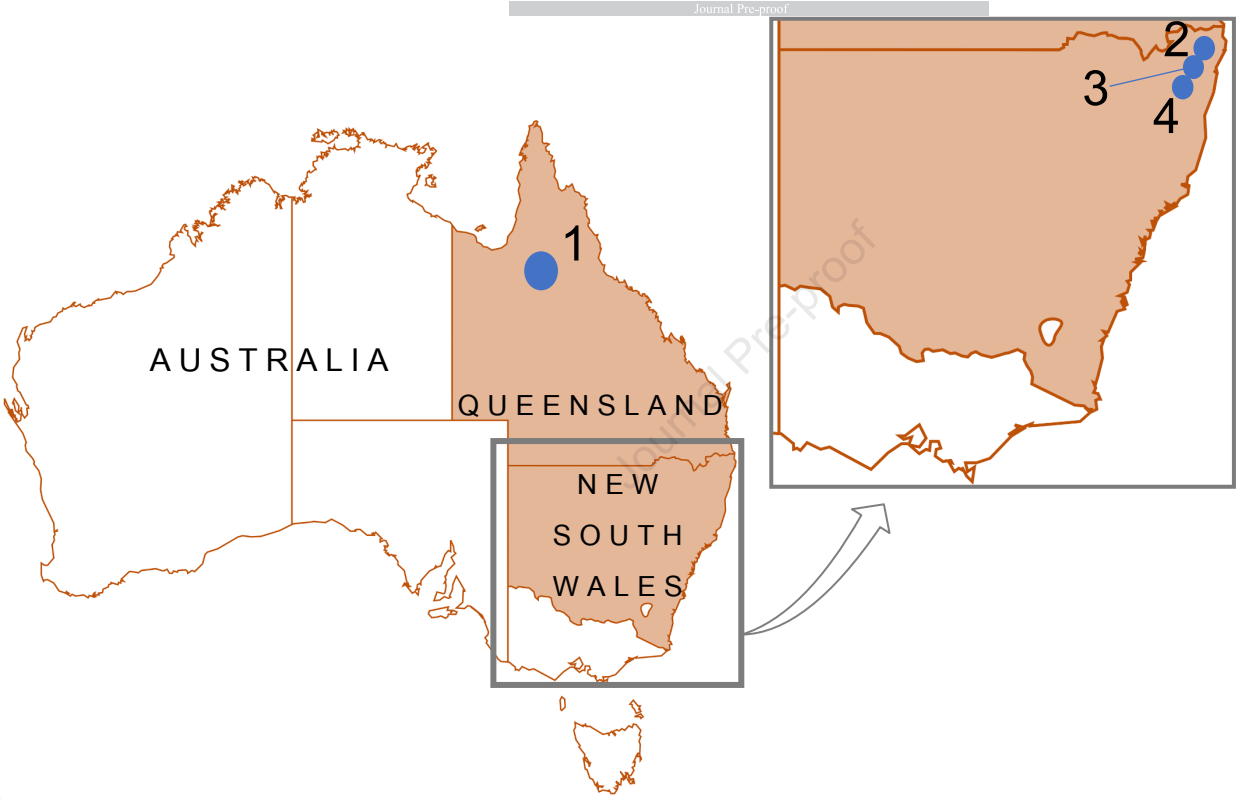
p,p'-DDD	6.76E-04	7.97E-04	1.47E-03	6.51E-05	1.11E-04	1.76E-04	5.00E-05	8.77E-05	1.38E-04
p,p'-DDT	1.01E-02	1.09E-02	2.10E-02	9.94E-04	1.53E-03	2.52E-03	7.43E-04	1.18E-03	1.92E-03
THQ	2.23E-02	2.49E-02		5.91E-03	3.54E-03		1.63E-03	2.73E-03	
		THI	4.72E-02		**THI**	9.44E-03		**THI**	4.36E-03

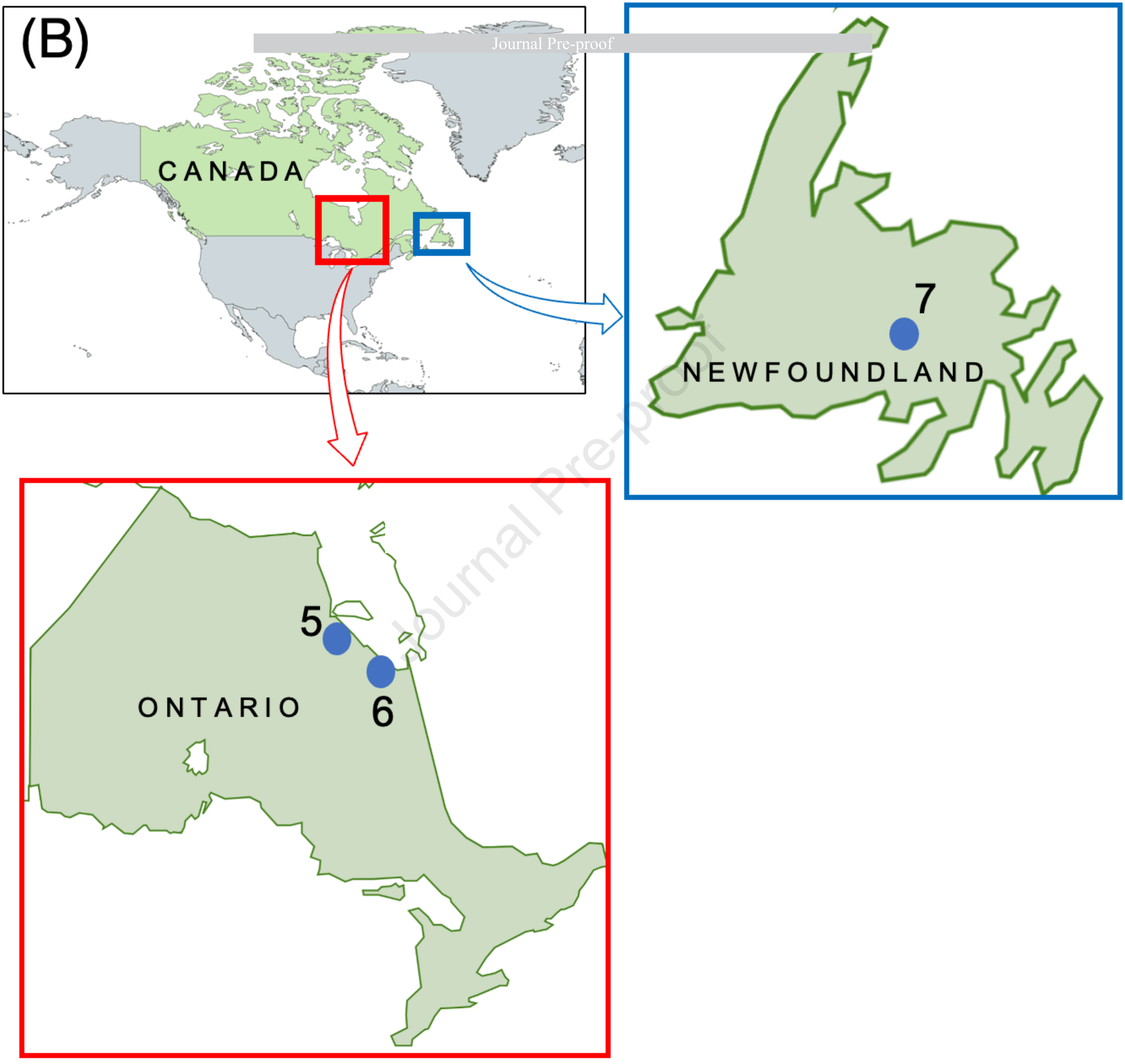
Key: HQ_D: dermal non-carcinogenic risk of exposure; HD_O: oral non-carcinogenic risk of exposure; HI: hazard index, THQ: total hazard, THI: total hazard index
Bold indicates HQ, HI, THQ or THI greater than 1.00.

Table 4. Carcinogenic risk of exposure to contaminants from soils intended for agricultural activities.

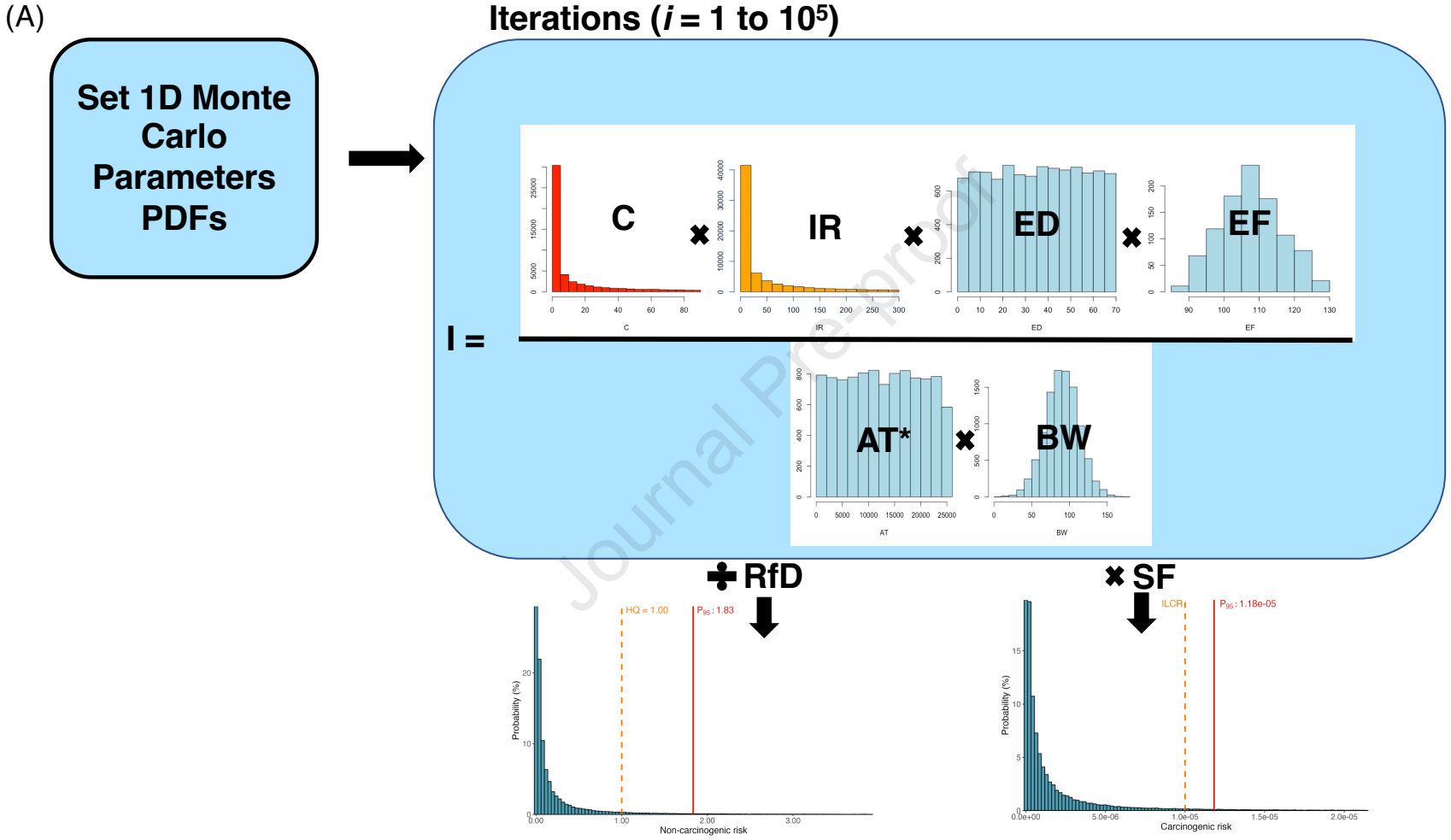
Location	Contaminant		Demographic								
			Children			Youth			Adult		
			R_D	R_O	ICR	R_D	R_O	ICR	R_D	R_O	ICR
Western James Bay	Metals	As	1.67E-07	8.15E-07	9.82E-07	2.46E-08	2.11E-07	2.36E-07	5.86E-07	1.00E-06	**1.59E-06[a]**
Newfoundland		As	4.25E-06	**1.68E-05[b]**	**2.11E-05[b]**	7.05E-07	4.29E-06	5.00E-06	1.92E-06	**1.18E-05[b]**	**1.37E-05[b]**
Western James Bay	Pesticides	Dieldrin	2.15E-07	2.53E-07	4.68E-07	3.73E-08	6.64E-08	1.04E-07	9.48E-08	1.84E-07	2.79E-07
		p,p'-DDE	2.85E-08	3.11E-08	5.96E-08	5.07E-09	7.99E-09	1.31E-08	1.34E-08	2.22E-08	3.56E-08
		p,p'-DDD	2.85E-09	5.00E-09	7.85E-09	4.99E-10	1.27E-09	1.77E-09	1.31E-09	3.61E-09	4.92E-09
		p,p'-DDT	6.95E-08	7.68E-08	1.46E-07	1.27E-08	1.97E-08	3.24E-08	3.24E-08	5.48E-08	8.72E-08
		TR	3.16E-07	3.66E-07		5.56E-08	9.54E-08		1.42E-07	2.65E-07	
				TICR	6.82E-07		TICR	1.51E-07		TICR	4.07E-07

Key: R_D: dermal carcinogenic risk of exposure; R_O: oral carcinogenic risk of exposure; TR: total risk, ICR: incremental cancer risk, TICR: total incremental cancer risk
Bold[a] indicates risk that exceeds the *de minimis* risk (1.00×10^{-6}) for Western James Bay only and bold[b] indicates a risk that exceeds the Incremental Lifetime Cancer Risk (1.00×10^{-5}) for Newfoundland only.





(A)



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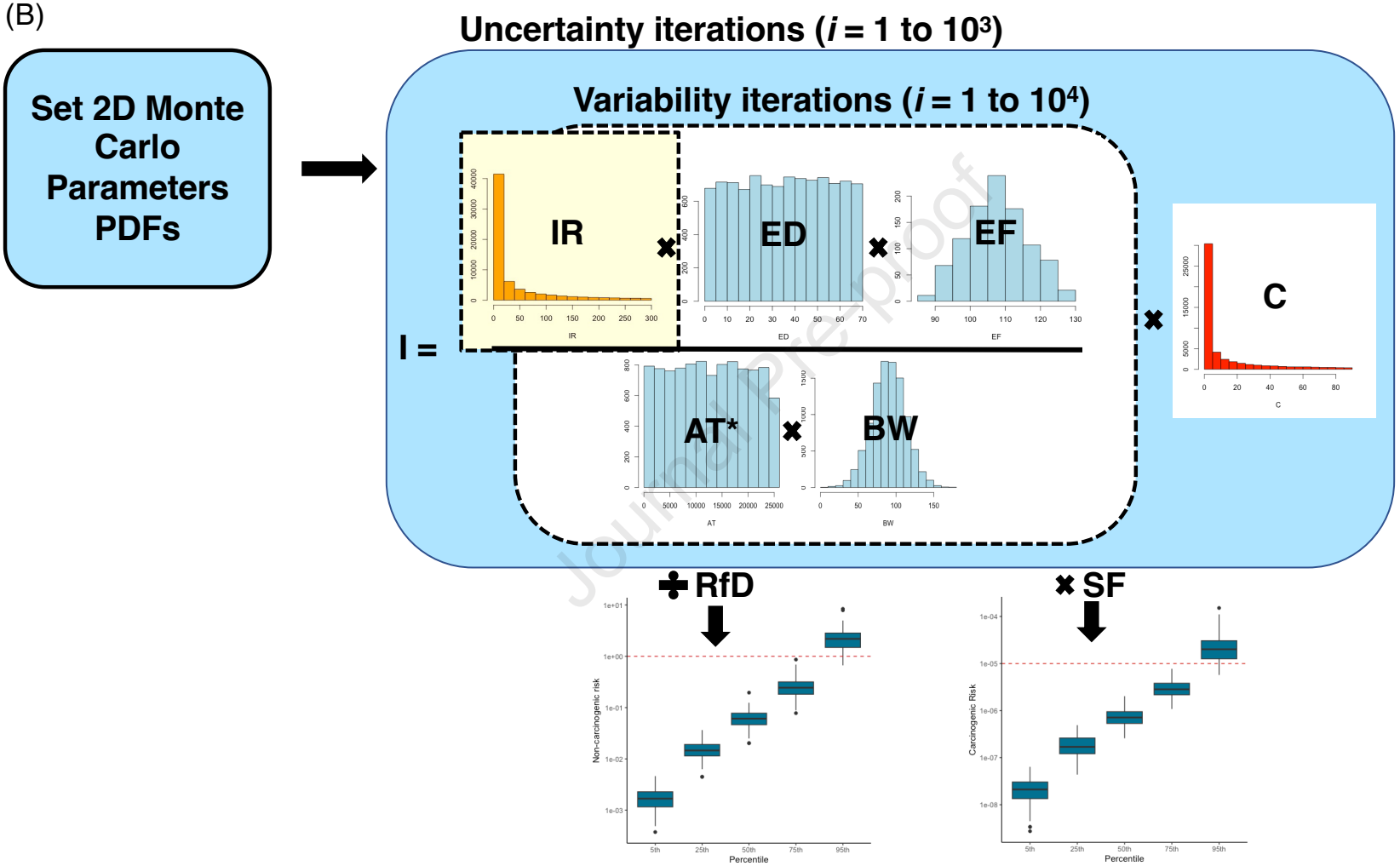

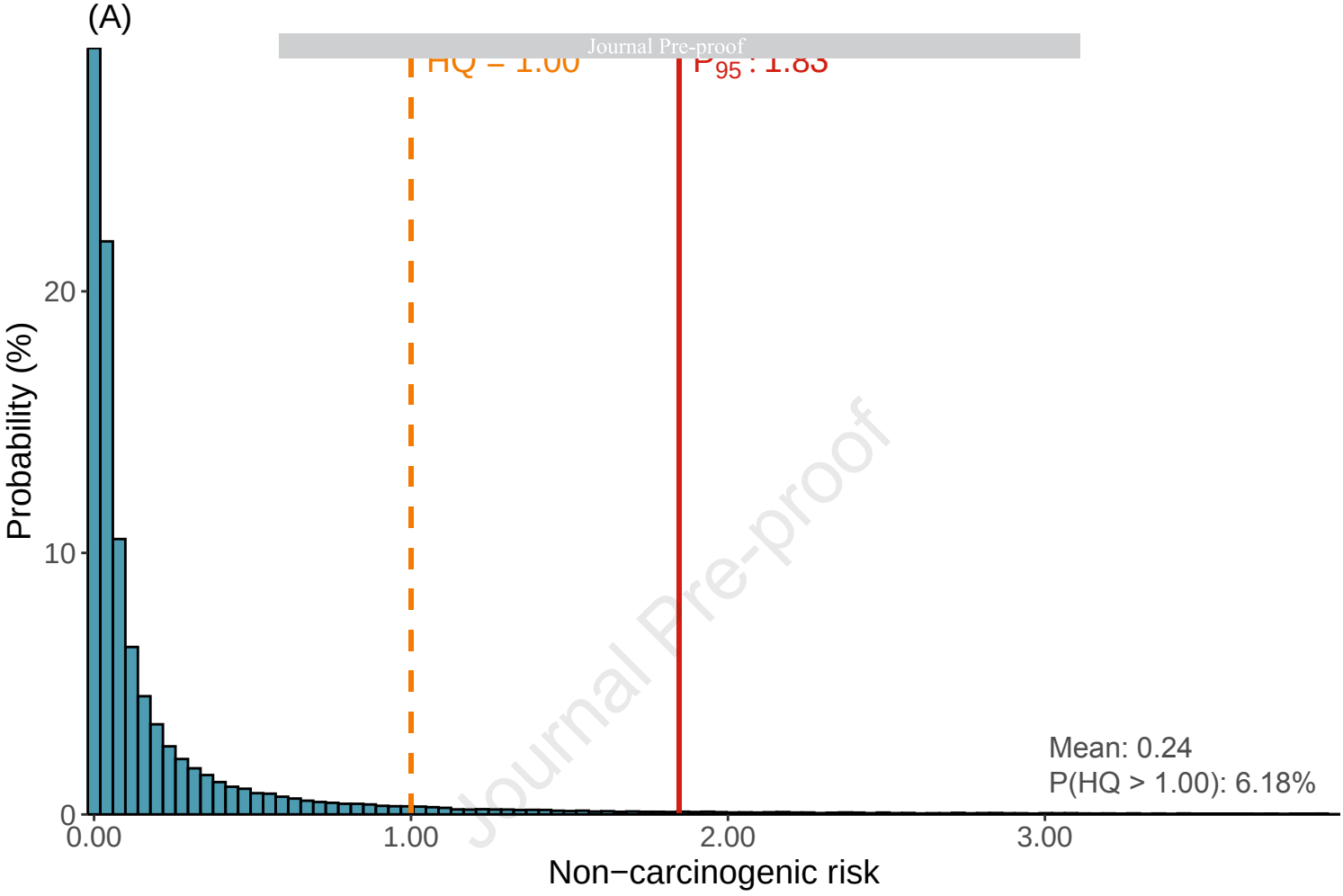

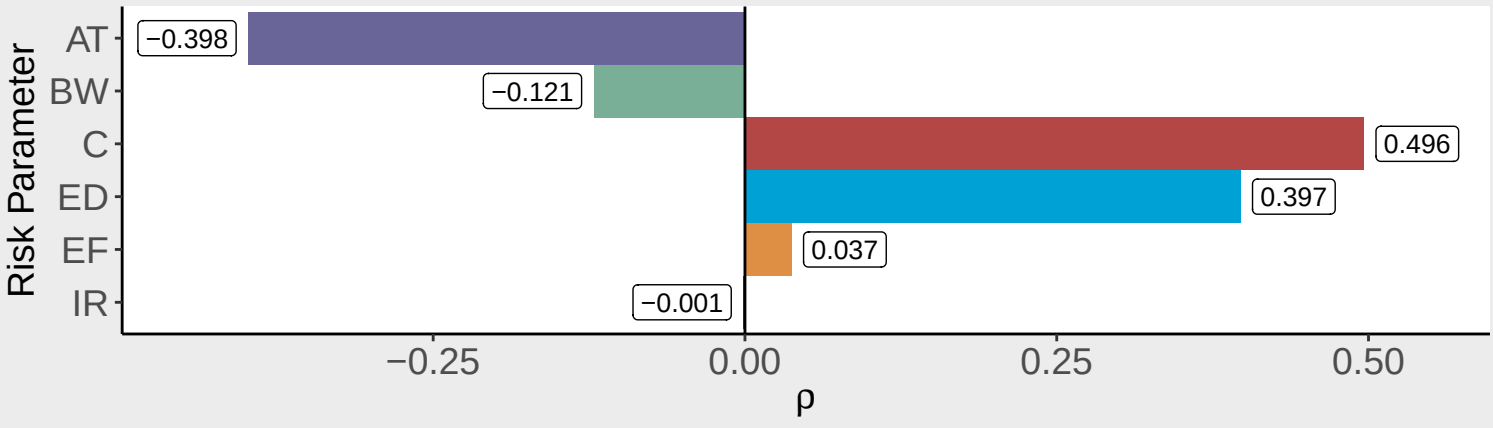

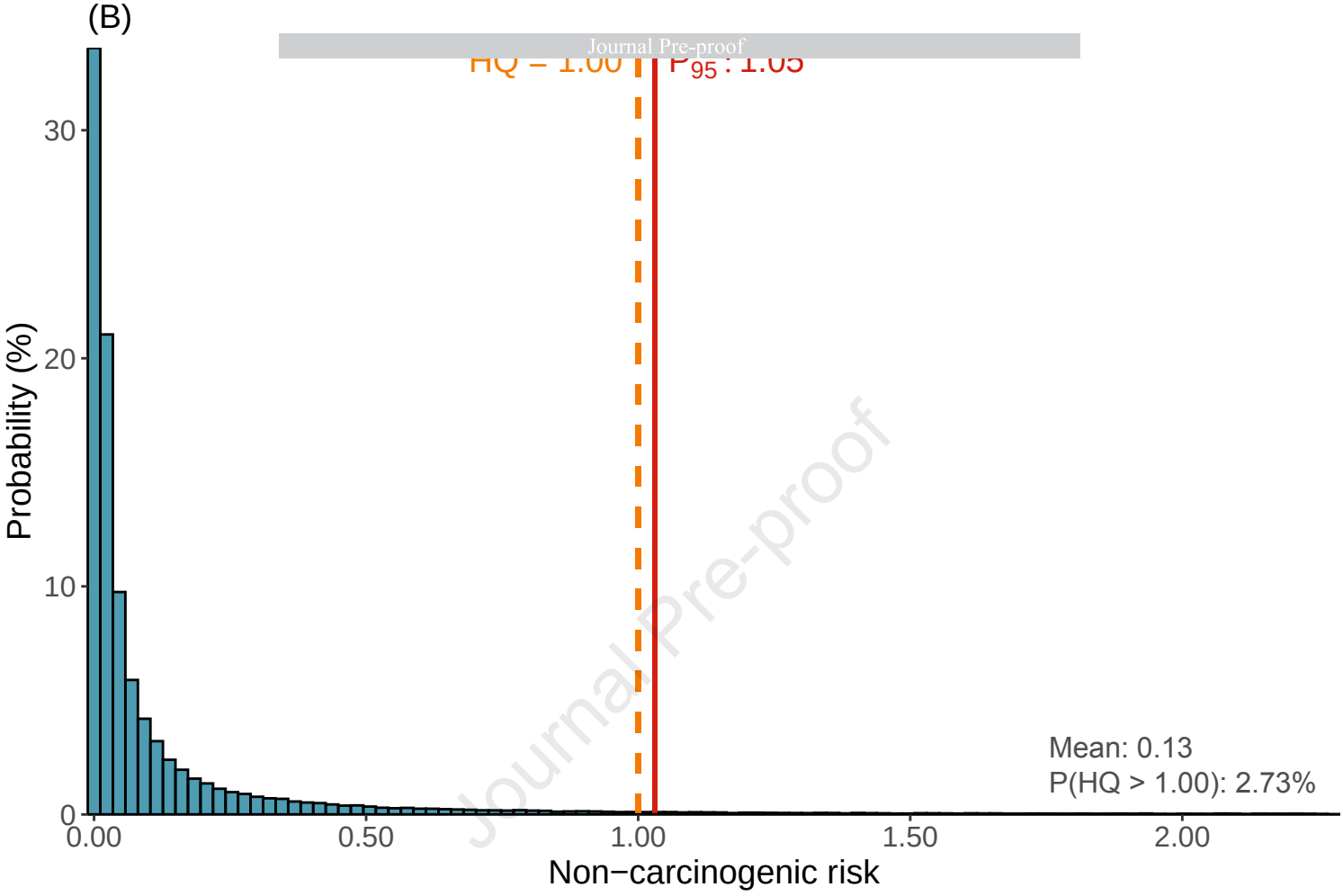

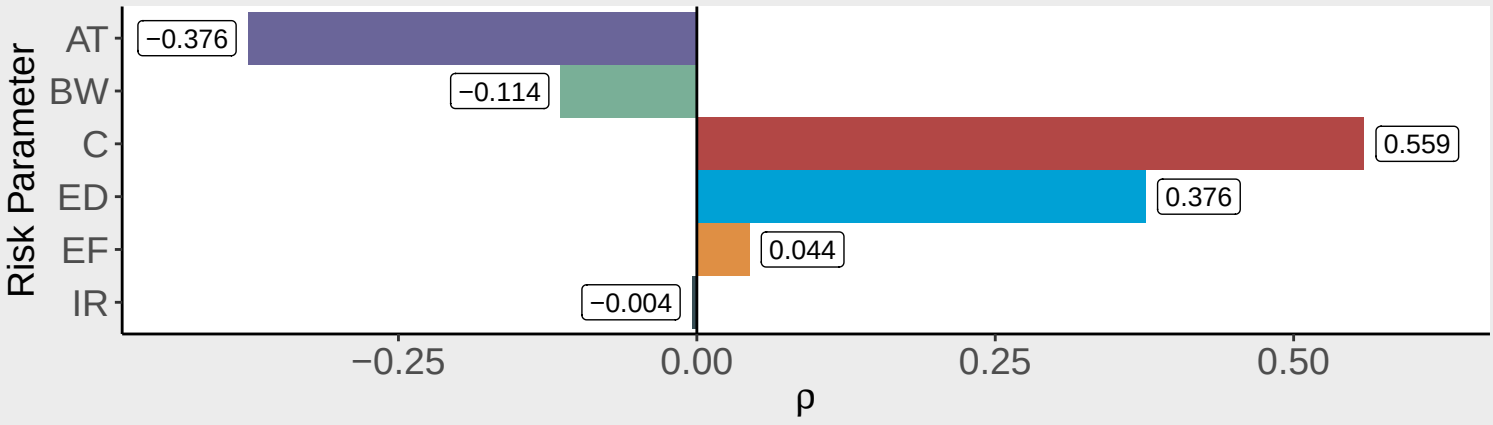

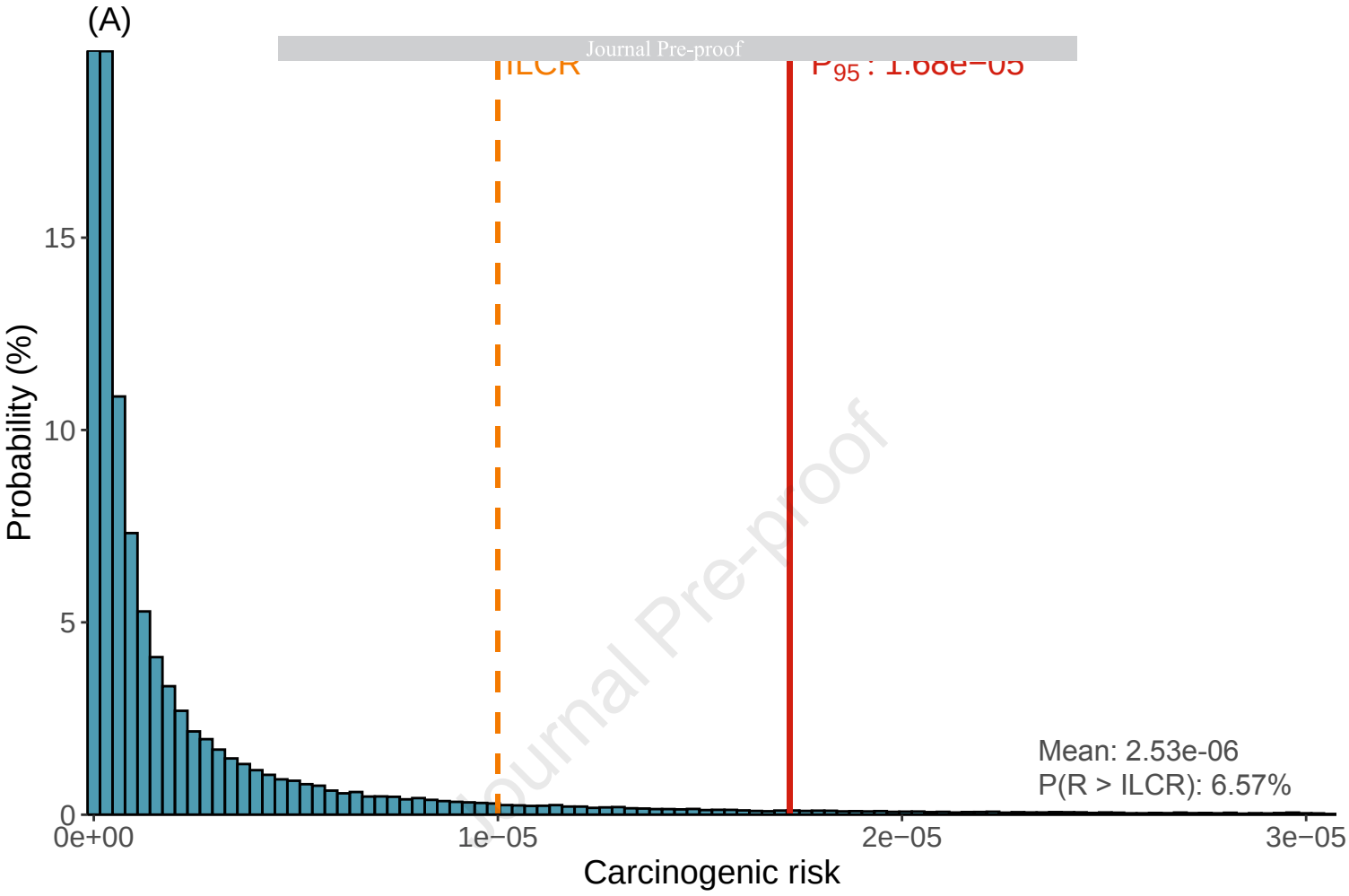

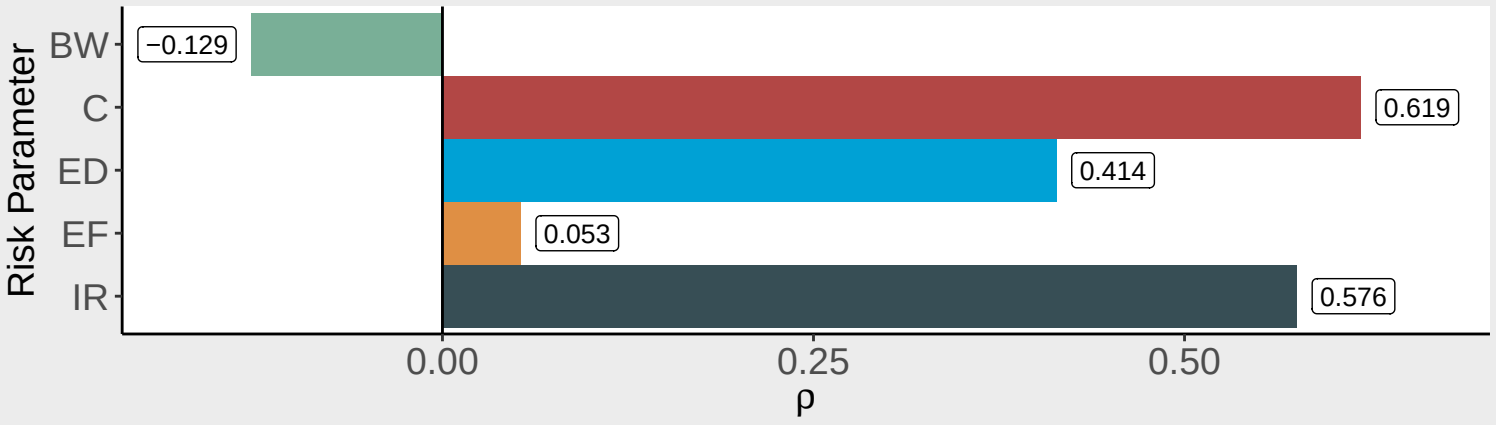

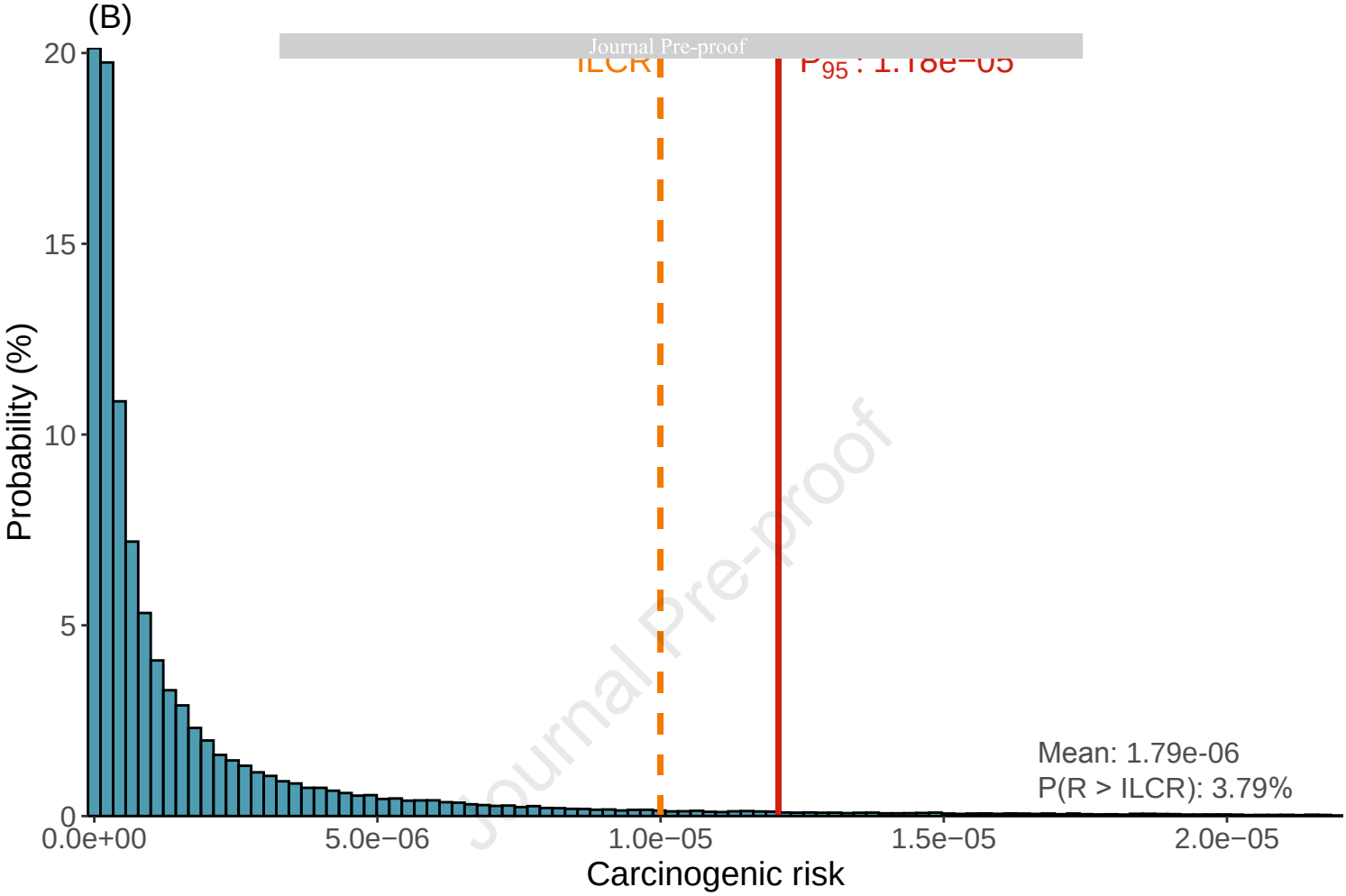

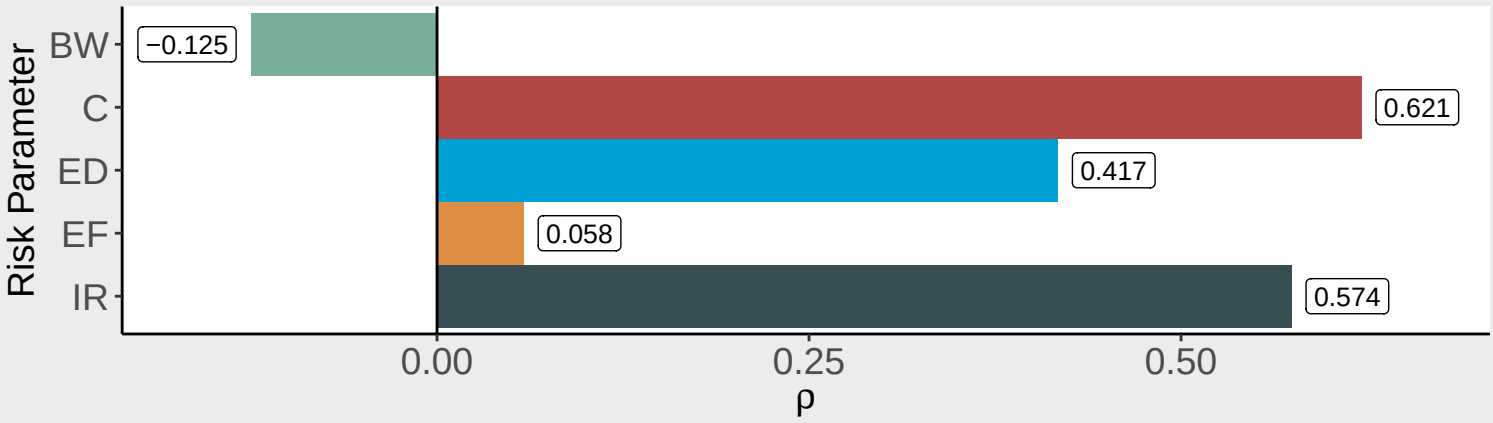

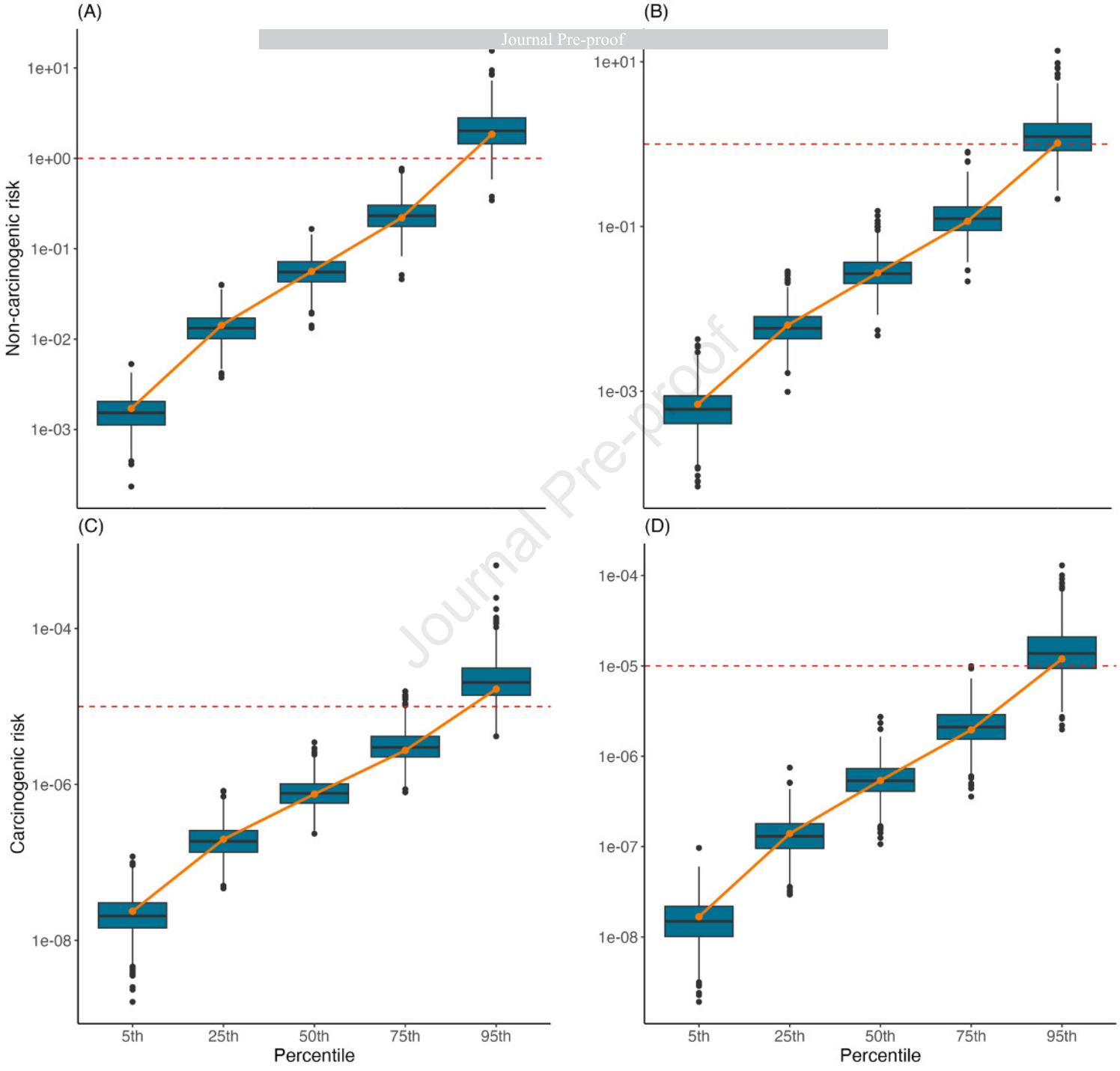

Highlights

- Monte Carlo simulations reveal risks from soils used for agricultural activities.
- Children in Australia face non-carcinogenic risk from Pb in agricultural soils.
- As in Newfoundland soils linked to carcinogenic risk for children and adults.
- Soil remediation and ongoing education efforts are recommended to reduce exposure risks.

Declaration of interests

☒ The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

☐ The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: