



Model-Based Assessment of Geogenic Radon Exposure in Chaco Canyon and Adjacent Ancestral Puebloan Residential Settings

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Abstract

Naturally occurring radon-222 and its short-lived alpha-emitting progeny are important sources of environmental radioactivity in enclosed, earth-contact spaces, yet their potential relevance to archaeological built environments has rarely been quantified. Here we evaluate chronic radon exposure in Chaco Canyon, where Ancestral Pueblo people occupied earth-contact and semi-subterranean rooms associated with Pueblo Bonito and other great houses during AD 850–1150. We developed a Chaco-specific source-to-risk model using an ensemble of uranium-miner radon risk functions to link uranium- and radium-bearing geology, soil-gas radon production, diffusion- and advection-driven indoor entry, ventilation, dust/smoke-enhanced progeny exposure, floor-level occupancy, premodern survival, and radon-associated lung-cancer mortality. The model converts indoor radon scenarios to working-level-month exposure and propagates risk through reconstructed birth cohorts and competing mortality. Under the reference population trajectory, modeled exposure produced notable conditional lifetime lung-cancer risk among long-lived residents and an age-structured mortality burden during AD 850–1150. Because survival into older adulthood was limited, realized population-level burden remained demographically constrained even where conditional individual risk was elevated. We do not propose radon as a primary cause of Chacoan depopulation, Pueblo Bonito decline, or regional abandonment. Rather, the results indicate that elevated radon in selected earth-contact microenvironments could have imposed a chronic, elder-concentrated health burden within an already stressed social and ecological system, making geogenic radioactivity a testable contributor to cumulative vulnerability.

Keywords: Radon-222; indoor radon; source-to-risk modeling; uranium-bearing strata; lifetime radiological risk; Chaco Canyon; Ancestral Pueblo.

1 Introduction

Naturally occurring radon-222 and its short-lived alpha-emitting progeny are important sources of environmental radioactivity in enclosed, earth-contact environments, including the semi-subterranean and floor-contact spaces documented in Chaco Canyon, New Mexico, USA (**Figure 1**). The monumental archaeological remains of Chacoan culture, crowned by Pueblo Bonito and other major great houses, together with more modest clusters of surrounding residential structures, testify to the achievements and sophistication of the Ancestral Pueblo people (Bustard, 1999; Crown, 2018; Mahoney and Kantner, 2000). Ancestral Puebloan communities thrived there from approximately AD 850 to 1150. Following that period, settlements within Chaco Canyon and the Chaco Regional System (CRS) experienced a relatively rapid decline (Judge, 1989; Lekson, 1984; Lekson and Cameron, 1995; Matteson, 2018; Sebastian, 1992; Vivian, 1990), and the Chaco Canyon site was eventually abandoned by AD 1300 (Kohler et al., 2025).

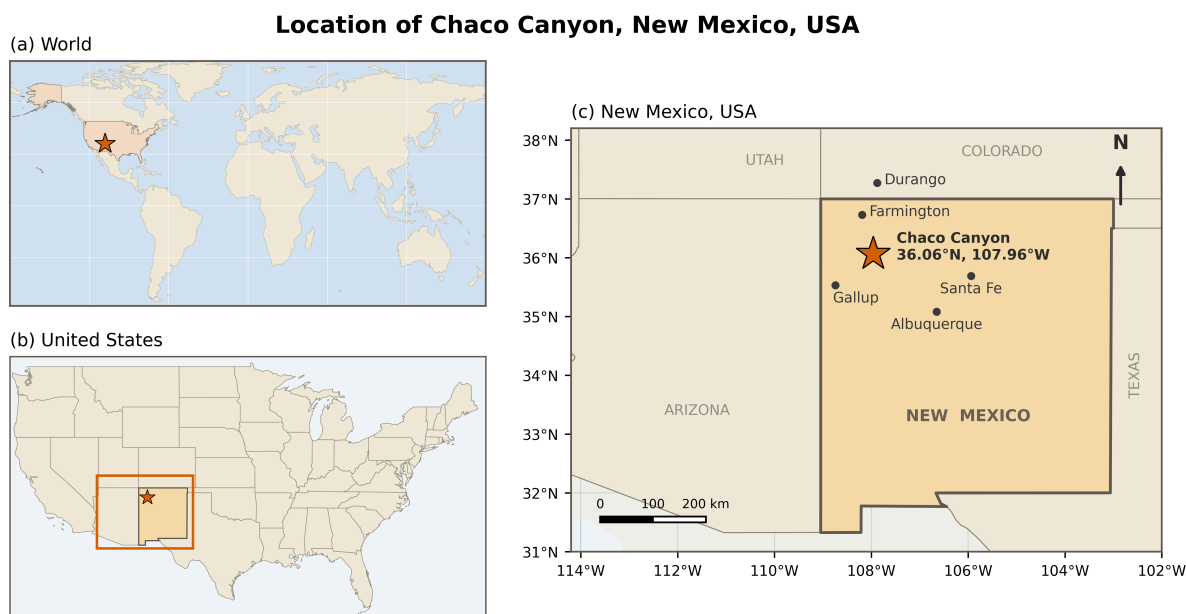


Figure 1: Location of Chaco Canyon, New Mexico, USA. (a) World, (b) the United States with New Mexico highlighted, and (c) the New Mexico / Four Corners region, with Chaco Canyon (36.06°N , 107.96°W) marked. This locator provides the geographic reference for the geologic, architectural, and demographic analyses.

Over more than a century of intensive archaeological research (Mills, 2002), substantial progress has been made in understanding the social organization, regional influence, and architectural complexity of Chacoan society. Archaeologists have also sought to explain the decline and eventual depopulation of this remarkable center of the American Southwest. Existing interpretations commonly emphasize climate, hydrology, agricultural productivity, resource depletion, migration, violence, trade, and social reorganization as intersecting factors that contributed to the collapse or reorganization of Chacoan society (Benson et al., 2019; Berger, 2009; Lekson, 2016; Lekson and Cameron, 1995; Plog, 2015; Van Dyke et al., 2016; Wills et al., 2024). Importantly, modern scholarship has generally rejected single-cause explanations.

The terminal Bonito-phase depopulation, conventionally centered around AD 1150 and completed by approximately AD 1300, remains among the most debated demographic transitions in North American archaeology. Mainstream interpretations now converge on a multicausal framework involving the AD 1130–1180 megadrought (Cook et al., 2010; Salzer and Kipfmüller, 2005; Williams et al., 2020), subsistence and agricultural stress in a marginal canyon environment (Benson et al., 2019; Vivian, 1990), sociopolitical reorganization (Lekson and Cameron, 1995; Plog and Heitman, 2010), episodic violence (LeBlanc, 1999; Turner and Turner, 1999), and broader Pueblo-world reorganization and continuity (Lekson and Cameron, 1995; Pinotti et al., 2025). Wills et al. (2014) further argued that no single proposed cultural or natural cause adequately explains the documented depopulation, leaving room for additional contributing factors. This underscores that significant uncertainties remain concerning the evolution, resilience, and eventual decline of Chacoan society.

One of the most important unresolved issues is population size. Published estimates for Chaco Canyon between AD 850 and 1150 vary substantially (e.g., Mills, 2004; Price et al., 2017; Kohler et al., 2025) and remain central to one of the most unresolved demographic transitions in North American archaeology. Peak Bonito-phase population estimates for the canyon proper range from 2,000 to 6,000 individuals, while the broader Chacoan regional system may have supported populations one to two orders of magnitude larger (Windes et al., 1984; Lekson et al., 1988; Benson et al., 2019; Kohler et al., 2025). This demographic uncertainty remains central to evaluating any proposed stressor, including environmental or geogenic hazards.

From this broader perspective, it is timely to ask whether additional environmental or geogenic

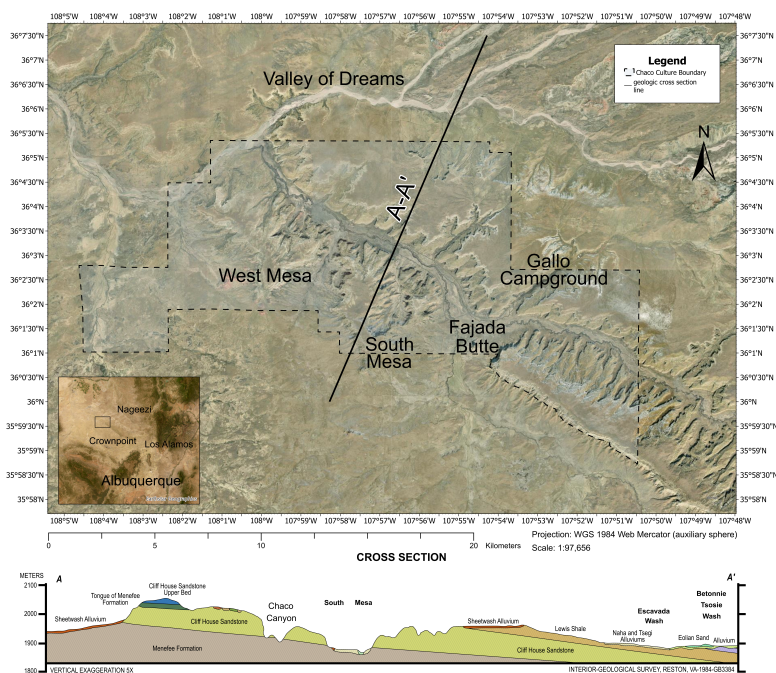


Figure 2: Chaco Canyon geographic setting and geologic cross-section. Satellite basemap showing the Chaco Culture boundary, major local geographic features, and the A–A' geologic cross-section transect, with inset location map for northwestern New Mexico. The lower panel shows the corresponding topographic-geologic cross-section across Chaco Canyon and adjacent mesas, including the Menefee Formation, Cliff House Sandstone, Lewis Shale, alluvial and eolian deposits, and related units relevant to potential geogenic radon source pathways.

mechanisms may have contributed to the cumulative burden on an already stressed Chacoan population. One such potential mechanism emerges from the geology and stratigraphic setting of Chaco Canyon (**Figure 2**; a detailed stratigraphic map is provided in Appendix A as **Figure A1**).

Chaco Canyon's great houses, including Pueblo Bonito, Chetro Ketl, Pueblo del Arroyo, Pueblo Alto, Casa Rinconada, and others, developed after earlier Basketmaker III-period settlements characterized by dispersed single-room pit houses (Bustard, 1999). These Chacoan structures are situated on the alluvial floor of Chaco Canyon beneath cliffs of Late Cretaceous Cliff House Sandstone (Crown, 2018), a shallow-marine sandstone deposited along the margin of the Western Interior Seaway (Bauer, 1916; Fassett, 1991; Hart et al., 2001). The underlying and nearby Menefee Formation includes siltstone, mudstone, sandstone, carbonaceous shale, and thin coal beds deposited in coastal-plain and swamp-like environments (Bristler and Price, 2002; Hall, 1977; Hart et al., 2001). This formation is known for uranium enrichment (McLemore, 2007, 1983; McLemore and Hawley, 1988; Ridgley et al., 1978). Selected coal beds have been reported to contain localized uranium concentrations up to 1.34 wt% (Cannon and Starrett, 1956; McLemore et al., 2013; McLemore and Hawley, 1988; Chenoweth, 1989; Vine et al., 1953). Carbonaceous shale and coal can also provide reducing environments favorable to uranium concentration at redox fronts (Chenoweth, 1989; Finch et al., 1989; McLemore et al., 2013; McLemore and Hawley, 1988).

The uranium decay series produces radon-222, an unstable radioactive gas with a half-life of 3.82 days. ^{222}Rn is the immediate alpha-decay daughter of radium-226, ^{226}Ra , in the uranium-238, ^{238}U , series. Because it is a noble gas, radon can migrate through pore spaces, fractures, and soil-gas pathways and can enter enclosed structures. Importantly, ppm-level uranium in porous, organic-rich, fractured shales and lignite beds can deliver substantial radon to overlying air, even when the host material is not ore grade. Radon availability depends not only on bulk uranium concentration but also on emanation efficiency, porosity, permeability, moisture, and ventilation (Silber et al., 2026; Nazaroff, 1992; Otton, 1992; Schumann and Gundersen, 1996; Sakoda et al., 2011).

When concentrated indoors, ^{222}Rn and its short-lived alpha-emitting decay products constitute a serious

health hazard. Radon is classified as a Group 1 human carcinogen (El Ghissassi et al., 2009; IARC, 2012) and is widely recognized as the second leading cause of lung cancer after smoking (Riudavets et al., 2022; Sheen et al., 2016; WHO, 2009). The primary health risk arises from short-lived radon progeny (^{218}Po , ^{214}Pb , ^{214}Bi , ^{214}Po). These progeny attach to indoor aerosols, deposit on the bronchial epithelium, and deliver localized alpha-particle doses capable of inducing DNA damage and somatic mutations that can initiate carcinoma (ICRP, 1994; National Research Council, 1999; Robertson et al., 2013).

Historically, this problem could have been especially relevant for pit-type Basketmaker-period and transitional earth-contact structures, such as those used in Ancestral Puebloan architecture during the Bonito phase. Chacoan architecture combined monumental above-ground masonry room blocks with a persistent and culturally central spectrum of semi-subterranean spaces, including kivas, pit structures, partially earth-contact storage rooms, and lower-tier enclosed rooms. Numerous mortar and stone residential structures in Chaco Canyon, beyond monumental great-house complexes, were also built with direct earth contact. The design of these spaces could mechanically couple soil-gas radon to occupied interior air. Internal hearths, especially during cold periods, could further enhance this coupling by producing pressure differentials that drive advective soil-gas entry. Dusty indoor environments would not necessarily increase radon gas concentration but could increase the inhalable radon-progeny burden by promoting progeny attachment to aerosols and increasing the equilibrium factor relative to cleaner modern residential air.

A common feature of these structures is low air exchange combined with direct or partial contact between interior air and the underlying soil or rock column. Comparable earth-contact and semi-subterranean architectures occur widely throughout the world, and studies document elevated indoor ^{222}Rn concentrations in such settings (Piedecausa and Chinchón, 2015; Köksal et al., 2004).

In principle, indoor radon commonly enters living spaces by pressure-driven advective flow through cracks, joints, floor pathways, and porous openings. Advection can dominate diffusion by one to two orders of magnitude (Nazaroff and Nero, 1988; Schumann and Owen, 1988; Nazaroff, 1992). At steady state, indoor concentration is controlled by the balance between entry rate and air-exchange rate. In a premodern Chaco Canyon-type room with a central hearth and limited ventilation, stack-effect depressurization at floor level could drive soil-gas ingress through any open pathway. Modern building studies demonstrate the same physical principle in contemporary settings, where increased airtightness and energy-efficiency measures can elevate indoor radon by reducing ventilation and increasing indoor accumulation. For example, double glazing, loft insulation, and wall insulation have been associated with elevated indoor radon in modern housing contexts (Symonds et al., 2019), and newer energy-efficient apartment buildings can show systematically higher indoor radon than conventional construction (Bossew et al., 2020).

A further issue for Chacoan residential and ceremonial structures is aerosol loading. Radon progeny attach to aerosols, and the equilibrium factor in smoky, dusty, low-ventilation interiors may differ substantially from modern residential assumptions. This process is well established in radon-progeny dosimetry and aerosol studies (Hopke et al., 1990; Porstendörfer, 1994; Porstendörfer, 2001; Vargas et al., 2000). In Chaco, hearth smoke, mineral dust, earthen floors, and ground-level sleeping could plausibly have increased effective radon-progeny exposure.

Despite these converging considerations, the chronic indoor radon burden of ancient populations inhabiting earth-contact architecture has received little systematic quantitative attention. Radiation epidemiology, geology, paleo-oncology, and archaeology have largely proceeded in parallel. The single direct ^{222}Rn measurement reported in an Ancestral Puebloan analog pit structure, dated to approximately AD 1120–1170, was 19.4–20.3 pCi/L, approximately five times the U.S. Environmental Protection Agency action level and approximately seven times the World Health Organization (WHO) reference level (Whitley and Boyer, 2018). However, that measurement has not been extended into a quantitative lifetime-dose model, integrated with the multi-stressor archaeology of Ancestral Puebloan decline, or developed into a broader research framework.

This paper posits that chronic indoor ^{222}Rn exposure in some semi-subterranean and earth-contact Chaco Canyon architectural settings may have constituted a long-duration health stressor that compounded the climatic, subsistence, environmental, and sociopolitical stressors already documented in the multicausal

approach described above. Specifically, we use quantitative and numerical analysis to test the hypothesis, not formally evaluated in the current literature, that chronic and multigenerational indoor ^{222}Rn exposure may have contributed to cumulative health and demographic vulnerability in Chaco Canyon. We do not propose radon as the sole or principal cause of Bonito-phase decline and depopulation, nor do we assume that every room in Pueblo Bonito or every Chacoan great house had excessive radon concentrations. Rather, we develop a defensible scientific and quantitative framework to evaluate the contribution and magnitude of a previously underexamined chronic-exposure pathway, not a causal explanation of an archaeological transition. Toward that end, we use numerical analysis to evaluate whether indoor ^{222}Rn and its short-lived alpha-emitting progeny could have constituted a chronic health stressor contributing to cumulative demographic vulnerability before and during the megadrought.

The analysis focuses on the population in residential settlements of Chaco Canyon, including Pueblo Bonito and other great houses, together with nearby dense settlement clusters over 10–12 square kilometers (Lekson, 1991; Lekson and Cameron, 1995). Chaco Canyon is emphasized because it provides the most tractable case: a well-studied population center with monumental architecture, semi-subterranean spaces, a constrained demographic chronology, and a relevant geologic setting. Extension of this analysis to the broader Chaco Regional System, including all three periods delineated by Kohler et al. (2025), may further clarify the regional relevance of this exposure pathway.

Therefore, the main purpose of this work is to quantitatively evaluate the potential magnitude and demographic timing of this exposure pathway within a multicausal interpretation of Chacoan transformation. We test whether geogenic radon could have constituted a plausible, previously underexamined chronic health and demographic stressor in a population already experiencing climatic, subsistence, and sociopolitical pressures.

2 Methods: Model Overview and Rationale

2.1 Initial considerations

In this expository study, we numerically model chronic indoor ^{222}Rn exposure in Chaco Canyon earth-contact and semi-subterranean microenvironments, including kivas, pit structures, lower-tier rooms, and broader great-house settings. The primary goal is to evaluate whether plausible radon exposure could have constituted a testable chronic health and demographic stressor. Low ventilation, hearth heating, cold high-desert nights, dust, floor-level sleeping, and earth-contact construction all increase the plausibility of elevated indoor radon and progeny exposure. The modeled population also had very low life expectancy; life expectancy at birth (e_0) was 18–25 years (Kohler and Reese, 2014), closely following the Coale-Demeny West level 3 model. Under such conditions, only a small proportion of the population survived into the later adult ages at which lung cancer would be expected to manifest, so modeled radon-associated mortality is concentrated in older adults.

We reconstructed radon-attributable lung-cancer burden for the Chaco Canyon core occupation during AD 850–1150 using a coupled physical, epidemiological, and demographic model derived from established methodologies. The epidemiological component applies miner-derived excess-relative-risk functions within the lifetime-risk approach described by Sommer et al. (2025), with model selection and sensitivity bounds described below. The calculation links radon-progeny exposure, premodern survival, and reconstructed Chaco demography to age-specific and calendar-year mortality while retaining separate physical and epidemiological components.

The calculation chain accounts for indoor radon-gas concentration (C_i), working level (WL), age-specific cumulative exposure in working-level months ($\text{WLM}(a)$), age-specific excess relative risk of lung-cancer death ($\text{ERR}(a)$), the radon-attributable lung-cancer death density at age a ($e(a)$) and the expected number of radon-attributable lung-cancer deaths in calendar year y ($D(y)$). This chain follows the general mass-balance approach used for indoor radon modeling and the lifetime-risk structure used in recent radon risk syntheses (Park et al., 2016; Sommer et al., 2025).

The calculation is defined by an explicit sequence of physical, exposure, epidemiological, survival, and demographic equations, with numerical assumptions and uncertainty terms specified below and in Appendix A. The lifetime-risk engine was benchmarked against the published [Sommer et al. \(2025\)](#) reference calculations. Numerical inputs are either sourced from the literature or identified as stated assumptions and propagated as appropriate.

2.2 Model selection and sensitivity bounds for Chaco

The epidemiological analysis emphasizes Biological Effects of Ionizing Radiation (BEIR) VI, the Pooled Uranium Miners Analysis (PUMA) full cohort, and the Joint Czech–French model as the principal miner-derived models. PUMA 1960+ is retained as an exploratory upper-bound sensitivity model because the post-1960 subcohort provides a lower-rate, better-characterized occupational comparison, but its published formulation does not include a separate exposure window beyond 34 years ([Richardson et al., 2022](#)). We also benchmark calculations against Wismut full, Wismut 1960+, and adjusted Jacobi methodologies. BEIR VI is retained because it is the canonical regulatory benchmark ([National Research Council, 1999](#)). PUMA full is retained because it represents the largest pooled miner data source ([Kelly-Reif et al., 2023](#); [Kreuzer et al., 2024](#)). The Joint Czech–French model provides an independent European low-exposure-rate comparison ([Tomasek et al., 2008](#)). Wismut full and Wismut 1960+ provide additional lower and midrange miner-derived bounds. These models are used collectively to establish epidemiological sensitivity bounds.

The use of a broad risk-model ensemble is particularly important because Ancestral Puebloan settings differ from both contemporary dwellings and adult miner cohorts. Principal differences include exposure that could begin in infancy for Chaco Canyon inhabitants ([Pinotti et al., 2025](#)), sleeping near the floor, indoor combustion and dust that could alter and enhance radon-progeny effects, and mortality evaluated under premodern survival rather than modern life expectancy ([Coale et al., 2013](#); [Kohler and Reese, 2014](#); [Lekson, 1984](#); [Nazaroff, 1992](#); [Porstendörfer, 2001](#)). The analytical methodology was designed to estimate demographic burden, not to diagnose individual archaeological cases. This distinction is important because radon-induced lung cancer does not leave skeletal markers or isotopic signatures uniquely tied to radon; after approximately 150 years, ^{210}Pb , the longest-lived relevant isotope, would not be reliably detectable ([Swarzenski, 2014](#)). The model is therefore used to evaluate implications for the wider sociodemographic structure.

2.3 Population trajectory and annual births

We reconstruct the Chaco Canyon residential population, with uncertainty, for AD 850–1150 from the existing archaeological literature (**Figure 3**). Published population reconstructions differ substantially according to spatial definition, archaeological proxy, room-occupancy assumptions, and estimates of local agricultural support. We therefore represent demographic uncertainty using low, reference, and upper-bound trajectories spanning the range of published estimates ([Benson et al., 2006](#); [Benson, 2010](#); [Bernardini, 1999](#); [Crown, 2018](#); [Harrod, 2017](#); [Nelson, 1995](#); [Plog and Heitman, 2010](#); [Plog and Watson, 2012](#); [Van Dyke et al., 2016](#); [Wills, 2001, 2009](#); [Wills and Dorshow, 2012](#)).

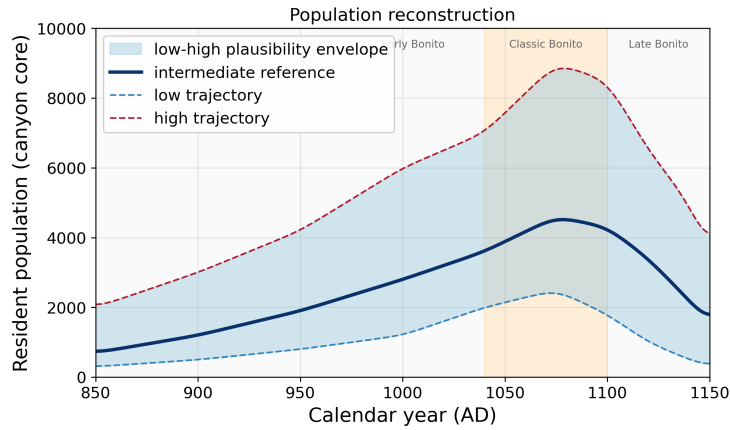


Figure 3: Reconstructed Chaco Canyon population. Model population trajectories and uncertainty envelope based on literature-derived Chaco Canyon residential estimates.

To simplify the approach, we retain the residential population model (Kohler et al., 2025) and use mean population estimates from the literature while carrying uncertainty through the calculations (Benson et al., 2006; Benson, 2010; Bernardini, 1999; Crown, 2018; Harrod, 2017; Plog and Heitman, 2010; Plog and Watson, 2012; Van Dyke et al., 2016; Wills, 2001, 2009; Wills and Dorshow, 2012).

Peak canyon-resident population estimates vary widely, from a few hundred under strict local-productivity interpretations, through commonly used estimates of ~2,100–2,700 and estimates near 5,900, to older archaeological reconstructions approaching 10,000 residents at the height of occupation (Altschul, 1978; Benson et al., 2019; Kohler et al., 2025; Lekson et al., 1988; Vivian, 1974). The three demographic trajectories used here therefore span this exceptionally broad literature range: the low and reference trajectories peak at approximately 2,500 and 4,600 residents, respectively, while the 9,000-resident high trajectory is retained as an upper-bound sensitivity scenario consistent with the upper range of historical archaeological estimates rather than as a preferred reconstruction.

Keeping the residential Chaco Canyon population model (Kohler et al., 2025; Pinotti et al., 2025) is justified because archaeogenomic and isotopic analyses document a multigenerational, locally rooted population, including an elite matrilineal lineage persisting at Pueblo Bonito from ~AD 800 to 1130 (Kennett et al., 2017; Price et al., 2017). The recent Picuris-Pueblo-led genomic study also confirms genetic continuity between descendant Pueblo communities and the Chacoan population (Pinotti et al., 2025). This evidence provides a strong demographic basis for considering chronic and multigenerational residential exposure. It does not demonstrate invariant occupancy of a single room or microenvironment, but neither does it imply frequent movement into systematically lower-exposure settings; movement within Chaco Canyon would generally remain within the same regional geologic setting, while indoor exposure magnitude would vary with local architectural and transport conditions (McLemore and Hawley, 1988; Ridgley et al., 1978). This is an important consideration because long-term, locally rooted residence provides the demographic setting in which sustained lifetime exposure is plausible for at least part of the population.

The model uses the reconstructed decadal Chaco Canyon population trajectory for AD 850–1150, interpolated to annual resolution. Let $P(y)$ denote the resident population in calendar year y . Annual births are calculated as:

$$B(y) = P(y)b. \quad (1)$$

Here, $B(y)$ is the number of births in year y , and b is the crude birth rate ($b = 0.049 \text{ yr}^{-1}$; Kohler and Reese, 2014). For example, at the modeled population peak in AD 1075, this translates into 225 annual births. Each annual birth cohort is followed through age-specific exposure, survival, and lung-cancer risk. This cohort structure is essential because radon-induced lung cancer is delayed by decades, and deaths in a given year are produced by births many years earlier.

Each annual birth cohort was propagated through age-specific exposure, premodern survival, and lung-cancer risk. Calendar-year deaths therefore arise from births many years earlier, not simply from the contemporaneous standing population (National Research Council, 1999; Sommer et al., 2025).

2.4 Indoor radon mass balance

No published indoor radon measurements exist for Chaco Canyon. Available regional background uranium values are typically in the range of 3–4 ppm, while uranium-bearing units of the Menefee Formation and the broader San Juan Basin contain localized enrichments that can exceed regional background by orders of magnitude (Finch et al., 1989; McLemore and Hawley, 1988; McLemore and Chenoweth, 1989; Ridgley et al., 1978). Selected carbonaceous and coal-bearing materials can contain much higher uranium concentrations, illustrating the strong spatial heterogeneity of the geogenic source term. Even assuming a low uranium concentration of 3 ppm U, variation over literature-supported radon emanation and air-filled porosity produces calculated soil-gas radon concentrations of 265–1860 pCi/L. Under the reference ground-coupled, low-ventilation configuration with $\Delta P = 3.6$ Pa, the forward mass-balance calculation yields an indoor radon concentration of ~ 23 pCi/L.

The most directly relevant archaeological-period analog for the prescribed Chaco exposure scenarios is the Ancestral Puebloan pit-structure measurement reported by Whitley and Boyer (2018), where indoor ^{222}Rn concentrations of ~ 19.4 – 20.3 pCi/L were measured. We therefore use 20 pCi/L as an analog-supported prescribed exposure scenario and 30 pCi/L as a higher exposure scenario for selected earth-contact Chacoan settings. Neither value is treated as a direct Chaco Canyon measurement or a canyon-wide average. The 30 pCi/L scenario reflects the greater geogenic potential possible where earth-contact structures are coupled to uranium-bearing carbonaceous strata and local soils, including documented uranium concentrations above regional background (Finch et al., 1989; McLemore, 1983; McLemore and Hawley, 1988). Importantly, these prescribed concentrations are not used to calibrate the physical model; the independent forward calculation described above places them within the physically attainable range under the evaluated Chaco source and transport conditions. Indoor radon is subsequently represented using a single-zone, well-mixed mass balance incorporating soil diffusion, pressure-driven soil-gas entry, building-material diffusion, outdoor-air exchange, ventilation removal, and radioactive decay (Park et al., 2016):

$$\begin{aligned} \frac{dC_i}{dt} = & \frac{S_g}{V} k_{d,s} (C_s - C_i) + \frac{S_g}{V} k_a \Delta P C_s \\ & + \frac{S_{bm}}{V} k_{d,bm} (C_{bm} - C_i) - \lambda_v (C_i - C_o) - \lambda C_i. \end{aligned} \quad (2)$$

In Eq. (2), C_i is indoor radon concentration, C_s is soil-gas radon concentration, C_{bm} is the effective radon source concentration in building material, and C_o is outdoor radon concentration. S_g is the ground-contact area, S_{bm} is the surface area of radon-bearing building material, and V is indoor air volume. $k_{d,s}$ and $k_{d,bm}$ are diffusion transfer coefficients for soil and building material, respectively. k_a is a pressure-normalized advective transfer coefficient, so that $k_a \Delta P$ has dimensions of velocity. ΔP is the soil–indoor pressure difference, λ_v is the ventilation rate, and λ is the ^{222}Rn decay constant.

The first term in Eq. (2) represents diffusive radon entry from soil, driven by $C_s - C_i$. The second term represents pressure-driven soil-gas inflow, which is central for hearth-heated, earth-contact structures. In this reduced formulation, $k_a \Delta P$ is treated as a source-entry coefficient rather than as an additional whole-room air-exchange term; bulk dilution is represented by λ_v . The third term represents radon entry from sandstone, adobe, mortar, or rubble fill. The fourth term represents ventilation exchange with outdoor air, and the final term represents radioactive decay. This reduced model follows the mass-balance framework of Park et al. (2016), who describe indoor radon concentration as governed by radon entry, air exchange, and decay, with soil, building material, water, outdoor air, and occupant behavior all potentially contributing to indoor exposure. Park et al. (2016) also note that model terms can be removed or added according to building-specific conditions, and that steady-state or numerical solutions may be used depending on

available data.

Here, we ignore the term for time-dependent changes in indoor radon concentration caused by radon in a building's water supply. Chaco-specific water-radon transfer parameters are unavailable, and the present model focuses on the indoor-air pathway from soil and building materials. Waterborne radon and dissolved uranium-series radionuclides remain relevant for future environmental sampling but are not required for the main air-inhalation model.

For simplicity, we also do not include the term describing radon exchange between compartments i and j , where $i \neq j$, because the model assumes that most Chaco Canyon residents, including those in great houses, occupied single-room dwellings or functionally single-zone indoor spaces for the purposes of this exposure calculation (Kohler et al., 2025).

Hearth heating in enclosed or semi-subterranean spaces can produce a stack-effect pressure differential that enhances advective soil-gas entry. The pressure difference can be expressed as:

$$\Delta P = \rho_o g H \left(\frac{T_{in} - T_{out}}{T_{in}} \right). \quad (3)$$

Here, ρ_o is outdoor air density, g is gravitational acceleration, H is the height of the warm indoor air column, T_{in} is indoor absolute temperature, and T_{out} is outdoor absolute temperature.

Hearth heating in enclosed or semi-subterranean spaces can produce a stack-effect pressure differential that enhances advective soil-gas entry. Using Eq. (3) with the reference Chaco room geometry and indoor-outdoor temperature contrast gives a reference pressure differential of $\Delta P = 3.6$ Pa. Across the range of heating and temperature conditions evaluated here, calculated pressure differences are 1.9–5.5 Pa. Based on recent radon-entry and ventilation studies (Gaskin et al., 2025; Matthews et al., 1987; Nazaroff, 1992; Turk, 1987), Pa-scale pressure differences of this magnitude are physically relevant for radon entry, while values approaching 10 Pa may occur under limiting conditions. Although the calculated pressure difference may be small in everyday terms, it is physically significant for radon because pressure differences of only a few pascals can enhance soil-gas entry through cracks, earthen floors, wall-floor joints, porous fill, and ventilator features (Nazaroff, 1992; Riley et al., 1994). A $\Delta P = 3$ –10 Pa case was retained as a stress-test scenario rather than as a central Chaco measurement.

For exposure intervals during which room use, ventilation, and source terms are approximately stable, we solve Eq. (2). Assuming steady state, $dC_i/dt = 0$ we obtain:

$$C_i = \frac{\frac{S_g}{V}(k_{d,s} + k_a \Delta P)C_s + \frac{S_{bm}}{V}k_{d,bm}C_{bm} + \lambda_v C_o}{\lambda_v + \lambda + \frac{S_g}{V}k_{d,s} + \frac{S_{bm}}{V}k_{d,bm}}. \quad (4)$$

The numerator is the total effective radon source term: diffusive soil entry, pressure-driven advective soil-gas entry, building-material contribution, and outdoor-air exchange. The denominator is the total removal and dilution term: ventilation, radioactive decay, and diffusive exchange terms.

Two complementary calculation pathways are used below. In the prescribed-concentration analysis, indoor radon concentration is fixed at $C_0 = 20$ or 30 pCi/L to evaluate conditional WLM and epidemiological outcomes. These scenario concentrations are not calculated from Eq. (4), and once C_0 is prescribed, source-side quantities such as C_s , λ_v , and ΔP do not independently enter the exposure-to-risk calculation. In the forward uncertainty analysis, by contrast, indoor concentration is generated directly by the mass-balance model. For Monte Carlo realization j , $C_0^{(j)} \equiv C_i^{(j)} = f(C_s^{(j)}, \lambda_v^{(j)}, \Delta P^{(j)}, \dots)$, where f denotes the steady-state solution in Eq. (4). The resulting $C_i^{(j)}$ is then used in the radon-progeny exposure and epidemiological calculations. Indoor concentration and its physical determinants are therefore not independently sampled within the same forward calculation.

The steady-state solution in Eq. (4) is used to evaluate the physical plausibility of the prescribed 20 and 30 pCi/L scenarios rather than to calibrate them. For a Menefee-derived substrate containing 3 ppm U, variation in radon emanation and air-filled porosity over the evaluated literature ranges produces

soil-gas radon concentrations of 265–1860 pCi/L. Using the reference $\Delta P = 3.6$ Pa ground-coupled, low-ventilation configuration yields an indoor concentration of ~ 23 pCi/L. Substantially lower or higher concentrations remain possible as source strength, emanation, porosity, ventilation, pressure coupling, and geometry vary. Dusty or smoky conditions are treated separately through the radon-progeny equilibrium factor because aerosol loading affects working level and WLM rather than radon gas concentration itself. The reference exposure calculation uses $F = 0.70$ to represent dusty, smoke-rich, low-ventilation Chacoan interiors, while $F = 0.40$ is evaluated separately as a modern residential comparator.

The physical mass-balance calculation and the prescribed-concentration health calculations serve related but distinct purposes. The mass-balance model evaluates whether indoor concentrations in the 20–30 pCi/L range are physically attainable under the evaluated source and architectural conditions. The deterministic WLM and epidemiological calculations are then conditioned on fixed indoor concentrations of 20 or 30 pCi/L; once a concentration scenario is specified, ΔP , soil-gas concentration, and ventilation do not independently enter that deterministic exposure-to-risk calculation. These source parameters are varied separately in the forward uncertainty and sensitivity analyses described in Appendix A4.

The mathematical exposure-conversion, lifetime-risk, survival, birth-cohort convolution, high-equilibrium subgroup, and uncertainty-propagation framework is provided in **Appendix A**.

3 Results

The 20–30 pCi/L indoor concentration values used below are treated as analog-supported exposure scenarios. The 20 pCi/L scenario is anchored by the 19.4–20.3 pCi/L measurement reported from an Ancestral Puebloan pit-structure analog (Whitley and Boyer, 2018), while both scenarios are evaluated independently against the indoor-radon mass-balance model described above.

The indoor-radon mass-balance model was evaluated using the source and transport parameters described above. For a substrate containing 3 ppm U, variation in radon emanation and air-filled porosity produces soil-gas radon concentrations of approximately 265–1860 pCi/L. The regional value of 890 pCi/L reported by Brookins (1991) lies within this physically derived range. Using the reference stack-effect pressure differential $\Delta P = 3.6$ Pa, the mass-balance model yields an indoor radon concentration of ~ 23 pCi/L under the reference ground-coupled, low-ventilation configuration. However, in limiting scenarios with low ventilation and high heating, pressure differences may approach 10 Pa in winter, as shown in domestic and Canadian basement studies (Gaskin et al., 2025; Matthews et al., 1987; Nazaroff, 1992; Turk, 1987).

These calculations demonstrate that indoor concentrations in the 20–30 pCi/L range are physically plausible in selected earth-contact, low-ventilation Chacoan microenvironments, while substantially lower or higher concentrations remain possible as source strength, emanation, porosity, ventilation, pressure coupling, and architectural geometry vary. The 20 and 30 pCi/L values are therefore treated as defined exposure scenarios rather than as direct measurements or canyon-wide estimates for Chaco Canyon.

The subsequent exposure and epidemiological calculations are evaluated conditionally at prescribed indoor radon concentrations of 20 and 30 pCi/L. Once indoor concentration is specified, source-side parameters such as soil-gas concentration, ventilation rate, and ΔP do not independently alter the deterministic WLM or mortality calculations. Their influence on source-derived indoor concentration and burden is evaluated separately through the physical-model and global sensitivity analyses described below.

The relationship between the prescribed-concentration and forward source-to-burden calculation pathways is summarized schematically in **Figure A2**.

Unless otherwise stated, the exposure calculations use the reference high-occupancy Chaco scenario described above, while epidemiological outcomes are evaluated across the radon risk-model ensemble. BEIR VI (National Research Council, 1999), PUMA full (Kelly-Reif et al., 2023; Kreuzer et al., 2024), and the Joint Czech–French model (Tomasek et al., 2008) are treated as the principal miner-derived models. PUMA 1960+ (Richardson et al., 2022) is retained as an exploratory upper-bound sensitivity model because

Table 1. Indoor radon mass-balance parameters, values, and references applicable to the Chaco Canyon environment.

Parameter	Definition in Chaco Canyon scenario	Value used, with reference
S_g	Earthen floor area in direct contact with soil gas	20 m ² ; representative small kiva/earth-contact room geometry based on Chaco great-house/kiva architectural descriptions, not a measured single room value (Lekson, 1984 ; Windes, 2014)
V	Interior air volume	40 m ³ ; derived from 20 m ² floor area $\times$ 2 m room height; representative scenario constrained by Chaco room/kiva architecture (Lekson, 1984 ; Lekson et al., 1988 ; Windes, 2014)
S_g/V	Soil-contact area-to-volume ratio	0.50 m ⁻¹ ; derived from 20/40 using modeled geometry above
$k_{d,s}$	Effective diffusive transfer velocity from soil/floor into room air	0.0036 m h ⁻¹ ; derived from $D_{\text{eff}} \sim 10^{-6}$ m ² s ⁻¹ over ~ 1 m diffusion length; order-of-magnitude soil-radon diffusion framework from (Nazaroff, 1992)
ΔP	Indoor depressurization relative to soil gas	Reference $\Delta P = 3.6$ Pa; modeled range ~ 1.9 – 5.5 Pa across evaluated heating conditions; values approaching 10 Pa are retained only as a limiting sensitivity case (Gaskin et al., 2025 ; Matthews et al., 1987 ; Nazaroff, 1992 ; Riley et al., 1994 ; Turk, 1987)
C_s	Soil-gas radon concentration beneath / near structure	9,805–68,820 Bq m ⁻³ (265–1860 pCi/L) for the 3 ppm U source case across the evaluated emanation/porosity range; the 890 pCi/L regional value reported by Brookins (1991) lies within this range
k_a	Effective advective transfer coefficient per Pa	0.00152 m h ⁻¹ Pa ⁻¹ ; derived from lower-end radon entry rate of 10 kBq h ⁻¹ into a dwelling in (Nazaroff, 1992), using $k_a = 10,000/(S_g \Delta P C_s)$
$k_a \Delta P$	Effective advective entry velocity	0.00547 m h ⁻¹ ; derived from 0.00152×3.6 Pa
S_{bm}	Interior exposed building-material surface: sandstone, mud mortar, plaster, masonry	32 m ² ; representative wall area for a ~ 5 m diameter, 2 m high circular room; Chaco masonry/kiva architecture from (Lekson, 1984 ; Windes, 2014)
S_{bm}/V	Building-material surface-to-volume ratio	0.80 m ⁻¹ ; derived from 32/40
$k_{d,bm}$	Effective diffusive transfer velocity from masonry/plaster pore space	0.0030 m h ⁻¹ ; effective modeling value chosen so $k_{d,bm} C_{bm} = 1.5$ Bq m ⁻² h ⁻¹ , matching reported upper ordinary building-material exhalation rates (Wentzel, 2018)
C_{bm}	Effective radon concentration in masonry/plaster pore space	500 Bq m ⁻³ ; effective value paired with $k_{d,bm}$; not independently measured for Chaco masonry. Product constrained by building-material exhalation literature (Wentzel, 2018)
$k_{d,bm} C_{bm}$	Radon exhalation flux from building materials	1.5 Bq m ⁻² h ⁻¹ ; reported upper ordinary building-material exhalation value (Wentzel, 2018)
λ_v	Moderate air-exchange / ventilation rate	0.15 h ⁻¹ ; reference low-ventilation case. Low-ventilated room literature commonly uses values around 0.1–0.3 h ⁻¹ ; 0.05 h ⁻¹ is retained as an extreme low-end sensitivity case (Abdallah et al., 2012 ; Akbari, 2009)
λ	Radioactive decay constant of ²²² Rn	0.00755 h ⁻¹ ; derived from ²²² Rn half-life of 3.8235 days: $\lambda = \ln 2/T_{1/2}$ (Nazaroff, 1992)
C_o	Outdoor radon concentration	10 Bq m ⁻³ ; typical outdoor-air order-of-magnitude value used in environmental radon assessments (United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), 2000 ; WHO, 2009)

application to exposure histories extending beyond 34 years requires continuation of the oldest available time-since-exposure coefficient. The physical indoor-radon and WLM calculations are independent of epidemiological risk-model choice. The risk engine follows the lifetime-risk architecture used by [Sommer et al. \(2025\)](#) and was benchmarked against the occupational reference calculations reported in that work. Six of seven miner-model reference values were reproduced to within approximately 2% (four within approximately 1%); PUMA full differed by approximately 6%, consistent with rounding of the published reference value. This benchmarking verifies the numerical implementation of the risk equations but does not validate the Chaco exposure assumptions.

Across the evaluated source and transport combinations, the mass-balance sensitivity calculations span substantially lower and higher indoor concentrations than the prescribed 20–30 pCi/L scenarios. These model bounds are not treated as a probability distribution or as representative canyon-wide exposure.

Indoor radon concentration was also evaluated under the same steady-state solutions for soil/rock uranium concentrations of 3, 6, and 9 ppm ([Finch et al., 1989](#); [McLemore and Chenoweth, 2003](#); [McLemore et al., 2013, 2016](#)). The calculations assume secular equilibrium with ^{226}Ra , a reference radon emanation fraction of 0.30 for the controlled uranium-content sensitivity calculation, soil grain density of 2650 kg m^{-3} , and air-filled porosity values of 0.30, 0.40, and 0.50. The secular-equilibrium conversion is a source-model assumption rather than a demonstrated property of all Meneffe materials; where U–Ra disequilibrium occurs, measured ^{226}Ra activity would provide the more direct radon-source constraint. The remaining geometric and transport parameters are listed in Table 1. **Figure 4** shows the resulting dependence of indoor radon on uranium content and ventilation, while **Figure 5** shows the corresponding increase in cumulative effective dose with increasing uranium content. These monotonic responses are sensitivity and model-consistency results, not independent validation of the assumed Chaco exposure scenarios.

The reference Chaco configuration uses $F = 0.70$ for radon-progeny equilibrium, $D_{\text{dust}} = 1.00$, $g_{\text{fl}} = 1.30$ for sleeping near the floor, $f_{\text{LC}} = 0.20$ for the scaled baseline lung-cancer hazard, sustained exposure from birth, Coale-Demeny West level 3 survival (**Figure 6**; [Coale et al., 2013](#); [Kohler and Reese, 2014](#)), and the reference Chaco Canyon population trajectory peaking at 4,600 residents in AD 1075. Life expectancy at birth, dominated by high childhood mortality, was approximately 22 years; 57.4% of the population did not reach age 15 and 26.9% did not reach age one ([Palkovich, 1980](#)). Only 25–30% reached age 45 and 4–10% reached age 65, while survival to age 50 in a Puebloan pithouse sample was approximately 6% ([Kohler and Reese, 2014](#)). Using the crude birth-rate coefficient $b = 0.049 \text{ yr}^{-1}$ ([Kohler and Reese, 2014](#)), integrated births over AD 850–1150 are 38,115 under the reference trajectory, bracketed by 16,969 and 79,821 under the low and upper-bound demographic reconstructions, respectively (**Table 2**).

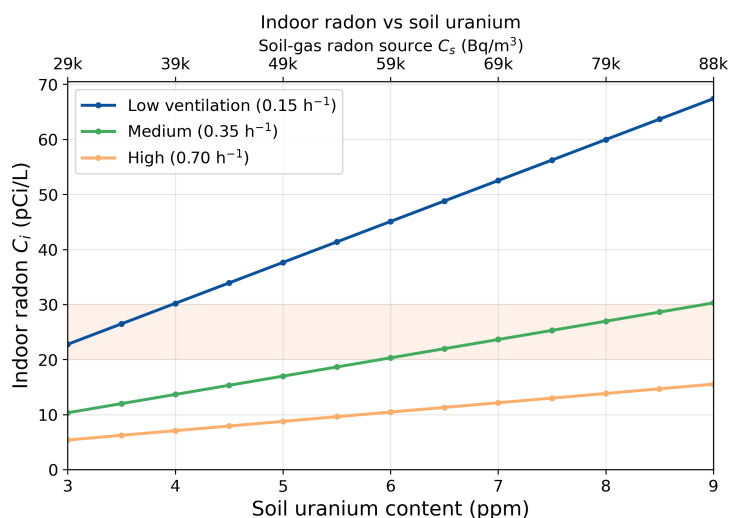
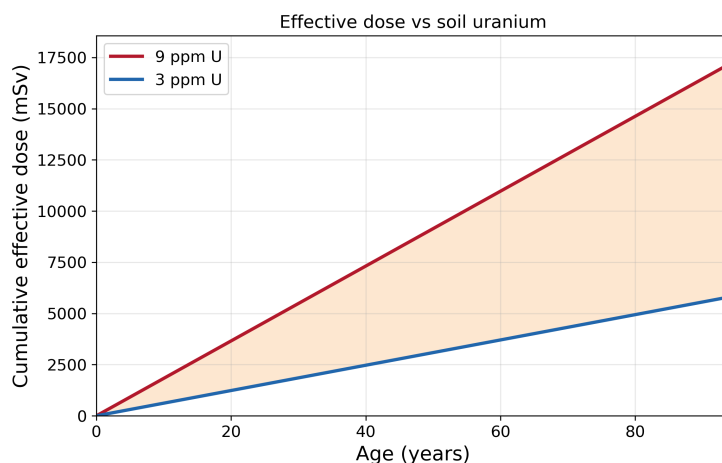
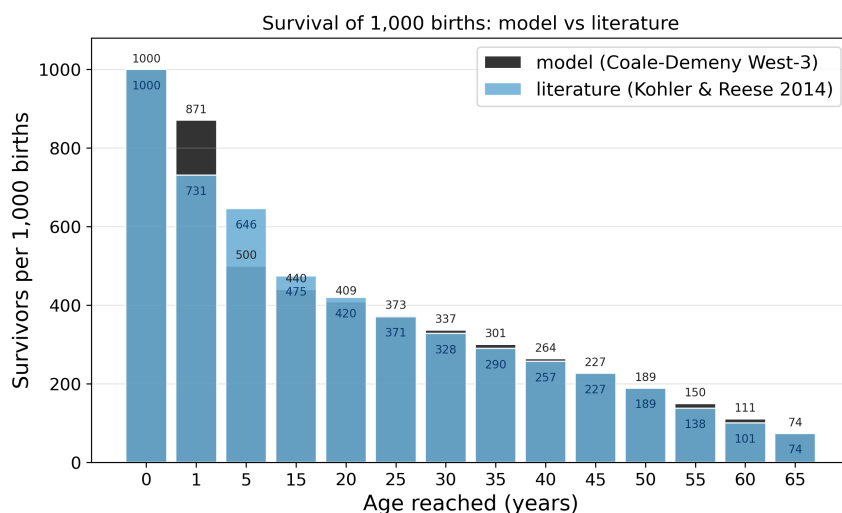


Figure 4: Indoor radon concentration by soil/rock uranium content. Modeled indoor radon concentration is shown as a function of uranium content under different ventilation conditions, with porosity fixed at 0.45.

Table 2. Reconstructed population anchors and integrated births.

Quantity	Low trajectory	Reference trajectory	Upper-bound trajectory
Residents, AD 850	300	700	2,000
Peak residents, AD 1075	2,500	4,600	9,000
Residents, AD 1150	300	1,500	3,500
Integrated births, AD 850–1150	16,969	38,115	79,821

**Figure 5:** Cumulative effective dose by age and uranium content. Cumulative effective dose is shown as a function of population age for the 3 and 9 ppm uranium scenarios.**Figure 6:** Premodern survival curve used in the Chaco model. Survival per 1,000 births in the Coale-Demeny West level 3 model is compared with reconstructed values from the literature (Kohler and Reese, 2014).

These parameter choices are reference scenario assumptions based on archaeological and demographic studies rather than direct measurements of a single Chaco population or room.

For the 20 pCi/L and 30 pCi/L scenarios, respectively, our Chaco-specific model yields 5.43 WLM yr⁻¹ and 8.14 WLM yr⁻¹. By age 50, these correspond to cumulative exposures of 271 and 407 WLM; by age 60, they correspond to 326 and 488 WLM. The influence of the equilibrium-factor assumption was evaluated separately using $F = 0.40$ as a modern residential comparator (**Figure 7**). At the Chaco-relevant reference value $F = 0.70$, annual exposures are 5.43 and 8.14 WLM yr⁻¹ for the 20 and 30 pCi/L scenarios, respectively, compared with 3.10 and 4.65 WLM yr⁻¹ at $F = 0.40$. By age 60, cumulative exposures decrease from 326 and 488 WLM at $F = 0.70$ to 186 and 279 WLM at $F = 0.40$. The comparison demonstrates the sensitivity of progeny exposure to F , while also showing that substantial

cumulative exposure is retained under the lower modern-residential assumption. The higher F case therefore represents the smoke- and dust-rich exposure environment considered in the Chaco reference configuration, while the $F = 0.40$ calculation provides a lower conventional comparator.

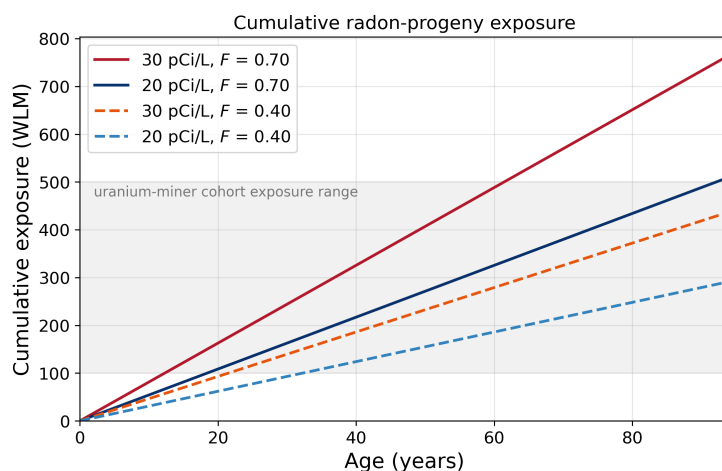


Figure 7: Cumulative radon-progeny exposure by age. Cumulative WLM is shown for the 20 and 30 pCi/L indoor radon scenarios using the Chaco-relevant reference equilibrium factor $F = 0.70$ and the modern residential comparator $F = 0.40$.

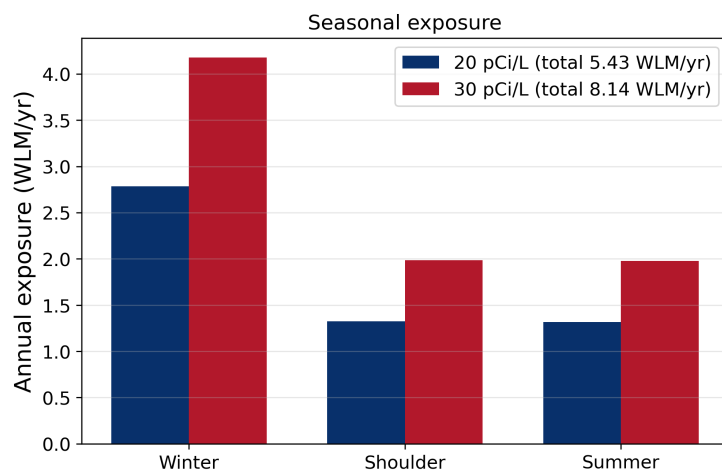


Figure 8: Seasonal variation in annual radon-progeny exposure. Seasonally adjusted annual working-level-month exposure is shown for the modeled Chaco exposure scenario.

Using the International Commission on Radiological Protection (ICRP) Publication 137 effective dose coefficient of $10\text{--}20 \text{ mSv WLM}^{-1}$ for inhaled radon progeny, the annual exposures of 5.43 and 8.14 WLM yr^{-1} correspond to annual effective doses on the order of $54\text{--}163 \text{ mSv yr}^{-1}$ across the modeled 20–30 pCi/L range (Harrison and Marsh, 2020; Paquet et al., 2017).

Seasonal variation in WLM is shown in **Figure 8**. Removing the seasonal modulation changes the 30 pCi/L annual exposure from 8.14 to 8.84 WLM yr^{-1} , an approximately 8% difference, indicating that the annual exposure magnitude is not strongly controlled by the adopted seasonal schedule.

The effective-dose comparison is provided as radiological context; mortality outputs are calculated from the epidemiological dose-response models rather than from effective dose directly.

Conditional lifetime lung-cancer mortality risk among individuals surviving into advanced adulthood is substantial across the three principal miner-derived models, BEIR VI, PUMA full, and Joint Czech–French (**Figure 9**). This conditional risk is evaluated separately from realized population burden because

Table 3. Annual and cumulative radon-progeny exposure and effective-dose equivalents. Annual exposure, cumulative exposure by ages 50 and 60, and effective-dose equivalents are shown for the 20 and 30 pCi/L indoor radon scenarios.

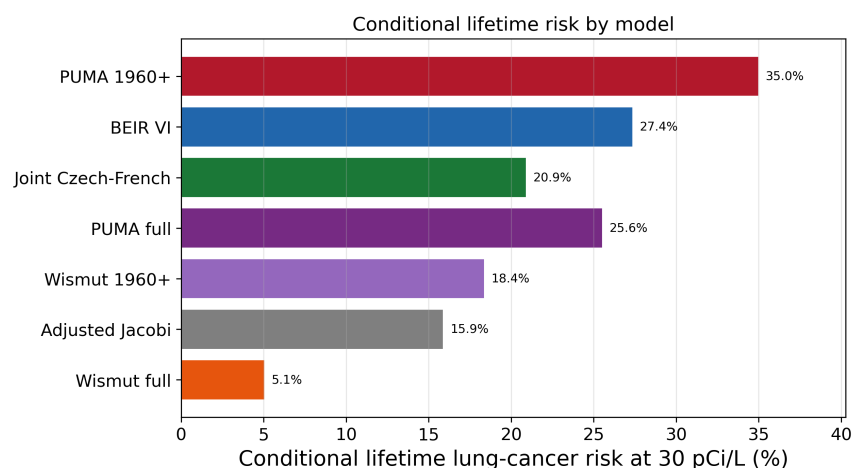
Indoor radon scenario [pCi/L]	Annual exposure [WLM/yr]	Cumulative exposure by age 50 [WLM]	Cumulative exposure by age 60 [WLM]	Effective dose at 10 mSv/WLM [mSv/yr]	Effective dose at 20 mSv/WLM [mSv/yr]
20	5.43	271	326	54.3	108.5
30	8.14	407	488	81.4	162.8

the premodern life table sharply limits survival into the ages at which radon-induced lung cancer is expressed.

The sustained from-birth exposure histories were propagated through the radon dose-response models and then evaluated under both modern-survival and premodern competing-risks assumptions. The distinction between these two quantities is important. Conditional lifetime risk estimates the lung-cancer mortality risk for an individual who survives into the cancer-prone age range. Realized per-birth risk incorporates the severe competing mortality of a premodern population, in which most individuals died before reaching older adulthood. **Figure 10** shows the relationship among exposure duration, conditional lifetime risk, and radon concentration. The exposure-duration analysis therefore explicitly evaluates shorter exposure histories in addition to the sustained from-birth case.

Across the full seven-model risk ensemble, conditional lifetime REID spans 5.1–35.0% at 30 pCi/L and is only marginally lower at 20 pCi/L. The exploratory PUMA 1960+ implementation gives the highest conditional lifetime risk, while the three principal miner-derived models occupy the main interpretive range (**Figure 9**). BEIR VI produces an intermediate-high conditional-risk estimate, and the Joint Czech–French model gives a lower conditional-risk estimate with earlier expression. In the separate realized-burden calculation summarized below, PUMA full defines the lower end among the principal miner-derived models.

When the age-specific excess-death density is convolved annually with the reconstructed birth series, the resulting realized-burden outputs are summarized by model and demographic scenario in **Figure 11** and **Table 5**. The low and high demographic trajectories scale the results approximately linearly. The realized population burden is smaller than the conditional lifetime risk because premodern survival truncates a long-latency disease process.

**Figure 9:** Conditional lifetime lung-cancer risk by dose-response model. Conditional lifetime risk of exposure-induced death (REID) is shown for the 30 pCi/L exposure scenario across the full seven-model risk ensemble. BEIR VI, PUMA full, and Joint Czech–French constitute the principal miner-derived models; PUMA 1960+ is retained as an exploratory upper-bound sensitivity model, while the Wismut and adjusted Jacobi formulations provide additional sensitivity comparisons.

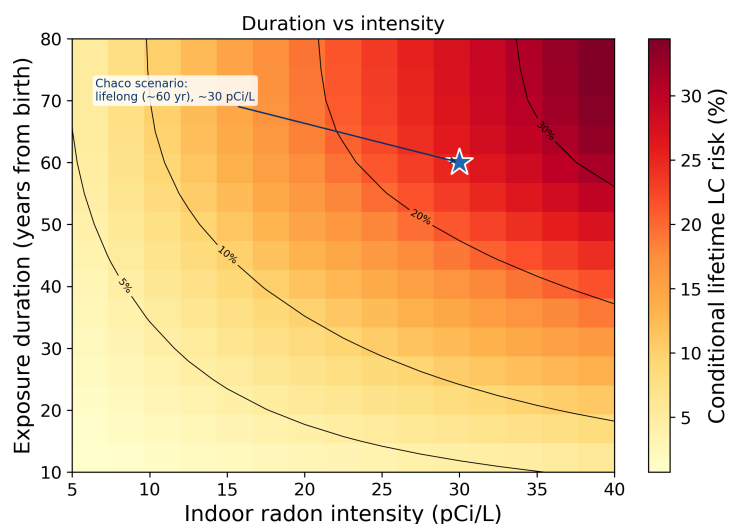


Figure 10: Exposure duration, radon concentration, and conditional lifetime risk. The relationship among exposure duration, conditional lifetime risk, and indoor radon concentration is shown for the Chaco Canyon population model.

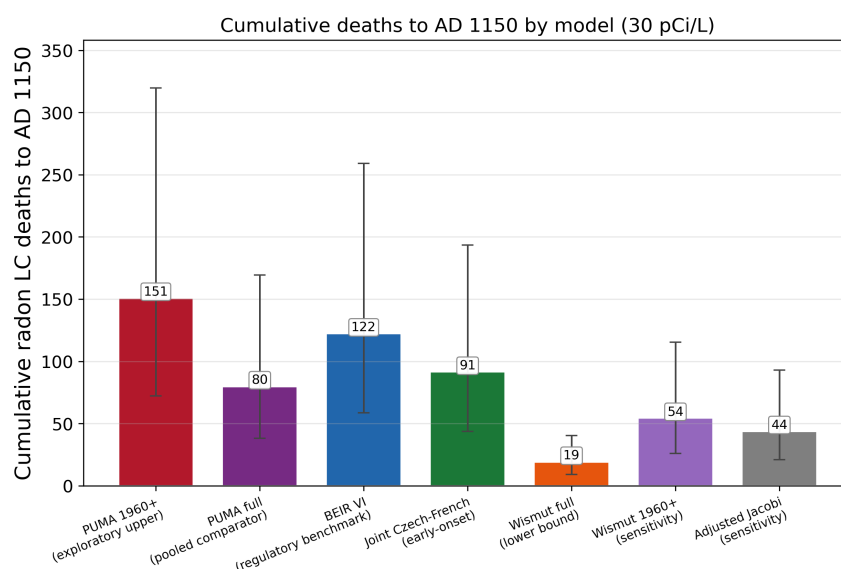


Figure 11: Cumulative radon lung-cancer burden during AD 850–1150. Cumulative radon lung-cancer burden occurring within the calendar window AD 850–1150 is shown for the 30 pCi/L scenario, with model and demographic uncertainty represented by the envelope.

For clarity, two analytically distinct burden measures are considered here. The first is the number of radon lung-cancer cases expressed within the modeled occupation interval. The second is the lifetime burden of cohorts born within that window, including cases expressed after the terminal date of the modeled occupation interval.

The exploratory PUMA 1960+ implementation produces the upper end of the model ensemble over the considered Chaco Canyon period, with the mean age of death at about 60 years (**Figure 12**). The lower tail of the age-at-death distribution indicates that the earliest onset of radon-induced cancer deaths occurs in the 40s. Annual radon-related mortality for Chaco Canyon is presented relative to the reconstructed population distribution in **Figure 13**.

The birth-cohort convolution produces a time-lagged mortality curve. The reference population reaches its peak in AD 1075, whereas radon-attributable lung-cancer deaths peak later, during the period shown in **Figure 13** and **Table 4**. This lag reflects the age distribution of the death density, which is concentrated in older adulthood. Across the model ensemble, mean age at radon-attributable lung-cancer death ranges

from 58.2 to 63.9 years (**Figure 12**).

The earliest onset is model-dependent. The Joint Czech–French model produces the youngest 5th-percentile onset age, approximately 40 years, and the lowest mean age, 58.2 years. Wismut full follows closely, with earliest onset around 41 years, while BEIR VI and the PUMA formulations place the earliest onset at ~45 years. These model contrasts show that the Joint Czech–French formulation contributes the strongest younger-adult tail, whereas the exploratory PUMA 1960+ implementation produces the largest total burden (**Figure 12**; Tables 4–5).

Increasing concentration, equilibrium factor, or occupancy increases the modeled burden but does not substantially shift the age of expression into childhood or early adulthood. For comparative purposes, **Figure 14** plots all-cause mortality and radon-related mortality across the Chaco Canyon Bonito period.

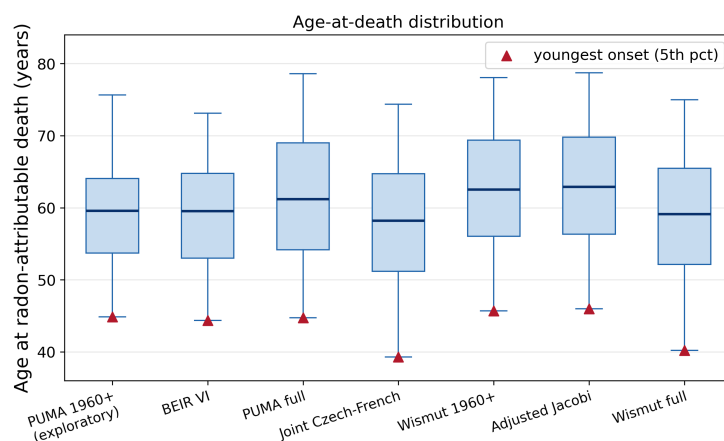


Figure 12: Age distribution of radon-induced lung-cancer mortality. Model-specific ages of radon-induced lung-cancer death are shown with uncertainty ranges. PUMA 1960+ is retained as an exploratory upper-bound sensitivity model.

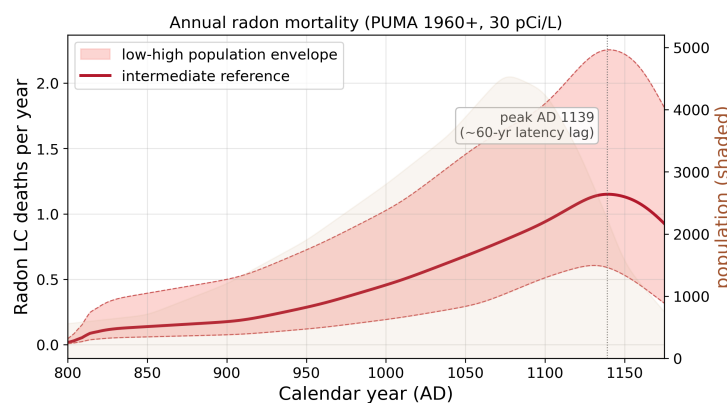


Figure 13: Annual radon-associated mortality relative to population history. Annual radon-induced mortality for Chaco Canyon is shown relative to the reconstructed population distribution. The annual series includes a short pre-AD 850 burn-in to avoid an artificial zero-boundary at the start of the published occupation window; the dotted line marks AD 850.

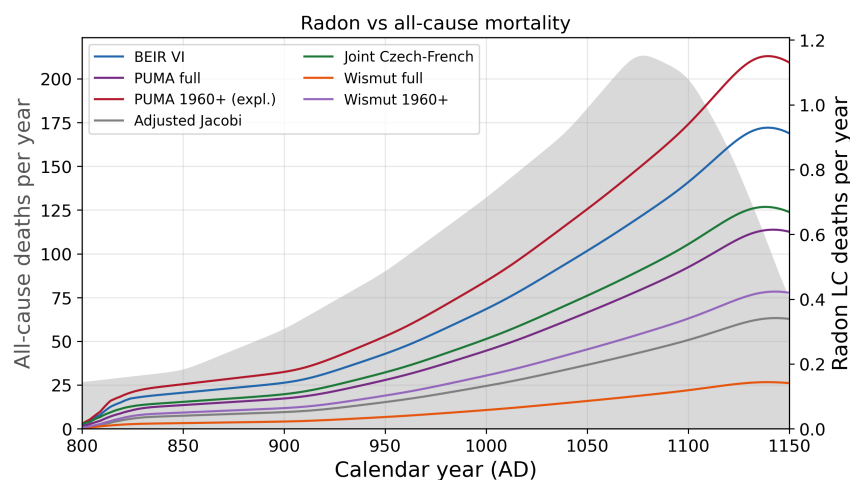


Figure 14: Radon-induced mortality compared with all-cause mortality. All-cause mortality and model-resolved radon-induced mortality are shown across the Chaco Canyon Bonito-period reconstruction. The short pre-AD 850 segment uses the same gentle pre-Bonito ramp as the annual mortality reconstruction to avoid an artificial zero-boundary at the start of the published occupation window; the dotted line marks AD 850.

The annual mortality series shows a pronounced latency-driven temporal displacement. Population size peaks near AD 1075, whereas modeled radon lung-cancer mortality peaks approximately six decades later, near AD 1138. This offset reflects the maturation of large eleventh-century cohorts into the cancer-prone ages as the standing population was declining.

For PUMA 1960+ at 30 pCi/L, annual modeled mortality remains low before AD 900 and peaks after the population maximum. The low–upper-bound demographic envelope follows the same timing, because the peak year is controlled mainly by cohort timing and latency rather than by the absolute level of the reconstructed population (**Table 4**).

The radon-associated share of mortality by age, conditional on survival into older adulthood, is shown in **Figure 15**.

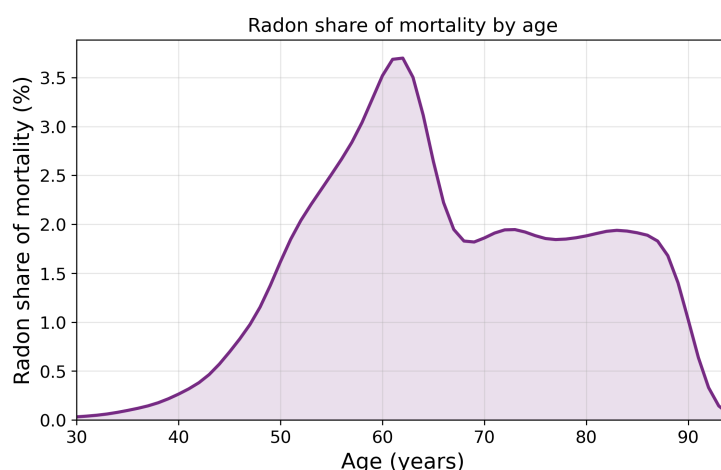


Figure 15: Radon-associated share of mortality by age. The age-specific share of mortality attributable to modeled radon-associated lung cancer is shown among individuals surviving into older adulthood.

Combining the low–upper-bound demographic envelope with the 20–30 pCi/L concentration interval produces the principal-model burden envelope summarized in **Figure 11** and **Table 5**. Among the principal miner-derived models, PUMA full defines the lower end, Joint Czech–French is intermediate, and BEIR VI defines the upper end. PUMA 1960+ is shown separately as an exploratory upper-bound sensitivity model.

Table 4. Annual radon-associated lung-cancer deaths at calendar-year milestones for the exploratory PUMA 1960+ case at 30 pCi/L. Population reconstruction uses the reference, low, and upper-bound trajectories, with the same pre-AD 850 burn-in used in the annual mortality reconstruction.

Year AD	Phase	Population, reference trajectory	Deaths yr ⁻¹ , reference trajectory	Low-upper-bound deaths yr ⁻¹	Rate per 1,000 yr ⁻¹
900	Early Bonito	1,200	0.17	0.07–0.50	0.15
950	Early Bonito	1,900	0.29	0.12–0.72	0.15
1000	Early Bonito	2,800	0.46	0.19–1.03	0.16
1050	Classic Bonito	3,886	0.68	0.29–1.46	0.17
1075	Classic Bonito, population peak	4,600	0.80	0.39–1.65	0.17
1100	Late Bonito	4,300	0.94	0.51–1.84	0.22
1130	Late Bonito	2,800	1.14	0.61–2.22	0.41
1138	Late Bonito, mortality peak	2,320	1.16	0.60–2.26	0.50
1150	Transition	1,500	1.13	0.53–2.23	0.76

Table 5. Total burden envelope by model, occurring deaths AD 850–1150.

Model	Reference-scenario burden at 30 pCi/L [deaths]	Full envelope, population × concentration
PUMA 1960+ (exploratory upper bound)	151	48–320
BEIR VI	122	39–259
Joint Czech–French	91	29–194
PUMA full	80	26–169
Principal miner-derived models ensemble	80–122	26–259

The absolute modeled burden is also sensitive to the assumed premodern baseline lung-cancer hazard. For the Joint Czech–French model at 30 pCi/L under the reference demographic trajectory, varying f_{LC} from 0.10 to 0.20 and 0.30 increases occurring deaths from 45.9 to 91.4 and 136.5, respectively; at 20 pCi/L, the corresponding values are 30.7, 61.1, and 91.4 deaths. This scaling is approximately linear, as expected from the multiplicative role of the baseline lung-cancer hazard. By comparison, conditional lifetime REID remains 20.9% across these f_{LC} values because f_{LC} scales the premodern absolute baseline hazard used in the realized-burden calculation rather than the modern-reference conditional-risk calculation.

The near linearity of burden with concentration provides an internal check. Across the principal miner-derived models, the 30 pCi/L to 20 pCi/L ratio in lifetime risk is 1.49–1.50, matching the working-level-month ratio. Across the evaluated uncertainty dimensions, the population envelope changes the burden by approximately a factor of four to five, concentration changes it by a factor of 1.5 across the 20–30 pCi/L interval, and risk-model choice changes the central 30 pCi/L burden by a factor of ~1.5 across the three principal miner-derived models.

Because late cohorts experience part of their radon-attributable mortality after AD 1150, calendar-window burden is lower than full lifetime cohort burden.

Imposing a positive correlation between indoor concentration and equilibrium factor, with correlation coefficient $\rho = 0.5$, leaves the reference burden essentially unchanged and slightly widens the upper uncertainty interval.

Monte Carlo propagation over exposure (soil-gas radon, stack pressure, ventilation, equilibrium factor), demographic, baseline-risk, lifetable, population, risk-model, and coefficient uncertainty gives a model-averaged realized Bonito-phase burden distribution with a median of 47 radon-associated lung-cancer deaths (mean 75) and a 2.5th–97.5th-percentile simulation interval of 5–326 deaths. Because the forward calculation derives indoor concentration from sampled source and transport parameters rather than fixing

it at 20 or 30 pCi/L, and integrates over the full risk-model ensemble rather than the principal models alone, this distribution is the marginal counterpart to the conditional concentration–population scenario envelope in **Table 5** rather than a restatement of it. The upper tail is produced by joint high-exposure, high-population realizations.

Variance-based global sensitivity analysis (**Figure 16**) shows that the dominant single uncertainty source is risk-model choice, followed by the indoor-exposure inputs (soil-gas radon concentration, ventilation rate, and stack pressure) and the archaeological population estimate. The total-order Sobol index is 0.40 for risk-model choice, 0.24 for soil-gas radon (C_s), 0.23 for ventilation, 0.14 for population, 0.13 for the risk coefficient (β), and 0.09 for stack pressure; the baseline lung-cancer scaling factor (0.07) and the equilibrium factor (0.06) are secondary, while occupancy and birth rate contribute approximately 0.01 and near-floor enrichment is near zero. When considered jointly, the mass-balance exposure inputs form the largest collective contributor, comparable to or exceeding risk-model choice.

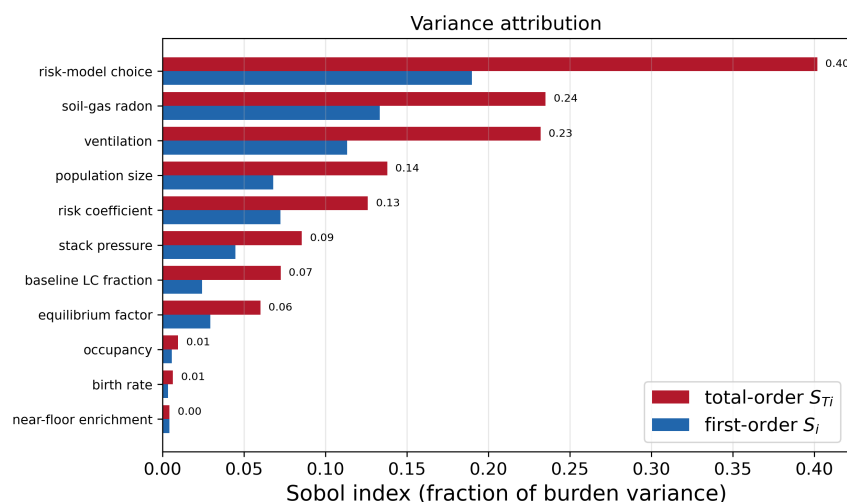


Figure 16: Variance attribution in the global sensitivity analysis. Variance attribution across model, exposure, demographic, and risk parameters is plotted against Sobol sensitivity indices.

Approximately 31% of the variance arises from interactions among inputs (the first-order indices sum to ~0.69), as indicated by the difference between total-order and first-order indices. The upper uncertainty interval is therefore driven by combinations of high-risk model choice, high indoor exposure (high soil gas / low ventilation), high population, and high-risk coefficient, rather than by any single parameter alone. The choice of lifetime-risk measure is much less important than risk-model choice or the exposure and population assumptions. For PUMA full, the excess lifetime risk (ELR), REID, and lifetime excess absolute risk (LEAR) cluster comparatively closely, whereas RADS is higher by construction. This confirms that the main uncertainty is not the formal lifetime-risk metric but the model, exposure, and population assumptions.

Additional architecture-specific sensitivity analyses evaluate near-floor enrichment associated with sleeping height and stack-effect pressure. These analyses show that higher near-floor enrichment increases conditional lifetime lung-cancer risk, while increasing stack-effect pressure raises modeled indoor radon concentration and the corresponding radon-associated mortality outputs (**Figures 17 and 18**).

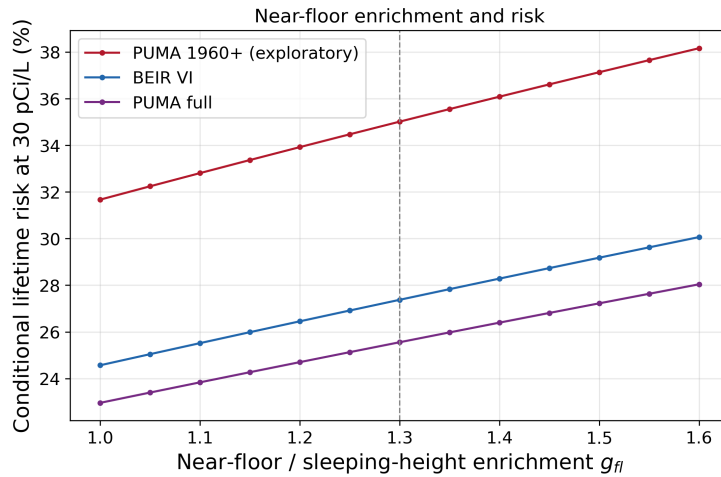


Figure 17: Near-floor enrichment and conditional lung-cancer risk. Sensitivity of conditional lifetime lung-cancer death risk is shown for the near-floor enrichment/sleeping-height factor g_{fl} . Lower sleeping height, represented by higher near-floor enrichment, increases modeled risk; the dotted line marks the reference value $g_{fl} = 1.30$.

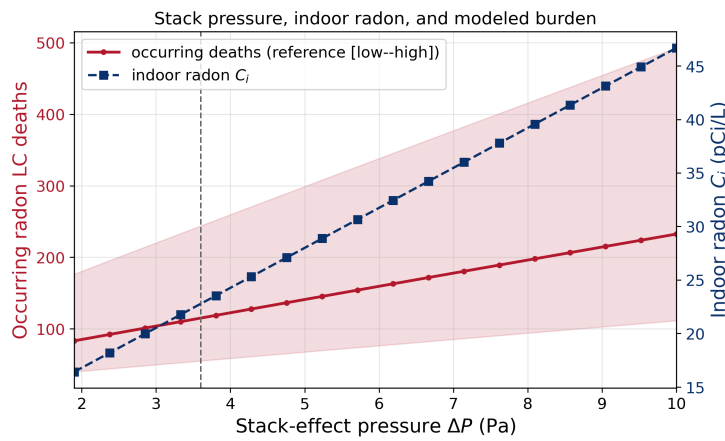


Figure 18: Stack-effect pressure, indoor radon, and modeled burden. Modeled indoor radon concentration C_i (blue dashed) and occurring radon lung-cancer deaths (red, with the low-high demographic envelope) are shown as functions of stack-effect pressure ΔP . The vertical dashed line marks the reference stack pressure $\Delta P = 3.6$ Pa.

4 Discussion

Steady-state solutions to the mass-balance equation, using published parameters and literature-supported uranium concentrations relevant to the regional geologic setting, especially the Menefee Formation beneath and adjacent to Chaco Canyon, indicate that selected Chacoan microenvironments could plausibly have produced chronic radon exposures of potential health and demographic relevance. The prescribed 20–30 pCi/L scenarios are intentionally modest relative to the full physical-model surface and are consistent with regional New Mexico radon conditions, where uranium content and local geology control source potential (e.g., [Brookins, 1991](#); [McLemore, 1995](#)). Under specific combinations of elevated source strength, low ventilation, and hearth-induced pressure coupling, the forward model also permits indoor concentrations approaching or exceeding 140 pCi/L. These higher values are not interpreted as representative Chaco concentrations; rather, they demonstrate the sensitivity of earth-contact interiors to local geology and indoor transport conditions and would imply substantially greater lung-cancer risk if such conditions occurred.

The physical model supports the plausibility of 20–30 pCi/L indoor radon in selected earth-contact, pressure-coupled, low-ventilation Chacoan microenvironments. This range is not asserted as a canyon-wide average. The scenarios are intended to represent higher-exposure architectural settings, including

rooms, pit structures, kivas, and other enclosed ground-coupled spaces in which limited ventilation, hearth heating, floor-level sleeping, dust, and sustained pressure coupling to soil gas could act together. Measured radon of 19.4–20.3 pCi/L in an Ancestral Puebloan pit-structure analog reported by [Whitley and Boyer \(2018\)](#) provides an independent architectural-period comparison for the lower scenario. The mass-balance calculations further show that lower ventilation, higher soil-gas radon, greater pressure differential, and lower porosity can raise indoor concentrations substantially above this range, whereas better ventilation or weaker source coupling can reduce them.

Across the evaluated exposure scenarios, the modeled health burden remains strongly dependent on the assumed exposure conditions. At the Chaco-relevant reference equilibrium factor $F = 0.70$, cumulative exposure by age 60 is 326 and 488 WLM for the 20 and 30 pCi/L scenarios, respectively; the corresponding values are 186 and 279 WLM using the modern-residential $F = 0.40$ comparator. Under the reference population trajectory and the 30 pCi/L scenario, the principal miner-derived models yield approximately 80–122 modeled radon-associated lung-cancer deaths during AD 850–1150, while the exploratory PUMA 1960+ continuation yields 151. Across the broader demographic and concentration sensitivity analysis, the full ensemble spans approximately 26–320 deaths. The sustained reference scenarios therefore produce cumulative exposures overlapping the empirical exposure domain of uranium-miner epidemiology. Conditional lifetime lung-cancer risk among individuals surviving into older adulthood is substantial across the principal miner-derived models; the higher 35% value is produced by the exploratory PUMA 1960+ implementation. In the high-mortality Chaco demographic setting, the resulting modeled burden is concentrated primarily among individuals who survive into later adulthood.

This age concentration arises because the realized population burden is moderated by premodern competing mortality. Convolving the age-specific excess-death density with the reconstructed Chaco birth series gives 80–122 radon-associated lung-cancer deaths across the principal miner-derived models during AD 850–1150 under the reference population trajectory at 30 pCi/L, with the exploratory PUMA 1960+ continuation producing 151 deaths and the broader demographic and concentration sensitivity envelope extending to 26–320 deaths across the full deterministic scenario ensemble. The corresponding lifetime burden of cohorts born within the occupation window is larger because late cohorts continue to experience risk after AD 1150. Even so, annual radon-associated mortality remains a small fraction of all-cause mortality. The model therefore supports radon as a plausible chronic health stressor in selected exposure environments, but not as a sufficient explanation for Chacoan depopulation.

Although numerically modest relative to all-cause mortality, an age-selective burden concentrated among older adults could have had consequences disproportionate to its absolute frequency. In Ancestral Puebloan society, accumulated knowledge was transmitted substantially through oral, experiential, ritual, familial, and intergenerational pathways; older adults may therefore have played important roles in maintaining agricultural, environmental, architectural, genealogical, and ceremonial knowledge. These potential social consequences are not quantified by the present model and are offered as an interpretive implication of the modeled age distribution rather than as a demonstrated historical outcome. This context is relevant because only 25–30% of the Ancestral Pueblo population may have reached age 45 ([Kohler and Reese, 2014](#); [Neitzel, 2018](#); [Palkovich, 1984](#)), while the modeled radon-associated share of mortality reaches approximately 3.6% near age 61 under the reference high-exposure scenario. The architectural setting is also important. Ancestral Puebloan great-house and residential units were built primarily of locally quarried or gathered rock and used mud mortar, rubble cores, and earthen plaster; softer Menefee Formation sediments, alluvial clays, and local soils were also used as mortar, plaster, and earthen construction materials. These practices created extensive contact between occupied spaces and the local geologic environment, although the model indicates that the highest indoor concentrations require soil-gas coupling and restricted ventilation rather than building-material exhalation alone.

The results presented here are best understood within a multicausal model of Chaco Canyon cultural transformation, not as a single-cause explanation for the decline or reorganization of Ancestral Pueblo society. The late eleventh- and twelfth-century reorganization of the Chaco Canyon world involved interacting pressures, including hydroclimatic instability, limits to local agricultural production, changing water-management constraints, dependence on extra-local food and construction resources, shifts in

ritual-political authority, labor mobilization, migration, and reconfiguration of regional networks. That interpretive frame is consistent with the reorganization model proposed by [Lekson and Cameron \(1995\)](#) and with recent work emphasizing population scale, subsistence constraints, and hydrologic change ([Kohler et al., 2025](#); [Wills et al., 2024](#)). In this setting, chronic indoor geogenic radon exposure is best interpreted as a biologically grounded, age-selective potential stressor embedded within a larger environmental, social, and ecological system, not as an independent demographic cause.

One important consideration is that the model does not include age-specific respiratory dosimetry for infants and children. Children differ from adults in airway dimensions, respiratory patterns, tissue geometry, and regional deposition, and microdosimetric modeling indicates that these differences can alter absorbed dose to radiosensitive airway cells ([Furi et al., 2025](#); [ICRP, 1994](#)). In particular, [Furi et al. \(2025\)](#) found higher absorbed dose rates in basal and secretory cell nuclei of a 5-year-old child than in a healthy adult under the same radon-progeny exposure, despite lower deposition rates, primarily because of smaller airway dimensions and thinner mucus layers. These effects do not, however, alter WLM as defined in Eq. (A3), because WLM is an environmental radon-progeny exposure metric and does not include breathing rate. Rather, childhood respiratory differences would affect inhaled activity, airway deposition, absorbed dose per unit exposure, or potentially age-specific biological risk. Incorporating such effects would therefore require a validated childhood-specific dosimetric or risk modifier applied downstream of the WLM calculation. Because no such Chaco-specific coefficient is available, age-specific childhood dosimetry is not included in the present model and remains an uncertainty in translating environmental exposure to biological effect.

The distinction is critical. Radon is capable of generating large lifetime risks among long-lived individuals, but lung cancer is a delayed disease. In a high-mortality premodern population, many individuals die of competing causes before radon-attributable lung cancer can be expressed. The model therefore separates conditional individual risk from realized population burden. This distinction resolves what would otherwise appear paradoxical: cumulative exposure can be miner-comparable for survivors, while the realized number of deaths remains demographically moderate.

The case for considering chronic residential and multigenerational exposure is unusually strong for Chaco. Archaeogenomic evidence from Pueblo Bonito indicates that an elite matrilineal kin group occupied the site across roughly three centuries, with multiple individuals sharing mitochondrial genomes and nuclear-genome kinship ties ([Kennett et al., 2017](#)). Isotopic analyses of Pueblo Bonito burials further support substantial local residence and population stability among the individuals studied ([Price et al., 2017](#)), while recent genomic evidence supports longer-term population continuity in the Chacoan context ([Pinotti et al., 2025](#)). Chaco was therefore not merely an episodic ceremonial gathering place; it also contained long-term residents embedded within a regional geologic environment capable of generating chronic radon exposure. This evidence does not demonstrate that an individual occupied an identical room or experienced an invariant indoor concentration throughout life. Movement among residences within Chaco Canyon would also not necessarily eliminate exposure to the underlying geogenic source, although indoor exposure magnitude would vary with earth contact, ventilation, permeability, pressure coupling, and room use. The sustained from-birth exposure schedule is therefore retained as a defensible long-term scenario for at least part of the locally resident population, while shorter exposure histories are evaluated separately in the exposure-duration sensitivity analysis.

The analysis yields several important findings. The risk framework follows the general architecture of radon lifetime-risk modeling, in which the exposed lung-cancer rate is represented as:

$$r_E(t) = r_0(t)[1 + ERR(t)]. \quad (5)$$

Here, $r_0(t)$ is the baseline age-specific lung-cancer mortality rate, and $ERR(t)$ is the excess relative risk derived from cumulative radon-progeny exposure. Recent comparative analyses show that lifetime-risk estimates are strongly shaped by three components: the selected risk model, the exposure scenario, and the baseline mortality structure ([Sommer et al., 2025](#)). The Chaco Canyon implementation changes all three relative to a modern occupational calculation because exposure begins at birth, occurs in residential and ritual microenvironments rather than mines, and is embedded in a high-mortality premodern survival

schedule.

The model is therefore not a modern residential radon calculation. Modern residential case-control models, including the European and North American pooled analyses, are invaluable for demonstrating radon-associated lung-cancer risk at household concentrations (Darby et al., 2005; Krewski et al., 2005). However, the Chaco exposure scenario produces cumulative WLM values that are more in line with occupational miner cohorts than with typical modern residential exposure. Conversely, the model is not a standard miner calculation, because exposure begins in childhood, continues through domestic and ceremonial spaces, and is filtered through a premodern life table.

The principal epidemiological interpretation emphasizes BEIR VI, PUMA full, and the Joint Czech–French model. BEIR VI provides the classic regulatory and epidemiologic benchmark for radon-associated lung-cancer risk (National Research Council, 1999). PUMA full is based on the largest pooled uranium-miner dataset and provides a high-statistical-power occupational comparator spanning a broad exposure range (Kelly-Reif et al., 2023; Kreuzer et al., 2024). The Joint Czech–French model provides an independent European comparison derived from uranium-miner cohorts exposed at relatively low exposure rates and incorporates time-since-exposure and age-related modifiers (Tomasek et al., 2008). PUMA 1960+ remains particularly informative because it focuses on miners hired in 1960 or later, whose exposures were generally lower and better characterized than those of earlier mining cohorts (Richardson et al., 2022). However, because the published PUMA 1960+ formulation does not include a separate exposure window beyond 34 years, its application to sustained from-birth exposure requires continuation of the oldest available time-since-exposure coefficient. We therefore retain PUMA 1960+ as an exploratory upper-bound sensitivity model rather than as a principal interpretive model. Wismut full, Wismut 1960+, and adjusted Jacobi formulations are retained as additional sensitivity comparators, allowing the influence of epidemiological model structure to be evaluated across the broader ensemble.

The resulting framework is a defensible hybrid because it incorporates mass-balance indoor-radon physics, miner-derived lung-cancer ERR functions, Chaco-specific exposure microenvironments, premodern survival, and annually reconstructed demography. Its principal advantage is that it does not confuse gas concentration, dose, conditional individual risk, and realized population mortality. Each is estimated separately and then connected through explicit assumptions.

Radon gas concentration is not the biologically complete exposure metric. The bronchial dose is delivered primarily by short-lived radon progeny, especially ^{218}Po and ^{214}Po , following inhalation and deposition in the respiratory tract. The conversion from radon gas to biologically effective progeny exposure therefore depends on the equilibrium factor, aerosol size distribution, attached/unattached fraction, ventilation, and breathing pattern (Porstendörfer, 2001). The equilibrium factor is not a universal constant but varies with ventilation, aerosol concentration and size distribution, indoor activities, and other environmental conditions, with substantial variation documented across indoor environments (Porstendörfer, 2001; Chen and Harley, 2018; Prasad et al., 2023; United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), 2024).

The model's reference equilibrium factor $F = 0.70$ represents dusty, smoke-laden, low-ventilation interiors rather than modern well-ventilated houses. Relative to the modern residential comparator $F = 0.40$, this increases working-level exposure by a factor of 1.75 at the same radon gas concentration. A value above the modern residential default is physically plausible for enclosed Chacoan spaces with hearth smoke, mineral dust, earthen floors, and limited air exchange, although F was not measured directly at Chaco. The explicit $F = 0.40$ comparison therefore provides a lower modern-residential sensitivity case rather than an alternative Chaco-specific reference.

Recent dose literature supports this emphasis. Makumbi et al. (2024) identify aerosol size distribution, unattached fraction, breathing rate, and ventilation as major uncertainties in the equivalent lung-dose coefficient for radon progeny. Kayouh et al. (2025) use computational fluid dynamics to model radon-progeny deposition in anatomically realistic airways and report attached-fraction dose-conversion factors of 6.55–10.72 mSv WLM⁻¹, broadly consistent with ICRP and United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR)-derived recommendations. These findings reinforce two

points central to the Chaco model: first, progeny physics and aerosol conditions matter; second, a scalar gas concentration alone understates the complexity of dose delivery.

This matters especially for childhood and ritual subgroups. Children have higher ventilation per unit body mass and may receive different regional airway deposition patterns. Ritual or craft specialists working in dusty, enclosed, smoke-rich spaces may experience higher equilibrium factors and greater attached-progeny exposure than the population mean. The model treats these as sensitivity dimensions rather than certainties, but the underlying physical mechanism is well supported.

The mass-balance model includes both soil-gas entry and a building-material term. The latter accounts for radon derived from sandstone, adobe, or masonry surfaces. However, the dominant pathway in the modeled Chaco settings is soil-gas entry driven by pressure coupling, not wall exhalation alone. This conclusion is consistent with the broader radon literature, which identifies soil gas as the dominant source in most elevated buildings and advective entry through foundations, cracks, and earthen contacts as the primary transport mechanism (Nazaroff, 1992; Park et al., 2016).

Recent building-material studies provide an important constraint. Caridi et al. (2025) measured ^{222}Rn exhalation from natural stones used in historical architecture and, using the Markkanen room model, found that ordinary stone exhalation under ordinary ventilation generally produced indoor radon below regulatory concern. That result supports the interpretation that Chaco's highest modeled concentrations require strong ground coupling, low ventilation, and geologic source strength rather than masonry exhalation alone.

The geologic source term must be considered in calculations. Nunes et al. (2023) and Silber et al. (2026) emphasize that indoor radon depends strongly on local geology, uranium/radium content, soil permeability, climate, and building conditions. Recent surveys and reviews further demonstrate that indoor radon can vary substantially with local geology, building characteristics, ventilation, and measurement setting (e.g., Chen and Harley, 2018; Hansen et al., 2023; Kuzmanović, 2026; Rey et al., 2024). Brookins (1991) provides a useful regional analog from Albuquerque, where soil uranium ranged from 1–6 ppm, soil-gas radon ranged from 40–890 pCi/L, and high indoor radon correlated with uranium-rich and bedrock-proximal settings. The Chaco model's soil-uranium sweep operationalizes this geology-first principle: uranium concentration, emanation, porosity, ventilation, and pressure coupling combine to determine the soil-gas source and its conversion to indoor concentration.

The model's sensitivity to soil uranium should therefore be treated as a first-order result. Under plausible assumptions, increasing soil uranium from 3 to 9 ppm approximately triples soil-gas radon, indoor concentration, WLM yr^{-1} , dose, and expected burden, holding other factors constant. Ventilation can offset or amplify this effect. A low-ventilation structure over high-U, low-porosity ground can produce indoor concentrations far above the 20–30 pCi/L scenario range, whereas a better-ventilated structure on lower-U substrate may remain much lower.

The annual mortality curve is delayed relative to the population curve. The reference population peaks around AD 1075, whereas radon-associated annual deaths peak approximately six decades later, near AD 1138. This is not an artifact. It is the expected result of applying a delayed carcinogenic hazard to birth cohorts. The large Classic Bonito cohorts must first survive into the ages at which radon-induced lung cancer is expressed.

This latency structure is interpretively important. The temporal overlap between the modeled mortality peak and Late Bonito depopulation should not be read as evidence that radon caused depopulation. Rather, radon mortality peaks late because the relevant cohorts age into the cancer-prone interval while the standing population is already contracting. The standing-population death rate therefore rises toward the end of the occupation window even when absolute annual deaths remain small.

This distinction is central to the argument. Radon is a delayed, age-selective stressor. It does not behave like famine, epidemic infectious disease, warfare, or abrupt migration. Its population signature is slow, elder-concentrated, and lagged. That is precisely why it can be biologically meaningful without appearing as an acute demographic shock.

The modeled population mean almost certainly conceals substantial exposure heterogeneity. Chaco

architecture included rooms, plazas, court kivas, great kivas, storage spaces, construction zones, and domestic interiors with very different ventilation, floor contact, dust loading, and occupancy patterns (Dyke, 2004; Windes, 2014). Earth-contact and partially subterranean spaces are the most important for radon because they combine proximity to the source, low ventilation, and potential pressure coupling.

Kivas and other ritual chambers are therefore plausible high-exposure microenvironments. They are enclosed, earth-contact architectural settings in which ventilation may have been restricted and smoke or dust loading elevated during use. The model's high-equilibrium sensitivity case, in which F approaches 1.0, is intended to represent such conditions. It should not be interpreted as applying to all residents or all rooms. It identifies a plausible upper-tail subgroup.

Tobacco introduces another potential heterogeneity. Archaeological and ethnographic evidence indicates long-standing Indigenous use of *Nicotiana* and other smoke plants in ceremonial contexts in North America (Adair, 2000; Tushingham and Eerkens, 2016; Tushingham et al., 2018; Winter, 2000). The evidence does not justify assuming that the entire Chacoan population smoked habitually in a modern sense. It does support the possibility that some ritual specialists or ceremonial participants had greater smoke exposure than others. Because radon and smoking interact in lung-cancer risk models, tobacco use could amplify risk within such subgroups. The best-supported modern evidence suggests a strong joint effect rather than simple additivity (Darby et al., 2005; National Research Council, 1999). The model's smoking sensitivity should therefore be interpreted as plausible subgroup amplification, not as evidence that the whole Chacoan population smoked habitually.

Under the evaluated exposure scenarios, radon risk would have been strongly influenced by architectural setting and patterns of occupancy. The highest modeled risks accrue to individuals with the longest lifespan, the greatest time in earth-contact low-ventilation spaces, the greatest exposure to dust and smoke, and, where applicable, tobacco use. If such exposure patterns corresponded to ritual specialists, elders, craft workers, construction laborers, or long-term occupants of kivas and other enclosed spaces, the burden could have been socially concentrated. That interpretation remains a hypothesis because the present model does not estimate the historical population fractions assigned to particular occupational, ritual, or architectural exposure classes.

The Chaco framework also provides a template for comparative archaeo-oncology: the study of how carcinogenic environments may have shaped disease burdens in past populations. The next most compelling class of sites is not necessarily another surface pueblo, but troglodytic or rock-cut habitation on radon-relevant substrates. Cappadocia is particularly important because its volcanic tuffs and rock-cut dwellings create a strong geological and architectural analog: source rock surrounds the dwelling volume, ventilation may be restricted, and multigenerational occupation is historically documented. Cappadocia also has an independent precedent for geogenic respiratory carcinogenesis through erionite-associated mesothelioma clusters in Turkish villages (Baris et al., 1987; Carbone et al., 2007, 2011).

A Cappadocian application would not require a new conceptual model. It would require site-specific inputs: tuff uranium and radium activity, emanation, exhalation rates, chamber geometry, ventilation, aerosol conditions, occupancy, and period-specific survival structure. Unlike Chaco, many Cappadocian spaces survive intact and can be instrumented directly with continuous radon monitors, tracer-gas ventilation measurements, RAD7 exhalation protocols, and aerosol/progeny measurements. Such work would test whether the Chaco model's central finding, such as high conditional risk but demographically limited realized burden, generalizes to other earth-contact societies or whether deeper, more enclosed, longer-lived populations generate larger realized burdens. The same logic extends to other radon-relevant earth-contact traditions: Mesa Verde cliff dwellings, Chinese loess yaodong dwellings, Mediterranean and North African cave houses, Petra's rock-cut spaces, and other tuff or karst settlements. The controlling variables are scalable, beginning with source-rock uranium/radium, emanation, porosity, ventilation, pressure coupling, progeny equilibrium, occupancy, and survival.

The principal empirical limitation is that the 20–30 pCi/L scenarios are analog-supported rather than measured directly in intact Chacoan living spaces. This site-specific limitation occurs within a broader statewide lack of representative, high-resolution indoor radon measurements in New Mexico (Silber et al.,

2026). This limitation does not preclude quantitative scenario analysis, but it means that the calculations should not be interpreted as a direct reconstruction of canyon-wide historical exposure. The model identifies a clear measurement program through which the physical assumptions and exposure ranges can be tested.

First, local soil-gas radon should be measured across major Chacoan lithologies, especially earth-contact architectural contexts and exposures of uranium-bearing or carbonaceous units. Second, uranium/radium activity, emanation fraction, porosity, moisture, and permeability should be measured directly for soils, floor sediments, mortars, masonry stones, and nearby bedrock. Third, ventilation rates should be measured in stabilized Chacoan rooms where preservation permits, and in reconstructed analog structures under seasonal conditions. Fourth, radon exhalation from building materials should be measured using closed-chamber RAD7 or equivalent methods. Fifth, equilibrium factor, aerosol loading, and unattached fraction should be measured in analog structures under dust and smoke conditions, because these control progeny dose more directly than radon gas concentration alone. Finally, all results should be propagated through the full ensemble of risk models rather than a single coefficient.

The irreducible uncertainty is epidemiologic: no modern model perfectly represents a premodern, from-birth, high-occupancy, mixed domestic-ritual exposure scenario. That uncertainty is not a reason to abandon the analysis. It is a reason to report model ensembles and to distinguish conditional individual risk from realized population burden.

The analysis supports a restrained but important conclusion. Chronic indoor radon progeny exposure in selected Chacoan microenvironments could have produced uranium-miner-comparable cumulative exposure among long-lived residents and a substantial conditional lifetime lung-cancer risk. When embedded in a premodern survival structure and convolved with the reconstructed Chaco birth series, that individual hazard translates into a realized burden of 80–122 radon-associated lung-cancer deaths across the principal miner-derived models during AD 850–1150 at 30 pCi/L. The exploratory PUMA 1960+ continuation produces the higher value of 151 deaths. These absolute burden estimates remain strongly dependent on the assumed premodern baseline lung-cancer hazard. Sensitivity to f_{LC} is approximately linear; for example, the Joint Czech–French model at 30 pCi/L yields 45.9, 91.4, and 136.5 occurring deaths for $f_{LC} = 0.10$, 0.20, and 0.30, respectively. By comparison, conditional lifetime REID remains 20.9% across these values because f_{LC} scales the premodern absolute baseline hazard rather than the modern-reference conditional-risk calculation. The absolute death counts should therefore be interpreted as scenario-dependent burden estimates, whereas the physical exposure calculation, conditional per-survivor risk, and latency structure are not controlled by this normalization.

This burden is too small to serve as a primary explanation for Chacoan depopulation. It is, however, large enough within the evaluated scenarios to warrant consideration as a chronic, age-selective stressor. Because the modeled excess deaths are concentrated in older adults, and because the highest exposures may have occurred in ritually or architecturally specialized microenvironments, the social consequences could have exceeded the raw death count. This is a conceptual implication rather than a modeled social outcome. If affected individuals held important roles in knowledge transmission, ritual practice, subsistence, caregiving, or community leadership, an age-selective health burden could have influenced continuity or adaptive capacity, but the present model does not quantify those effects. Radon-attributable lung cancer remains the health endpoint quantified here; other chronic respiratory outcomes remain outside the present burden calculation and should be treated as hypotheses for future work. The methodological contribution of the study is that it links geologic source strength, building physics, radon-progeny dosimetry, miner-derived epidemiology, premodern life tables, and annually reconstructed demography in a single quantitatively specified approach. That approach is directly testable at Chaco through soil-gas, ventilation, exhalation, and progeny measurements and is transferable, with site-specific parameterization, to other earth-contact and rock-cut archaeological settings where natural radiation may have been a neglected component of disease ecology.

5 Conclusions

This study develops and applies a quantitative model-based assessment of chronic indoor radon exposure as a previously underexamined geogenic health stressor in Chaco Canyon. The analysis does not replace established archaeological explanations for Chaco Canyon reorganization or assign radon a singular or primary causal role. Rather, it asks whether plausible Chacoan earth-contact and semi-subterranean microenvironments could have generated radon-progeny exposures sufficient to produce a modeled chronic disease burden within a population already affected by hydroclimatic, agricultural, hydrologic, social, and political stress.

The methodological novelty of this study is that it links geologic source strength, soil-gas radon production, pressure- and diffusion-driven indoor transport, building-material exhalation, ventilation, radon-progeny equilibrium, seasonal occupancy, floor-level exposure, working-level-month accumulation, miner-derived excess-relative-risk models, premodern survival, and annually reconstructed Chaco demography into a quantitatively specified and testable source-to-burden model. This integration keeps source, exposure, epidemiological risk, and realized demographic burden distinct while connecting them through explicit assumptions.

The physical results indicate that selected Chacoan microenvironments could plausibly have supported elevated indoor radon concentrations under specific combinations of uranium/radium-bearing soil or rock, soil-gas transport, low ventilation, pressure coupling, and high occupancy. The modeled 20–30 pCi/L scenarios should not be interpreted as reasonable canyon-wide averages for Chaco-specific residential structures. Rather, they should be considered high-exposure scenarios for vulnerable architectural settings: ground-coupled rooms, pit structures, kivas, lower-tier rooms, enclosed sleeping or storage spaces, and other interiors where direct earth contact, limited air exchange, hearth heating, smoke, dust, and floor-level occupation could have increased both radon entry and radon-progeny availability. The measured Ancestral Puebloan pit-structure analog and the mass-balance calculations together show that these concentrations are physically plausible rather than speculative.

At the individual level, the reference high-occupancy, high-aerosol scenario yields 5.43–8.14 WLM yr⁻¹ at 20–30 pCi/L and cumulative exposure of 326–488 WLM by age 60. These scenario-based values overlap the empirical exposure domain of uranium-miner epidemiology, and conditional lifetime lung-cancer risk among individuals surviving into older adulthood is substantial across the principal miner-derived models. They should not be interpreted as independently reconstructed historical exposures.

Demographic interpretation is necessarily more constrained. When the same age-specific excess-death densities are embedded within a high-mortality premodern survival schedule and convolved with reconstructed annual births, realized population burden is much smaller than conditional individual risk alone would imply. This distinction is fundamental. In a population with high infant, juvenile, and young-adult mortality, many individuals died of competing causes before reaching the older ages at which radon-induced lung cancer is most likely to be expressed. Under the reference population trajectory and the 30 pCi/L scenario, the principal miner-derived models yield approximately 80–122 modeled radon-associated lung-cancer deaths occurring during AD 850–1150. The exploratory PUMA 1960+ continuation produces a higher upper-bound estimate of 151 deaths. Annual radon-attributable mortality peaks late, during the Late Bonito contraction, because large eleventh-century birth cohorts reached cancer-prone ages decades after the population maximum. This latency structure is expected for a delayed carcinogenic hazard and should not be mistaken for evidence of an acute demographic event.

The model does not support radon as a primary driver of decline. It identifies radon as a plausible, quantifiable chronic health stressor in selected Chacoan residential and ceremonial microenvironments. Any broader social consequences of an older-adult mortality burden remain speculative and are not outputs of the model.

The model also suggests that exposure was unlikely to have been evenly distributed. The highest risks would have accrued to those who lived longest, spent the most time in ground-coupled low-ventilation interiors, slept or worked near floors, and experienced the greatest combined exposure to radon progeny,

dust, smoke, and reduced air exchange. Ritual specialists, craft workers, construction laborers, or long-term occupants of kivas and other enclosed ceremonial spaces may therefore have occupied the upper tail of the exposure distribution. This possibility remains a hypothesis, but it is physically and epidemiologically coherent and should be evaluated through microenvironment-specific sampling.

A key implication is that radon's archaeological signature is expected to be indirect. Radon-induced lung cancer leaves no diagnostic skeletal lesion, and short-lived radon progeny cannot persist across Chacoan timescales. The relevant evidence is therefore not likely to be osteological in the narrow sense. It is expected to be environmental, architectural, spatial, and statistical. This distinguishes radon from other archaeologically documented stressors and explains why it could have been overlooked despite being biologically consequential.

The empirical path forward is clear. The present scenario model should be strengthened, wherever possible, by site-specific exposure reconstruction. The highest-priority measurements are soil-gas radon across Chacoan lithologies and architectural contexts; uranium and radium activity in soils, floor sediments, masonry, mortar, and bedrock; radon emanation and exhalation rates; porosity, permeability, and moisture; ventilation rates in preserved or reconstructed rooms; pressure differentials under seasonal heating and wind conditions; and equilibrium factor, aerosol loading, and unattached progeny fraction under dust- and smoke-rich conditions. These measurements would directly test the central physical assumptions and substantially reduce the largest exposure-side uncertainties.

In sum, the study identifies a plausible chronic geogenic exposure pathway that can be evaluated quantitatively but remains conditional on reconstructed source, architectural, epidemiological, and demographic assumptions. Whether and to what extent radon affected historical Chacoan health remains to be tested with site-specific environmental measurements. The broader significance is methodological: the approach links natural radiation, architecture, indoor-air physics, epidemiologic risk transfer, and paleodemography in a form that can be applied to other enclosed earth-contact or rock-cut settings.

Data availability

The mathematical formulation, parameter definitions, assumptions, uncertainty treatment, and computational sequence used in this study are provided in the main text and Appendix A, with numerical results reported in the figures and tables. The computational implementation used for the analysis is proprietary and is not publicly available at this time. Figures 2 and A1 were generated in ArcGIS (<https://www.esri.com>); the data figures and the underlying numerical analyses were produced in Python 3.10.5 using NumPy 2.1.2 and SciPy 1.14.1.

Declaration of competing interests

The authors declare no conflict of interest.

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Author contributions: CRediT

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Appendix A. Supplementary Stratigraphic Context and Mathematical Framework

Appendix A provides supporting material for the source-to-burden model used in the manuscript. It begins with a detailed stratigraphic map and legend of the Chaco Canyon area (**Figure A1**), which expand the geologic context summarized in **Figure 2** and identify mapped units, contacts, structural features, and Menefee Formation coal-bearing intervals relevant to potential geogenic radon source pathways. The appendix then presents the mathematical treatment used to translate this spatial and stratigraphic context, together with architectural and demographic assumptions, into indoor radon concentration, radon-progeny exposure, cumulative WLM, lifetime-risk metrics, premodern survival, cohort-level burden, and uncertainty propagation.

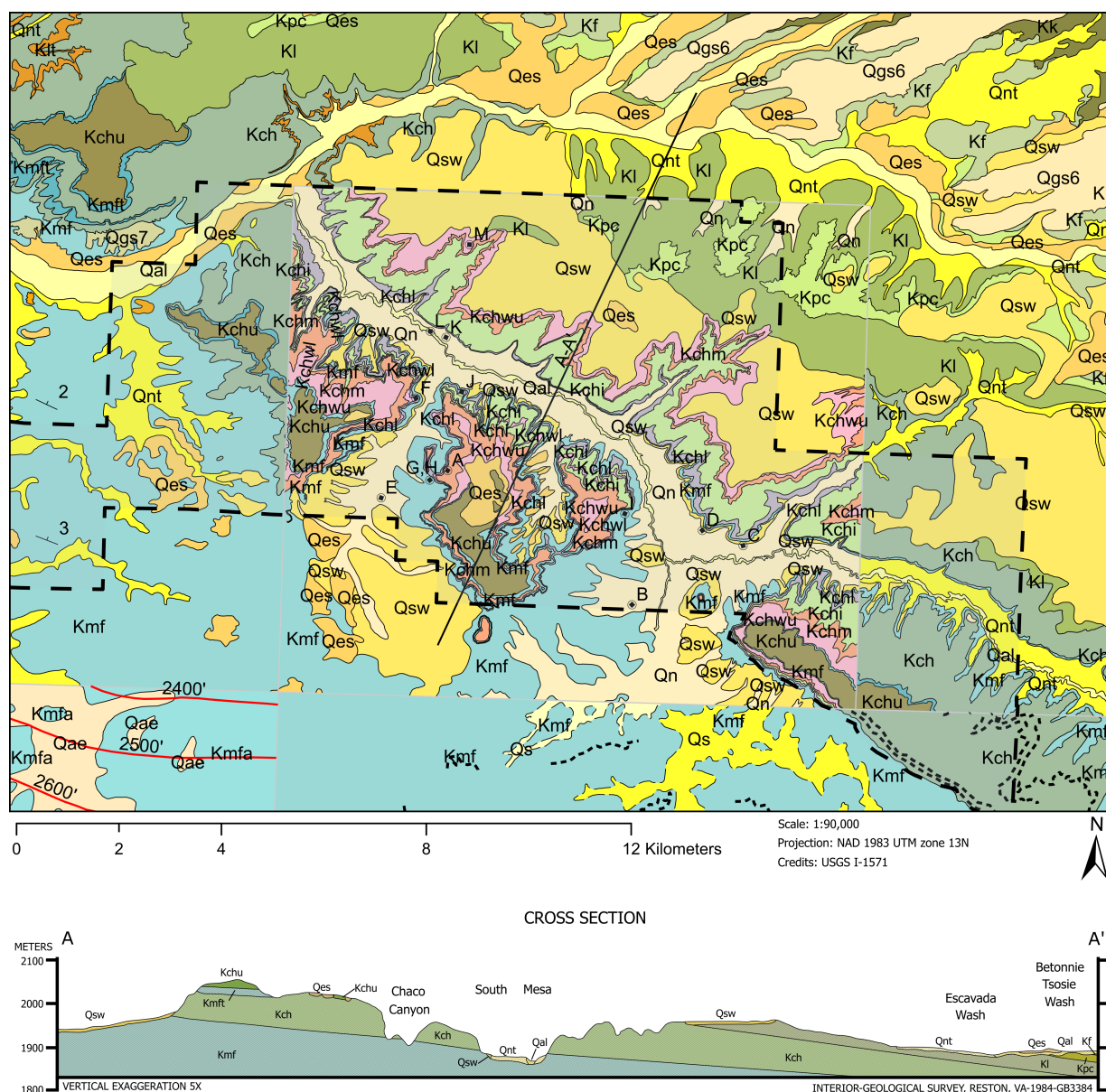


Figure A1: Detailed stratigraphic map and legend for Chaco Canyon. Supplementary geologic map showing stratigraphic units, contacts, structural features, the Chaco Culture boundary, and the A–A' geologic cross-section transect used to contextualize **Figure 2**. The legend appears on the following page and identifies mapped Quaternary deposits, Cretaceous stratigraphic units, Menefee Formation coal-bearing intervals, Cliff House Sandstone subdivisions, contacts, faults, folds, lineaments, and map symbology relevant to evaluating potential geogenic radon source pathways. Map credit: United States Geological Survey (USGS) I-1571; projection: NAD 1983 UTM Zone 13N. Map geology and symbology are reproduced from USGS I-1571; minor cartographic registration offsets inherent to the source map are retained. Apparent color discontinuities between adjacent areas result from aggregating USGS surveys that were conducted with different objectives and at different mapping scales, which yielded higher- or lower-resolution coverage in different areas rather than true geological boundaries.

Figure A1 legend. Map symbols and stratigraphic-unit definitions corresponding to **Figure A1**.

The mathematical framework below provides the quantitative treatment used to support the Methods and Results sections, including exposure conversion, near-floor enrichment, annual and cumulative WLM, lifetime-risk metrics, premodern survival, birth-cohort convolution, high-equilibrium subgroup calculations, and uncertainty propagation. **Table A1** lists all symbols and variables appearing in this appendix.

Table A1. Symbols used in Appendix A. Definitions are limited to symbols appearing in the appendix equations and accompanying text.

Symbol	Definition	First use
WL	Working level; radon-progeny potential alpha-energy concentration.	Eq. (A1)
C_0	Prescribed indoor radon-gas concentration used for exposure calculations.	Eq. (A1)
C_s	Soil-gas radon concentration varied in uncertainty propagation.	Text before Eq. (A27)
F	Equilibrium factor between radon gas and short-lived progeny.	Eq. (A1)
D_{dust}	Optional dust-resuspension multiplier; held at 1.00 in the reference model.	Eq. (A1)
C_{floor}	Radon concentration at the floor-level sleeping breathing zone.	Eq. (A2)
C_{room}	Room-average radon concentration.	Eq. (A2)
g_{fl}	Near-floor enrichment factor applied to sleeping exposure.	Eqs. (A2)–(A3)
WLM_{yr}	Annual exposure in working-level months per year.	Eq. (A3)
s	Season index.	Eq. (A3)
κ_s	Seasonal concentration fraction.	Eq. (A3)
$h_{s,sl}$	Indoor sleeping hours per day in season s .	Eq. (A3)
$h_{s,wk}$	Indoor waking hours per day in season s .	Eq. (A3)
d_s	Number of days in season s .	Eq. (A3)
D_{eff}	Annual effective dose used only for radiation-protection comparison.	Eq. (A4)
D_C	Dose coefficient in mSv/WLM.	Eq. (A4)
$w(a)$	Annual WLM received at age a .	Eq. (A5)
a	Age or attained age in years.	Eqs. (A5)–(A23)
A	Attained-age cutoff for cumulative exposure.	Eq. (A6)
$W(A)$	Cumulative exposure through attained age A .	Eq. (A6)
W_{50}, W_{60}	Cumulative exposure through ages 50 and 60, respectively.	Text after Eq. (A6)
χ_a	Optional age-specific dosimetric/risk modifier factor.	Text after Eq. (A6)
$r_E(a)$	Exposed lung-cancer mortality rate at age a .	Eq. (A7)
$r_0(a)$	Baseline lung-cancer mortality rate at age a .	Eq. (A7)
$ERR(a)$	Model-specific radon excess relative risk at age a .	Eq. (A7)
β	Leading excess-relative-risk exposure coefficient.	Eq. (A8)
$W_{5-14}(a)$	Cumulative WLM received 5–14 years before attained age a .	Eq. (A8)
$W_{15-24}(a)$	Cumulative WLM received 15–24 years before attained age a .	Eq. (A8)
$W_{25+}(a)$	Cumulative WLM received 25 or more years before attained age a .	Eq. (A8)
$\theta_{15-24}, \theta_{25+}$	Weights applied to older time-since-exposure windows.	Eq. (A8)
$\phi_{age}(a)$	Attained-age modifier.	Eq. (A8)
γ_z	Exposure-rate modifier.	Eq. (A8)
$S_0(a)$	Baseline survival to age a .	Eq. (A9)

Symbol	Definition	First use
$q_0(u)$	Baseline all-cause mortality hazard at age u .	Eq. (A9)
u	Age index used in survival summations.	Eqs. (A9)–(A10), (A15)
$S_E(a)$	Exposure-affected survival to age a .	Eq. (A10)
LEAR	Lifetime excess absolute risk.	Eq. (A11)
REID	Risk of exposure-induced death.	Eq. (A12)
ELR	Excess lifetime risk.	Eq. (A13)
RADS	Radiation-attributed decrease of survival.	Eq. (A14)
$S_{Chaco}(a)$	Premodern Chaco survival to age a .	Eq. (A15)
$q_{Chaco}(u)$	Premodern Chaco all-cause mortality hazard at age u .	Eq. (A15)
$r_0^{Chaco}(a)$	Chaco baseline lung-cancer hazard at age a .	Eq. (A16)
f_{LC}	Scaling factor for the premodern baseline lung-cancer hazard.	Eq. (A16)
$r_0^{light}(a)$	Reference light-smoker age-specific lung-cancer mortality curve.	Eq. (A16)
$e(a)$	Age-specific radon-attributable lung-cancer death density per birth.	Eq. (A17)
$REID_{birth}$	Per-birth risk of exposure-induced death.	Eq. (A18)
$\bar{A}$	Mean age at radon-induced death.	Eq. (A19)
$D(y)$	Expected radon-attributable lung-cancer deaths in calendar year y .	Eq. (A20)
y	Calendar year.	Eqs. (A20)–(A23)
$B(y - a)$	Number of births a years before calendar year y .	Eq. (A20)
$D_{decade}(Y)$	Decade-scale burden beginning in year Y .	Eq. (A21)
Y	Decade-start calendar year in Eq. (A21); generic model output in Sobol expressions.	Eqs. (A21), (A28)–(A29)
$\rho(y)$	Standing-population death rate per 1,000 persons in year y .	Eq. (A22)
$P(y)$	Resident population in calendar year y .	Eq. (A22)
λ_v	Ventilation rate varied in uncertainty propagation.	Text before Eq. (A27)
ΔP	Soil-indoor pressure difference varied in uncertainty propagation.	Text before Eq. (A27)
b	Crude birth rate used to calculate annual births.	Text before Eq. (A27)
D_{total}	Total Bonito-phase radon-attributable burden.	Eq. (A23)
Θ	Risk-model parameter vector.	Eq. (A25)
Θ_0	Maximum-likelihood estimate of the parameter vector.	Eq. (A25)
Σ_0	Covariance matrix for the parameter vector.	Eq. (A25)
$\beta^{(j)}$	Sampled leading exposure coefficient in Monte Carlo draw j .	Eq. (A26)
Z_j	Standard normal variate for Monte Carlo draw j .	Eq. (A26)
GSD	Geometric standard deviation inferred from published uncertainty intervals.	Eq. (A26)
$Q_{0.025}, Q_{0.975}$	2.5th and 97.5th percentiles of the simulated burden distribution.	Eq. (A27)
S_i	First-order Sobol index for input parameter i .	Eq. (A28)
S_{Ti}	Total-order Sobol index for input parameter i .	Eq. (A29)
X_i	Input parameter i in the Sobol analysis.	Eqs. (A28)–(A29)
$X_{\sim i}$	All input parameters except X_i .	Eq. (A29)

The carcinogenic exposure is not radon gas alone, but the short-lived radon progeny deposited in the respiratory tract. Radon gas concentration is generally measured in pCi/L, where 1 pCi/L = 37 Bq m⁻³, and was converted to working level:

$$WL = C_0 F 10^{-2} D_{dust}. \quad (A1)$$

Here, WL is working level, C_0 is radon gas concentration in pCi/L, F is the equilibrium factor between radon gas and short-lived progeny, and D_{dust} is an optional additional dust-resuspension multiplier. The 10^{-2} factor appears because 100 pCi/L at full secular equilibrium corresponds to 1 WL (National Research Council, 1999).

The choice $F = 0.70$ represents a smoke- and dust-rich, low-ventilation indoor environment. This does not mean that dust creates more radon gas; rather, aerosol loading changes radon-progeny attachment and airborne potential alpha energy at a given radon gas concentration (National Research Council, 1999; Paquet et al., 2017; Porstendörfer, 2001). Relative to the modern residential comparator $F = 0.40$, the working-level exposure is larger by a factor of 1.75. Because aerosol effects are represented through F , D_{dust} is held at 1.00 to avoid double counting. The $F = 0.40$ calculation is used as a comparator rather than as a Chaco-specific reference because the dusty, smoke-rich, earthen, and poorly ventilated environments considered here differ from typical modern residential conditions. At $F = 0.70$, WL is 0.140 and 0.210 for the 20 and 30 pCi/L scenarios, respectively; both remain below 0.5 WL, placing the reference Chaco exposure in the lowest exposure-rate category of BEIR VI/PUMA-type models.

Because radon enters from soil and floors, and because Chaco residents could sleep on mats on or near the ground surface, the sleeping breathing zone may have been enriched relative to the room-average concentration. This was represented by a near-floor enrichment factor:

$$g_{fl} = \frac{C_{floor}}{C_{room}}. \quad (A2)$$

The reference value is $g_{fl} = 1.30$, with a sensitivity range of $1.0 \leq g_{fl} \leq 1.6$. The value $g_{fl} = 1$ corresponds to a completely mixed room. Values above 1 represent floor-level enrichment during sleeping. The enrichment factor is applied only to sleeping hours, not to all indoor hours.

Annual exposure was calculated by summing seasonal and posture-specific contributions:

$$WLM_{yr} = \frac{FD_{dust}10^{-2}}{170} \sum_s \left[(\kappa_s C_0) (g_{fl} h_s^{sl} + h_s^{wk}) d_s \right]. \quad (A3)$$

Here, s indexes season, C_0 is the prescribed indoor radon scenario, κ_s is the seasonal concentration fraction, $h_{s,sl}$ is sleeping hours per day indoors, $h_{s,wk}$ is waking hours per day indoors, d_s is the number of days in the season, g_{fl} enriches sleeping exposure near the floor, and 170 h defines 1 WLM.

Table A2. Seasonal and posture budget used for the reference exposure scenario.

Season	Days d_s	Waking		κ_s	Effective weighted hours $g_{fl} h_s^{sl} + h_s^{wk}$
		Sleeping hours h_s^{sl}	indoor hours h_s^{wk}		
Winter	151	8	12	1.00	22.4
Shoulder	92	8	9	0.90	19.4
Summer	122	8	6	0.80	16.4

This structure reflects cold high-desert nights and seasonal heating/ventilation differences. Indoor exposure is highest in winter, but summer is not treated as exposure-free because nighttime cooling can still lead to indoor sleep and hearth use. Because Eq. (A3) is linear in C_0 , total annual exposure is 5.43 WLM yr⁻¹ and 8.14 WLM yr⁻¹ for the 20 and 30 pCi/L scenarios, respectively.

For radiation-protection comparison only, annual effective dose was calculated as:

$$D_{eff} = \text{WLM}_{\text{yr}} D_C, \quad (\text{A4})$$

where D_C is the dose coefficient in mSv/WLM. ICRP reports approximately 10 mSv/WLM for most circumstances and approximately 20 mSv/WLM for poorly ventilated or physically demanding work conditions (Paquet et al., 2017; Marsh et al., 2021). Thus, the 20 pCi/L scenario corresponds to 54–109 mSv yr⁻¹, and the 30 pCi/L scenario corresponds to 81–163 mSv yr⁻¹. These values communicate exposure magnitude only. Lung-cancer mortality was calculated from epidemiological risk models using WLM, not from effective dose.

For the sustained from-birth exposure scenario, exposure was modeled as beginning at birth:

$$w(a) = \text{WLM}_{\text{yr}}, \quad a = 0, 1, \dots, 94. \quad (\text{A5})$$

Cumulative exposure to attained age A is:

$$W(A) = \sum_{a=0}^{A-1} w(a) = A \text{ WLM}_{\text{yr}}. \quad (\text{A6})$$

For 20 pCi/L, $W_{50} = 271$ WLM and $W_{60} = 326$ WLM. For 30 pCi/L, $W_{50} = 407$ WLM and $W_{60} = 488$ WLM. These cumulative exposures overlap the range of occupational miner exposures used in radon risk models, although the exposure context is domestic/ritual rather than occupational and begins earlier in life (Kelly-Reif et al., 2023; National Research Council, 1999; Sommer et al., 2025). An optional age-specific modifier χ_a can be applied at the dosimetric or risk stage to represent potential age-dependent differences in respiratory deposition, absorbed dose, or susceptibility. It does not modify WLM as defined in Eq. (A3). The main analysis sets $\chi_a = 1$ because no validated childhood-specific coefficient is available for the present exposure context.

All radon risk models were applied as multiplicative excess-relative-risk models:

$$r_E(a) = r_0(a) [1 + \text{ERR}(a)]. \quad (\text{A7})$$

where $r_E(a)$ is exposed lung-cancer mortality at age a , $r_0(a)$ is baseline lung-cancer mortality, and $\text{ERR}(a)$ is the model-specific radon excess relative risk. This projection structure is used in BEIR VI, PUMA, and Sommer-style lifetime-risk calculations (National Research Council, 1999; Kelly-Reif et al., 2023; Kreuzer et al., 2024; Sommer et al., 2025). The risk model ensemble included BEIR VI, PUMA full, PUMA 1960+, Joint Czech-French, Wismut full, Wismut 1960+, and adjusted Jacobi formulations. Coefficients were taken from the published risk-model tables or from the Sommer et al. (2025) synthesis. Residential pooled models were retained only as lower-bound sensitivity models because they use long-term residential radon concentration rather than WLM with explicit time-since-exposure windows (Darby et al., 2005; Krewski et al., 2005).

The PUMA full model was implemented as a primary pooled-miner comparator because it is based on the largest pooled miner dataset (Kelly-Reif et al., 2023; Kreuzer et al., 2024).

PUMA- and BEIR VI-type models assign different weights to WLM accumulated in time-since-exposure windows and modify the ERR by attained age and exposure rate (Sommer et al., 2025). The general form is:

$$\text{ERR}(a) = \beta [W_{5-14}(a) + \theta_{15-24} W_{15-24}(a) + \theta_{25+} W_{25+}(a)] \phi_{age}(a) \gamma_z. \quad (\text{A8})$$

Here, $W_{5-14}(a)$, $W_{15-24}(a)$, and $W_{25+}(a)$ are cumulative WLM received 5–14, 15–24, and 25 or more years before attained age a , respectively. The θ terms weight older exposure windows, ϕ_{age} modifies risk by attained age, and γ_z modifies risk by exposure rate.

PUMA 1960+ was retained as an exploratory, low-exposure-rate sensitivity model because the post-1960 subcohort better approximates lower-rate, better-characterized, protracted occupational exposure than early high-exposure mining (Richardson et al., 2022). However, the published PUMA 1960+ formulation does not include a separate 35+ year exposure window and is therefore ambiguous for sustained from-birth domestic exposure histories extending beyond 34 years. For the exploratory continuation case, the final available older-exposure coefficient is applied to exposure older than 34 years. This continuation is not treated as an independently validated extension of the published PUMA 1960+ model; its outputs are therefore interpreted only as upper-bound sensitivity results. At age 60 under the 30 pCi/L exposure scenario, the resulting weighted term is 394.3. The corresponding exposed age-60 lung-cancer mortality rate is approximately 18.6 times baseline.

Baseline survival and lung-cancer hazard calculations

The model calculates four lifetime-risk measures following the Sommer et al. framework. Premodern competing mortality was represented by a Coale-Demeny West level 3 model life table (Coale et al., 2013). Annual survival was calculated as:

$$\tilde{S}_0(a) = \exp \left[- \sum_{u < a} q_0(u) \right]. \quad (\text{A9})$$

where $q_0(u)$ is the all-cause mortality hazard. This life table was selected to represent high premodern mortality and to prevent modern survival from inflating the number of people reaching late lung-cancer-prone ages. Archaeological demographic work indicates that prehistoric Southwestern populations had high mortality and low proportions surviving into older adulthood (Kohler and Reese, 2014).

Exposure-affected survival is:

$$\tilde{S}_E(a) = \tilde{S}_0(a) \exp \left[- \sum_{u < a} r_0(u) ERR(u) \right]. \quad (\text{A10})$$

The lifetime excess absolute risk (Sommer et al., 2025) is:

$$LEAR = \sum_a r_0(a) ERR(a) \tilde{S}_0(a). \quad (\text{A11})$$

The risk of exposure-induced death is:

$$REID = \sum_a r_0(a) ERR(a) \tilde{S}_E(a). \quad (\text{A12})$$

The excess lifetime risk is:

$$ELR = \sum_a r_0(a) [1 + ERR(a)] \tilde{S}_E(a) - \sum_a r_0(a) \tilde{S}_0(a). \quad (\text{A13})$$

The radiation-attributed decrease of survival is:

$$RADS = 1 - \exp \left[- \sum_a r_0(a) ERR(a) \right]. \quad (\text{A14})$$

Sommer et al. define REID, ELR, and RADS in relation to baseline and exposed survival and show that ELR, REID, and LEAR are generally close at low-to-moderate exposure, while RADS is larger in most situations. In this Chaco model, REID is the primary lifetime-risk measure because it weights excess lung-cancer mortality by exposed survival. This is important when cumulative exposure is high enough

that exposure-affected survival is no longer negligible. LEAR is retained for comparison with [Sommer et al. \(2025\)](#) and the PUMA dose-conversion literature.

A1. Premodern survival and Chaco baseline lung-cancer hazard

The realized Chaco burden was calculated with a premodern competing-risks survival structure. Baseline survival is:

$$\tilde{S}_{Chaco}(a) = \exp \left[- \sum_{u < a} q_{Chaco}(u) \right]. \quad (A15)$$

The all-cause mortality curve $q_{Chaco}(u)$ was represented by a Coale-Demeny West level 3 life-table structure, with life expectancy at birth of approximately 22 years and only a small fraction of births surviving into the highest lung-cancer-risk ages.

The Chaco baseline lung-cancer hazard was defined as:

$$r_0^{Chaco}(a) = f_{LC} r_0^{light}(a). \quad (A16)$$

where $r_0^{light}(a)$ is the reference light-smoker age-specific lung-cancer mortality curve and f_{LC} scales its absolute magnitude for a non-cigarette premodern population with biomass-smoke and dust exposure. The light-smoker curve is used as an age-specific hazard-shape reference and is not intended to imply cigarette smoking in the Chaco population. A modern never-smoker curve would likewise not constitute a directly measured premodern Chaco baseline; consequently, uncertainty in the absolute baseline hazard is represented explicitly through f_{LC} . The reference value is $f_{LC} = 0.20$. This is a model assumption, not a directly measured Chaco disease rate, and it is varied in sensitivity analysis because the absolute population burden is approximately proportional to f_{LC} . Because the radon excess is multiplicative on the baseline lung-cancer rate, increasing f_{LC} approximately proportionally increases realized excess deaths, all else equal.

A2. Age-specific radon-attributable death density

The age-specific radon-attributable lung-cancer death density per birth was:

$$e(a) = \tilde{S}_E(a) r_0^{Chaco}(a) ERR(a). \quad (A17)$$

Here, $e(a)$ is the probability that a person born into the modeled exposure regime dies of radon-induced lung cancer at age a . The per-birth risk of exposure-induced death is:

$$REID_{birth} = \sum_{a=0}^{94} e(a). \quad (A18)$$

The mean age at radon-induced death is:

$$\bar{A} = \frac{\sum_a a e(a)}{\sum_a e(a)}. \quad (A19)$$

The earliest lower-tail onset age was calculated from the lower percentile of the normalized distribution $e(a)/\sum e(a)$. Calendar-year radon-attributable deaths were calculated by convolving annual births with the age-specific death density:

$$D(y) = \sum_{a=0}^{94} B(y-a) e(a). \quad (A20)$$

Here, $D(y)$ is radon-attributable lung-cancer deaths in calendar year y , $B(y-a)$ is the number of births a years earlier, and $e(a)$ is the probability of radon-induced death at age a . This convolution places deaths

in their proper calendar year and naturally produces a delayed mortality peak after the population peak. Decade-scale burden is:

$$D_{decade}(Y) = \sum_{y=Y}^{Y+9} D(y), \quad Y \in \{850, 860, \dots, 1140\}. \quad (\text{A21})$$

The standing-population death rate is:

$$\rho(y) = \frac{1000D(y)}{P(y)}. \quad (\text{A22})$$

The total Bonito-phase burden is:

$$D_{total} = \sum_{850 \leq y \leq 1150} D(y). \quad (\text{A23})$$

Because $e(a)$ peaks in later adulthood, $D(y)$ is expected to lag the birth and population curves by several decades. The model reports both deaths occurring within AD 850–1150 and full lifetime deaths among cohorts born within that interval. These differ because cohorts born late in the Chaco occupation window may experience radon-attributable mortality after AD 1150.

A3. High-equilibrium subgroups

The reference model uses $F = 0.70$. Some microenvironments may approach secular equilibrium, $F \rightarrow 1$, especially dust- and smoke-saturated kivas or construction contexts. Since WL is linear in F , and the reference Chaco exposure is in the low-WL category where $\gamma_z = 1$, the exposure and most risk outputs scale approximately with F . The exposure scaling is:

$$\text{WLM}_{\text{yr}}(F) = \text{WLM}_{\text{yr}}(0.70) \left(\frac{F}{0.70} \right). \quad (\text{A24})$$

For $C_0 = 30$ pCi/L and $F = 1.0$, $\text{WLM}_{\text{yr}} = 11.63 \text{ WLM yr}^{-1}$. Subsequently, cumulative exposure to age 60 becomes $W(60) = 11.63 \times 60 = 698 \text{ WLM}$.

This is toward the upper range of uranium-miner cumulative exposure. Under PUMA 1960+, the conditional risk for such a subgroup reaches approximately 45%, while under BEIR VI it reaches approximately 36%. These values are not canyon-wide estimates; they represent high-equilibrium subgroup envelopes.

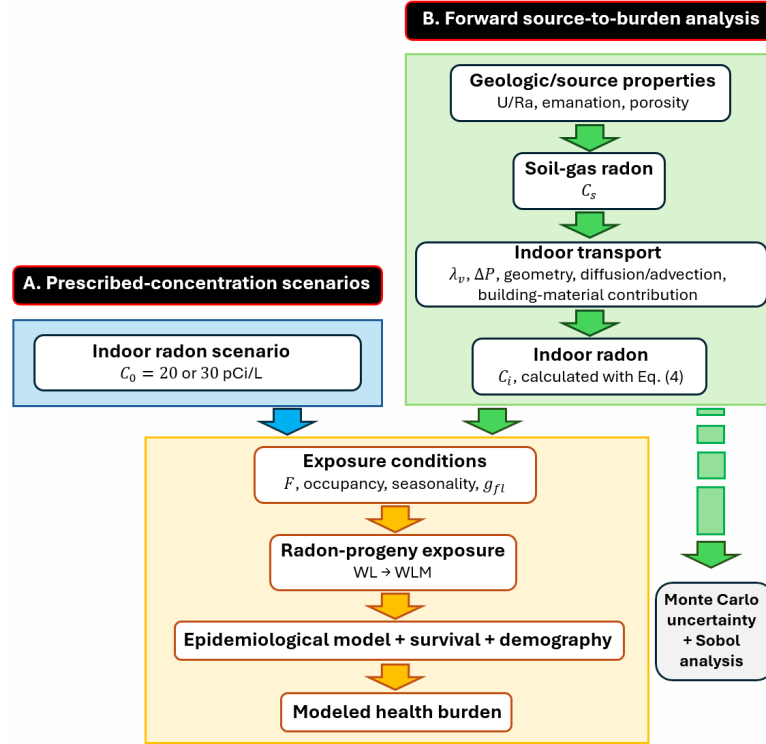


Figure A2: Dependency structure of the prescribed-concentration and forward source-to-burden calculations. (A) The prescribed-concentration analysis evaluates exposure and epidemiological outcomes conditionally at fixed indoor radon concentrations of 20 and 30 pCi/L. (B) In the forward analysis, indoor radon concentration C_i is calculated from geologic source and indoor transport parameters using the steady-state mass-balance model in Eq. (4). Indoor concentration and its physical determinants are therefore not independently sampled within the same calculation. Both pathways subsequently use the same exposure, epidemiological, survival, and demographic calculations to estimate modeled health burden. Monte Carlo uncertainty propagation and Sobol sensitivity analysis use the forward pathway only.

A4. Uncertainty propagation

Uncertainty was propagated following the Sommer uncertainty framework. Sommer et al. describe lifetime risk as a composite quantity and use Monte Carlo methods to propagate uncertainty in risk-model parameters and mortality rates. Their Approximate Normality Assumption represents risk-model parameter estimates as:

$$\hat{\Theta} \sim \mathcal{N}(\hat{\Theta}_0, \hat{\Sigma}_0), \quad (\text{A25})$$

Here, Θ_0 is the maximum-likelihood estimate and Σ_0 is the covariance matrix. Sommer et al. report that risk-model parameter uncertainty is the largest contributor to lifetime-risk uncertainty, while baseline lung-cancer mortality uncertainty is also substantial.

For the Chaco implementation, because ERR is approximately linear in the leading exposure coefficient in the low-WL regime, the reduced-ANA implementation samples:

$$\beta^{(j)} = \hat{\beta} \exp[Z_j \ln(\text{GSD})], \quad Z_j \sim \mathcal{N}(0, 1). \quad (\text{A26})$$

Here, $\beta^{(j)}$ is the sampled coefficient in Monte Carlo draw j , $\hat{\beta}$ is the published central value, and GSD is the model-specific geometric standard deviation inferred from published uncertainty intervals. This reduced-ANA method propagates uncertainty in the leading slope but does not fully propagate covariance among all effect modifiers; it is therefore a practical uncertainty approximation, not a full covariance reconstruction for every model.

Numerical uncertainty propagation and variance-based sensitivity analysis were performed in Python 3.10.5 (Python Software Foundation, 2022) using NumPy 2.1.2 (Harris et al., 2020) and SciPy 1.14.1 (Virtanen et al., 2020). Sobol sampling used two independent $n \times k$ base matrices A and B together with k cross-matrices AB_j , with $n = 2000$ and $k = 11$, resulting in 26,000 model evaluations per risk model. First-order Sobol indices were calculated using the Saltelli et al. (2010) estimator and total-order indices using the Jansen estimator (Jansen, 1999). Sampling was implemented directly in NumPy using random seed 20260611.

In the forward uncertainty calculation, indoor radon concentration C_i was calculated from soil-gas radon C_s , ventilation λ_v , pressure differential ΔP , and the remaining physical terms in Eq. (4) rather than sampled independently. The uncertainty distribution of the leading epidemiological coefficient β was conditional on the sampled risk model. All other uncertain factors were sampled independently in the principal analysis. A separate calculation evaluated a positive dependence between C_i and F using $\rho = 0.5$.

In the forward Monte Carlo simulation, C_s , F , g_{fl} , λ_v , ΔP , f_{LC} , b , $P(y)$, risk-model choice, and β were varied; for each realization, C_i was calculated from the steady-state mass-balance solution in Eq. (4) rather than sampled independently. The fixed $C_i = 20$ and 30 pCi/L exposure scenarios were evaluated separately and were not part of this joint source-parameter sampling.

The forward uncertainty analysis used 11 uncertain factors. Risk-model choice was sampled as an equal-weight categorical factor across the seven implementations. Population was represented by triangular interpolation across the person-year totals of the low, reference, and upper-bound trajectories. The crude birth rate was sampled as triangular (0.040, 0.049, 0.055), soil-gas radon as lognormal with median 40,000 Bq m⁻³ and GSD 1.6, ventilation as triangular (0.10, 0.30, 0.80) h⁻¹, ΔP as triangular (1.0, 3.6, 10.0) Pa, F as triangular (0.40, 0.70, 1.00), g_{fl} as triangular (1.00, 1.30, 1.60), f_{LC} as triangular (0.10, 0.20, 0.35), and the occupancy multiplier as triangular (1.00, 1.00, 1.40). The leading epidemiological coefficient β was sampled lognormally using model-specific GSDs of 1.36, 1.25, 1.30, 1.35, 1.25, 1.42, and 1.35 for PUMA 1960+, PUMA full, BEIR VI, Joint Czech–French, Wismut full, Wismut 1960+, and adjusted Jacobi, respectively.

The uncertainty interval was defined as:

$$95\% \text{ UI} = [Q_{0.025}, Q_{0.975}], \quad (\text{A27})$$

Here, $Q_{0.025}$ and $Q_{0.975}$ are the 2.5th and 97.5th percentiles of the simulated burden distribution. Variance-based global sensitivity was summarized using first-order and total-order Sobol indices:

$$S_i = \frac{\text{Var}_{X_i}(E[Y|X_i])}{\text{Var}(Y)}, \quad (\text{A28})$$

and:

$$S_{T_i} = 1 - \frac{\text{Var}_{X_{-i}}(E[Y|X_{-i}])}{\text{Var}(Y)}. \quad (\text{A29})$$

The quantities S_i and S_{T_i} measure the independent and total contributions of parameter i , respectively. This analysis is important because model burden is approximately multiplicative in exposure, population size, risk coefficient, and baseline lung-cancer hazard.

In summary, the model used in this study is an integrated framework consisting of the Park indoor-radon mass balance, the Sommer/PUMA lifetime-risk engine, a multi-model epidemiological ensemble, Chaco sustained from-birth exposure, dust-attached progeny correction, premodern survival, and birth-cohort convolution. The principal empirical limitation is the absence of direct Chaco Canyon measurements for soil-gas radon, room-specific indoor radon, equilibrium factor under hearth smoke and dust, radon exhalation from local masonry and fill, and ventilation rates in reconstructed or experimental Chacoan

rooms. The model should therefore be interpreted as a testable exposure-and-risk calculation against which future empirical data from Chaco Canyon can be evaluated, not as definitive proof of causation.

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