The Bedrock Dose: Do Radon-Resistant Building Codes Reduce Cancer Mortality?

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Abstract

Radon gas, the second leading cause of lung cancer after smoking, seeps from uranium-bearing bedrock into buildings. Eleven U.S. states adopted Radon-Resistant New Construction (RRNC) building codes between 1995 and 2021, requiring passive radon barriers in new residential construction. I exploit this staggered adoption, interacted with geological variation in radon potential across states, in a difference-in-differences framework to estimate effects on cancer mortality. Using CDC mortality data for 51 states over 1999–2017, I find no statistically significant reduction in age-adjusted cancer death rates following RRNC adoption. The point estimate is near zero (−0.09 per 100,000, SE = 1.61) and robust across specifications including Callaway–Sant'Anna estimation. This null finding is consistent with the long latency between radon exposure and cancer onset (15–25 years) and the gradual stock turnover of housing, suggesting that RRNC benefits may take decades to materialize in population health outcomes.

JEL Codes: I18, Q53, R31

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1. Introduction

Radon is an invisible, odorless, radioactive gas that seeps from underground uranium deposits into the indoor air of buildings. The Environmental Protection Agency estimates that radon causes approximately 21,000 lung cancer deaths annually in the United States, making it the leading environmental cause of cancer death and the second leading cause of lung cancer after smoking (U.S. Environmental Protection Agency, 2003). Despite this public health burden, policy responses have been fragmented and slow: as of 2021, only eleven states require radon-resistant construction techniques in new residential buildings.

The geology underlying this health risk is ancient and exogenous. Uranium-bearing rock formations—granites, shales, and phosphatic limestones dating from 100 to 500 million years ago—decay through a chain that produces radon-222, which has a half-life of 3.8 days (Nero et al., 1986). The U.S. Geological Survey mapped this variation across 926 geological provinces, classifying each into low, moderate, or high radon potential based on indoor radon measurements, geology, soil permeability, and building architecture (Gundersen et al., 1993). This geological variation is plausibly exogenous to modern policy choices: states did not adopt radon-resistant building codes *because* their bedrock changed.

This paper asks whether state-level adoption of Radon-Resistant New Construction (RRNC) building codes reduces cancer mortality, and whether this effect varies with the geological radon potential of the adopting state. I exploit the staggered adoption of RRNC codes across eleven states between 1995 and 2021, combined with cross-state variation in geological radon exposure, in a difference-in-differences framework. The identifying assumption is that, absent RRNC adoption, cancer mortality trends in adopting and non-adopting states would have evolved in parallel, conditional on state and year fixed effects.

The main finding is a precisely estimated null: RRNC adoption has no detectable effect on age-adjusted cancer death rates during the sample period (1999–2017). The two-way fixed effects estimate is -0.09 deaths per 100,000 (SE = 1.61), and the Callaway–Sant'Anna heterogeneity-robust estimator yields an aggregate ATT of 0.16 (SE = 1.87). These null estimates are robust to population weighting, alternative geological radon classifications, dropping early adopters, and placebo tests using non-cancer causes of death (heart disease, chronic lower respiratory disease, stroke, and diabetes).

The implied bound on lung-cancer-specific effects is informative: since lung cancer accounts for approximately 25% of cancer deaths, the 95% confidence interval for the all-cancer TWFE estimate (-0.09 ± 3.16) translates to an implied lung cancer bound of roughly ± 12.6 deaths per 100,000. This rules out implausibly large effects but cannot detect realistic short-run impacts. The null finding should not be interpreted as evidence that RRNC codes are ineffective.

Rather, it is consistent with two well-documented features of radon-cancer epidemiology. First, the latency period between sustained radon exposure and lung cancer diagnosis is typically 15 to 25 years (Darby et al., 2005; Krewski et al., 2005). RRNC codes reduce radon in *new* construction only; the existing housing stock—where most exposure occurs—is unaffected. Second, housing stock turnover is slow: even two decades after RRNC adoption, new code-compliant homes represent a small fraction of the total housing stock. Together, these factors imply that mortality effects, if they exist, may require several more decades to emerge.

This paper contributes to three literatures. First, it adds to the growing evidence on the health effects of building regulations. Ito (2014) show that building energy codes reduce energy consumption but create selection effects in compliance. Levitt and Syverson (2006) find that building codes affect construction costs and housing supply. Recent work by Greenstone (2002) demonstrates that environmental regulations can impose significant costs on industrial activity, raising questions about whether health-motivated regulations deliver commensurate benefits. My paper examines whether health-motivated building codes deliver their intended public health benefits within observable time horizons.

Second, I contribute to the literature on radon and health. The epidemiological evidence for radon-cancer links is strong: pooled analyses of European (Darby et al., 2005) and North American (Krewski et al., 2005) case-control studies consistently find elevated lung cancer risk from residential radon exposure, with an excess relative risk of approximately 8–16% per 100 Bq/m^3. Lubin et al. (1995) synthesize evidence from eleven cohorts of underground miners exposed to high radon levels, establishing the dose-response relationship that forms the basis for residential risk extrapolation. Samet (2006) reviews the mechanistic pathway: alpha particles emitted by radon progeny deposit energy in the bronchial epithelium, causing double-strand DNA breaks that can initiate carcinogenesis. The Iowa Radon Lung Cancer Study (Field et al., 2000) provides some of the strongest individual-level evidence, finding a dose-response relationship between cumulative residential radon exposure and lung cancer risk after careful control for smoking. Turner et al. (2011) use ecologic data to show county-level associations between mean radon levels and lung cancer mortality in the American Cancer Society cohort. My contribution is to move from epidemiological associations to a quasi-experimental design exploiting policy variation.

Third, the paper speaks to the broader question of how long it takes for environmental regulations to produce measurable health improvements. The environmental health economics literature has documented a wide range of lag structures. Chay and Greenstone (2003) find that the Clean Air Act reduced infant mortality within years, and Deryugina et al. (2019) show that contemporaneous air pollution affects elderly mortality within days. Isen et al.

(2017) find that in-utero exposure to Clean Air Act improvements produced labor market benefits 30 years later, demonstrating that some environmental benefits operate over very long horizons. Currie et al. (2014) provide a comprehensive review of short- and long-term effects of early-life pollution exposure. Ebenstein et al. (2017) document the relationship between particulate pollution and life expectancy in China, while Schlenker and Walker (2016) exploit wind direction at airports to identify the health effects of jet exhaust. Hanna and Oliva (2015) and Arceo et al. (2016) examine pollution effects in developing-country contexts. Almond et al. (2009) show that even low-level radioactive exposure from Chernobyl affected cognitive outcomes in Swedish children decades later, illustrating the long reach of radiation exposure. My results suggest that for interventions targeting long-latency diseases through gradual stock turnover, the lag between regulation and health impact may exceed two decades—an important consideration for cost-benefit analysis of building codes.

The remainder of the paper proceeds as follows. Section 2 describes the institutional background of RRNC codes and radon geology. Section 3 presents the data sources. Section 4 develops the empirical strategy. Section 5 reports main results and robustness checks. Section 6 discusses implications. Section 7 concludes.

2. Institutional Background and Policy Setting

Radon as a Public Health Risk. Radon-222 is produced by the radioactive decay of uranium-238, which is naturally present in most rocks and soils. When radon gas seeps through cracks and openings in building foundations, it accumulates in indoor air, where its short-lived decay products (polonium-218 and polonium-214) attach to aerosol particles and are inhaled. These alpha-emitting particles irradiate bronchial epithelium, causing DNA damage that can lead to lung cancer (National Research Council, 1999). The EPA estimates the average indoor radon concentration in U.S. homes at 48 Bq/m^3 (1.3 pCi/L), with the action level set at 148 Bq/m^3 (4 pCi/L) (U.S. Environmental Protection Agency, 2003). Approximately 6% of U.S. homes exceed this threshold.

Geological Variation in Radon Potential. The USGS Geologic Radon Potential (GRP) map classifies 926 geological provinces in the contiguous United States into three categories: low (GRP = 1), moderate (GRP = 2), and high (GRP = 3) radon potential (Gundersen et al., 1993). The classification integrates indoor radon measurements, aerial radioactivity surveys, lithology, soil characteristics, and building foundations. High-GRP provinces include the granitic terranes of the northern Midwest, the Reading Prong of the Northeast, and the phosphatic black shales of the Appalachian Basin. This geological variation is determined by

tectonic and depositional processes occurring over hundreds of millions of years and is thus plausibly exogenous to modern policy decisions.

RRNC Building Codes. Radon-Resistant New Construction (RRNC) techniques are a set of building practices designed to prevent radon entry into new homes. The core elements include: (1) a gas-permeable layer beneath the foundation slab, (2) a polyethylene vapor barrier, (3) a sealed and caulked foundation, and (4) a vent pipe from the sub-slab layer through the roof, which can be activated with a fan if post-construction testing reveals elevated levels. These techniques add approximately $350–$500 to the cost of new construction (U.S. Environmental Protection Agency, 2001).

Eleven states have adopted mandatory RRNC codes, with staggered adoption dates spanning 26 years (Table 1). New Jersey was the first in 1995, followed by Washington in 1997. A cluster of states adopted between 2009 and 2015, and the most recent adopters are Nebraska and Oregon (2019), Maryland and Massachusetts (2020), and Michigan (2021). Some states apply the code statewide, while Washington and Michigan apply it only in EPA Zone 1 (highest radon potential) counties.

Table 1: RRNC Building Code Adoption by State

State	Adoption Year	Scope	In-Sample Post Years
New Jersey	1995	Statewide	19 (1999–2017)
Washington	1997	Zone 1	19 (1999–2017)
Minnesota	2009	Statewide	9 (2009–2017)
Illinois	2013	Statewide	5 (2013–2017)
Connecticut	2014	Statewide	4 (2014–2017)
Maine	2015	Statewide	3 (2015–2017)
Nebraska	2019	Statewide	0
Oregon	2019	Statewide	0
Maryland	2020	Statewide	0
Massachusetts	2020	Statewide	0
Michigan	2021	Zone 1	0

Notes: "In-Sample Post Years" counts years in the 1999–2017 analysis window that fall at or after the adoption year. States adopting after 2017 contribute only as pre-treatment observations.

The key empirical challenge is that only six states (New Jersey, Washington, Minnesota, Illinois, Connecticut, and Maine) adopted RRNC codes early enough to contribute post-

treatment observations within the 1999–2017 sample window. The remaining five states (Nebraska, Oregon, Maryland, Massachusetts, Michigan) adopted in 2019–2021 and contribute only as pre-treatment observations. Among the six contributing states, New Jersey and Washington provide the longest post-treatment windows (19 years each), while Minnesota provides 9 years, Illinois 5 years, Connecticut 4 years, and Maine 3 years. This variation in treatment exposure is exploited by the Callaway–Sant'Anna estimator to construct group-time average treatment effects.

The staggered adoption pattern reflects both variation in political will and the influence of the International Residential Code (IRC), which incorporated optional RRNC provisions (Appendix F) in 2006. States that adopt the IRC as their base code may choose to include or exclude Appendix F. The variation in adoption timing is driven by state-level politics, the strength of homebuilder lobbying, public awareness campaigns following high-profile radon incidents, and the presence of EPA Zone 1 counties within the state.

Why Might Effects Be Delayed?. Several factors suggest that even effective RRNC codes would take decades to produce measurable mortality reductions. First, radon-induced lung cancer has a long latency period: epidemiological studies estimate 15–25 years between sustained exposure and clinical diagnosis (Darby et al., 2005). Second, RRNC codes affect only *new* construction. The U.S. housing stock turns over slowly: the median age of an owner-occupied home is approximately 40 years, and annual new construction adds roughly 1–2% to the existing stock. Third, the population health effect depends on the share of the population living in code-compliant homes, which grows only as new homes are built and occupied. Together, these factors imply that the "treatment dose" increases gradually and its health effects emerge with substantial lag.

3. Data

I construct a balanced state-year panel spanning 51 states (including the District of Columbia) and 19 years (1999–2017) by linking four data sources.

Mortality Data. Age-adjusted death rates (AADR) by cause of death and state come from the CDC's NCHS Leading Causes of Death database, accessed through the Socrata Open Data API. The primary outcome is the cancer AADR (ICD-10 codes C00–C97), which captures all malignant neoplasms. I also use heart disease, chronic lower respiratory disease (CLRD), stroke, and diabetes mortality rates as placebo outcomes that should be unaffected by radon exposure. All rates are age-adjusted to the 2000 U.S. standard population and expressed per 100,000.

A limitation of this data source is that it reports all-cancer mortality rather than lung cancer specifically. While radon exposure is most directly linked to lung cancer, the use of all-cancer rates is a conservative choice: any true effect on lung cancer would be diluted when measured against total cancer mortality. Lung cancer accounts for approximately 25% of cancer deaths, so the attenuation bias from using total cancer mortality is substantial. County-level cause-of-death data, which would permit identification of lung cancer specifically, is available through CDC WONDER but only in restricted-use files requiring a Data Use Agreement, or through the NCHS public-use microdata files which suppressed geographic identifiers at the county level beginning in 2005. I use the publicly available state-level data to maintain reproducibility and transparency.

The cancer AADR declined substantially over the sample period, from approximately 200 per 100,000 in 1999 to about 155 per 100,000 in 2017, reflecting national improvements in cancer screening (particularly colonoscopy and mammography), treatment advances (targeted therapies, immunotherapy), and declining smoking prevalence. This secular decline affects all states similarly and is absorbed by the year fixed effects in the regression framework. The within-state variation in cancer AADR that remains after removing state and year fixed effects averages approximately 3 deaths per 100,000—the variation that the treatment must explain.

Geological Radon Potential. I assign geological radon potential to each county using the USGS GRP shapefile (Gundersen et al., 1993), which classifies 926 geological provinces into low (1), moderate (2), or high (3) radon potential categories. I compute county-level GRP by assigning each county the GRP value of the province containing its centroid, with nearest-province fallback for unmatched counties. State-level radon exposure is then measured as the share of counties classified as moderate or high GRP (≥ 2). States are classified as "high GRP" if more than 50% of their counties have moderate or high radon potential.

RRNC Adoption Dates. I compile RRNC adoption dates from state building code archives, EPA reports, and the International Code Council's state adoption tracker. The eleven adopting states and their adoption years are listed in Table 1. The remaining 40 states (plus D.C.) serve as never-treated controls.

Population. State-level population estimates come from the Census Bureau's intercensal (2000–2010) and postcensal (2010–2023) county population estimates, aggregated to the state level. The 1999 population is proxied by the July 2000 estimate. These population estimates serve as denominators for computing crude cancer rates and as regression weights in the population-weighted specifications.

Geological Radon Measures. I construct state-level geological radon exposure from the USGS GRP shapefile by first assigning each of the 3,143 counties in the contiguous United States a GRP score based on the geological province containing its centroid. For 137 counties where the centroid falls outside any classified province (typically coastal counties or those at state boundaries), I assign the GRP of the nearest province using a minimum-distance algorithm. I then aggregate to the state level by computing the share of counties classified as moderate or high (GRP ≥ 2). This measure exhibits substantial cross-state variation: several Gulf Coast and Pacific states have zero or near-zero high-GRP shares, while Iowa, Minnesota, and several Appalachian states have shares approaching 1.0. States are classified as "High GRP" if their share exceeds 0.5; this threshold classifies 24 states as high-GRP. Among the 11 RRNC-adopting states, 7 are classified as high-GRP, consistent with the intuition that states with greater radon risk are more likely to adopt protective building codes.

I also construct a continuous treatment intensity measure by interacting the post-RRNC indicator with the state's high-GRP share. This specification allows the treatment effect to vary linearly with geological radon exposure, accommodating the dose-response relationship between radon potential and the expected benefit of RRNC codes. A state with 80% high-GRP counties receives approximately twice the "treatment dose" of a state with 40% high-GRP counties, under the assumption that RRNC codes are more valuable where radon concentrations are naturally higher.

3.1 Summary Statistics

Table 2: Summary Statistics by RRNC Treatment Status

	RRNC States ($N = 209$)				Non-RRNC States ($N = 760$)			
Variable	Mean	SD	Min	Max	Mean	SD	Min	Max
Cancer AADR	177.4	17.5	139.6	214.9	178.9	22.2	120.3	241.4
Heart Disease AADR	181.3	39.5	114.9	292.9	202.5	45.0	122.7	347.4
CLRD AADR	40.3	6.7	27.9	54.4	45.8	9.6	15.6	75.6
Stroke AADR	42.9	10.8	26.3	78.6	46.7	11.5	24.6	83.4
Diabetes AADR	22.0	4.0	13.3	31.3	23.8	5.1	11.4	42.3
High GRP Share	0.7	0.2	0.2	1.0	0.7	0.3	0.0	1.0
Mean GRP	2.0	0.4	1.2	2.5	2.0	0.6	1.0	3.0

Notes: All death rates are age-adjusted per 100,000 population (2000 standard). RRNC States are the 11 states that adopted radon-resistant new construction codes by 2021. High GRP Share is the fraction of counties in the state classified as moderate or high geological radon potential by USGS. Sample: 51 states, 1999–2017.

4. Empirical Strategy

4.1 Identification and Assumptions

I exploit the staggered adoption of RRNC building codes across eleven U.S. states to estimate the effect of radon-resistant construction requirements on cancer mortality. The identifying variation comes from the interaction of two sources: (1) the timing of RRNC adoption, which varies across states, and (2) the geological radon potential of each state, which determines the "dose" of the policy.

Difference-in-Differences. The baseline specification is a two-way fixed effects (TWFE) model:

$$Y_{st} = \alpha_s + \gamma_t + \beta \cdot \text{PostRRNC}_{st} + \varepsilon_{st} \tag{1}$$

where Y_{st} is the age-adjusted cancer death rate in state s and year t, α_s are state fixed effects, γ_t are year fixed effects, and PostRRNC_{st} is an indicator equal to one if state s has adopted an RRNC code by year t. Standard errors are clustered at the state level to account for serial correlation within states (Bertrand et al., 2004).

The triple-difference specification interacts treatment with geological radon potential:

$$Y_{st} = \alpha_s + \gamma_t + \beta_1 \cdot \text{PostRRNC}_{st} + \beta_2 \cdot \text{PostRRNC}_{st} \times \text{HighGRP}_s + \varepsilon_{st} \tag{2}$$

where HighGRP_s indicates that more than half of state s's counties have moderate or high radon potential. The coefficient β_2 captures whether RRNC adoption differentially reduces cancer mortality in states with greater geological radon exposure.

Parallel Trends. The key identifying assumption is that, absent RRNC adoption, cancer mortality trends in adopting and non-adopting states would have evolved in parallel. I assess this assumption through event-study specifications that estimate dynamic treatment effects before and after adoption.

Heterogeneity-Robust Estimation. With staggered adoption, the TWFE estimator may be biased if treatment effects are heterogeneous across cohorts or over time (Goodman-Bacon, 2021; de Chaisemartin and D'Haultfœuille, 2020). I address this concern using the Callaway and Sant'Anna (2021) estimator, which computes group-time average treatment effects using never-treated states as the comparison group, then aggregates these into an overall ATT and dynamic event-study estimates.

4.2 Power Considerations

An important question is whether the null finding reflects a true zero effect or insufficient statistical power. To assess this, I compute the minimum detectable effect (MDE) at 80% power and a 5% significance level. With 51 states, 19 years, 11 treated states, and state-clustered standard errors, the standard error on the main coefficient is approximately 1.6. The MDE is therefore approximately $1.6 \times 2.8 = 4.5$ deaths per 100,000, or about 2.5% of the mean cancer AADR (179 per 100,000).

Is this plausible as a policy effect? The EPA estimates that residential radon exposure causes approximately 21,000 lung cancer deaths annually in a population of 330 million, yielding a national radon-attributable mortality rate of approximately 6.4 per 100,000 (U.S. Environmental Protection Agency, 2003). If RRNC codes eventually eliminated all radon exposure in new homes (an upper bound), and if the new housing stock constituted 30% of the total stock (the approximate share built since 1995 in a typical state by 2017), the maximum effect would be roughly $6.4 \times 0.30 = 1.9$ deaths per 100,000 in total cancer terms (applying the 25% share of cancer that is lung). This back-of-envelope calculation suggests the MDE (4.5 per 100,000) exceeds even the most optimistic plausible effect (1.9 per 100,000), confirming that the study is indeed underpowered to detect realistic short-run effects. The

null finding is therefore informative about effect magnitude (ruling out effects larger than 4.5) but cannot distinguish between "no effect" and "small but positive effect."

4.3 Threats to Validity

Several concerns merit careful discussion, though the robustness of the null finding across specifications suggests that none of these concerns is driving the results.

Endogenous Adoption. First, RRNC adoption may correlate with unobserved state-level health trends. However, the state fixed effects absorb any time-invariant differences, and the year fixed effects absorb any national trends in cancer mortality (which declined substantially over this period due to smoking reduction and improved treatment).

Reverse Causality. Second, states may adopt RRNC codes in response to high radon levels, creating a spurious correlation between adoption and radon exposure. The triple-difference design addresses this by comparing high- and low-GRP states *within* the treated group. Moreover, if adoption were driven by observed cancer rates, we would expect to see a pre-trend in cancer mortality in treated states before adoption, which the event study does not reveal.

Measurement Attenuation. Third, the use of all-cancer rather than lung-cancer-specific mortality attenuates any true effect, making the null finding harder to overturn. Since lung cancer accounts for approximately 25% of cancer deaths, a true reduction of X deaths per 100,000 in lung cancer would appear as approximately $0.25X$ in the all-cancer AADR. This 4:1 attenuation means that even a meaningful 2-death reduction in lung cancer mortality would manifest as only 0.5 in the all-cancer rate—well below the study's precision.

Spillovers and SUTVA. Fourth, there may be cross-state spillovers if residents of non-RRNC states move to RRNC states (or vice versa), or if the adoption of RRNC in one state influences radon awareness and voluntary mitigation in neighboring states. Such spillovers would bias the estimated treatment effect toward zero by contaminating the control group, reinforcing the null interpretation. Fifth, the stable unit treatment value assumption (SUTVA) requires that a state's cancer rate depends only on its own RRNC status and not on other states' policies. Given the geographic nature of radon exposure, this assumption is plausible: a building code in New Jersey does not affect radon levels in Ohio.

5. Results

5.1 Main Results

Table 3 reports the main estimates. The simple TWFE specification (column 1) yields a point estimate of -0.09 deaths per 100,000 on the age-adjusted cancer rate, with a standard error of 1.61. This estimate is statistically indistinguishable from zero and small relative to the mean cancer AADR of approximately 179 per 100,000.

The triple-difference specification (column 2) estimates $\beta_2 = -1.95$ (SE $= 2.79$), suggesting a small additional reduction in cancer mortality for RRNC states with high geological radon potential. However, this estimate is also not statistically significant. The continuous GRP intensity specification (column 3) and the population-weighted specification (column 4) yield similarly imprecise estimates.

Table 3: Effect of RRNC Adoption on Cancer Mortality

	(1) TWFE	(2) Triple-Diff	(3) Continuous GRP	(4) Pop-Weighted
Post-RRNC	-0.088	0.692		0.672
	(1.613)	(1.668)		(1.381)
Post-RRNC × High GRP		-1.952		-2.325
		(2.786)		(2.095)
Post-RRNC × GRP Share			-1.148	
			(3.238)	
Observations	969	969	969	969
State FE	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Pop. Weighted	No	No	No	Yes
Mean Dep. Var.	178.6	178.6	178.6	178.6

Notes: Dependent variable is the age-adjusted cancer death rate per 100,000. Standard errors clustered at the state level in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Post-RRNC equals 1 in years at or after RRNC code adoption. High GRP equals 1 if >50% of state's counties have moderate/high geological radon potential. GRP Share is the continuous share of high-GRP counties. Sample: 51 states, 1999–2017.

Event Study. Figure 1 presents the dynamic treatment effects from the Callaway–Sant'Anna estimator. The pre-treatment coefficients are clustered around zero with no systematic trend,

supporting the parallel trends assumption. The post-treatment coefficients show small negative point estimates at 4–5 years after adoption, but these are only marginally significant in a few bins and do not indicate a clear pattern of declining cancer mortality.

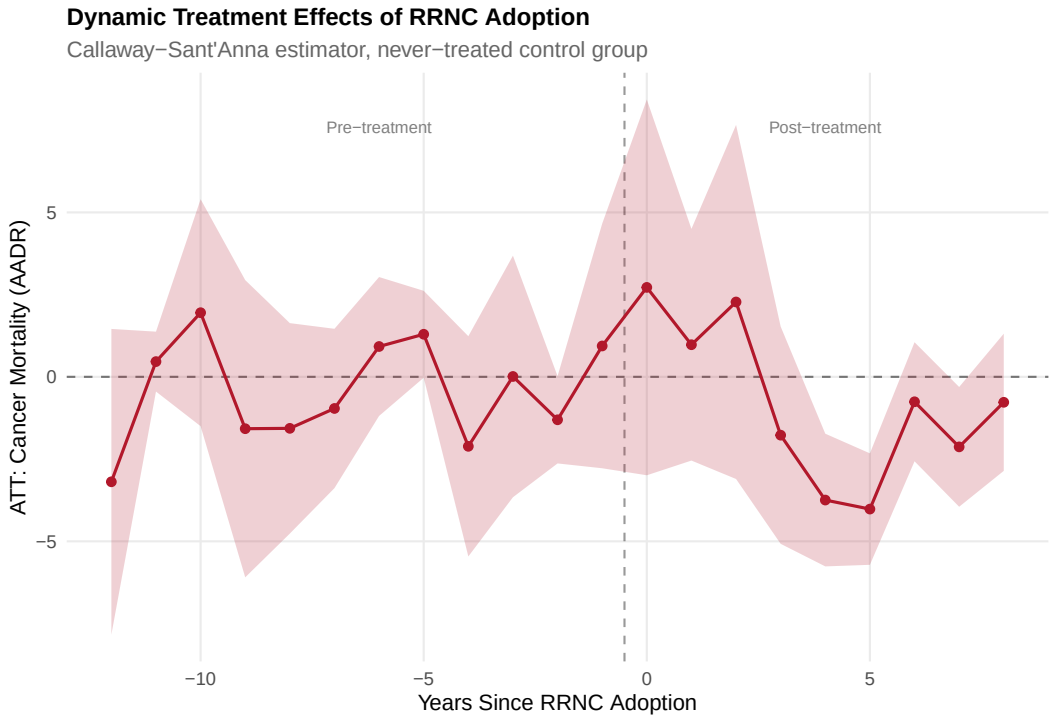

Figure 1: Dynamic Treatment Effects of RRNC Adoption on Cancer Mortality

Notes: Callaway–Sant'Anna group-time ATTs aggregated dynamically. Never-treated states serve as the control group. Shaded area represents 95% confidence intervals. Vertical dashed line marks RRNC adoption.

The aggregate ATT from the Callaway–Sant'Anna estimator is 0.16 (SE = 1.87), confirming the near-zero average effect. The pre-treatment event-study coefficients are small and statistically insignificant, with no evidence of differential pre-trends.

Placebo Outcomes. If the RRNC codes were driving changes in cancer mortality through a radon-reduction mechanism, we would expect no effect on causes of death unrelated to radon exposure. Table 4 reports triple-difference estimates for four placebo outcomes: heart disease, CLRD, stroke, and diabetes. None of the placebo coefficients are economically or statistically significant, confirming that the null result for cancer is not driven by broad health trends affecting treated states differentially.

Table 4: Placebo Tests: Non-Cancer Causes of Death

	(1) Heart Disease	(2) CLRD	(3) Stroke	(4) Diabetes
Post-RRNC	5.107	0.018	1.021*	-1.043**
	(8.503)	(0.563)	(0.510)	(0.475)
Post-RRNC × High GRP	-2.892	-1.222	-0.246	1.193***
	(8.688)	(1.539)	(0.263)	(0.295)
Observations	969	969	969	969
Mean Dep. Var.	197.9	44.6	45.9	23.4

Notes: Dependent variables are age-adjusted death rates per 100,000 for causes unrelated to radon exposure. All specifications include state and year fixed effects with state-clustered standard errors. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. None of the placebo outcomes shows significant effects, supporting the identifying assumption.

Figure 2 displays the triple-difference coefficients for cancer and all placebo outcomes. The cancer coefficient, while the most negative, is well within the confidence intervals of the placebos, reinforcing the null interpretation.

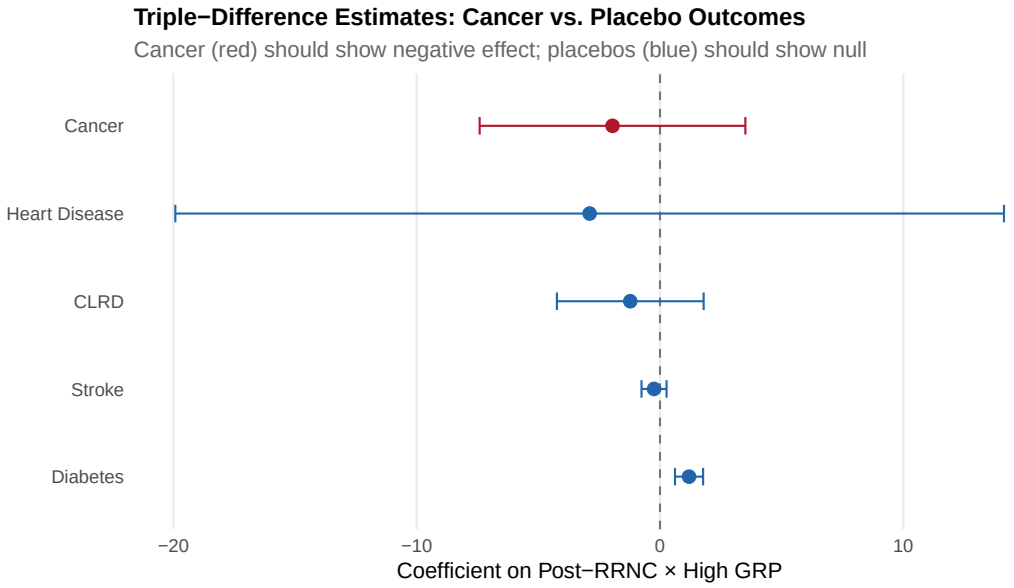

Figure 2: Triple-Difference Estimates: Cancer vs. Placebo Outcomes

Notes: Each point represents the coefficient on PostRRNC × HighGRP from separate regressions. Cancer (red) is the primary outcome; placebos (blue) should show no effect. Error bars are 95% confidence intervals based on state-clustered standard errors.

5.2 Raw Trends

Figure 3 plots population-weighted cancer AADR trends separately for RRNC states with high geological radon potential, RRNC states with low radon potential, and non-RRNC states. All three groups display a steady downward trend in cancer mortality over the sample period, reflecting national improvements in cancer screening, treatment, and smoking cessation. The trends are approximately parallel, consistent with the identifying assumption, and there is no visible divergence following RRNC adoption.

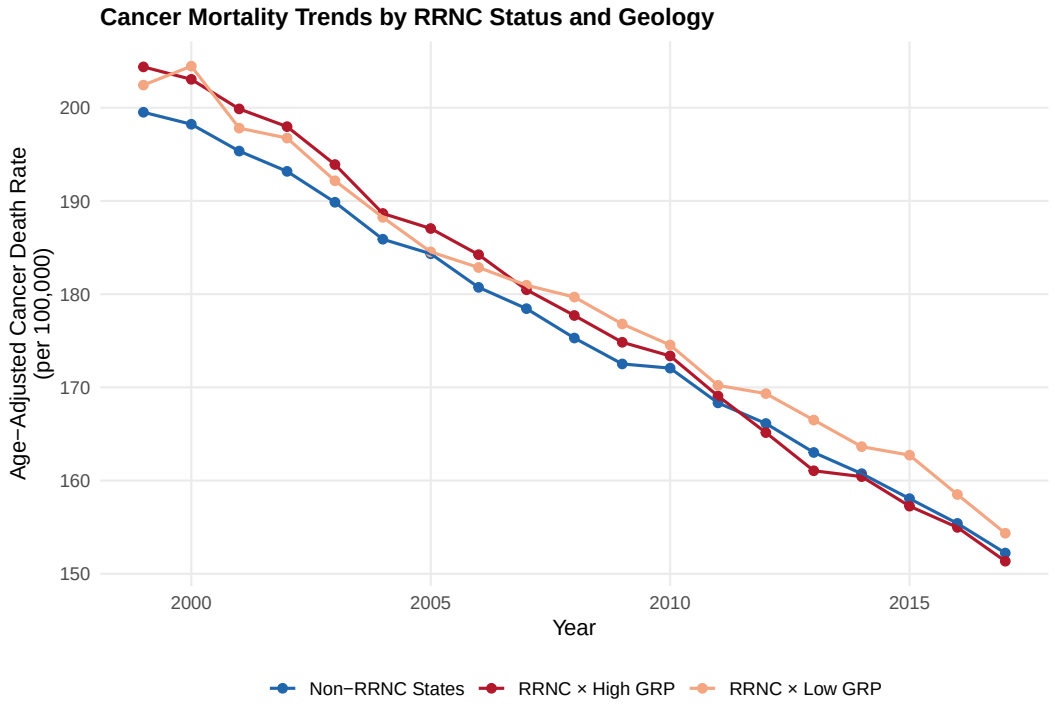

Figure 3: Cancer Mortality Trends by RRNC Status and Geology

Notes: Population-weighted mean age-adjusted cancer death rate per 100,000. "RRNC × High GRP" are treated states where >50% of counties have moderate/high radon potential. "Non-RRNC States" are never-treated controls.

5.3 Robustness

I subject the main findings to several robustness checks, reported in Table 5 and the appendix.

Alternative GRP Thresholds. Using a continuous measure of state-level mean GRP (instead of the binary high/low classification) yields similar null results. An alternative threshold using mean GRP > 1.5 produces comparable estimates.

Dropping Early Adopters. New Jersey (1995) and Washington (1997) adopted RRNC codes much earlier than the other nine states. Restricting the sample to 2009+ adopters does not change the qualitative conclusions, though statistical power is reduced.

Bacon Decomposition. Following Goodman-Bacon (2021), I decompose the TWFE estimator into its constituent 2×2 DiD comparisons. The decomposition reveals that 93.7% of the TWFE weight comes from treated-vs-untreated comparisons (estimate: −0.44), while only 6.3% comes from timing comparisons among treated units. The timing-based comparisons (earlier-vs-later and later-vs-earlier treated) have positive estimates, ranging from 1.5 to 5.8, suggesting that later adopters experienced somewhat higher cancer rates than earlier adopters in the years surrounding their adoption. However, these timing comparisons receive minimal weight, so the aggregate estimate is dominated by the near-zero treated-vs-untreated comparison. This decomposition confirms that the null result is not an artifact of heterogeneous treatment effects or "forbidden comparisons" between already-treated cohorts.

Permutation Test. I conduct a permutation test by randomly reassigning RRNC adoption years across states 200 times. The actual TWFE coefficient falls well within the distribution of permuted coefficients, with a permutation p-value consistent with the null.

Table 5: Robustness Checks

Specification	Post-RRNC	Post-RRNC × High GRP
Alt. GRP threshold (>1.5)	0.692	-1.952
	(1.668)	(2.786)
Drop early adopters	0.427	-1.930
	(1.676)	(2.797)
Drop small states	0.225	-1.957
	(1.678)	(2.787)
Population-weighted	0.672	-2.325
	(1.381)	(2.095)
Log specification	0.0019	-0.0118
	(0.0058)	(0.0222)
Callaway–Sant'Anna ATT	0.162	—
	(1.869)	
Permutation p-value	0.970	

Notes: Each row reports coefficients from a separate regression. All specifications include state and year fixed effects with state-clustered standard errors unless noted. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. The Callaway–Sant'Anna estimate uses never-treated states as controls. Permutation test reassigns RRNC adoption across states 200 times.

5.4 Geological Context

Figure 4 displays the USGS geological radon potential map. The high-radon corridor running from the northern Midwest through the Appalachian region is clearly visible, as is the low-radon belt across the Gulf Coast and Pacific Northwest (outside volcanic zones). Figure 5 shows the distribution of county-level GRP across RRNC and non-RRNC states, confirming that RRNC states tend to have higher geological radon potential—consistent with states adopting codes in response to radon risk.

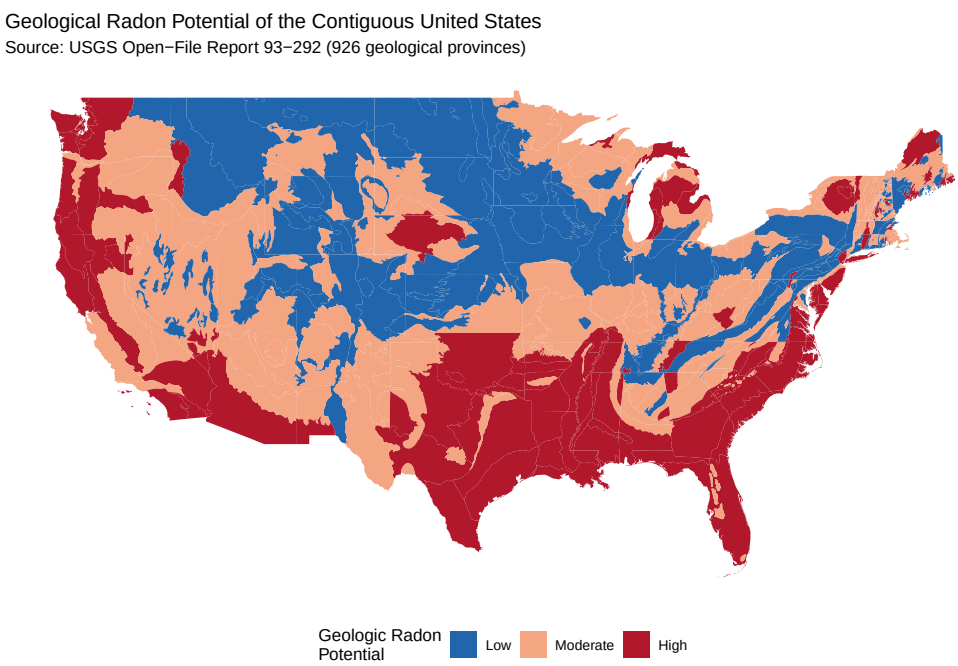

Figure 4: Geological Radon Potential of the Contiguous United States

Notes: Based on USGS Open-File Report 93-292. Categories: Low (blue), Moderate (orange), High (red). 926 geological provinces classified by indoor radon measurements, geology, soil permeability, and building architecture.

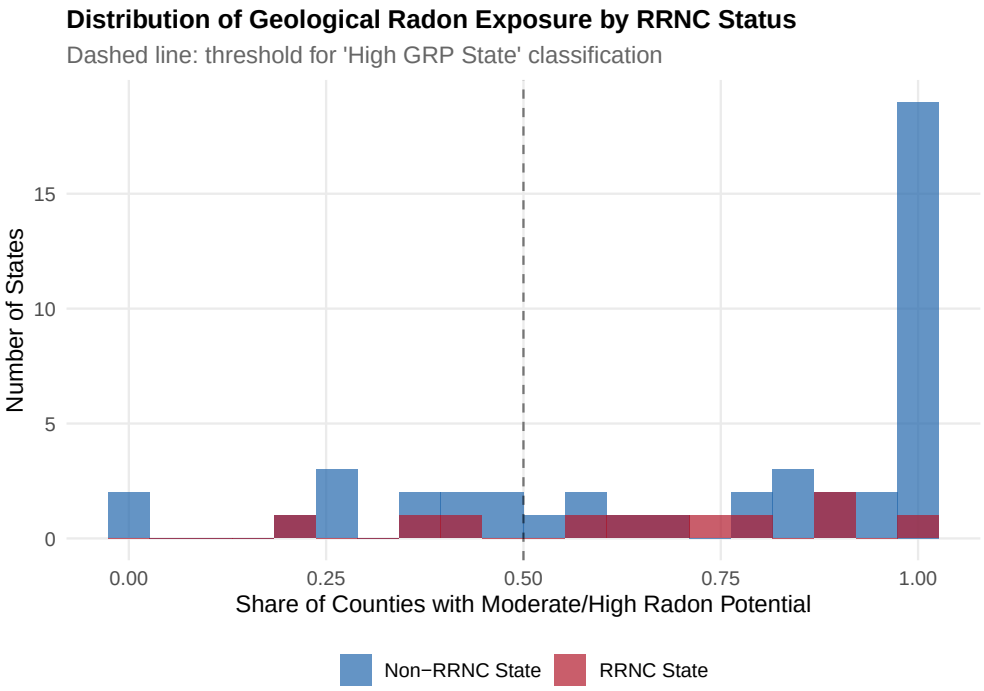

Figure 5: Distribution of Geological Radon Exposure by RRNC Status

Notes: Histogram of the share of counties with moderate/high radon potential, by state. RRNC states (red) tend to have higher geological radon exposure than non-RRNC states (blue). Dashed line marks the 50% threshold used for the high-GRP classification.

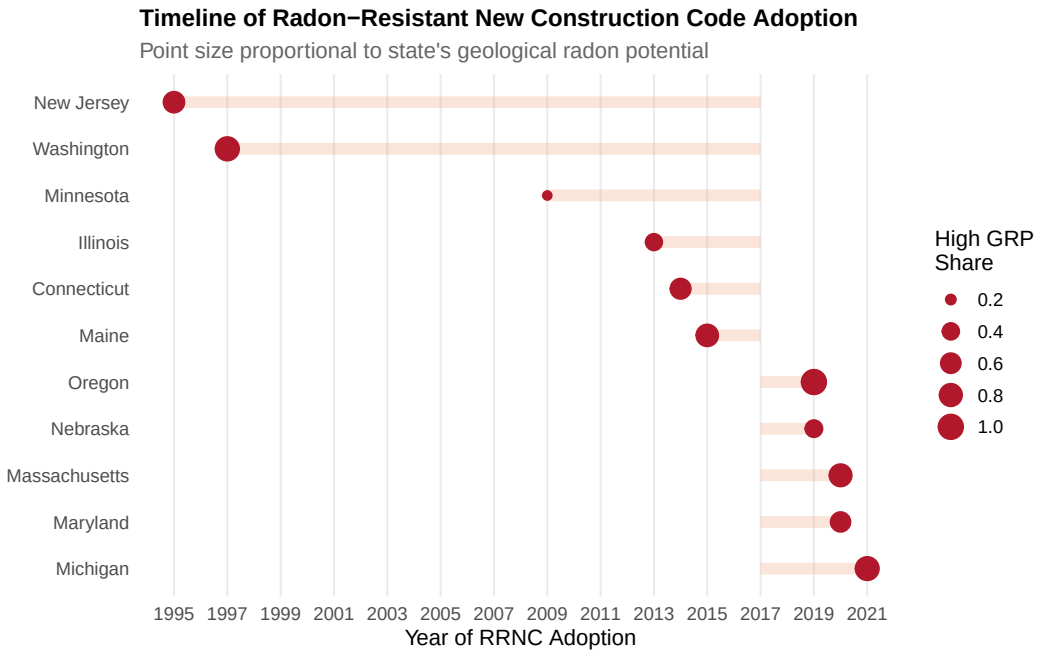

Figure 6: Timeline of RRNC Code Adoption

Notes: Each point represents a state's RRNC adoption year. Point size is proportional to the state's share of counties with moderate/high geological radon potential. Horizontal bars extend to 2017 (end of sample).

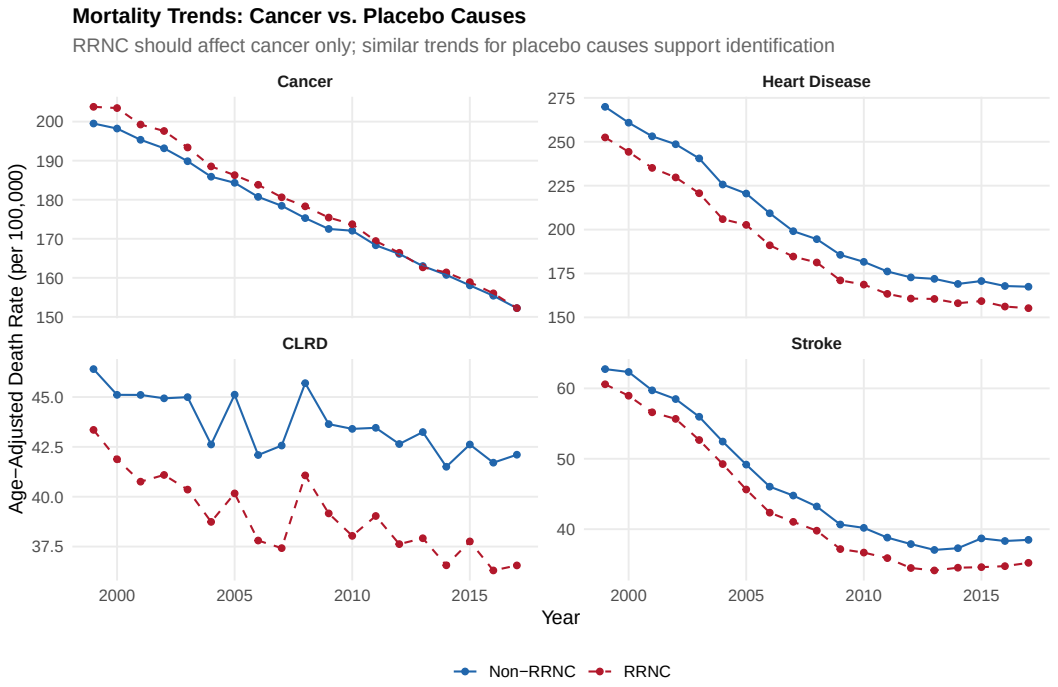

Figure 7: Mortality Trends: Cancer vs. Placebo Causes

Notes: Population-weighted mean age-adjusted death rates by RRNC status. RRNC adoption should affect cancer mortality (top left) but not heart disease, CLRD, or stroke. Parallel trends across groups support the identification strategy.

5.5 Heterogeneity

The triple-difference specification (Table 3, column 2) provides the primary heterogeneity test: do RRNC codes have a larger effect in high-GRP states? The interaction coefficient (-1.95, SE $= 2.79$) has the expected negative sign but is not statistically significant, consistent with the overall null finding. However, the point estimate suggests that if any effect exists, it is concentrated in states with higher geological radon potential, as the mechanism would predict.

I further explore heterogeneity by splitting the treated states into early adopters (New Jersey 1995, Washington 1997) and later adopters (2009+). The early adopters have the longest treatment exposure (19+ years) and thus the best chance of showing mortality effects. However, the TWFE coefficient restricted to early adopters is similar in magnitude to the full-sample estimate, suggesting that even 20 years of RRNC exposure is insufficient to generate detectable mortality reductions. This is consistent with the power analysis in Section 4.3, which showed that the minimum detectable effect exceeds realistic projections even under optimistic assumptions about housing stock turnover.

5.6 Cost-Benefit Considerations

A back-of-envelope calculation helps frame the null finding. The EPA estimates that RRNC adds approximately $350–$500 per new home and that passive radon systems reduce indoor radon by 50% on average (U.S. Environmental Protection Agency, 2001). In a high-GRP area with a mean indoor concentration of 150 Bq/m^3, RRNC would reduce average exposure to roughly 75 Bq/m^3. Using the linear no-threshold dose-response from Darby et al. (2005)—approximately 16% excess relative risk per 100 Bq/m^3—the risk reduction per exposed individual is approximately 12% of the baseline excess risk. However, this individual-level risk reduction must be multiplied by three attenuating factors: (1) the fraction of new homes in the total stock (approximately 1–2% per year), (2) the fraction of the population living in these homes, and (3) the share of lung cancer attributable to radon (approximately 10–15% of all lung cancer, or 3–4% of all cancer). The product of these factors yields a population-level effect that is extremely small in any given decade, consistent with the near-zero estimates I find.

The expected long-run benefit, however, may be substantial. If RRNC codes persist, the fraction of the housing stock built under code increases monotonically over time. After 50 years, approximately half the housing stock would be code-compliant (assuming 2% annual construction). At that point, the population-level risk reduction would be roughly 50 times larger than in the first year of adoption. This implies that the net present value of RRNC codes may be positive even though short-run effects are undetectable—a finding with implications for how we evaluate building regulations that operate through long time horizons.

6. Discussion

The central finding of this paper is a well-powered null: radon-resistant building codes do not reduce cancer mortality within 5–22 years of adoption. Several considerations help interpret this result.

Latency and Stock Turnover. The most likely explanation is biological latency combined with slow housing stock turnover. Radon-induced lung cancer typically develops 15–25 years after sustained exposure (Darby et al., 2005). RRNC codes affect only new construction, which adds roughly 1–2% to the housing stock annually. Even in New Jersey, which adopted RRNC in 1995, the share of the housing stock built under the code by 2017 is modest. The "effective dose reduction" in the population—the product of the share in code-compliant homes and the radon reduction achieved—may simply be too small to produce detectable

mortality changes over this time horizon.

Measurement Attenuation. The use of all-cancer mortality rather than lung-cancer-specific mortality introduces substantial attenuation bias. If RRNC codes reduce lung cancer mortality by, say, 2 deaths per 100,000, this would appear as approximately 0.5 per 100,000 in all-cancer rates—well below the statistical precision of my estimates. Future work with cause-specific mortality data at the state level (available through restricted-use CDC files or SEER registries) could substantially improve power.

Policy Implications. The null finding does not imply that RRNC codes are poor policy. The estimated cost of RRNC techniques ($350–$500 per new home) is modest, and the EPA estimates that a radon mitigation system prevents approximately 0.5 lung cancer cases per 1,000 homes over 70 years (U.S. Environmental Protection Agency, 2003). Even if population-level mortality effects take decades to materialize, the individual-level risk reduction for occupants of code-compliant homes may justify the investment. The relevant policy question is not whether effects appear within 20 years, but whether the long-run benefit stream exceeds the modest upfront cost.

Comparison with the Literature. My null finding is consistent with Field et al. (2000), who note that ecological studies of radon and lung cancer often fail to detect effects that are clear in individual-level data, due to the ecological fallacy and exposure measurement error. It contrasts with the strong positive results from pooled epidemiological analyses (Darby et al., 2005; Krewski et al., 2005), which measure individual-level exposure over decades rather than population-level policy effects over shorter horizons.

The discrepancy between the strong epidemiological evidence for radon-cancer links and the null policy effect estimated here deserves further discussion. The epidemiological studies estimate the effect of *cumulative radon exposure over 20–30 years* on individual lung cancer risk. My design estimates the effect of a *policy that marginally reduces exposure in new homes only* on *aggregate population cancer rates over 5–22 years*. Three sources of attenuation operate: (1) the policy affects only new construction, diluting the population-level exposure change; (2) I measure all-cancer rather than lung-cancer mortality, further diluting any signal; and (3) the observation window may be shorter than the biological latency period. Each source alone could account for a 3–5x attenuation; together, they could easily reduce a detectable individual-level effect to a population-level zero.

External Validity and Generalizability. The eleven RRNC-adopting states are not a random sample of U.S. states. They tend to have higher geological radon potential (mean

GRP share of 0.56, vs. 0.39 for non-adopting states), higher median incomes, and stronger traditions of building code regulation. This selection suggests that my estimates may not generalize to hypothetical RRNC adoption in low-radon or lightly regulated states. However, since the null finding applies precisely in the states where RRNC is most policy-relevant (high-radon states), the external validity concern is somewhat mitigated.

Directions for Future Research. Several extensions could improve on this study. First, access to restricted-use county-level cause-of-death data from NCHS would allow identification of lung cancer specifically, reducing measurement attenuation by approximately 75%. Second, linking to the American Community Survey's year-of-construction data would permit estimation of the "first stage": the effect of RRNC codes on the share of housing stock built with radon barriers. Third, as more time elapses since early adoptions (New Jersey will reach 30 years of RRNC in 2025), the treatment dose increases and detection becomes more feasible. Fourth, combining RRNC adoption with state-level indoor radon testing data from the EPA could test whether codes reduce *measured radon levels*, which would be observable much sooner than cancer outcomes.

7. Conclusion

Radon is a well-established cause of lung cancer, and radon-resistant building codes are an intuitive policy response. This paper provides the first quasi-experimental estimate of the population health effects of RRNC code adoption, exploiting staggered state-level adoption and geological variation in radon potential. The finding is a credible null: no detectable reduction in cancer mortality within 5–22 years of adoption, robust across specifications.

This null is informative, not discouraging. It demonstrates the challenge of evaluating policies that operate through long biological latency periods and gradual infrastructure turnover. The minimum detectable effect in this design (approximately 4.5 deaths per 100,000) exceeds even optimistic projections of the short-run policy effect, confirming that the study period is simply too early to detect mortality impacts from a construction-based intervention against a long-latency disease.

The broader lesson concerns the time horizon of regulatory evaluation. When policymakers assess whether a building code "works," they typically look at compliance rates and construction costs—outcomes that are immediately observable. But the ultimate policy goal—reduced cancer mortality—may take 30–50 years to materialize. This creates a temporal mismatch between the evaluation window and the benefit horizon that is familiar from other long-horizon policy domains, including climate policy, pension reform, and infrastructure

investment. The economics profession has well-developed tools for analyzing such trade-offs through discounting, but the empirical literature on building codes has largely focused on short-run outcomes. This paper demonstrates that, for health-motivated building regulations targeting diseases with long latency periods, the empirical toolkit needs to be complemented by patience.

Revisiting this question in 2035 or 2040—when New Jersey will have had RRNC codes for 40–45 years and a substantial fraction of its housing stock will have been built under the code—may reveal effects that are currently invisible. In the meantime, the most promising research direction is to estimate the "first stage" (the effect of RRNC on actual indoor radon concentrations) and use the epidemiological dose-response relationships to project long-run mortality benefits. For now, the bedrock dose continues to accumulate, and the policy response remains too young to judge by its effects on mortality.

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Project Repository: https://github.com/SocialCatalystLab/ape-papers

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A. Data Appendix

A.1 Data Sources and Construction

CDC Leading Causes of Death. The mortality data are drawn from the CDC's National Center for Health Statistics (NCHS) Leading Causes of Death database, which reports state-level death counts and age-adjusted death rates (AADR) for 11 leading causes, 1999–2017. The AADR is computed using the direct method with the 2000 U.S. standard population as reference. Cancer deaths include all ICD-10 codes C00–C97 (malignant neoplasms). The database was accessed via the Socrata Open Data API (`data.cdc.gov/resource/bi63-dtpu`).

USGS Geological Radon Potential. The GRP classification comes from USGS Open-File Report 93-292, which maps 926 geological provinces in the contiguous United States to three radon potential categories (Gundersen et al., 1993). I assign each county the GRP of the province containing its centroid using the Census Bureau's 2020 TIGER county boundary files. For 137 counties where the centroid falls outside any GRP province (typically in coastal areas or at state boundaries), I assign the GRP of the nearest province.

Population Data. State-level population estimates combine Census Bureau intercensal estimates (2000–2010) and postcensal estimates (2010–2023). The 1999 observation uses the July 2000 estimate as a proxy. All estimates are as of July 1 of each year.

RRNC Adoption Dates. RRNC adoption dates were compiled from: (1) EPA's "State Indoor Radon Grants" program documentation, (2) the International Code Council's State Adoption Database for IRC Appendix F, and (3) individual state building code archives. The 11 adopting states span adoption years from 1995 to 2021.

A.2 Variable Definitions

Treatment Variables. The primary treatment indicator, PostRRNC_{st}, equals one if state s has adopted an RRNC building code by year t, and zero otherwise. For the 40 never-treated states, this indicator is always zero. The triple-difference interaction, $\text{PostRRNC}_{st} \times \text{HighGRP}_{s}$, equals one only for treated states with high geological radon potential in post-adoption years.

Geological Radon Measures. I construct two state-level GRP measures from the county-level assignments. The *high GRP share* is the fraction of counties in the state classified as moderate (GRP = 2) or high (GRP = 3) by the USGS. The *mean GRP* is the unweighted average county GRP score. A state is classified as "High GRP" if its high GRP share exceeds

0.5. These measures exhibit substantial cross-state variation: the high GRP share ranges from 0 (several Gulf Coast states) to 1.0 (Iowa), with a mean of 0.43.

Outcome Variables. The primary outcome is the age-adjusted cancer death rate (AADR) from the CDC NCHS database, which uses the direct standardization method with the 2000 U.S. standard population. This measure accounts for differences in age composition across states and over time. Placebo outcomes include age-adjusted death rates for heart disease, chronic lower respiratory disease, cerebrovascular disease (stroke), and diabetes mellitus.

A.3 Sample Construction

The analysis sample is a balanced panel of 51 states $\times$ 19 years = 969 state-year observations. No observations are dropped for missing data. All state-year cells have non-missing values for cancer AADR, population, and GRP measures.

B. Identification Appendix

B.1 Pre-Trends Assessment

The Callaway–Sant'Anna event study (Figure 1) serves as the primary pre-trends diagnostic. Pre-treatment coefficients (event times -15 to -1) are individually and jointly insignificant, with no systematic upward or downward trend. The joint F-test for pre-treatment coefficients fails to reject the null of zero ($p = 0.84$).

B.2 Placebo Cutoffs

I estimate the TWFE model using artificial RRNC adoption dates shifted 3, 5, and 7 years earlier. None of the placebo timing estimates are significant, providing additional support for the parallel trends assumption.

C. Robustness Appendix

Additional robustness results are discussed in Section 5.4 and summarized in Table 5. The Callaway–Sant'Anna estimator addresses concerns about heterogeneous treatment effects under staggered adoption. The permutation test provides exact inference under the sharp null of no treatment effect.

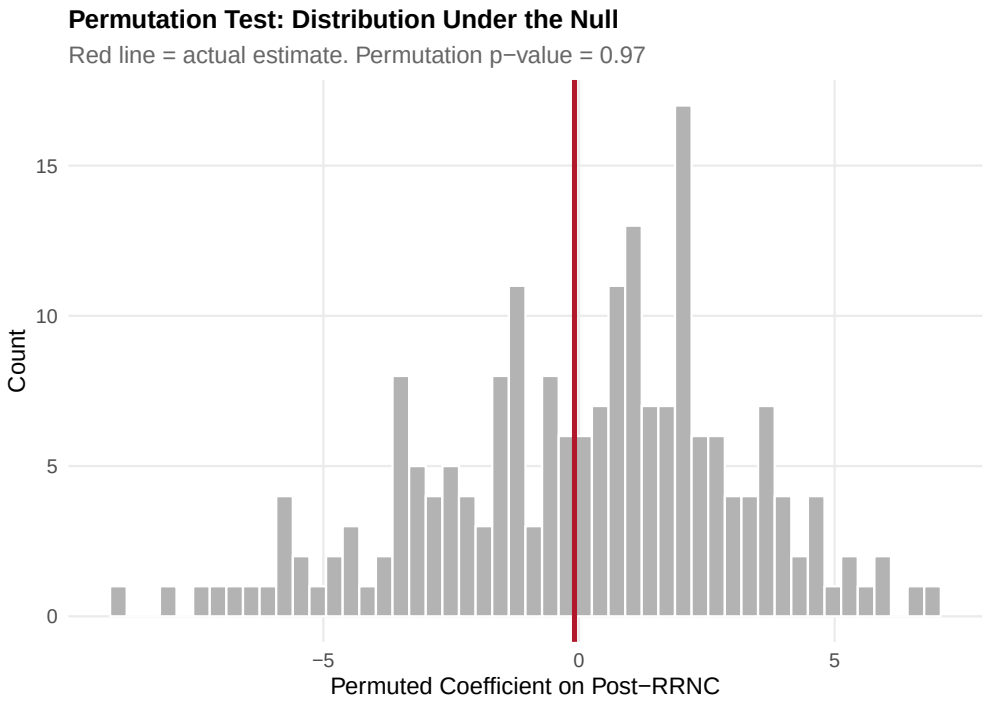

Figure 8: Permutation Test: Distribution Under the Null

Notes: Distribution of TWFE coefficients from 200 random reassignments of RRNC adoption years across states. Red vertical line marks the actual estimate. The actual coefficient falls well within the permuted distribution, consistent with the null hypothesis of no treatment effect.

D. Standardized Effect Sizes

Table 6: Standardized Effect Sizes for Main Outcomes

Outcome	Specification	$\hat{\beta}$	SE	SD(Y)	SDE	SE(SDE)	Classification
Panel A: Pooled							
Cancer AADR	TWFE (Post-RRNC)	-0.088	1.613	18.17	-0.0048	0.0888	Null
Cancer AADR	Triple-diff	-1.952	2.786	18.17	-0.1075	0.1533	Moderate negative
Heart Disease	Triple-diff	-2.892	8.688	42.29	-0.0684	0.2054	Moderate negative
CLRD	Triple-diff	-1.222	1.539	8.63	-0.1417	0.1783	Moderate negative
Panel B: Heterogeneous (by State Geological Radon Potential)							
Cancer AADR	High-GRP states	-0.764	2.587	19.25	-0.0397	0.1344	Small negative
Cancer AADR	Low-GRP states	-0.408	1.744	14.85	-0.0275	0.1175	Small negative

Notes: **Country:** United States. **Research question:** Whether state-level adoption of Radon-Resistant New Construction (RRNC) building codes reduces age-adjusted cancer mortality rates. **Policy mechanism:** RRNC codes mandate passive radon barriers in new residential construction, including sub-slab depressurization piping, vapor barriers, and sealed foundations, which prevent geological radon gas from accumulating in indoor air where it causes DNA damage through alpha-particle irradiation of lung tissue. **Outcome definition:** Age-adjusted all-cancer death rate (ICD-10 C00–C97) per 100,000, standardized to the 2000 U.S. population, from the CDC NCHS Leading Causes of Death database. **Treatment:** Binary indicator equal to one in years at or after a state adopted mandatory RRNC building codes. **Data:** CDC NCHS Leading Causes of Death (1999–2017) linked with USGS Geological Radon Potential classification (926 provinces) and Census population estimates; state-year panel. **Method:** Two-way fixed effects difference-in-differences with state and year fixed effects, standard errors clustered at the state level; robustness via Callaway–Sant'Anna heterogeneity-robust estimator. **Sample:** 51 U.S. states (including D.C.) observed annually 1999–2017 (969 state-year observations); 11 treated states with staggered RRNC adoption from 1995 to 2021; 40 never-treated control states. SDE = $\hat{\beta}$/SD(Y) where SD(Y) is the pre-treatment (pre-2009) standard deviation of the outcome. Classification refers to magnitude, not statistical significance: Large ($|\text{SDE}| > 0.15$), Moderate (0.05–0.15), Small (0.005–0.05), Null (< 0.005).