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Review

Residential Radon Exposure and Lung Cancer Prevention: From Environmental Risk to Public Health Implementation

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ABSTRACT: Residential radon is an established environmental carcinogen and preventable cause of lung cancer, yet it remains incompletely integrated into routine prevention. Because radon is imperceptible and indoor concentrations vary between buildings, quality-assured dwelling-specific measurement is essential; however, a household concentration does not by itself determine cumulative exposure, radiation dose, or individual cancer risk. This Perspective examines radon prevention at the interface of radiation biology, risk communication, environmental health literacy, and implementation. We propose a six-stage Radon Prevention Continuum linking risk recognition and prioritization, quality-assured measurement, interpretation and risk communication, mitigation planning, mitigation implementation, and verification with longitudinal follow-up. The framework is iterative, allowing return to earlier stages when measurements are uncertain, mitigation is ineffective, or building conditions change. Primary health care and environmental health surveillance may support recognition, communication, referral, and continuity, while measurement quality assurance and structural mitigation remain specialized functions. Brazil is examined as an implementation case in transition, where a recently adopted residential reference level coexists with developing measurement, quality-assurance, and institutional capacity. The framework aims to connect established evidence with coordinated health-system and built-environment action, using verified, sustained reductions in indoor radon concentration as a proximal implementation endpoint.

Keywords: Residential radon; Lung cancer; Radiation protection; Environmental health; Risk communication; Primary health care; Cancer prevention; Implementation science

1. Introduction

Residential radon presents a distinctive challenge for cancer prevention: its carcinogenicity is firmly established, yet exposure remains largely invisible in everyday life and is inconsistently incorporated into routine preventive practice. Radon-222 (222Rn) is a naturally occurring radioactive gas generated within the uranium-238 decay series. After entering buildings through cracks, construction joints, service penetrations, and soil-foundation interfaces, it may accumulate indoors, particularly under conditions of

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limited air exchange. Its short-lived decay products can be inhaled and deposited in the respiratory epithelium, where alpha-particle irradiation contributes to DNA damage and carcinogenesis [1,2].

The epidemiological association with lung cancer is supported by substantial evidence. The pooled analysis of 13 European case-control studies demonstrated an approximately linear increase in lung-cancer risk with increasing long-term residential radon concentration, including concentrations encountered in ordinary homes [3]. Subsequent reviews have confirmed the relevance of residential exposure among both smokers and never-smokers and have emphasized that tobacco use substantially modifies absolute risk without negating radon as an independent and preventable carcinogenic exposure [4,5].

Indoor radon concentration is determined by interacting geological, structural, environmental, and behavioral factors rather than by geology alone. Building characteristics, pressure differentials, soil-building interfaces, ventilation patterns, occupant behavior, and thermal or energy-efficiency interventions can modify radon entry, retention, and dilution. Our previous integrative review found that reduced air exchange may favor indoor accumulation, whereas energy-efficient construction does not necessarily increase exposure when airtightness is combined with appropriate soil sealing, sub-slab depressurization, and controlled ventilation [6]. A complementary analysis of radon, vulnerability, and sustainable development emphasized that the capacity to test and remediate dwellings is socially distributed, making exposure reduction a problem of both radiation protection and environmental equity [7].

At the biological level, the prevention rationale rests on a focused mechanistic sequence: inhaled radon progeny deposit within the respiratory tract, their alpha emissions deliver densely ionizing high-linear-energy-transfer (LET) radiation to bronchial epithelial cells, and this energy deposition can produce complex DNA and chromosomal damage relevant to carcinogenesis [2,8–10]. Molecular, inflammatory, and other cellular responses associated with radon exposure remain areas of active investigation, but they are not required to justify current prevention strategies [9,11,12]. Accordingly, radiobiology is used here to establish biological plausibility for exposure reduction rather than to support molecular screening or individual causal attribution.

Despite strong biological and epidemiological evidence, recognition of carcinogenicity does not automatically translate into effective prevention. Radon cannot be detected by human senses, indoor concentrations vary within and between buildings, and exposure produces no immediate clinical signature. Measurement is therefore essential, but testing is only one component of a longer prevention pathway: individuals and institutions must recognize the risk, obtain a quality-assured measurement, interpret the result, identify an appropriate response, implement mitigation when indicated, and verify that indoor radon concentration has been reduced [1,13,14].

This Perspective examines residential radon at the intersection of radiation biology, lung-cancer prevention, environmental health, risk communication, and implementation. It is not intended as a systematic or exhaustive review; rather, it integrates established international guidance and peer-reviewed evidence relevant to the translational problem in order to develop and critically examine an implementation-oriented conceptual framework. The central question is not whether radon is carcinogenic, which is already supported by extensive evidence, but how established knowledge of radon-related lung-cancer risk can be translated into an actionable prevention pathway. We use the term Radon Prevention Continuum to describe a six-stage framework linking risk recognition, quality-assured measurement, interpretation, mitigation, and verified reduction in indoor radon concentration. Brazil is examined as an implementation case in transition because a recently adopted residential-radon provision has deferred implementation, public technical initiatives are developing measurement and quality-assurance capacity, and the SUS provides a territorially organized health-system context in which referral and surveillance functions could be evaluated [15–20].

2. Radon and Lung Cancer: From Alpha-Particle Exposure to Carcinogenesis

2.1. Radon Progeny and Alpha-Particle Deposition in the Respiratory Tract

The carcinogenic potential of residential radon arises predominantly from its short-lived radioactive progeny rather than from chemically inert 222Rn itself. Radioactive decay generates radionuclides including 218Po, 214Pb, 214Bi, and 214Po. These solid progeny may remain unattached or associate with airborne aerosols and, after inhalation, deposit along the respiratory tract. Alpha-emitting progeny, particularly 218Po and 214Po, therefore account for a substantial fraction of the radiation dose delivered to radiosensitive cells of the bronchial epithelium [2,8–10].

Alpha particles have a short range in tissue but deposit energy densely along their tracks because of their high LET. When radon progeny decay on or near bronchial epithelial surfaces, highly localized energy deposition can generate complex lesions within a small number of traversed cells. Experimental and microdosimetric evidence supports the biological plausibility of DNA and chromosomal damage produced by radon-progeny alpha particles [8–10].

Four related quantities are distinguished throughout this Perspective. Indoor radon concentration is the activity concentration measured in air (Bq/m^3) at a defined location over a defined period. Personal exposure reflects the concentrations encountered over time and therefore depends on occupancy and temporal variation. Radiation dose to the respiratory tract additionally depends on factors including the equilibrium between radon and its short-lived progeny, aerosol characteristics and attachment, breathing pattern, airway deposition, and the dosimetric model applied [9,11,21]. Lung-cancer risk is an epidemiological probability associated with cumulative exposure and modified by factors such as smoking; it cannot be inferred directly from a single household concentration measurement or a single dose estimate.

Building characteristics act upstream of this biological pathway by influencing the concentration available for inhalation. Soil permeability, pressure gradients, cracks and service penetrations, foundation design, ventilation, and air-exchange rates all affect radon entry and accumulation [1,22]. The exposure pathway therefore extends from geology and the built environment to inhalation, microscopic energy deposition, molecular damage, and ultimately carcinogenesis. Importantly, several determinants along this pathway are modifiable before disease develops.

2.2. DNA Damage and Biological Plausibility of Radon Carcinogenesis

The central mechanistic link between radon exposure and lung cancer is the capacity of high-LET alpha radiation to generate complex DNA damage. Dense ionization tracks can produce single- and double-strand DNA breaks, base damage, DNA-protein crosslinks, chromosomal aberrations, deletions, and rearrangements [2,8,9]. Clustered lesions are particularly important because they are more difficult to repair accurately than isolated damage and may increase the probability of misrepair, mutation, or persistent genomic instability.

Radon-focused reviews and experimental studies have also reported oxidative, inflammatory, epigenetic, and signaling changes, as well as candidate alterations involving DNA-repair pathways and genes such as *TP53*, *RASSF1*, *KRAS*, and *EGFR* [9,12]. These findings require cautious interpretation: the evidence is heterogeneous, many alterations are not specific to radon, and substantial overlap exists with responses to tobacco smoke and ionizing radiation more broadly. No molecular alteration or biomarker is currently validated for diagnosing residential radon exposure or for predicting radon-attributable lung cancer in an individual [9,12].

For the prevention framework developed here, the mechanistic implication is deliberately limited but important. Radon progeny can deliver biologically damaging alpha-particle radiation to respiratory epithelium, providing mechanistic support for the established epidemiological association. Prevention, however, does not depend on identifying a molecular signature in exposed residents. Environmental

measurement and mitigation remain the actionable interventions; accordingly, verified and sustained reduction in indoor radon concentration, as a proximal indicator of reduced exposure, rather than biomarker detection, is the immediate endpoint of the Radon Prevention Continuum [1,2,8,10].

2.3. Epidemiological Translation: Smoking, Never-Smokers, and Lung-Cancer Risk

The biological plausibility of radon carcinogenesis is supported by decades of epidemiological evidence. Initial evidence arose from underground miners exposed to high concentrations of radon progeny, and subsequent residential studies demonstrated that the association extends to the lower concentrations encountered in homes. The pooled European analysis by Darby et al. remains a cornerstone: after correction for uncertainty in measured residential concentrations, lung-cancer risk increased by approximately 16% for each 100 Bq/m^3 increase in long-term radon concentration, with an approximately linear exposure-response relationship and no evidence of a risk-free threshold within the residential range [3]. This estimate is population based and should not be interpreted as permitting causal attribution of an individual tumor to a specific household measurement.

More recent literature continues to support this association. A 2024 review integrating epidemiology, cell biology, dosimetry, and risk modeling concluded that residential radon remains a major preventable contributor to lung cancer and highlighted persistent gaps between scientific evidence and effective risk reduction [11]. Smoking strongly modifies absolute risk by increasing the underlying probability of lung cancer. WHO risk estimates illustrate the magnitude of this interaction: at a long-term radon concentration of 800 Bq/m^3, the modeled cumulative risk of lung-cancer death by age 75 is approximately 22% for continuing smokers and 0.9% for lifelong never-smokers; radon nonetheless increases risk in both groups [1]. These population-level estimates are useful for risk communication and prevention but are not individual predictions.

The interaction between tobacco and radon has two practical implications. First, smoking cessation remains central to lung-cancer prevention and substantially reduces absolute risk among people who smoke. Second, smoking history does not determine whether radon measurement is warranted. A never-smoker living in a dwelling with elevated radon remains exposed to a preventable carcinogen, whereas a smoker in the same environment experiences the combined effect of two major pulmonary carcinogenic exposures [3–5,23].

These findings support a conceptual shift: radon should not be considered merely as an alternative explanation for lung cancer once smoking has been excluded. Rather, smoking and radon should be understood as distinct, potentially interacting, and simultaneously preventable risk factors. Epidemiological association, however, does not permit reliable retrospective attribution of an individual lung cancer to a specific residential concentration. Historical exposure is often uncertain, concentrations vary across time and dwellings, and carcinogenesis is multifactorial. Population-level prevention is therefore more tractable than individual causal attribution.

3. Why a Well-Established Carcinogen Remains an Invisible Public Health Risk

Residential radon presents a public health paradox: strong scientific evidence of carcinogenicity coexists with limited recognition and inconsistent preventive action. Unlike tobacco smoke, asbestos, or visible outdoor air pollution, radon produces no sensory warning. It is colorless, odorless, and tasteless, causes no immediate symptoms, and may accumulate in buildings that appear structurally adequate. We use the term radon prevention gap to describe the implementation gap between established evidence of cancer risk and the actions required to achieve a measurable and sustained reduction in indoor radon concentration.

The first component of this gap is informational. Public understanding of radon remains limited in many settings, particularly with respect to its origin, health effects, testing methods, interpretation of results,

and mitigation options [24]. Recognition of the term radon is not equivalent to understanding why testing is relevant to a particular dwelling or what action should follow an elevated result.

The second component is behavioral. Awareness alone does not reliably produce preventive action. In a randomized trial of 3500 households, behaviorally informed communication increased uptake of free home radon testing from approximately 22% to 33% [25]. Behavioral responses after testing also influence realized exposure: among households receiving high radon results, economic barriers and delayed mitigation can prolong exposure even after the environmental hazard has been identified [26].

The third component is operational. Household radon concentration cannot be characterized without environmental measurement, while individual cumulative exposure additionally depends on time, occupancy, and changes in the environments in which a person lives or works. Measurement itself depends on access to an appropriate detector or service, correct placement and duration, technically valid interpretation, and follow-up. Attrition can occur at every transition, from awareness to testing, testing to interpretation, interpretation to mitigation, and mitigation to verification.

The fourth component is socioeconomic. Households do not enter the prevention pathway with equivalent resources. Testing requires information, access, and sometimes payment; mitigation may require structural modification, landlord cooperation, specialized contractors, and substantial financial expenditure. Our previous analysis of environmental vulnerability emphasized that socioeconomic position, housing conditions, and tenure may determine whether knowledge about radon can be converted into effective protection; independent evidence likewise shows that social and behavioral factors influence whether households act on radon test results [7,26]. An environmental carcinogen generated within the built environment cannot be adequately addressed through health education alone.

Finally, sensory invisibility may be compounded by institutional invisibility. If environmental exposures are not considered in clinical or public health practice, radon may remain absent from routine histories and prevention discussions. If health and regulatory systems do not provide a route to quality-assured measurement and technical follow-up, individuals may not know how to act. If household results remain fragmented across universities, commercial laboratories, public programs, and residents, surveillance systems cannot readily generate comparable territorial intelligence. A self-reinforcing cycle may result: limited measurement produces limited data, limited data reduce institutional priority, and low priority constrains measurement infrastructure.

We therefore conceptualize the radon prevention gap as the cumulative loss of preventive potential between recognizing radon as a carcinogen and verified reductions in indoor radon concentration, with the intended consequence of reduced residential exposure. The following sections examine the communication, literacy, technical, and institutional functions required to reduce this loss across the prevention pathway.

4. Risk Communication and Environmental Health Literacy: Moving from Information to Action

4.1. Environmental Health Literacy as a Prerequisite for Prevention

For residential radon, communication is itself a component of prevention. Individuals need to understand that radon is an imperceptible environmental exposure, recognize why measurement is necessary, interpret concentrations reported in Bq/m^3, and understand the actions that may follow an elevated result. These requirements place radon prevention within the domain of environmental health literacy, which encompasses the ability to access, understand, evaluate, and use environmental health information in decision making [27–29].

Environmental health literacy extends beyond factual knowledge by linking environmental exposures, health literacy, risk communication, and community engagement [27,28]. Gray distinguished basic acquisition of knowledge from capacities that support change at individual and community levels [29]. In

the context of radon, knowing that the gas exists is not equivalent to knowing whether a specific dwelling has elevated concentrations, how a result should be interpreted, or how exposure can be reduced.

Citizen-science initiatives illustrate this progression. In a home-radon testing program, participants demonstrated improvements in environmental health literacy, health-information efficacy, and testing self-efficacy, whereas confidence and capacity related to mitigation developed more slowly [30]. Testing and mitigation are therefore distinct behavioral and operational steps. Environmental health literacy should be linked to an actionable pathway; otherwise, awareness may increase without producing mitigation or measurable reduction in indoor radon concentration [26,30].

4.2. Designing Communication for Action Rather Than Awareness Alone

Conventional models of risk communication may imply a simple progression from information to knowledge, concern, and protective behavior. Radon research instead suggests a non-linear pathway in which knowledge, perceived susceptibility, trust, self-efficacy, cost, social norms, and feasibility interact [24–26]. Communication should therefore connect four elements: the hazard, its personal relevance, a measurable action, and an available response. Research on news-based communication further indicates that source selection, narrative framing, and contextualization influence how radon risk is understood [31].

Experimental evidence indicates that message design can influence testing behavior. In the randomized trial by Timmons and Lunn, behaviorally informed communication increased household uptake of radon testing [25]. Digital communication has also been evaluated: the 2025 Radon Buster campaign across Austria, Belgium, Ireland, and Slovenia found that exposure to and appreciation of campaign messages were associated with intentions to seek radon information, although effects on testing intentions were more complex [32]. In 2026, an experiment involving 1716 residents of British Columbia found that a radon-risk map increased testing intentions in medium-high and high-risk areas but slightly reduced intentions among some non-smokers in the lowest-risk category [33].

These findings underscore a central communication challenge: geographic risk maps can support population prioritization but can also generate false reassurance if a lower-risk category is interpreted as evidence that a specific dwelling does not require testing. Geographic susceptibility is only one determinant of household concentration; foundation design, pressure differentials, ventilation, renovation, and occupant behavior also contribute [1,22,33]. Risk maps should therefore be used for population-level prioritization rather than as substitutes for dwelling-specific measurement.

4.3. Communicating Numbers Without Creating False Thresholds

Communication becomes particularly challenging once numerical results and regulatory values are introduced. Reference levels are operational radiological-protection benchmarks for optimization and action; they are not biological thresholds, dose limits, or boundaries between risk and no risk. WHO recommends a national residential reference level of 100 Bq/m^3 where feasible and advises that it should not exceed 300 Bq/m^3 under country-specific conditions [1,13]. ICRP similarly recommends an integrated optimization strategy based on a national reference level, with 300 Bq/m^3 as the upper value for annual mean indoor radon concentration [14]. Brazil's current regulatory framework incorporates a 300 Bq/m^3 reference level for residences and other high-occupancy environments, with deferred implementation of the relevant provision [15].

Activity concentration and radiation dose should not be treated as interchangeable quantities. Bq/m^3 describes radon activity concentration in indoor air. A dose estimate derived from a given concentration depends on assumptions or measurements concerning occupancy time, equilibrium between radon and its progeny, aerosol characteristics, breathing and deposition, and the dose coefficient or dosimetric model applied [9,14,21]. For most homes and buildings, ICRP guidance allows optimization of protection directly

from measured air concentrations without requiring calculation of an individual dose [14,21]. Accordingly, a concentration such as 300 Bq/m^3 should not be translated into a single fixed dose for every resident. Epidemiological evidence also does not indicate that risk begins abruptly at a regulatory value [3]. Interpretation of a measurement therefore requires context, including the method, duration, location, applicable reference level, uncertainty, need for confirmation, and recommended next action.

The practical principle is therefore straightforward: measurement contributes to prevention only when the result is technically interpretable and linked to an actionable response.

4.4. Communicating the Radon-Smoking Interaction Without Blame

Combined exposure to radon and tobacco produces substantially greater lung-cancer risk, and smoking cessation remains one of the most effective means of reducing absolute risk among people who smoke [1,3–5]. Risk communication should nevertheless avoid implying either that radon is relevant only to smokers or that people who smoke are responsible for the environmental component of their risk. Both interpretations are misleading.

A clearer communication strategy distinguishes the exposures while explaining their interaction: tobacco smoking increases lung-cancer risk; radon independently increases lung-cancer risk; combined exposure produces a substantially greater absolute risk; and the two exposures are addressed through different but complementary preventive interventions [3–5,23]. This framing allows radon measurement to complement, rather than compete with, tobacco-control strategies.

4.5. From Individual Communication to Institutional Responsibility

Communicating an environmental carcinogen without providing a realistic route to action may shift responsibility from institutions to individuals. A household receiving an elevated radon result needs an operational pathway: whether the measurement should be confirmed, who can validate and interpret the result, which mitigation options are technically appropriate, who is qualified to assess the building, how remediation can be financed, how rented housing is addressed, and how effectiveness will be verified.

Environmental health literacy should strengthen the capacity of individuals and communities to participate in prevention, not serve as a mechanism for transferring responsibility to residents. Individual agency is necessary, but individual responsibility alone is insufficient [26]. Communication therefore operates across the full prevention pathway: before testing, during measurement, after interpretation, during mitigation planning and implementation, and again when post-mitigation measurement determines whether indoor radon concentration has been reduced.

5. From Measurement to Mitigation: The Radon Prevention Continuum

Existing international guidance provides the scientific and technical foundations for radon control. WHO guidance addresses population assessment, measurement, reference levels, risk communication, prevention in new buildings, mitigation of existing buildings, professional training, and program evaluation [1,13]. ICRP recommends an integrated and graded approach based on optimization, national reference levels, measurement, prevention and mitigation, stakeholder responsibilities, and national action planning [14]. RadoNorm extends this knowledge base through multidisciplinary research on exposure assessment, dosimetry, health effects, mitigation, risk communication, stakeholder engagement, and societal dimensions [22,34].

The Radon Prevention Continuum does not claim novelty for these individual technical components. Its contribution is organizational and implementation oriented: it reconfigures recurrent elements from international guidance and implementation literature into an explicit pathway with defined transition points, potential attrition, distributed institutional responsibilities, and a proximal endpoint of verified and

sustained reduction in indoor radon concentration as an indicator of reduced exposure. WHO and ICRP primarily provide policy and radiological-protection guidance, whereas RadoNorm contributes multidisciplinary research across the radiation-risk management cycle; this framework focuses on what must occur operationally between recognizing possible residential exposure and confirming that corrective action has reduced indoor radon concentration.

The six stages were derived by mapping recurrent actions across international guidance and supporting literature onto the practical decision sequence faced by residents and institutions: (1) risk recognition and prioritization; (2) access to quality-assured measurement; (3) interpretation and risk communication; (4) mitigation planning; (5) mitigation implementation; and (6) verification and longitudinal follow-up. The primary sequence is logically ordered, but the pathway is not strictly linear. Invalid or uncertain measurements may require repeat testing; unsuccessful or incomplete mitigation may return the process to planning or implementation; major renovation or changes in building use may require reassessment; and later increases in radon concentration may trigger renewed measurement and mitigation. These feedback loops are represented in Figure 1. Environmental health literacy, equity, quality assurance, risk communication, governance, and intersectoral coordination operate across multiple stages rather than as separate sequential steps. Table 1 summarizes the stages, outputs, common points of failure, and illustrative implementation indicators.

Implementation science is used here as an evaluative lens rather than to imply that the Continuum is an empirically validated implementation-science framework. Established implementation-outcome taxonomies distinguish acceptability, adoption, appropriateness, feasibility, fidelity, implementation cost, penetration, and sustainability, whereas the RE-AIM framework emphasizes reach, effectiveness, adoption, implementation, and maintenance [35,36]. These concepts allow each stage of the Continuum to be paired with observable indicators, including reach of risk communication and testing offers; uptake and completion of quality-assured measurement; acceptability and appropriateness of interpretation and referral; feasibility, time, and cost of mitigation planning; mitigation adoption and fidelity; and post-mitigation effectiveness and maintenance. The indicators are illustrative rather than prescriptive and require adaptation to the regulatory, housing, socioeconomic, and health-system context in which the pathway is evaluated.

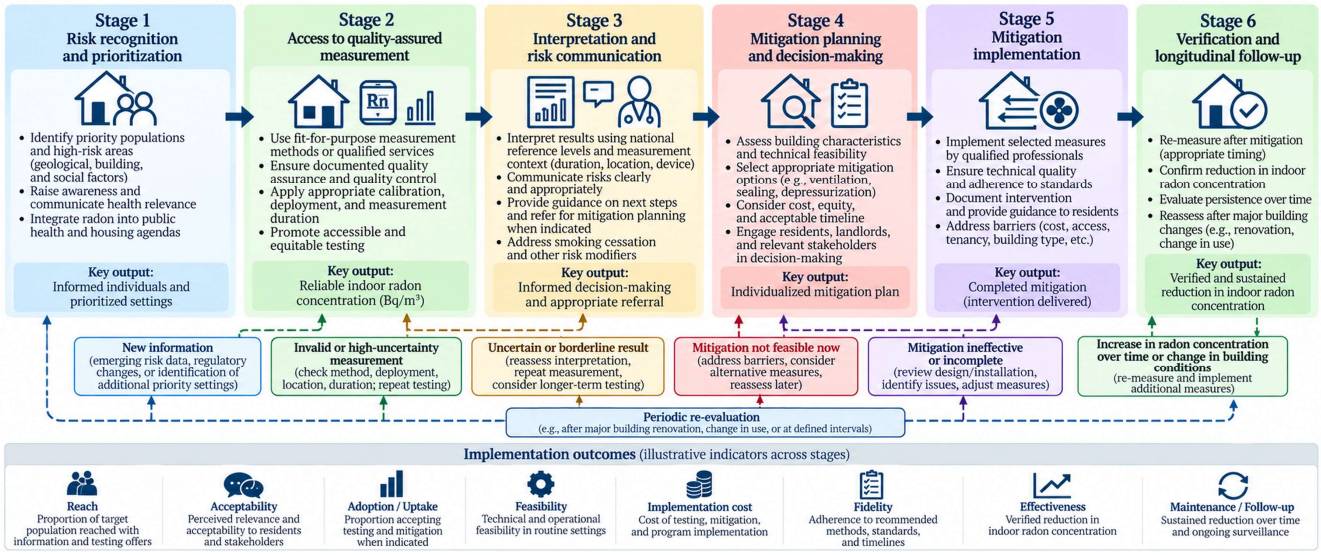


Figure 1. The Radon Prevention Continuum: a six-stage implementation pathway with feedback loops. Solid arrows indicate the primary sequence from risk recognition to verified and sustained reduction in indoor radon concentration. Dashed feedback loops indicate return to earlier stages when measurements are invalid or uncertain, mitigation is infeasible or ineffective, building conditions change, or radon concentrations increase during follow-up. Implementation outcomes are illustrative and should be adapted to local regulatory, housing, and health-system contexts. Abbreviations: Rn, radon; Bq/m^3, becquerels per cubic meter.

Table 1. Proposed Radon Prevention Continuum.

Stage	Core Question	Primary Output	Potential Point of Failure	Illustrative Implementation Indicator
1. Risk recognition and prioritization	Who or what should be assessed?	Decision to measure	Risk remains unrecognized or maps generate false reassurance	Reach of prioritized populations/settings; proportion offered measurement
2. Quality-assured measurement	What is the actual indoor concentration?	Quality-assured and interpretable indoor-radon measurement	Limited access, inappropriate detector, or invalid measurement protocol	Testing uptake and completion; feasibility, turnaround time, accessibility, and cost
3. Interpretation and communication	What does the result mean?	Interpreted result linked to a decision pathway	Numerical result without understandable or actionable guidance	Proportion receiving an interpretable result and documented next step; acceptability and comprehension
4. Mitigation planning	How can indoor radon concentration be reduced?	Technically appropriate mitigation plan	No qualified referral, infeasible plan, or inappropriate intervention	Proportion of actionable results linked to a mitigation plan; feasibility, time, and projected cost
5. Mitigation implementation	Can the intervention be completed?	Completed radon-mitigation intervention	Cost, tenure, access, logistical, or technical barriers	Mitigation uptake and completion; implementation fidelity, actual cost, and barriers to completion
6. Verification and follow-up	Did the intervention work?	Verified and sustained reduction in indoor radon concentration	No repeat measurement, ineffective mitigation, or loss of effectiveness over time	Change in indoor radon concentration after intervention; proportion with sustained reduction at follow-up

Cross-cutting domains: environmental health literacy, equity, quality assurance, risk communication, intersectoral coordination, governance, and evaluation.

5.1. Stage 1—Risk Recognition and Prioritization

Stage 1 establishes whether assessment should be pursued and where initial resources should be directed. At the population level, geological information, previous measurements, building characteristics, occupancy patterns, and high-occupancy settings such as schools, childcare facilities, hospitals, and workplaces may inform prioritization [1,13,33]. Prioritization should not be confused with individual exposure determination: neighboring dwellings may have substantially different concentrations, and a lower-risk map category does not establish that a particular home is radon-free [1,33].

Within health care, an environmental history may identify concern, previous testing, residence in a higher-potential area, prolonged occupancy in ground-contact spaces, or relevant occupational exposure. Its purpose is to recognize context and facilitate appropriate referral, not to diagnose radon exposure from clinical information alone. The primary output of Stage 1 is a justified decision to proceed to measurement.

5.2. Stage 2—Access to Quality-Assured Measurement

Measurement converts an imperceptible environmental hazard into quantifiable information and is therefore the critical transition from generalized risk to dwelling-specific assessment. Measurement quality depends on detector performance, calibration, placement, duration, environmental conditions, data handling, and adherence to standardized procedures [1,14,22]. Longer-duration measurements are generally more informative for estimating annual mean concentration because indoor radon varies over time [1]. Throughout this Perspective, quality-assured measurement denotes a result obtained using a fit-for-purpose detector or measurement service under documented quality-assurance and quality-control procedures, including appropriate calibration, deployment, measurement duration, procedural adherence, and traceable

data handling. The term does not imply that a single measurement method is appropriate for every monitoring objective.

Access is as important as technical quality. Even a rigorous measurement system has limited population impact if detectors or services are difficult to obtain, unaffordable, or geographically inaccessible. Access models may include public or subsidized detector programs, accredited laboratories, quality-assured commercial measurement services, university-community partnerships, citizen science, and targeted monitoring of public buildings [22,30]. The primary output of Stage 2 is a technically interpretable, quality-assured measurement, not detector distribution alone.

5.3. Stage 3—Interpretation and Risk Communication

A radon result requires interpretation. Individuals need to understand the measured concentration, its relation to the applicable reference level, the limitations and uncertainty of the measurement, and whether additional action is warranted. Reference levels should be communicated as tools for optimization and action rather than as biological thresholds, dose limits, or boundaries between risk and no risk [1,14]. Interpretation should integrate the concentration, measurement duration, detector and sampling location, applicable reference level, uncertainty, need for confirmation, and recommended next step. It should not imply that a household concentration alone defines an individual's cumulative exposure, radiation dose, or cancer risk.

An elevated result does not indicate that an occupant has lung cancer and does not, by itself, create an indication for radon-specific clinical testing. It identifies a preventable environmental exposure that should be evaluated and, when appropriate, reduced. The primary output of Stage 3 is a correctly understood result linked to an appropriate decision pathway.

5.4. Stage 4—Mitigation Planning

When mitigation is indicated, effective intervention requires identification of the relevant radon-entry and accumulation mechanisms within the specific building; simply advising occupants to increase ventilation is insufficient. Potential strategies include sealing soil-building interfaces, improving under-floor ventilation, installing radon sumps or soil-depressurization systems, modifying mechanical ventilation, and combining engineering approaches [1,14,22].

Mitigation planning requires expertise distinct from clinical risk communication. Health professionals may recognize and communicate the problem, but structural diagnosis and corrective intervention generally require building professionals, engineers, architects, radiation-protection specialists, or trained mitigation practitioners. The primary output of Stage 4 is a technically appropriate and feasible mitigation plan.

5.5. Stage 5—Mitigation Implementation

A technically appropriate mitigation plan does not reduce indoor radon concentration until it is implemented. Households may be unable or unwilling to proceed because of cost, limited access to qualified professionals, landlord-tenant relationships, disruption associated with construction, uncertainty about effectiveness, or competing priorities [26,37]. Programs should therefore evaluate not only where elevated concentrations are identified but also which households can realistically complete mitigation.

Mitigation is also linked to energy renovation. Some thermal-retrofit interventions may alter indoor radon concentrations when air exchange and soil-building pathways are not simultaneously managed [6,22]. Radon protection should therefore be integrated into building-energy policy rather than treated as being in conflict with energy efficiency and indoor environmental quality. The primary output of Stage 5 is a completed radon-mitigation intervention of appropriate technical quality.

5.6. Stage 6—Verification and Longitudinal Follow-Up

The continuum is not complete until intervention effectiveness has been evaluated. Post-mitigation measurement determines whether indoor radon concentration has decreased to the intended extent and converts mitigation from a presumed intervention into a measurable prevention outcome [1,22]. Counting remediation systems installed may overestimate success if their performance is not verified.

Longitudinal reassessment may be warranted after major structural modifications, ventilation changes, energy renovations, mitigation equipment failure, or subsequent increases in radon concentration. The primary outcome of Stage 6 is verified and sustained reduction in indoor radon concentration, used here as a proximal indicator of reduced residential exposure. This endpoint is more informative than awareness, testing, or mitigation considered in isolation because cumulative exposure, rather than participation in a program, is ultimately associated with lung-cancer risk.

6. Integrating Primary Health Care, Environmental Surveillance, and Radiation Protection

The Radon Prevention Continuum requires explicit division and coordination of responsibilities across sectors. Residential radon lies at the intersection of cancer prevention, environmental health, radiation protection, housing, and building science; no single professional group can reasonably perform all functions. A distributed model is therefore more appropriate: primary health care may contribute recognition, communication, referral, and continuity; environmental health surveillance may provide population and territorial intelligence; radiation-protection services and laboratories provide technical quality assurance; and built-environment professionals undertake structural assessment and mitigation [14,22,34].

The purpose of integration is not to expand professional scopes indefinitely, but to ensure continuity across sectors and prevent residents from being lost between disconnected services.

6.1. Primary Health Care as an Entry Point, Not a Radon Laboratory

Primary health care has characteristics that may make it a useful entry point into prevention, including longitudinal contact, preventive counseling, knowledge of household and occupational context, community outreach, and the ability to connect environmental concerns with established cancer-prevention strategies. Radon, however, remains rarely incorporated into routine primary-care communication. In a survey of family physicians in a high-radon US state, 67% did not inform patients about radon and 80% had never discussed the combined risk of radon and smoking [38].

The role of primary care should therefore remain bounded and practical. Depending on local epidemiology and infrastructure, professionals may identify environmental concerns or previous elevated measurements, ask selected environmental-history questions when relevant, explain the relationship between radon and lung cancer, reinforce that environmental measurement rather than symptoms determines household concentration, direct individuals to quality-assured testing, support smoking cessation, and maintain continuity while technical interventions are undertaken [38].

These functions are distinct from environmental diagnosis. A clinician cannot determine household radon concentration from symptoms, physical examination, housing appearance, or geographic location. Primary-care services should likewise not be expected to select calibration procedures, certify measurement validity, inspect foundations, or design depressurization systems. Primary care can recognize, communicate, refer, and follow; it should not replace radiological measurement, technical assessment, or building-mitigation expertise.

6.2. Environmental History as a Bridge Between Clinical Care and Prevention

A brief environmental history can connect clinical care with environmental prevention without estimating concentration. Relevant elements may include place and duration of residence, previous radon

testing, measurement duration and result, use of basements or other ground-contact rooms, major renovation, occupational exposure history, smoking status, and whether other household members share the same environment. These questions identify context and potential need for information or referral; they do not establish exposure [38].

Clinical documentation should preserve this distinction. Terms such as reported elevated household radon measurement, previous radon test, or radon measurement recommended are preferable to an unverified environmental diagnosis. This distinction is particularly important for patients with lung cancer who have never smoked: radon is an established etiological factor [4,5,23], but retrospective attribution of an individual tumor to a specific residence is usually not possible with confidence.

6.3. Environmental Health Surveillance as the Coordinating Layer

Environmental health surveillance can provide the population and territorial perspective that individual clinical care cannot. Potential functions include aggregating technically comparable data, identifying spatial patterns, prioritizing, providing technical guidance, setting communication standards, coordinating referrals, and evaluating preventive outcomes. Isolated household measurements have limited public health value if they remain fragmented; appropriately governed and quality-assured data may support geographic prioritization, assessment of public buildings, and prevention planning.

Data quality and governance are essential. Detector type, calibration, measurement duration, room location, season, building characteristics, and quality-control procedures influence interpretation [1,22]. Residential measurements may also reveal information about specific properties and affect housing perceptions or tenancy. Public-health systems should therefore protect identifiable household data and clearly distinguish area-level radon potential from the measured concentration of an individual dwelling.

6.4. Radiation Protection and the Built Environment

Radiation-protection expertise provides the technical backbone of the continuum, including measurement protocols, detector validation and calibration, quality assurance, uncertainty interpretation, dosimetric considerations, application of reference levels, criteria for repeat measurement, and regulatory guidance [14,22]. This technical layer reduces the risk of both underestimation and overinterpretation of exposure.

Reduction in indoor radon concentration is often achieved through intervention in the built environment. Radon mitigation may involve combinations of sealing, sub-slab or sub-membrane depressurization, radon sumps, crawl-space interventions, pressure management, and ventilation [1,14,22]. The appropriate strategy depends on the building and the dominant radon-entry pathway; no single intervention is universally applicable. The health objective is cancer prevention, but the effective intervention may occur in the building foundation or ventilation system.

6.5. Closing the Referral Gap and Avoiding Medicalization

Even where relevant expertise exists, prevention may fail if residents and professionals lack a clear pathway between sectors. An effective system should identify who validates a measurement, whether confirmation is required, who assesses the building, who designs and implements mitigation, whether financial support is available, who verifies effectiveness, and where data are recorded. Individuals may enter the continuum through different routes, including health care, occupational services, universities, schools, independent testing, or public campaigns, but these entry points should connect to an interoperable pathway [34].

Integration should also avoid medicalizing an environmental exposure. An elevated household measurement is an exposure finding, not a medical diagnosis. It does not establish that an occupant has lung cancer, and no radon-specific clinical test can determine whether a future malignancy will develop. Clinical

symptoms and eligibility for lung-cancer screening should continue to follow established clinical pathways. Radon prevention is primarily upstream: the objective is to reduce exposure before disease develops.

7. Brazil as an Implementation Case: From Regulatory Recognition to Population Prevention

Brazil is examined here as an implementation case in transition, not as a representative or mature national radon-control model. The rationale is analytical: Brazil combines (i) a recently adopted residential-radon reference-level provision with deferred implementation; (ii) public technical initiatives aimed at standardizing measurement, quality assurance, and national assessment; and (iii) a universal health system whose primary-care and surveillance policies are territorially organized. This combination provides a useful setting for examining what must occur between regulatory recognition and an operational prevention pathway. The case is illustrative rather than evaluative: the present Perspective does not assess a mature national radon program because the cited regulatory and institutional sources instead describe a system that is still under development [15–20].

Brazil also has substantial geological, climatic, socioeconomic, and housing heterogeneity. These features increase the need for context-sensitive implementation while limiting straightforward extrapolation from regional measurements or radon-control models developed elsewhere. The Brazilian case is therefore used to examine the logic and operational requirements of the Radon Prevention Continuum, not to imply that current Brazilian practice already follows the proposed pathway.

7.1. Current Regulatory Status of Residential Radon in Brazil

The current ANSN 3.01—Basic Requirements for Radiation Protection and Radiological Safety of Radiation Sources, established through Resolution CNEN 344/25, includes a specific provision addressing public exposure to radon in indoor environments. Article 139 establishes a reference level of 300 Bq/m^3 for residences and other high-occupancy areas, including childcare facilities, hospitals, and workplaces, and provides that actions to reduce radon concentrations should be justified and optimized, and that relevant information on exposure and health risks should be made available to the public and other stakeholders [15].

A critical regulatory nuance is that this provision is not yet in force. Resolution CNEN 344/25 was published in July 2025, and Article 151 states that Article 139 will take effect four years after publication, in July 2029 [15]. Brazil in 2026 should therefore be characterized as being in a regulatory transition period. The 300 Bq/m^3 residential reference level has been adopted in regulatory text, but its relevant provision has deferred effect; this should not be interpreted as evidence that a comprehensive operational residential-radon program is already in place.

Regulatory recognition is necessary but does not specify how residents obtain quality-assured measurements, how elevated results are confirmed and communicated, who assesses buildings, how mitigation is financed and delivered, or how post-mitigation effectiveness is verified. These operational transitions constitute the implementation problem addressed by the Radon Prevention Continuum.

7.2. Current Technical and Institutional Foundations

Public technical initiatives indicate active capacity building. A CNEN project entitled Plano de Ação Nacional de Radônio identifies objectives that include defining a national strategy for indoor-radon surveys, harmonizing previous Brazilian measurements, establishing a quality-assurance and quality-control chain, and making an ISO/IEC 17025-accredited procedure for indoor-radon measurement available in Brazil [19]. A related CDTN/CNEN project describes development of a standardized indoor-monitoring protocol, pilot monitoring in public childcare facilities, socioeconomic evaluation of reference levels, a measurement quality-control system, and a network of laboratories trained in radon measurement [20].

These sources provide evidence of institutional activity but should be interpreted as infrastructure-building objectives and pilot initiatives rather than as evidence of nationwide operational coverage. The cited projects focus primarily on measurement standardization, quality assurance, laboratory capacity, and pilot assessment. They do not demonstrate universal access to testing, a nationally integrated referral route after elevated results, nationwide mitigation capacity, or established financing mechanisms for residential remediation. The current status is therefore better characterized as development of technical foundations for a future national strategy [19,20].

7.3. Health-System Context: Relevance and Limits of the SUS

The Brazilian Unified Health System (SUS) has organizational characteristics potentially relevant to environmental cancer prevention, particularly territorial primary care and health surveillance. The National Primary Care Policy defines primary care as encompassing individual, family, and collective actions involving health promotion, prevention, protection, care, and surveillance within defined territories, while the National Health Surveillance Policy describes a continuous process of collecting, analyzing, and disseminating information to guide measures addressing determinants, risks, and health events [16,17]. Federal guidance also emphasizes integrating primary care and health surveillance and using territorial planning [18].

These policies provide an organizational rationale for considering the SUS as a potential entry point for communication, referral, and territorial coordination; they do not constitute a radon-specific care pathway and should not be interpreted as evidence that primary-care services currently perform residential-radon assessment. Brazilian studies have reported limited familiarity with environmental health surveillance and fragile integration between Family Health teams and surveillance structures in routine practice [39,40]. Any future role for primary care in radon prevention would therefore require training, defined referral protocols, access to technical reference services, intersectoral agreements, and explicit consideration of workload and local capacity.

7.4. Proposed Application of the Radon Prevention Continuum in Brazil

The architecture described in this subsection is proposed for future pilot testing and is not presented as an existing national policy or established SUS pathway. The six stages of the Continuum could be adapted to Brazilian institutions without creating a parallel health care structure, provided that responsibilities remain distributed across health, radiation-protection, laboratory, housing, engineering, and local-government sectors.

Stage 1 could integrate existing measurements, geological information, building characteristics, occupancy patterns, and research evidence to prioritize territories and high-occupancy settings. Primary-care and community-health teams in selected pilot areas could support risk communication and identify concerns or previous measurements but should not classify dwellings by presumed radon concentration.

Stage 2 would require standardized protocols, fit-for-purpose detectors or measurement services, qualified laboratories, quality assurance, and accessible testing. Current CNEN/CDTN initiatives address several of these technical foundations [19,20]. Initial pilots could prioritize settings in which technical support already exists and where the complete pathway, rather than detector distribution alone, can be evaluated.

Stage 3 would require a technically defined route for interpretation and communication. Primary-care professionals should not be expected to validate detector performance or conduct dosimetric assessment independently; technically significant or uncertain results should be linked to designated radiation-protection, laboratory, university, or other reference services. Health professionals could then support communication of health relevance, smoking cessation when applicable, and continuity through subsequent steps.

Stages 4 and 5, mitigation planning and implementation, would require coordination with engineering, architecture, construction, housing, labor, education, and local government. The appropriate structural intervention depends on the building, whereas feasibility depends on access to qualified expertise, cost, tenure, household resources, and local construction practices. Stage 6 would require repeat measurement after intervention and, with appropriate governance, could contribute anonymized information to surveillance and program evaluation.

7.5. Implementation Constraints and Prerequisites

The feasibility of this proposed architecture should not be assumed. First, Brazil does not yet have nationally representative residential-radon data to characterize exposure distributions across all regions and housing types; the technical projects cited above are directed toward harmonization, protocol development, pilot monitoring, and expanding measurement capacity [19,20]. Access, turnaround time, quality assurance, and geographic distribution of measurement services therefore remain implementation variables rather than established conditions.

Second, measurement capacity must be matched by a realistic pathway to mitigation. The public documents cited here describe substantial work on measurement and quality infrastructure but do not specify a nationwide residential-mitigation referral system, financing mechanism, or uniformly available technical workforce for building remediation. This distinction is critical because expanding testing without parallel capacity to interpret and reduce elevated concentrations could identify hazards while leaving households unable to act. Costs, landlord-tenant relationships, housing informality, building typologies, and unequal household resources may therefore generate attrition at precisely the stages where mitigation is required [26,37].

Third, implementation would require governance across institutions whose current mandates and workflows are not organized around a single residential-radon pathway. Radiation-protection authorities, laboratories, health surveillance, primary care, housing and building sectors, municipalities, universities, and private technical services may each hold part of the required capacity. Data governance and protection of household-level information would also require explicit definition. Within the SUS, adding radon-related communication or referral without training, technical support, and workload planning could create responsibility without operational capacity. These constraints support empirical testing of the Continuum rather than an assumption of immediate national scalability.

7.6. Pilot Testing Before Scale-Up

A national strategy need not begin with universal household testing. Large-scale screening before measurement quality, referral structures, mitigation capacity, financing mechanisms, and data governance are established could generate demand that existing systems cannot adequately address. A more defensible approach is regional pilot implementation selected according to existing radon evidence, geological susceptibility, measurement infrastructure, population characteristics, housing context, and institutional capacity [19,20].

Each pilot should evaluate the entire Continuum rather than detector distribution alone. Relevant outcomes include reach of priority populations; availability and cost of quality-assured measurement; proportion completing quality-assured measurement; time from an elevated result to technical interpretation; proportion proceeding to building assessment and mitigation; mitigation completion and actual cost; change in indoor radon concentration after intervention; maintenance of concentration reduction; socioeconomic differences in completion; unintended effects such as anxiety or housing stigma; and feasibility of referral and data-sharing arrangements. Such pilots would convert the Radon Prevention Continuum from a conceptual pathway into an empirically testable implementation model.

7.7. Transferability Beyond Brazil

Brazil should not be assumed to represent other countries. Potentially transferable observations are process based rather than institutional: regulatory recognition may advance faster than implementation capacity; a reference level alone does not create access to measurement or mitigation; testing has limited preventive value unless it is connected to interpretation, mitigation, verified reduction in indoor radon concentration, and follow-up; cost and equity influence whether households complete the pathway; and pilot evaluation should examine the complete sequence rather than a single intervention.

Other features are specifically Brazilian and should not be transferred without adaptation. These include the territorial organization of the SUS and Family Health Strategy, the relationship between primary care and health surveillance, the ANSN/CNEN regulatory structure and deferred entry into force of Article 139, the distribution of national laboratory and technical capacity, and Brazilian housing, tenure, and governance arrangements [15–20,39,40]. Countries with different health systems, building stocks, rental regulation, laboratory infrastructure, or mitigation markets may require different entry points and allocation of responsibilities. The continuum is therefore intended as an adaptable implementation logic, not a universal institutional blueprint.

8. Research and Implementation Priorities for the Next Decade

The evidence linking residential radon to lung cancer is sufficiently mature to justify prevention, but important uncertainties continue to limit precision, implementation, and equity. Future research should move beyond repeatedly establishing carcinogenicity and increasingly address the mechanisms, measurements, behaviors, and systems that determine whether exposure is actually reduced [9,11,12,22,34].

8.1. Improving Individual Exposure Assessment and Biological Dosimetry

A persistent challenge is the distinction between environmental concentration and individual cumulative exposure. A measurement obtained in one room over a limited period cannot reconstruct lifetime exposure: individuals move between residences, workplaces, schools, and other buildings; concentrations vary seasonally; occupancy patterns differ; and structural or ventilation changes alter concentrations over time [1,11]. Research priorities therefore include repeated and longer-duration measurements, residential histories, time-activity patterns, uncertainty quantification, and methods that integrate environmental, behavioral, and dosimetric information.

Biological dosimetry remains exploratory. Candidate approaches include chromosomal and DNA-damage markers, oxidative or epigenetic signals, and other molecular signatures [9]. Priorities include validation, reproducibility, exposure specificity, dose-response characterization, temporal stability, and clinical relevance. Until these criteria are met, biomarker research should complement, not replace, environmental measurement and should not be used to determine whether a household requires testing or mitigation.

8.2. Clarifying Biological Susceptibility

Individuals exposed to similar radon concentrations may not experience identical biological consequences. Smoking is the best-established modifier of absolute lung-cancer risk, whereas proposed genetic, DNA-repair, oxidative, inflammatory, and other susceptibility pathways remain incompletely characterized [12,41]. Future studies should link well-characterized environmental exposure with smoking history, biological markers, and long-term outcomes before such factors are incorporated into risk stratification. At present, these mechanisms define research priorities rather than clinical criteria for testing, mitigation, or individual causal attribution.

8.3. Improving Risk Models for Never-Smokers and Combined Exposures

Risk models should increasingly account for population heterogeneity, including never-smoking status, former smoking, smoking intensity, age at exposure, sex, occupational exposures, air pollution, and other environmental carcinogens. An exposome perspective may be particularly useful because lung-cancer risk arises from interacting environmental, occupational, behavioral, and biological determinants rather than from isolated exposures. Radon prevention remains relevant regardless of smoking status and should complement tobacco-control strategies [3–5,11,23].

8.4. Moving Mitigation Research from Technical Efficacy to Real-World Effectiveness

Radon mitigation technologies are well established, but technical efficacy and population-level effectiveness are not equivalent. Public-health impact depends on whether households complete testing, receive and understand results, obtain appropriate mitigation, can afford and implement the intervention, and verify effectiveness. Research should therefore evaluate real-world completion, time from testing to intervention, magnitude and persistence of concentration reduction, maintenance and failure rates, interactions with energy-efficiency retrofits, household acceptance, and barriers to implementation [22,26,37].

8.5. Embedding Health Economics and Equity

Economic evidence will become increasingly important as countries determine the scale and financing of testing and mitigation. Cost-effectiveness depends on baseline radon prevalence, intervention thresholds, participation rates, mitigation uptake, housing characteristics, costs, and modeling assumptions. A Canadian analysis found mitigation in existing housing a plausible policy option and showed that cost-effectiveness improves as testing and mitigation rates increase [37]. Future analyses should compare universal and targeted testing, prevention in new construction versus remediation of existing homes, public subsidies versus household financing, and alternative reference-level strategies.

Equity should be treated as an explicit dimension of economic evaluation. A program may appear efficient while systematically excluding households that cannot afford testing or mitigation. Distributional outcomes should therefore be considered alongside conventional cost-per-QALY or life-year metrics.

8.6. Developing Implementation Science and Evidence from Underrepresented Regions

A major remaining gap is understanding how radon-prevention systems perform under real-world conditions. RadoNorm demonstrates the value of stakeholder engagement, co-creation, tailored communication, citizen science, and interaction among researchers, regulators, building professionals, and communities [34]. In this Perspective, implementation science is used in a bounded and testable sense: to determine whether the linked components of radon prevention reach intended populations, are adopted by relevant organizations and households, are delivered with appropriate quality, remain feasible and affordable, produce verified reduction in indoor radon concentration, and are sustained over time [35,36]. Future evaluation of the Radon Prevention Continuum should therefore measure both technical outcomes and implementation outcomes, including reach, acceptability, appropriateness, adoption or uptake, feasibility, fidelity, implementation cost, penetration into routine systems, and sustainability. Stage-specific assessment can identify where attrition occurs and which implementation strategies are required to improve pathway completion.

Geographic representation is another important limitation of the evidence base. Much of the strongest epidemiological and implementation evidence derives from Europe and North America, whereas Latin America, Africa, and other regions often lack nationally representative residential datasets, long-term cohorts, standardized mitigation studies, and integrated surveillance systems. Brazil has meaningful regional evidence, but available measurements cannot yet be interpreted as a complete national exposure

distribution [6]. International research should prioritize locally generated exposure and implementation data rather than assume direct transferability of policy models across climates, building typologies, socioeconomic conditions, and health systems.

8.7. Defining Meaningful Endpoints

The field requires greater precision in defining successful prevention. Awareness, intention to test, completed testing, elevated measurements identified, mitigation recommended, mitigation completed, post-mitigation concentration, cumulative exposure reduction, and ultimately prevented lung-cancer burden are related but non-equivalent outcomes. A campaign may increase awareness without reducing exposure; a testing program may identify many elevated homes but achieve limited prevention if mitigation uptake is low; and a mitigation program may install systems without demonstrating effectiveness if follow-up measurement is absent.

For this reason, the Radon Prevention Continuum identifies verified, sustained reductions in indoor radon concentration as the most immediate meaningful implementation endpoint and a proximal indicator of reduced residential exposure. Lung-cancer incidence and mortality remain the ultimate health outcomes, but their long latency makes them impractical as the sole measures of short- and medium-term program effectiveness.

These priorities are summarized in Table 2.

Table 2. Priority research agenda for translating radon evidence into lung-cancer prevention.

Research Domain	Key Unresolved Question	Priority Research Outcome
Exposure science	How can long-term individual exposure be estimated more accurately?	Improved cumulative-exposure assessment
Dosimetry	How do environmental concentrations translate into biologically effective dose?	Better dose models
Biomarkers	Can reproducible markers of internal dose or early biological effect be identified?	Validated biomarker panels
Radiobiology	Which molecular pathways modify susceptibility to alpha-particle carcinogenesis?	Mechanistic risk stratification
Epidemiology	How does risk vary by smoking status, genetics, age, sex, and co-exposures?	Refined risk models
Measurement	How can quality-assured testing become broadly accessible?	Scalable and quality-assured measurement
Mitigation	What works under real-world building and socioeconomic conditions?	Sustained reduction in indoor radon concentration
Communication	Which strategies move people from awareness to action?	Completion of the prevention pathway
Health economics	Which strategies provide efficient and equitable prevention?	Context-specific economic models
Implementation science	Where and why are people lost along the pathway?	Reduced attrition across stages
Equity	Who remains unable to test or mitigate?	Equitable access
Surveillance	How can household data inform population action while protecting privacy?	Actionable territorial intelligence

9. Limitations of the Proposed Framework

The Radon Prevention Continuum should be interpreted as a conceptual and implementation-oriented framework rather than as an empirically validated model. Its six stages were derived through narrative synthesis of international guidance and supporting literature, not through a systematic review, formal consensus process, Delphi procedure, or prospective validation study. The proposed stage boundaries, sequence, feedback loops, and implementation indicators therefore require empirical testing before they can be treated as validated components of a radon-prevention program.

The implementation indicators adapted from established implementation-science constructs are illustrative rather than a radon-specific validated implementation-outcome set. Their relevance, definitions, denominators, and thresholds will vary according to regulatory arrangements, housing stock, measurement infrastructure, mitigation capacity, financing, data systems, and health-system organization. Likewise, the institutional roles described in this Perspective are intended to support coordination and should not be interpreted as universal professional responsibilities.

Transferability is further limited by the geographic concentration of the evidence base. Much of the available epidemiological, communication, and implementation literature derives from Europe and North America, whereas many other regions have less complete residential-exposure data and fewer evaluations of mitigation pathways. Finally, verified and sustained reduction in indoor radon concentration is a proximal implementation endpoint and an indicator of reduced exposure rather than direct evidence of prevented lung-cancer cases; the long latency of lung cancer limits the feasibility of using incidence or mortality as short- or medium-term indicators of program effectiveness. The Continuum is therefore intended as a testable organizing framework that complements, rather than replaces, national technical standards, regulatory requirements, and locally validated implementation strategies.

10. Conclusions

Residential radon is an established but incompletely addressed cause of lung cancer. Radiobiological, dosimetric, and epidemiological evidence demonstrates that inhaled radon progeny expose respiratory epithelial cells to high-LET alpha radiation capable of producing complex DNA damage and contributing to pulmonary carcinogenesis [1–5,9,11,12]. The principal translational question is therefore no longer whether radon is a relevant environmental carcinogen, but how accumulated evidence can be converted into effective and equitable prevention.

This challenge extends beyond environmental measurement. Radon is imperceptible to human senses, varies substantially between buildings, and cannot be inferred reliably from geography or construction characteristics alone. Measurement is essential, but testing in isolation does not reduce cancer risk. Prevention requires a connected pathway from recognition to quality-assured measurement, interpretable results, appropriate mitigation, and verification that indoor radon concentration has been reduced [1,14,22].

The Radon Prevention Continuum organizes this pathway into six linked stages: risk recognition and prioritization, quality-assured measurement, interpretation and risk communication, mitigation planning, mitigation implementation, and verification with longitudinal follow-up. The model is iterative rather than strictly linear; feedback loops allow return to earlier stages when measurements are uncertain, mitigation is ineffective, building conditions change, or concentrations increase during follow-up. Environmental health literacy, equity, quality assurance, governance, and intersectoral coordination operate across the continuum. The framework therefore shifts the immediate endpoint of prevention from awareness or detector distribution to verified, sustained reductions in indoor radon concentration as a proximal indicator of reduced exposure.

This approach necessarily distributes responsibility across sectors. Health professionals may contribute environmental history, risk communication, referral, and continuity, but should not replace specialists responsible for radiological measurement or building mitigation. Radiation-protection authorities provide standards and quality assurance; laboratories support technically valid exposure assessment; environmental surveillance can integrate territorial information; and built-environment professionals translate exposure data into structural interventions [14,22,34,38–40].

Brazil illustrates a regulatory and institutional transition rather than a completed model. A residential reference-level provision has been adopted, but Article 139 is scheduled to take effect in 2029, while public technical initiatives are developing measurement, quality-assurance, and assessment capacity [15,19,20]. These developments create an opportunity for pilot implementation, but substantial uncertainties remain

regarding national measurement coverage, referral and mitigation capacity, financing, professional training, intersectoral governance, and integration with territorial health structures. The Brazilian case therefore underscores the need to distinguish regulatory recognition from operational prevention and to evaluate the complete pathway before scale-up.

Ultimately, radon-prevention performance should not be judged solely by awareness, detector distribution, or the number of elevated measurements identified. The most meaningful proximal outcome is whether indoor radon concentration has been reliably measured, appropriately interpreted, reduced through effective mitigation, and subsequently verified, thereby providing evidence that residential exposure has been reduced before disease develops.

Statement of the Use of Generative AI and AI-Assisted Technologies in the Writing Process

During the preparation of this manuscript, the authors used ChatGPT (GPT-6, OpenAI) for structural organization and language refinement and OpenAI image-generation tools to assist in preparing the graphical representation of the authors' conceptual framework in Figure 1. After using these tools, the authors reviewed, edited, and verified the scientific content, references, interpretations, and graphical elements as needed and take full responsibility for the content of the published article.

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Author Contributions

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Ethics Statement

Not applicable. This Perspective does not involve human participants, identifiable personal data, animal experimentation, or intervention research.

Informed Consent Statement

Not applicable.

Data Availability Statement

No original datasets were generated for this Perspective. The evidence discussed derives from published scientific literature and publicly available regulatory and institutional documents cited in the References.

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Declaration of Competing Interest

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

References

1. World Health Organization. *WHO Handbook on Indoor Radon: A Public Health Perspective*; World Health Organization: Geneva, Switzerland, 2009.
2. International Agency for Research on Cancer. Radiation. In *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 100D*; IARC: Lyon, France, 2012.
3. Darby S, Hill D, Auvinen A, Barros-Dios JM, Baysson H, Bochicchio F, et al. Radon in homes and risk of lung cancer: Collaborative analysis of individual data from 13 European case-control studies. *BMJ* **2005**, *330*, 223. DOI:10.1136/bmj.38308.477650.63
4. Urrutia-Pereira M, Chatkin JM, Chong-Neto HJ, Solé D. Radon exposure: A major cause of lung cancer in nonsmokers. *J. Bras. Pneumol.* **2023**, *49*, e20230210. DOI:10.36416/1806-3756/e20230210
5. Ruano-Ravina A, Martin-Gisbert L, Kelsey K, Pérez-Ríos M, Candal-Pedreira C, Rey-Brandariz J, et al. An overview on the relationship between residential radon and lung cancer: What we know and future research. *Clin. Transl. Oncol.* **2023**, *25*, 3357–3368. DOI:10.1007/s12094-023-03308-0
6. Gabriel C, Marins DSS, Costa JG, Ruano-Raviña A, Campos RFF, Adami ER. Building airtightness and radon exposure: International evidence and implications for socio-environmental management in Brazil. *Rev. Gest. Soc. Ambient.* **2026**, *20*, e014602. DOI:10.24857/rgsa.v20n8-011
7. Marins DSS, Costa JG, Campos RFF, Godoy CMT, Somensi LB, Baldicera AK, et al. Environmental exposure, vulnerability and public health: Radon as a challenge to sustainable human development. *Aposta Rev. Cienc. Soc.* **2026**, *24*, e906. DOI:10.23900/ra.v24i114.906
8. National Research Council. *Health Effects of Exposure to Radon: BEIR VI*; National Academies Press: Washington, DC, USA, 1999. DOI:10.17226/5499
9. Rathebe PC, Kholopo M. Radon-Induced Radiation Biomarkers: A Scoping Review from Exposure Dosimetry to Early Biological Effects on the Lung. *Int. J. Mol. Sci.* **2026**, *27*, 4391. DOI:10.3390/ijms27104391
10. Hofmann W, Crawford-Brown DJ, Fakir H, Caswell RS. Energy deposition, cellular radiation effects and lung cancer risk by radon progeny alpha particles. *Radiat. Prot. Dosimetry* **2002**, *99*, 453–456. DOI:10.1093/oxfordjournals.rpd.a006830
11. Liu Y, Xu Y, Xu W, He Z, Fu C, Du F. Radon and lung cancer: Current status and future prospects. *Crit. Rev. Oncol. Hematol.* **2024**, *198*, 104363. DOI:10.1016/j.critrevonc.2024.104363
12. Kashkinbayev Y, Kazhiyakhmetova B, Altaeva N, Bakhtin M, Tarlykov P, Saifulina E, et al. Radon Exposure and Cancer Risk: Assessing Genetic and Protein Markers in Affected Populations. *Biology* **2025**, *14*, 506. DOI:10.3390/biology14050506
13. World Health Organization. *Compendium of WHO and Other UN Guidance in Health and Environment, 2024 Update*; World Health Organization: Geneva, Switzerland, 2024. ISBN 978-92-4-009538-0
14. International Commission on Radiological Protection. Radiological Protection against Radon Exposure. ICRP Publication 126. *Ann. ICRP* **2014**, *43*, 5–73. DOI:10.1177/0146645314542212
15. Autoridade Nacional de Segurança Nuclear. *Norma ANSN 3.01—Requisitos Básicos de Radioproteção e Segurança Radiológica de Fontes de Radiação. Resolução CNEN 344/25*; Diário Oficial da União: Brasília, Brazil, 2025. Available online: https://www.gov.br/cnen/pt-br/acesso-rapido/normas/grupo-3/ansn-3-01-requisitos-basicos-de-radioprotecao-e-seguranca-radiologica-de-fontes-de-radiacao.pdf (accessed on 11 September 2026).
16. Brasil. Ministério da Saúde. *Portaria nº 2.436, de 21 de Setembro de 2017. Aprova a Política Nacional de Atenção Básica*; Diário Oficial da União: Brasília, Brazil, 2017. Available online: https://bvs.saude.gov.br/bvs/saudelegis/gm/2017/prt2436_22_09_2017.html (accessed on 11 September 2026).
17. Brasil. Conselho Nacional de Saúde. *Resolução nº 588, de 12 de Julho de 2018. Institui a Política Nacional de Vigilância em Saúde*; Diário Oficial da União: Brasília, Brazil, 2018. Available online: https://www.gov.br/conselho-nacional-de-saude/pt-br/atos-normativos/resolucoes/2018/resolucao-no-588.pdf/view (accessed on 11 September 2026).
18. Brasil Ministério da Saúde, Secretaria de Atenção à Saúde, Secretaria de Vigilância em Saúde. *Guia Política Nacional de Atenção Básica: Módulo 1—Integração Atenção Básica e Vigilância em Saúde*; Ministério da Saúde: Brasília, Brazil, 2018.
19. Comissão Nacional de Energia Nuclear. Plano de Ação Nacional de Radônio. In *Portfólio de Projetos para Captação de Recursos*; CNEN: Rio de Janeiro, Brazil, 2025.

20. Comissão Nacional de Energia Nuclear, Centro de Desenvolvimento da Tecnologia Nuclear. Desenvolvimento de Metodologias e Estratégias de Monitoramento do Gás Radônio em Ambientes Fechados Para Bases de um Plano de Ação Nacional de Controle de Radônio. Relação de Projetos do Subprograma de Capacitação Institucional (PCI). CNEN/CDTN: Brazil. 2024. Available online: https://www.gov.br/cdtn/pt-br/assuntos/noticias/cnen-publica-edital-pci-cnen-ndeg-01-2024-do-programa-de-capacitacao-institucional-pci/anexo-i-relacao-dos-projetos.pdf (accessed on 11 September 2026).
21. International Commission on Radiological Protection. Occupational Intakes of Radionuclides: Part 3. ICRP Publication 137. *Ann. ICRP* **2017**, *46*, 1–486. DOI:10.1177/0146645317734963
22. John W, Birschwilks M, Février L, Madas B, Naarala J, Nieminen V, et al. Contributions of the RadoNorm project to European and international radiation protection research. *Radiat. Environ. Biophys.* **2025**, *64*, 561–579. DOI:10.1007/s00411-025-01156-w
23. Cheng ES, Egger S, Hughes S, Weber M, Steinberg J, Rahman B, et al. Systematic review and meta-analysis of residential radon and lung cancer in never-smokers. *Eur. Respir. Rev.* **2021**, *30*, 200230. DOI:10.1183/16000617.0230-2020
24. Cori L, Curzio O, Donzelli G, Bustaffa E, Bianchi F. A systematic review of radon risk perception, awareness, and knowledge: Risk communication options. *Sustainability* **2022**, *14*, 10505. DOI:10.3390/su141710505
25. Timmons S, Lunn PD. Behaviourally-informed household communications increase uptake of radon tests in a randomised controlled trial. *Sci. Rep.* **2023**, *13*, 20401. DOI:10.1038/s41598-023-47832-z
26. Irvine JL, Simms JA, Cholowsky NL, Pearson DD, Peters CE, Carlson LE, et al. Social factors and behavioural reactions to radon test outcomes underlie differences in radiation exposure dose, independent of household radon level. *Sci. Rep.* **2022**, *12*, 15471. DOI:10.1038/s41598-022-19499-5
27. Finn S, O'Fallon L. The emergence of environmental health literacy—From its roots to its future potential. *Environ. Health Perspect.* **2017**, *125*, 495–501. DOI:10.1289/EHP.1409337
28. Lindsey M, Chen SR, Ben R, Manoogian M, Spradlin J. Defining environmental health literacy. *Int. J. Environ. Res. Public Health* **2021**, *18*, 11626. DOI:10.3390/ijerph182111626
29. Gray KM. From content knowledge to community change: A review of representations of environmental health literacy. *Int. J. Environ. Res. Public Health* **2018**, *15*, 466. DOI:10.3390/ijerph15030466
30. Stanifer S, Hoover AG, Rademacher K, Rayens MK, Haneberg W, Hahn EJ. Citizen Science Approach to Home Radon Testing, Environmental Health Literacy and Efficacy. *Citiz. Sci. Theory Pract.* **2022**, *7*, 26. DOI:10.5334/cstp.472
31. Negreira-Rey MC, Vázquez-Herrero J, Forja-Pena T. Radon Risk Communication through News Stories: A Multi-Perspective Approach. *Int. J. Environ. Res. Public Health* **2024**, *21*, 1302. DOI:10.3390/ijerph21101302
32. Apers S, Symons M, Vandebosch H, Perko T. From Attention to Intention: Evaluating the Effectiveness of Social Media and Website Strategies in the Radon Buster Campaign. *J. Health Commun.* **2025**, *30*, 488–504. DOI:10.1080/10810730.2025.2562844
33. Slavik CE, Chapman DA, Trieu J, McVea DA, Fish CS, Peters E. Do radon risk maps encourage residential testing behaviour? Evidence from an experimental study in Canada. *Public Health* **2026**, *251*, 106103. DOI:10.1016/j.puhe.2025.106103
34. Železnik N, Horvat B, John W, Schofield PN. Lessons learned on stakeholder engagement in radiation protection, and communication and dissemination of results from the RadoNorm project. *Radiat. Environ. Biophys.* **2026**, *65*, 121–133. DOI:10.1007/s00411-025-01184-6
35. Proctor E, Silmere H, Raghavan R, Hovmand P, Aarons G, Bunger A, et al. Outcomes for implementation research: Conceptual distinctions, measurement challenges, and research agenda. *Adm. Policy Ment. Health* **2011**, *38*, 65–76. DOI:10.1007/s10488-010-0319-7
36. Glasgow RE, Vogt TM, Boles SM. Evaluating the public health impact of health promotion interventions: The RE-AIM framework. *Am. J. Public Health* **1999**, *89*, 1322–1327. DOI:10.2105/AJPH.89.9.1322
37. Gaskin J, Whyte J, Coyle D. An assessment of uncertainty using two different modelling techniques to estimate the cost effectiveness of mitigating radon in existing housing in Canada. *Sci. Total Environ.* **2020**, *724*, 138092. DOI:10.1016/j.scitotenv.2020.138092
38. Schmitz D, Klug MG, Schwartz GG. Radon knowledge and practices among family physicians in a high radon state. *J. Am. Board Fam. Med.* **2021**, *34*, 602–607. DOI:10.3122/jabfm.2021.03.200553
39. Ivancko GM, Querino RA, Silva GCS, Borges RD, Limongi JE. Estratégia Saúde da Família e Vigilância em Saúde: Conhecimento de médicos de família e comunidade sobre Vigilância Sanitária, Vigilância em Saúde Ambiental e Vigilância em Saúde do Trabalhador. *Rev. Bras. Med. Fam. Comunidade* **2021**, *16*, 2733. DOI:10.5712/rbmfc16(43)2733
40. Silva GCS, Querino RA, Borges RD, Ivancko GM, Silva MI, Limongi JE. Relações entre Estratégia Saúde da Família e Vigilância em Saúde na perspectiva de médicos de equipes de saúde da família em Minas Gerais, Brasil: Uma análise qualitativa. *Saúde Soc.* **2020**, *29*, e191007. DOI:10.1590/S0104-12902020191007

41. Lesbek A, Omori Y, Bakhtin M, Kazymbet P, Tokonami S, Altaeva N, et al. Systematic Review and Meta-Analysis of Inflammatory Biomarkers in Individuals Exposed to Radon. *Biomedicines* **2025**, *13*, 499. DOI:10.3390/biomedicines13020499