RADON

CHARACTERISTICS, SMOKING IMPACTS, EXPOSURE & POLICY IMPLICATIONS ON PUBLIC HEALTH VS. ASBESTOS

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Nov. 17, 2017

Radon Characteristics

World-wide, radon is believed to be the second leading cause of lung cancer after tobacco smoking(1,2), responsible for approximately 10% of all lung cancers in America (1,3) and 16% in Canada(4). Statistical studies of underground miners have identified causal links between radon exposure and lung cancer risk(5). In research from North America, Europe and China, meta-analysis revealed that for every 100 Bq/m^3 increase in radon level exposure, lung cancer risk increased by 10%(6), 8.4% (16% after irregularity adjustments)(3), and 13.3%(7) respectively(8). Studies suggest that long-term exposure presents greater risk over short term exposure, irrespective of exposure dose(8), with the risk of lung cancer increasing for every 10 years one resides in a high-risk geological zone(4). Despite US and Canadian authorities respectively setting the action level for exposure to radon gas at 148Bq/m^3(8) and 200 Bq/m^3(4), it remains unclear if lower concentrations increase lung cancer risk(8).

Radon is a colorless, odorless(4), natural occurring gas(9) that is considered largely harmless. However, its decay products are not, and are deemed responsible for its deadly effects. Radon gas is formed through the natural radioactive disintegration of Uranium-238(10). Found in rocks and soil, Uranium-238 is natures heaviest natural occurring element(10). As it decays, it produces a series of approximately two dozen different radioactive elements, eventually disintegrating into radioactive radium-226(10). As disintegration continues, radium-226 decays into radioactive radon-222(8,10). This radon gas breaks down into approximately 6 different radioactive “radon decay products” (RDP)(9) dubbed as “radon daughters” or “radon progeny”(10). Two such particles

include polonium-218 and polonium-214(8). These particles settle into lung tissue, emitting alpha radiation, unleashing their carcinogenic potential(8). (See Appendix)

Indoor radon accumulates as a function of pressure disparities between outdoor soil gas and indoor air. Pressure differences create a vacuum, drawing radon gas indoors where it can accumulate to dangerous levels(1). As radon is drawn inside its concentration builds as a function of indoor air flow(1). Accumulation of radon to dangerous levels is negatively associated with the breathability of homes(8), resulting in potentially dangerous concentrations when sealed(1). This is particularly true in winter months when air circulation is low(8). In two separate studies, one by Barros et al. and the other by Casey et al, it was determined that 9.8% and 39% of indoor spaces respectively tested in Missouri and Pennsylvania exceeded the actionable limit set by the US EPA of 148 Bq/m^3(1,2). Conversely, no outdoor spaces exceeded this limit(2). Radon gas is not known to present outdoor risk as concentrations in open air are too low to present harm(11).

Although radon sources include water, natural gas and ambient air, it is believed that the primary source of indoor radon comes from the subsoil(8). As such, the geological dynamic of the region(1) dictates exposure to this gas. Accordingly, risk has been assessed as guarded, moderate, or high with the latter presenting the greatest degree of radon risk(4). When present, it can accumulate indoors, seeping through foundation materials, foundation cracks and through other open-air passages between the subsoil and the interior of the home(11). As radon gas is heavier than air, it tends to remain low to the ground(10) with highest concentrations nearest the source. Hence, basements and

crawlspaces are subject to the greatest radon levels(11), with concentrations decreasing with increased floor level(8). Indoor radon gas accumulation has been associated to lung pathogenesis in humans(8).

Radon vs Asbestos Exposure and Respective Lung Cancer Pathogenesis

Radon-222 gas is the sixth-generation decay product of Uranium-238(12), sourced from ore containing bedrock(8). Radon gas moves freely into subsoils and, to a lesser extent, is dissolved into water systems(8). Although US geological measurements show significant variations in outdoor radon gas concentrations, none are significant enough to present health concerns(2,11). Average concentrations sit at a nominal 7.4Bq/m^3(13). It is the seepage and accumulation of radon gas indoors that forms the primary source for dangerous exposure levels among populations(8). This, via the movement of gas through foundation cracks, structural openings and absorption across foundation materials. Long term, indoor exposure from residing in high-risk zones presents the greatest risk for radon-related lung cancer within populations(8).

Radon gas is rarely absorbed into body tissues upon inhalation rendering it virtually harmless(8). Concerning are Radon decay products (RDP) formed from its disintegration, specifically polonium 214 and 218 whose emission of alpha particles inside the lung tissue is believed linked to the mutagenesis associated with 3-15% of all lung cancer(8). RDP attached to particulate matter is inhaled, settling into air passages and lung tissue(14). Here, RDP decay further, emitting alpha radiation(8), generating free radicals, and causing injury to lung epithelial cells(14). The release of this radiation is targeted, with penetration of energy unable to transverse distance(8). This localized ionization acts

directly on DNA resulting in DNA damage that includes double stranded breaks(8,15). It is also suggested that alpha particle ionization results in the formation of reactive oxygen species (ROS) in the cytoplasm, causing DNA sequence mutations (chromosomal injury) and DNA copy abnormalities (including gene deletions)(8). It is speculated that the byproducts of alpha-particle decay during the formation of Reactive oxygen species (ROS) cause even greater chromosomal damage to neighboring cells through a "bystander effect"(8,16). Essentially, irradiated cells damage neighboring cells, hitting them with the ionization energy released from ROS formation(17). This results in mutation(8) and is speculated to be the most predominate mode of chromosomal injury tied to radon-related lung carcinoma(18). Alpha-particle decay is also associated with DNA methylation damage(14). Hence, lung tumor formation(14). Point mutations to tumor suppressors, p53 and p16(8) that correct for chromosomal alterations are theorized to be impaired by RDP decay(12). Multiple, complex cytogenic pathways are associated with radon related pathogenesis(8).

Asbestos are natural occurring mineral fibers found in rock that include, chrysotile, amosite, anthophyllite and crocidolite(14). These fibers are deemed responsible for 8% of all lung cancers in Canada(19). Once used by industry in products such as insulations, predominant exposure today results from the interface between humans and the disturbance of this product in homes and other facilities(20), resulting in 3 times more lung cancers in exposed over unexposed persons(14). Due to its disease latency of 10-50 years(19), most diagnosis today are link to occupational exposures prior to implementation of restrictions in the 1980s(20,21). Lung damage is correlated with

frequency of exposure, dosage(21), and inhaled fiber characteristics, with more redox reactive iron rich fibers presenting greater harm(22-24).

Phagocytosis plays a key role in asbestos-related lung cancer as cellular phagocytes are unable to ingest and breakdown asbestos fibers >5µm in length, resulting in asbestos-related inflammation, fibrosis and carcinogenesis(14,25,26). Without phagocytosis, reactive oxygen or reactive nitrogen is produced in the presence of H_2O(15) via catalytic processes(27). Damaging ROS, and RNS are released, being particularly pronounced in the company of iron rich fibers such as crocidolite and amosite(14). These reactions results in DNA strand breakage and alterations in cell signaling(14). In the latter, mutated gene codes debilitate cell messaging that controls cellular activities(28). Other effects impact the electron transport system in mitochondrial DNA, whose own sensitivity to ROS processes can result in oxygen based damage to DNA, single-stranded DNA fractures along with interference of normal programmed cellular death (apoptosis)(29-31). It is thought that the collective link between oxidative stress by ROS and chronic inflammation escalates lung carcinogenesis(32). Additionally, incomplete phagocytosis can lead to alterations in microRNA expression and DNA methylation, both of which regulate gene expression(14,33,34). Complete phagocytosis of fibers ≤5µm can lead to lung carcinogenesis through “chromosomal mis-segregation” during mitosis, whereby replicated cells end up with too few or too many chromosomes(14,35).

Historically, asbestos and radon exposure was largely occupationally based prior to legislative controls to protect workers that were respectively introduced in the 1980’s

and 1970's(36,37). Low-dose, long-term risks associated with asbestos exposure through the natural environment, industry, and through unintentional contact in buildings continues to present concern (36,38,39). Meanwhile, long-term exposure to radon that is predominantly linked to high indoor concentrations, presents the greatest concern today(4) although, occupational exposure still occurs(40).

Radon and Tobacco Smoking: Implications and Policy Provisions

There is an interesting relationship between long-term exposure to radon and lung cancer risk among "never smokers" and "ever smokers", with the latter group at greater risk, as evidenced through epidemiological testing that includes BEIR VI model calculations(41). BEIR VI is a statistical formula developed from data collected on mine workers describing the relative risk of contracting lung cancer from RDP exposure in "never smokers' versus "ever smokers"(13,42). Although, created using occupational data of miners, thus raising concerns regarding its generalizability to describe residential radon-related risk, population case-control studies have confirmed its statistical validity for residential use(13,42). BEIR VI calculations can assess radon-related risk for lung cancer(41) as having an additive effect--whereby the relative risk would be greater than 13 in "ever smokers" over "never smokers"--or multiplicative effect--with risk equal between "never smokers" and "ever smokers"(42). A superadditive/submultiplicative effect has been observed(8,42), finding ever smokers at increased risk for radon-related lung cancer. As reported by the EPA, lifelong exposure at ≥4pCi/L (148 Bq/m^3) results in 62/1,000 cases of lung cancer in smokers and 7/1,000 cases in non-smokers(8,42). This equates to a relative risk ratio of 8.86:1, suggesting that smokers exposed to radon are

almost 9 times more likely to contract lung cancer than never smokers exposed(42). It is speculated that this “submultiplicative” effect may also affect those exposed to second hand smoke(43).

Tobacco smoking confounds the effects radon exposure, exacerbating lung cancer rates(8). In part, this is due to the mode in which RDP enter lung tissue, with negatively charged radon daughters attaching to particulate matter prior to inhalation(14). As tobacco smoke increases indoor particulate matter, it increases the dose of RDP entering the lung tissue, increasing the degree of ionization damage to lung epithelial cells(14,44). The result, increased potential for mutations to DNA and RNA strands along with DNA methylation alterations(14). Additionally, physiological impairments to air-transfer processes and reduced ventilation capacity common to smokers amplifies the RDP dose by a factor of two(44). Further, it is speculated that the duplication of carcinogenetic exposures from RDP and tobacco smoke complement one another, confounding risk(41). Also, inflammation(45) and ROS(8) are associated with both smoking and radon exposure and are thought to collectively increase risk of lung cancer(8).

Policies must support the delivery of targeted health promotion strategies and programs to maximize cost-efficiencies, cost-benefits and impacts(46,47). Utilization of statistical data provides key insights for the formulation of strategies that yield optimal outcomes(46). This is especially crucial for chronically underfunded public health systems(47). Every dollar must be effectively allocated, in efforts to address long term health and economic implications associated with poor population health(47). Given the 8.86-fold risk of lung cancer in smokers over non-smokers exposed to radon(42), logic

demands focused efforts to address radon exposure in the former subpopulation to maximize population level gains(47). Using baseline data from Mendez et al., we can see that efforts to achieve complete radon remediation compliance among the public would result in a 21% drop in radon-related lung cancer(42). Conversely, reductions in smoking that are realistically based on current trends (reductions from 24% to 10%), with no changes to radon remediation, would see these cancers drop by 42%(42). Collectively, radon remediation and efforts to reduce smoking would improve outcomes by 54%(42). However, only 22% of these gains would be attributable to remediation compliance with 78% of gains tied to smoking reduction(42). Hence, the greatest impact in reducing radon related lung cancer demands that efforts place increased emphasis towards targeting smokers(47). Additionally, collaboration between those working to reduce tobacco use, with those working to reduce radon-related lung cancer is logical, optimizing outcomes and efficiency of dollars(47).

Radon vs. Asbestos: Implications of Public Messaging/Intervention on Chronic Disease Prevention in Canada

To date, cautionary radon provisions at the national level continue to be limited to recommendations rather than mandated action(48). These soft measures include sharing information related to radon testing, remediation guidelines, building code recommendations, along with some public education(48,49). Similar messaging has been echoed at provincial levels(48) to varying degrees, as evidenced by informational disparities among respective governmental websites(50-56). Where provincial legislation exists, it is largely limited to building codes for new constructions, ignoring meaningful remediation efforts that target the broader population(48). International safety threshold

guidelines by the WHO, the EPA and Health Canada are set at 100Bq/m^3, 148 Bq/m^3 and 200 Bq/m^3 respectively(48). In the few legislative instances where controls exist to regulate indoor air quality in Canada, such as those for federal buildings, none set limits at or below Health Canada's action level of 200Bq/m3(48). Although, legislative authority is applicable to all three tiers of government, no single governing body has enacted a comprehensive model of strict regulations to reduced radon exposure on a grand scale(48).

Although Asbestos imports/exports will not be fully banned until 2018 in Canada(57), its use has been strictly regulated since the 1980s(11). What distinguishes public health outcomes between radon and asbestos may be largely attributable to the presence or absence of comprehensive regulatory restrictions, both locally and internationally. With respect to both radon and asbestos, all three levels of government hold legislative powers to enact provisions to support reductions in exposures(48). Implementation of federal asbestos restrictions, by default, led to the enactment of provincial and municipal regulations that ensured alignment and compliance with national policy. In kind, this multitiered legislation held implications for numerous industries who were accountable to uphold asbestos legislation(48). This affected institutions such as, real-estate, occupational health and safety, education etc.(48).

Arguably, comprehensive multilayered restrictions work to support increased public awareness(13) with most Canadians aware of asbestos dangers (58). International bans along with strict legislative controls, particularly when tied to deadly consequences,

create a sense of fear based urgency, inciting public attention and calls to action(59). New legislation attracts media, further influencing awareness. As such, it appears that international and national legislation surrounding the controlled use of asbestos(57), coupled with increased media attention over that of radon, appears to be positively associated with both public interest and public awareness. Despite the fact that 7% of Canadian homes exceed the 200Bq/m^3 thresholds, and that radon is the seconding leading cause of lung cancer in Canada(8), >95% of Canadians lack awareness(60), and are left apathetic to its harmful effects(48). The absence of comprehensive legislation to protect the public from radon-related harm appears to have played a role in the continuation of inadequate public knowledge, rooted in indifference(48). Without awareness, apathy prevails rendering radon remediation inconsequential to the public(8). This in contrast to public awareness, attention and concern paid to asbestos.

Public health outcomes are improved through comprehensive, action oriented legislation. Indirectly, laws to protect health further elevate public concern and urgency, working to counter apathy. Legislative actions change cultural attitudes, creating captive audiences that are more receptive to receiving vital information and more apt to implement protective measures that promote health. Additionally, media commentary that naturally follows governmental changes further builds momentum towards action. Thus, restrictive laws pertaining to the controlled use and disposition of asbestos versus soft measure associated with radon may explain why so few understand the significance of radon in their lives. Until extensive efforts to combat radon exposure are mandated through law, similar to the impending full ban on the import/export of asbestos(57), we

will continue to see action based gaps that allow for complacency, apathy, continued radon exposure and diagnosis of radon-related disease; a public health failure for radon.

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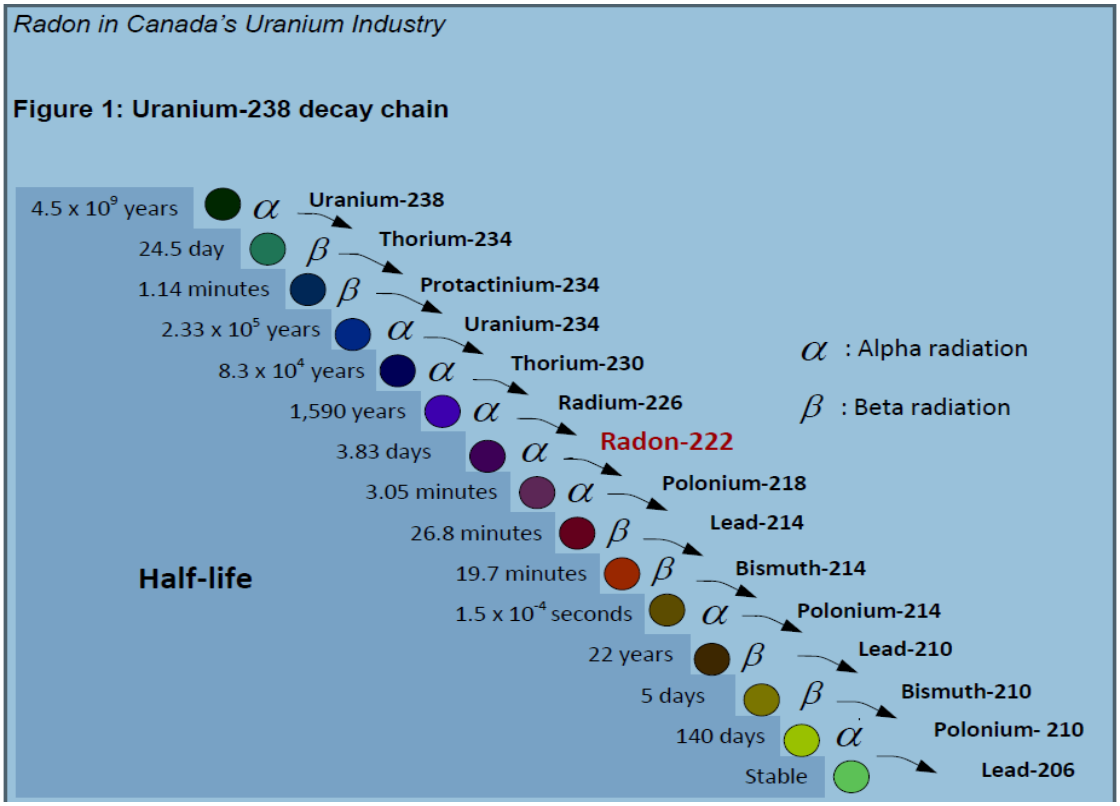
Appendix 1



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