



Rectifying Radon's Record: An Open Challenge to the EPA

Jeffrey A Siegel¹, Charles W Pennington², Bill Sacks³ and James S Welsh^{4*}

¹Nuclear Physics Enterprises, Marlton, NJ, USA

²Executive Nuclear Energy Consultant, NAC International, Norcross, GA, USA

³Emeritus Medical Officer, FDA Center for Devices and Radiological Health, Rockville, MD, USA

⁴Dept of Radiation Oncology, Stritch School of Medicine Loyola University, Chicago, USA

*Corresponding author: James S Welsh, MS, MD, FACRO, Director of Clinical & Translational Research, Department of Radiation Oncology, Stritch School of Medicine Loyola University, Chicago, 2160 S 1st Ave, Maguire Center, Rm 2932, Maywood, IL 60153, USA, Tel: 708-216-2561, E-mail: james.welsh@lumc.edu

Abstract

The American Lung Association has recently led a national workgroup to develop *The National Radon Action Plan: A Strategy for Saving Lives*. The U.S. Environmental Protection Agency (EPA) is the lead governmental organization projected to implement this plan. The stated intent of the plan is to address the "radon problem" in the United States, with the aim of saving 3,200 lives by the year 2020 through preventing at least a portion of the lung cancer mortality that is assumed to arise from inhaling modest doses of radon in homes, offices, and buildings. The plan identifies a number of actions that government can take in the spirit of saving lives by avoiding the inhalation of radon and its progeny. We are among a growing number of investigators who recognize the substantial body of evidence demonstrating that the radiation doses associated with indoor radon inhalation are not harmful. Radon, at these doses, is unlikely to be a cause of lung cancer, and, on the contrary, may be beneficial in various ways, including its paradoxical tendency to protect against lung cancer. In the present paper, we review and critique the past policies of the EPA with respect to indoor radon and the very impetus for the plan. We indicate that the plan should not be implemented because a preponderance of the evidence indicates an unintended consequence: implementation of the plan is likely to *increase*, rather than decrease, the risk of lung cancer.

Keywords

EPA, Radon, Lung cancer, Radiation carcinogenesis, Linear no-threshold model, LNT, Low-dose radiation exposure risk

Introduction

Twelve organizations have recently issued a pamphlet outlining *The National Radon Action Plan: A Strategy for Saving Lives* (<http://www.lung.org/assets/documents/healthy-air/national-radon-action-plan.pdf>). They include "American Lung Association, American Association of Radon Scientists and Technologists, American Society of Home Inspectors, Cancer Survivors Against Radon, Children's Environmental Health Network, Citizens for Radioactive Radon Reduction, Conference of Radiation Control Program Directors, Environmental Law Institute, National Center for Healthy Housing, U.S. Environmental Protection Agency, U.S. Department of Health

and Human Services, U.S. Department of Housing and Urban Development [emphasis ours]."

While this is an impressive list of organizations, the pamphlet states on page 3, "The American Lung Association led a national radon workgroup to develop this Plan." So the American Lung Association took the initiative, while the U.S. Environmental Protection Agency (EPA) is the lead governmental organization projected to implement this plan, among nine governmental agencies listed on the last page of the pamphlet. These agencies, comprising the membership of the Federal Radon Action Plan Workgroup, include the Department of Agriculture, Department of Defense, Department of Energy, Department of Health and Human Services, Department of Housing and Urban Development, Department of the Interior, Department of Veterans Affairs and the General Services Administration.

In the present paper, we carefully review and critique the very impetus for the plan and indicate that the preponderance of the evidence reveals an unintended consequence that makes implementation of the plan likely to *increase*, rather than decrease, the risk of lung cancer. Thus, in our view, the plan should not be implemented.

The Consequences of Error

To be clear, the position of the authors of the present paper is that ionizing radiation (hereafter radiation, for brevity) is no different from almost every other agent in our environment - from water to oxygen to sunlight, etc. In that, as with the other agents, radiation is harmful at too high a level *or at too low a level*, but necessary for health in an optimal middle range. It is quite likely that over billions of years, natural selection has eliminated all organisms, if there ever were any, for which radiation is/was harmful at all levels. For purposes of the first part of this paper, however, we will temporarily suspend our advocacy of that position until later, and at first focus on the following question to the EPA and all other organizations and agencies who propose to put into effect the new National Radon Action Plan (NRAP):

Do you not think it necessary before you implement such a plan that you have the predominance of evidence behind your belief that

radon contributes to raising, rather than lowering, the risk of lung cancer at the levels found in homes? After all, if you are wrong, and if home and office radon levels are within a middle range that is beneficial to health, in particular in the lowering of the risk of lung cancer, and if you plan to reduce the levels below that middle range to a level that is too low, or at least less appropriate, for the promotion of health, and instead you inadvertently increase the risk of lung cancer, you will be accomplishing the precise opposite of your intended outcome, not to mention creating new, astronomical costs involved in promoting such harm. Furthermore, you would then be culpable of and liable for deliberately adopting a policy and action plan that worsens public health, particularly when you have neglected to review objectively the various relevant scientific documentation, and instead have approached it *a priori* with a biased position, forcing rejection, out of hand, of the substantial evidential basis for the opposite conclusion rather than for your favored one. Such unintended consequences can be avoided with prior intent.

So before you embark on such a plan, it behooves you to call for and encourage an open public debate to determine which estimate of radon's effect is true and which is false, and to commit to educating the public, as well as members of regulatory and other organizations, regarding the outcome of this objective search for the truth about radon. The difference between these two opposite positions, after all, is literally a matter of life and death.

The Plan (NRAP)

The stated intent of the plan is to address the “radon problem” in the U.S., with the aim of saving 3,200 lives by the year 2020 through preventing at least a portion of the lung cancer mortality that the EPA and its eleven partner organizations assume arises from inhaling modest doses of radon in homes, offices, and buildings. The plan calls for three components: 1) all existing homes to be tested for radon, 2) all high-radon homes to undergo fixes to lower the levels, and 3) all new homes to be built to specifications minimizing avoidable accumulations of radon in the first place.

The plan identifies a number of actions that government can take in the name of saving lives by avoiding the inhalation of radon and its progeny: impose new building standards; require more extensive radon testing; stipulate specific technology alternatives; and impose real estate sales covenants, obligations, and requirements.

On page 2 the pamphlet boldly asserts, without supporting evidence, “Radon causes 21,000 lung cancer deaths every year.” Elsewhere (<http://www.lung.org/lung-health-and-diseases/lung-disease-lookup/lung-cancer/symptoms-causes-and-risk-factors/what-causes-lung-cancer.html>) the American Lung Association states, “Radon exposure is the second-leading cause of lung cancer [after smoking].” This fear-inspiring number and the unsupported attribution are based, not on any direct epidemiological or experimental evidence, but rather on a hypothetical relationship between radon levels and lung cancer - a relationship derived from an extrapolation downward from observations of lung cancer rates in uranium miners, most of whom were exposed to far higher levels than are found in homes and offices. It is known as the linear no-threshold hypothesis (LNTH), which is a mathematically convenient relationship, but empirically unsupported and indeed unsupportable (because of the immense sample sizes required), as admitted by LNTH advocates.

The number “21,000” is taken from a 1999 report of the committee of the National Academy of Sciences (NAS) that goes by the acronym BEIR, which stands for Biological Effects of Ionizing Radiation [1]. BEIR VI, as the report is known, modestly states, however, that a threshold of exposure, below which there is no risk, could exist, yet not be identifiable from the available epidemiologic data (see next section for further discussion). This modest admission from the originating source is entirely omitted by the EPA and the other sponsoring organizations in their pamphlet. Thus, the number of “21,000 lung cancer deaths every year” is made to appear as factual rather than as the hypothetical estimate it is, based on an unsupported

mathematical model derived from assumption. Therefore, the large and frightening figure is built upon a veritable house of cards, though one that promotes mass radiophobia to stimulate the public's predictable tendency to support, even give thanks for, any mitigation plan that the EPA proposes, no matter the risk of higher rates of lung cancer or higher cost to the public - of which risk and cost the EPA steadfastly keeps this same public uninformed.

For example, Puskin of the EPA [2] has stated that there is unequivocally a risk even at the lowest doses of ionizing radiation and, therefore, large economic costs can legitimately be imposed. He then asserts that denials only fuel distrust and it is better to acknowledge that the science, so far, is consistent with a non-zero risk at low doses, even if direct verification is lacking. However, there is a plethora of evidence contradicting this claim [3-6].

Mass phobias of any sort interfere with rational consideration of any proposal to relieve an alleged hazard. Despite that obstacle to rationality, the EPA is, nonetheless, committed to the LNTH, and the “hazard” of radon inhalation is now inviolable doctrine within the EPA. Furthermore, reversing mass phobias is far more difficult than instilling them, and opponents of the exaggerated, if not entirely false, claims are easily mistaken for the enemy, while the proponents of the claim are easily mistaken for the saviors and protectors.

The EPA Policies and the Plan's Goal of Saving Lives is not Supported by the Evidence

To explain this assertion we begin with a brief radiation dosimetry primer. The extent to which lung cancers are radiation-induced must be related to the magnitude of the absorbed radiation dose, a point to which both sides of the argument agree *at high enough doses and dose rates*. The dosimetry of inhaled radon and its decay products must, therefore, be known in the region of the cells of the tracheo-bronchial epithelium (TBE).

According to a model given in an UNSCEAR report (2000), the annual effective dose H (mSv) received by individuals due to indoor radon and its progeny can be estimated as follows [7]:

$$H = C \times F \times O \times T \times D$$

where,

C = weighted average indoor radon (^{222}Rn) activity concentration (Bq/m^3) = $148 \text{ Bq}/\text{m}^3$ (= 4 pCi L^{-1} = 0.15 Bq L^{-1});

F = the equilibrium equivalent concentration of ^{222}Rn = 0.4 (indoors);

O = the occupancy factor = 0.8 (per UNSCEAR report);

T = time = 8760 h; and

D = the dose conversion factor for ^{222}Rn and its radioactive progeny = $9 \times 10^{-6} \text{ mSv}/\text{h}$ per Bq/m^3

Rather than basing its action level - above which mitigation is called for - on a chosen value of the effective dose H , the EPA bases its action level on a chosen value for the radioactive material activity concentration C , for the measurement of either of which instruments are available [8]. Furthermore, according to the EPA (Puskin and Pawel 2014), its radon “action level was not chosen on a health-risk basis, but it was driven by the technical feasibility of achieving reliable and verifiable reductions by homeowners [9].” The parameter values for F , O , and D are taken from the UNSCEAR 2000 report [7]. The chosen value of the dose conversion factor D is the estimated central value for this parameter; the dosimetric evaluation of the absorbed dose to the basal cells of the TBE per unit exposure gives values for D in the range of $5 - 25 \times 10^{-6} \text{ mSv}/\text{h}$ per Bq/m^3 . The typical value assumed for F is 0.4; indoor measurements show a range from 0.1 to 0.9, but most values are within 30% of 0.4 (i.e., 0.4 ± 0.12). As noted in the UNSCEAR report [7], a recent study in seven North American houses has shown that the equilibrium factor in any particular building exhibits a significant variation with time, typically of a few tens of percent. Thus, it is important to recognize that use of 0.4 may

be in error, frequently by several tens of percent and occasionally, though rarely, by as much as a factor of 2.

Using the equation and all parameter values as above, the estimated annual effective dose H , at the chosen action level for C , is 3.73 mSv. This value is in excellent agreement with the annual effective dose of 3.70 mSv resulting from the same assumed indoor radon activity concentration of 0.15 Bq L^{-1} derived from a representative lung-absorbed-dose value given in table 4-1 of the BEIR VI report [1]. However, there is a range of possible values of H , given the ranges of the constituent variables. While the actual values would more likely be close to the midrange, it is worth evaluating the extreme values to understand the possible range and to see that a chosen value for C (activity) leaves open widely different possible values for H (annual dose). It is H that should be the parameter of interest for those who believe that the LNT assumption is a valid approach to regulatory policy.

If we were to use the *maximum* value for D of 25×10^{-6} mSv/h per Bq/m^3 , instead of the *central* value 9×10^{-6} , and the maximum value for $F = 0.9$, instead of 0.4, H would be 23.3 mSv. Conversely if we were to use the *minimum* value for D of 5×10^{-6} along with $F = 0.1$, H would be 0.52 mSv. Thus, the range of possible values of H extends from 0.52 to 23.3 mSv, a range covering a ratio of almost 45 to 1. Therefore, EPA's choice of the radon activity concentration C , instead of annual dose H , as the target for its action level - assumed by the agency to be based on the principle of ALARA (as low as is reasonably achievable) - is associated with a very wide range of potential annual effective doses H . This ALARA approach, based on the LNTH, is extraordinarily inconsistent and would demonstrate a failure to protect many people if the EPA's own assumptions were correct. That the assumptions are not correct does not exonerate the EPA's policy in this case from a charge of failure to achieve its professed goal, which is to save lives.

Furthermore, the EPA has fought for years to counter independent proof that, with respect to radon inhalation, the LNTH is patently false. Let us return to the claim in the NRAP pamphlet, and subscribed to by the EPA [10], that radon causes more than 21,000 lung cancer deaths annually in the U.S. This figure is the basis of the EPA's claim that "Radon is the number one cause of lung cancer among non-smokers...Overall, radon is the second leading cause of lung cancer (<http://www.epa.gov/radon/health-risk-radon>)," and is generated from the BEIR VI report [1], based on data from 11 cohorts of miners fit to empirical risk models. In this report, estimating lung cancer mortality from indoor radon was admittedly associated with large uncertainties (the analysis demonstrates that the number of radon-related lung-cancer deaths could be as low as 3,000), including those in the parameter estimates in the exposure-response model, in specifying the form of the model, and in its application to the general U.S. population. For the latter, the most important sources of uncertainty relate to problems in extrapolating results from data on miners, many of whom received large radon exposures at high exposure rates, to people in homes with low exposure rates. An LNT model was selected for this purpose, but, as mentioned above, the BEIR VI committee recognized and stated that a threshold - meaning a level of exposure below which there is no risk beyond that associated with everyday background radiation - could exist and not be identifiable from the available epidemiologic data.

To evaluate the actual effects of protracted exposures of the general population to the much smaller concentrations of radon occurring in residential dwellings requires epidemiologic studies under the conditions of relevance, rather than by simply assuming knowledge of the effects gained by extrapolating downward from the much higher doses found in many mines. Such a study was done in the early 1990s by Bernard Cohen (1990, 1995), who first established, defended, and validated the *falsity* of the LNTH [11,12]. Cohen performed a large ecological study of over 1,700 U.S. counties containing more than 90% of the country's population. He reported what was at the time a surprisingly strong *negative* correlation between lung cancer mortality and measured average home radon levels in each county.

The two most obvious possible explanations for this finding were (1) confounding by a negative association between radon and other risk factors for lung cancer, particularly cigarette smoking, which is a causal factor in the great majority of lung cancer deaths, or (2) a protective effect of low dose-rate radiation exposure. Although Cohen did find some evidence of confounding by smoking, he showed that, even under extremely improbable conditions, smoking could, at best, explain only a part of the negative correlation.

In response, one of the EPA's experts, Puskin (2003), reexamined Cohen's data and countered Cohen's conclusions, claiming that smoking was indeed an adequate confounder to explain the entire negative correlation between lung cancer and radon levels, and denying that there is substantial evidence for a protective effect of low level radon exposure [13]. Cohen rebutted Puskin's claim regarding the adequacy of this confounding with a clear mathematical demonstration that there is no possible set of smoking prevalences that could explain the entire negative causal association of radon levels and lung cancer mortality. Further, he remarked on the impossibly low probability that smoking could be strongly correlated at all, whether positively or negatively, with radon levels throughout the entire set of counties.

The low probability of such a correlation was based on the absence of any causal link between the two (nor did Puskin suggest any causal link), so that any such correlation would have to be due to sheer coincidence. Considering the size of Cohen's sample - over 1,700 U.S. counties containing over 90% of the U.S. population - this impossibly low probability is manifest. Thus, Cohen reinforced his earlier conclusion that the explanation for the negative correlation was indeed the protective effect of radon and its progeny [14]. Puskin's response ignored Cohen's mathematical demonstration and simply reiterated that confounding by smoking appears to be the likely cause of the negative correlations while confining his comments to denying the *plausibility* (based on speculation without any scientific evidence) that it might rather be due to a protective (hormetic) effect of radiation from inhaled radon progeny [15]. Puskin's reasoning is commonly known as the argument from incredulity, a well known logical fallacy.

Indeed, Cohen and a colleague analyzed his data for more than 500 possible combinations of confounding factors, but the conclusion remained firm that the LNTH fails decisively, not only by grossly overestimating the cancer risk from low-level radiation, but by actually reversing it [16]. Upon scrutinizing the data, the conclusion is inescapable that there is a protective effect of increasing radon exposure levels over the range of residential exposure levels. Given this conclusion, it would mean that actions to reduce moderate radon levels in homes are "misguided," if not worse. In fact, such actions would be likely to lead to the opposite of the desired effect and, by lowering the radon concentrations, would paradoxically increase rather than reduce rates of lung cancer.

An NCRP Scientific Committee, like Puskin, also concluded that Cohen had not accounted for possible confounding by smoking, as well as other possible deficiencies [17]. But, as was the case for Puskin, Cohen's rebuttal again showed that these conclusions were totally without merit [18]. Thus, neither the EPA nor any other person or organization has been able to rebut the Cohen study. It stands as scientifically sound research, demonstrating that low-to-moderate radon concentrations do not "cause" lung cancer, but rather mitigate it.

When directly confronted with the proposition that the LNTH may grossly overestimate cancer risks associated with radon inhalation [19], Puskin and Pawel of the EPA responded [9] that rejection of the LNTH is "indefensible when it comes to radon," citing the study by Darby *et al.* (2005) as "proof" that the LNTH provides a reasonable estimate of risk at radon levels only slightly above the EPA action level [20]. However, the Darby study has been shown to be based on circular reasoning; their model assumed *a priori* a linear association between radon and lung cancer and assigned the comparison baseline

for risk calculations to the lowest level in their study rather than to zero dose-rate, which carries a higher, rather than lower, risk than the lowest dose-rate in their study. So, given their circular reasoning that assumes linearity down to zero, it is no wonder that their result is consistent with the LNTH and that the actual negative risk (positive benefit) region is hidden from view. A more recent pooled Bayesian meta-analysis of the 28 international radon studies compared the linear and six other dose-response models [21]. They demonstrated that there was no evidence of such a linear dependence or even a monotonically rising relationship in the low dose/dose-rate range, even when Cohen's data were excluded.

A further observation involves early studies on uranium miners in North America, in which lung cancer mortality was compared with the occupational exposure to radon over time, measured by Working Level Months (WLM), of such miners, as summarized in BEIR IV [22]. Several separate global studies were performed, but two in North America, the Colorado Plateau Study (CPS) by Lundin *et al.* (1971) and the Ontario Uranium Miners Study (OUMS) by Muller *et al.* (1983), were performed with account generally taken of proper data collection and possible confounders [23,24]. These studies offer interesting insight into large-scale, dose-response experience. Each study offered cumulative WLM vs. lung cancer mortality data for large numbers of miners and high levels of person-years. In each study, there was a large group of miners with cumulative WLM exposure roughly comparable to in-home radon exposures of the general public. Only the CPS was adjusted for the confounding influence of smoking by miners, which is reported in data within BEIR IV (1988) to be in the range of 80% (with one-pack-per-day and greater smoking around 60%) [22]. The CPS results show that for a cumulative exposure of < 120 WLM, the OTE ratio (observed lung cancers divided by expected lung cancers) was 0.55. For the OUMS, smoking was not taken into account, but even at that, for cumulative exposures of < 100 WLM, the OTE was only 1.05, and with similar adjustments for smoking as in the CPS, the ratio would be less than 1.0. Such data are entirely consistent with Cohen's demonstration of a negative association between typical in-home radon exposures and lung cancer mortality.

Other epidemiologic studies assessing the lung cancer risk due to indoor radon exposure have yielded conflicting results. A study by Pershagen *et al.* (1994) indicated evidence of increased lung cancer risk due to radon in homes [25]. However, seven other studies up to 1996, as referenced by Auvinen *et al.* (1996), involving at least 200 case subjects with individual radon measurements, did not find a statistically significant association between indoor radon exposure and lung cancer [26]. A more recent study by Turner *et al.* (2011) concluded that lung cancer risk increased with increasing radon concentrations, and that there was no significant departure from a linear relationship [27]. However, upon review of their reported data and graphical representations of the data, one can see that this conclusion is not justified. One major criticism is that most of their point estimates fail to lie statistically significantly above a hazard ratio of 1 (no effect), and there appears to be a clear dose threshold before the points begin to rise above 1. Whether those points at higher doses are valid is a different question, but the study, while claiming to demonstrate linearity and the absence of a threshold, in fact does the precise opposite. This error, which is not uncommon in this type of research, underscores the ready willingness of some investigators to attribute a significance to their data that simply does not exist. Such willingness reflects a grasp by a powerful and prevalent paradigm that will require honest and determined reevaluation to overcome.

The continuing uncertainty and conflicting results regarding the cancer risk from indoor radon exposure are probably due to the fact that it is very difficult to prove or disprove the small risk associated with exposure to low levels of radon in any single study. Pooled analyses are, therefore, likely necessary. But the heterogeneity among studies in the accuracy of radon measurements, as well as assessment of confounding factors (above all, smoking), may bias the results of such exercises, even if stringent selection criteria are

employed. The examination conducted by Fornalski and Dobrzyński (2011) was based on the group of what constituted at that time the only 28 reported international studies [21]. To resolve the uncertainties among them, Bayesian statistics were applied, as these are best suited for analysis of inconsistent and scattered data. Many of these 28 reported studies rested their evaluations on the *a priori*, paradigm-based assumption that the relative risk (RR) of lung cancer incidence cannot be less than 1 (when compared to that at zero dose), i.e., that there could not be a protective effect of radon that would lower the risk of lung cancer below that at zero dose. But instead of the response at zero dose they employed the response at the lowest observed doses in their studies as the reference point for relative risk (RR = 1). This *a priori* approach has, in many epidemiological studies, raised all the estimates of RR at doses above the lowest dose to RR > 1, by definition rather than by observation - a logical fallacy based on circular reasoning. This approach will often lead to the conclusion that risk increases with dose, whether in a manner consistent with a linear (LNT) or a curved model. As a consequence, such a risk dependence, while not necessarily linear, will generally exhibit a monotonic increase with dose, but only because of their *a priori* assumptions. In fact, as we have reported [28], the appearance of validity in epidemiological studies that claim to confirm the LNT model, in addition to resting on circular reasoning, rests on cherry picking, faulty experimental design, and/or misleading inferences from weak to statistically insignificant evidence.

Conclusion

We are among a growing number of investigators who recognize the substantial evidence that the radiation doses associated with indoor radon inhalation are not harmful, let alone a cause of lung cancer, and, on the contrary, are beneficial in various ways, including their tendency to protect against lung cancer. The radon record simply needs to be rectified. Stunningly, for example, over the last 12 years, the EPA has never provided any analysis that demonstrates what Puskin *et al.* (2004) simply claimed was true (while casually dismissing the statistically sound proof of radon's benefit in Cohen (2004)) and presented it for peer review [14,15]. We point this out to suggest that agencies and regulators take an objective and thorough further look at the data and realize how contrary to the empirical data their recommendations and regulations truly are, and that their continued refusal to take into account and credit these data constitutes the real risk related to radon. We recommend that agencies stop regulating and attempting to lower doses that are in fact safe and even beneficial, as they essentially have done in the case of their proposed action level for indoor radon exposure.

An important and beneficial outcome of such deregulation and the complete abandonment of The National Radon Action Plan would be to permit the longer term mitigation of the public's misplaced *radiophobia*, thus alleviating the great harm that it does. Finally, the foundations for the EPA's health policies on indoor radon and its progeny have been based on unsupported and biased opinion and promote unwarranted fears on the part of the public. Indeed, given the EPA's intention of protecting the public's health, the agency should instead be educating the public as to the safety of home radon levels and the hazards of lowering it.

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