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# Topics under Debate

## DOES EXPOSURE TO RESIDENTIAL RADON INCREASE THE RISK OF LUNG CANCER?

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### INTRODUCTION

The Biological Effects of Ionizing Radiation (BEIR) VI Report *The Health Effects of Exposure to Indoor Radon*, concluded that radon exposure represents a significant cause of lung cancer which was second only to cigarette smoking. Humans as a species have been exposed to various concentrations of radon for about as long as they have existed on Earth. Certain parts of our planet, because of their geology, have rather high naturally occurring concentrations of radon, and many people have been immersed in those high concentrations with seemingly little deleterious effect. On the other hand, it is well known that mine workers exposed to high concentrations of radon do indeed suffer health effects. When modern homes are constructed they are quite well sealed against air infiltration, thus by symmetry they are also resistant to the outflow of gases. In situations where homes are constructed in areas of high natural radon concentrations, it is logical to ask whether residents may or may not experience serious health effects. Our two participants have considered this question and have divergent views. They are both well qualified to present their arguments regarding this topic, and we are fortunate to have them share those views with us in this debate.

Professor R. William Field is a cancer epidemiologist in the Department of Epidemiology and a member of the Graduate Faculty in the College of Public Health at the University of Iowa. He also serves as an Adjunct Professor in the Department of Occupational and Environmental Health. Dr Field received his doctorate degree in preventive medicine from the University of Iowa in 1994. Prior to that, he was involved in environmental research following the Three Mile Island accident and worked both as a health physicist at the University of California, Berkeley and as an environmental consultant. He serves as a reviewer, and occasional guest editor, for numerous scientific journals.

Professor Klaus Becker spent more than 15 years in dosimetry research at the Oak Ridge National Laboratory Health Physics Research Division and international projects in Germany. He became director of the German Nuclear Standards Committee at DIN, and Secretary of the International Organization for Standardization (ISO) Technical Committee 85 on 'Nuclear Energy'. He has authored several books and about 300 publications in scientific journals. He holds patents for various radiation detection methods (e.g. radon dosimetry by track etching), and has served as a member of numerous national and international bodies, and as a technical expert for various organisations, such as the IAEA, WHO, EU, ISO and ICRP. He also was founding President of the German/Swiss Radiation Protection Society, is an Editorial Board Member of *Radiation Protection Dosimetry*, and Vice-President of Radiation, Science & Health, Inc.

FAVOURING THE PROPOSITION: R.William Field

**Argument**

The fact that prolonged exposure to radon decay products (radon) causes lung cancer is well documented[1,2]. Radon's carcinogenicity is supported by an unparalleled occupational data set that clearly links prolonged radon exposure and lung cancer[3]. Approximately 20 epidemiological studies of radon-exposed underground miners have been performed, 11 of which have provided exposure response relationships between radon progeny exposure and lung cancer[3]. The findings from these studies show a substantial degree of consistency, even though their methodologies differ. Lubin *et al*[4] pooled the data for the 11 studies of underground uranium miners in the United States (Colorado, New Mexico), France, Australia, the Czech Republic, Canada (Ontario, Port Radium, Beaverlodge), metal miners in Sweden, fluorspar miners in Canada and tin miners in China. The data set included 65,000 miners and more than 2700 lung cancer deaths. The mean exposure among exposed miners was 162 WLM (working level months), which is approximately three times the exposure from lifetime residence at 148 $Bq.m^{-3}$. The study found that the cumulative radon progeny exposure was consistently linear in relation to lung cancer deaths in the range of exposures to miners. Based on their findings, the authors stated that, "exposures at lower levels, such as in homes, would carry some risk".

To examine the risk posed by radon exposures at lower levels, Lubin *et al*[5] performed a subset analysis of lung cancer risk on miner data restricted to the lower exposure categories (either <50 WLM or <100 WLM). These ranges clearly extend down into the levels experienced by some homeowners. Significant departures from a linear excess relative risk model in cumulative exposure were not found. Relative risks for exposure categories exhibited a statistically significant increasing trend with exposure in each of the restricted data sets. Both the restricted and unrestricted data yielded general patterns of declining excess risk per WLM with attained age, exposure rate, and time since exposure.

The findings of the 11 pooled studies of underground miners served as the foundation for the National Academy of Sciences (NAS) BEIR VI report[3]. The NAS report is the most definitive accumulation of scientific data on the health effects of radon to date. The committee developed risk models that were used to project the lung-cancer risk, both for individuals and for the entire population of the United States, associated with radon exposure. The committee used a linear no-threshold relationship relating exposure to risk for indoor radon. To support their linear model, the committee provided mechanistic information on alpha-particle-induced carcinogenesis along with other corroborating evidence for linearity such as the pooled miner studies described above[3]. The NAS Committee's two preferred risk models projected that residential radon exposure was responsible for approximately 18,600 lung cancer deaths each year in the United States.

In addition to the underground miner studies, numerous ecological and case–control epidemiological studies have been performed in an attempt to directly examine the association between residential radon exposure and lung cancer. Because the ecological study design relies on summary measures, it has major limitations and cannot assess an individual's current or retrospective radon exposure. In fact, the 1989 Study Design Group of the International Workshop on Residential Radon Epidemiology concluded that ecological studies should not be used for the assessment of residential radon risk[6]. However, direct evidence indicating that residential radon produces lung cancer is accumulating from residential radon case–control studies. These studies do not require projections from underground miner data. Residential case–control studies have been performed in Canada, China, Finland, Germany, Sweden, the United Kingdom, and the United States. A 1997 report by National Cancer Institute (NCI) researchers[7] directly examines the effects of residential radon exposure on lung cancer risks by using data from eight of the previously published large scale case–control studies that were performed independently in Canada, China, Finland, Sweden, and the United States. The results of the meta-analysis showed an increased summary excess risk of 14% at 148 $Bq.m^{-3}$. Additional documentation that exposure to residential radon concentrations as low as 148 $Bq.m^{-3}$ causes lung cancer is mounting. Major studies in Germany[8] and the United Kingdom[9] are in close agreement with the risk estimates obtained from the meta-analysis performed by the NCI.

In the recent residential case–control study performed in Missouri, a statistically significant lung cancer risk was found for radon concentrations estimated using glass based detectors[10]. The Iowa Study[11], which used enhanced dosimetry methods, also found a statistically significant excess risk at average radon exposures of 148 $Bq.m^{-3}$. The Iowa and Missouri risk estimates are generally higher than the reported risk estimates for the previously published residential radon studies. The higher risk estimates are likely attributable to reduced radon exposure misclassification[11]. In fact, Alavanja *et al*[12] recently suggested that the estimated 18,600 lung

cancer deaths attributed to residential radon exposure each year in the United States may be an underestimate.

When examined as a group, the projections from miners and the major residential radon epidemiological investigations provide overall consistent and convincing evidence that prolonged exposure to residential radon, even at residential concentrations as low as 148 $Bq.m^{-3}$, increases the risk of lung cancer.

**Rebuttal**

Professor Becker's evidence against the proposition is based primarily on selected anecdotal reports and undocumented claims rather than a firm scientific foundation. In response, I will focus on a few of his major arguments.

*1. Professor Becker indicates that several studies performed in the region of Saxony, Germany support his argument.*

Professor Becker fails to present even one published peer-reviewed study examining the association between radon exposure and lung cancer for the Saxony area. He does provide references to conference proceedings, which included a description of an ecological epidemiological study entitled*, Saxony – a Little Known Case Study*. As noted below, ecological epidemiological studies have major design limitations.

*2. Professor Becker states that epidemiologists studying radon prefer the expensive and slow case–control studies – even if other approaches (cohort and ecological) have more power and are considered satisfactory for regulatory purposes.*

He appears to favour ecological studies, such as the Cohen reference, for examining the risk posed by radon exposure since prospective cohort studies are even more expensive and time consuming than case–control studies. Ecological epidemiological studies are faster and generally cheaper, but they have the least *a priori* validity for risk assessment as compared to case–control and prospective cohort studies. Over 20 ecological radon studies have been published since 1981. The majority of ecological studies attempted to correlate geographically based lung cancer rates with the summary radon concentrations from that geographical area. The ecological study design should be reserved for generating hypotheses since it has major limitations including ecological confounding and cross-level bias[13-17].

*3. Professor Becker suggests that case–control studies are invalid, because they may suffer from tobacco usage misclassification. He theorises that a misjudgement of only one cigarette/day may falsify most case–control studies.*

Case–control residential radon epidemiological studies can control for smoking at the level of the individual, unlike ecologic studies. For example, in a residential radon case–control study like the Iowa Radon Residential Case–Control Study[11], detailed individual smoking histories were obtained. The participants' smoking histories do not need to match the smoking histories of the controls since the effect of smoking can be adjusted for using standard statistical methods. We have previously shown that increasing radon exposure measurement error reduces the lung cancer risk estimates[11]. Misclassification of smoking status would also bias the study findings toward no association in the majority of cases. In fact, in order to get a radon dose-response relationship attributable to poor recall of smoking as suggested by Professor Becker, the misclassification of smoking would have to correlate with radon exposure. In other words, individuals would have to independently know their radon concentrations in their home and increasingly under-report their smoking habits as the residential radon concentrations increase. Since most people in a study do not know the radon concentrations in their home at time of interview, this is a very unlikely scenario. In addition, recent studies have suggested that tobacco usage recall is fairly accurate with the exception of certain sub-populations[18]. It is also of interest that a recently published residential case–control study in Germany of non-smokers demonstrated an excess relative risk of 43% for average radon exposures of 100 $Bq.m^{-3}$[19].

*4. Professor Becker states that randomised double-blind clinical trials have demonstrated that radon exposure has beneficial health effects.*

In a search of the medical literature, no references were found indicating that clinical trials have been performed demonstrating that radon exposure prevents or cures lung cancer.

In summary, the preponderance of scientific evidence to date suggests that prolonged residential radon exposure increases the risk of lung cancer.

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OPPOSING THE PROPOSITION: K. Becker

**Argument**

I grew up in a region in Germany where a lung disease among miners was first described by Paracelsus (who said "It is the dose which makes the poison!") almost five centuries ago. This area is close to the later-famous radon spa of Bad Schlema, which enjoyed great popularity among patients with rheumatism, arthritic

diseases, etc. as 'the world's strongest radon source' until 1945, when Russia started mining 220,000 tons of uranium there. The spa successfully reopened a few years ago, while at the same time billions of Deutschmarks in German tax revenues are spent on radon evaluation and remediation programmes in this area. In that area, 12% of the houses exceed the US Environmental Protection Agency (EPA) intervention level by a factor of more than 100. It is, therefore, an excellent natural laboratory for testing the question of residential radon risks, the LNT hypothesis, ICRP lung models, and the cost benefit ratio of the global multibillion US dollar radon industry.

Certainly during the 'Klondike years' after the war, radon in mines in Saxony at concentrations exceeding two million $Bq.m^{-3}$ (which according to ICRP corresponds to a dose equivalent of several $Sv.y^{-1}$) contributed to the increase in lung cancer among miners, apparently ranking fourth after smoking, silica, and arsenic inhalation in a complex synergistic process, but ahead of uranium and mineral dusts, nitrous gases, diesel exhaust fumes and other confounders. Obviously, such conditions cannot be extrapolated down over many orders of magnitude for comparison to residential radon. Indeed, no detrimental health effects have been observed within the rather stable population, with some families living for centuries in the same houses, not only regarding lung cancer, but also other solid cancers and leukaemia[1]. This also applies to employees in other high radon environments, such as Bavarian public water plants with radon levels up to 700,000 $Bq.m^{-3}$. Furthermore, animal experiments demonstrated that it is the dose rate, and not the integral dose, which is important for the lung cancer incidence[2].

Various experts argued that epidemiology in general may work for strong correlations such as smoking and lung cancer (a factor *ca*. 30), or salmonella infections and ice cream[3], but is a weak tool for relative risks below about 2, a region that makes it easy to artificially create statistical diseases. The three approaches, namely cohort, correlation (ecological), and case–control studies, each have advantages and disadvantages regarding power and significance, the influence of confounders, etc. However, some epidemiologists studying radon prefer the expensive and slow case–control studies – even if the other approaches have more power and are considered satisfactory for other regulatory purposes in radiation protection or toxicology, and the results do not always meet the established criteria for 'good epidemiology'.

The current situation is confusing. The most frequently quoted meta-analysis of case–control studies by Lubin and Boice shows the very different results of studies in three continents. Of about 30 data points with very large vertical error bars (but no horizontal bars indicating the large uncertainties in retrospective radon dosimetry), only one is slightly above the control line. Even this single indication of a slight lung cancer increase at 450 $Bq.m^{-3}$ has recently been shown by the same group to decrease, similar to those of Cohen and others, if an ecological evaluation is used, leading to controversies about the interpretation[4].

Future meta-analysis studies may show similar inconclusive results. For example, in the high radon areas of the former East Germany, one expensive case–control study claims a slightly positive trend, while another, supported by the EU and restricted to non-smoking females, finds clear indications of an initially negative trend and a threshold around 1000 $Bq.m^{-3}$[3(5)]. As an explanation of all this confusion appears to emerge, a trivial old problem which will soon be responsible for half of all exogenous human cancers, namely cigarette smoking.

Because of the extensive mining in the area, lung cancer was readily diagnosable in Saxony 150 years ago, and in a large hospital in the capital, Dresden, 20,000 autopsies were performed in following decades. Lung cancer was extremely rare among non-miners, so only 0.06% of the autopsies showed lung cancer during 1852-1876, slowly rising to 0.43% between 1885 and 1894[6]. The first German cigarette factory started production in Dresden in 1862. It is well established that in 'retrospective smoking dosimetry', smokers notoriously underestimated their past and present smoking habits (even more than other drug addicts), in particular after lung cancer has been diagnosed[7].

There have been many attempts to establish an equivalent between the assumed risks of residential radon and of smoking. Numbers between 500 WLM equivalent to one pack of cigarettes per day and as little as one cigarette per day have been discussed[8], thus reaching the realm of passive smoking and corresponding to an assumed doubling risk of 400 $Bq.m^{-3}$, which is almost twice the currently discussed EU 250 $Bq.m^{-3}$ limit for new buildings. Obviously, a misjudgement of only one cigarette per day may thus falsify most case–control studies, unless they are restricted to never-smokers (and assuming that their lifetime radon dose is sufficiently accurately known). Incidentally, it should be noted that in the Iowa Lung Cancer Study by Field *et al* 86% of the lung cancer cases were smokers, but only 32% of the controls.

In summary, the residential radon issue appears to remain 'hidden behind a dense smoke screen'[7]. With a U-shaped dose effect relationship, as explained in a mechanistic model[9], with a threshold around 1000 $Bq.m^{-3}$ likely, there is no reason to waste public or private funds on residential radon reduction measures in all but perhaps very few exceptional cases of very high radon levels in the homes of heavy smokers. It has been correctly pointed out that a glass of good red wine would provide more life expectancy and fun, and cost

less. No wonder that, despite all the propaganda about 'dangerous radon', only very few people seem to be eager to spend private money on the reduction of the radon levels in their homes.

Moreover, much indirect evidence such as the results in lung cancer reduction for external low-LET exposures with 1 Gy and a threshold at 2 Gy[10], or the RBE for alpha emitting inhaled aerosols determined to be 2 instead of 20[11] and studies demonstrating basic differences in the biological response between low dose/dose-rate and high dose/dose-rate radiation, supports an important conclusion, namely that residential radon is one of the many pseudo-problems in radiation protection, confounding more serious public health problems. Under all but perhaps a few exceptional conditions, we do not have to worry about residential radon risks.

As randomised double-blind clinical studies have demonstrated, long term beneficial radon health effects (e.g. among the about 75,000 patients treated annually with radon inhalation, drinking, or bathing, in German and Austrian clinics) far exceed any hypothetical small risks at the mGy levels involved[12]. Even ICRP now seriously reconsiders the radon 'problem' (such as permitting ten times as much public exposure to radon than to external radiation), as well as the reduction of the RBE for alpha particles from 20 to 10 — last but not least because of serious consequences of restrictive radon regulations for important conventional industries (phosphate, mineral and coal mining, etc.) in many national economies – without evidence of adverse effects from those practices (for graphs and more references, see reference 12).

**Rebuttal**

Professor Field's argument projects the image of a firm and scientifically sound basis, and surprising precision with statements such as: "The results of the meta-analysis showed an increased summary excess risk of 14% at 148 $Bq.m^{-3}$", and the (also repeated) 18,600 estimated lung cancer deaths/year in the USA. Unfortunately, such magical 'consensus' numbers are taken seriously by many people in the radon industry to provide funds for further research and measurements, expensive remediation programmes, etc. Unfortunately, limited space permits mentioning here only a few counter arguments, which do not support what became the 'official' point of view in various countries.

There is no question about radon being a carcinogen in humans. The question is whether the relatively low residential radon levels may indeed increase the lung cancer risk. In addition to the lack of power in most radon studies[13], we are faced with, among many other problems, that of 'forgotten doses' among the miners.

In attempting to extrapolate underground miners' data to residential radon situations (besides confounders such as arsenic, silica, etc.), there are differences in aerosol size and daughter product attachment distributions, the contributions of $^{220}Rn$, external gamma radiation, etc.[14], which, in combination, lead to an underestimate in the actual bronchial tract dose of miners by at least a factor of 2, and perhaps up to 10. This implies a corresponding overestimate in residential lung doses. Besides, it has been shown in single-cell alpha microbeam experiments that low (single-hit) alpha exposures are much less carcinogenic than high multi-hit exposures[15].

The validity of ecological studies should not be neglected. On the one hand, apparently many of the case–control studies lack sufficient power[13]. On the other hand, many arguments against ecological studies are not very convincing[4].

In BEIR VI, it is stated that the lung cancer risk from smoking amounts to a factor of 10-20 (other estimates are in the 30-40 region), compared to indoor radon with 0.2-0.3. Obviously, such an overwhelming confounder can only be considered with a sufficiently correct self-reporting of lung cancer cases, about 90% of which are smokers[16]. We know that under-reporting of only one cigarette/day results in a 13% error in radon risk estimates and invalidates most population studies. Many other weaknesses of BEIR VI have been pointed out, also in an editorial in this journal[17].

Even BEIR VI admits that there may be a threshold. Therefore, we should probably focus future studies and debates on the question where exactly this threshold is hidden in the murky waters of statistical background noise, probably somewhere around 1000 $Bq.m^{-3}$.

In addition, we should keep in mind the substantial social and economical costs involved in the implementation of low radon limits. For example, accidents in radon reduction programmes (e.g. in the former uranium mining areas in East Germany) involve huge overground soil movements and cause real, not hypothetical, human casualties. Probably, serious studies on non-smokers with a wide exposure range from < 50 to >3000 $Bq.m^{-3}$, are most likely to obtain a less biased view of the real situation.

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## SUMMARY

In assessing the risks of exposure to ionising radiation, it is important to neither overstate nor understate the effects of the hazard. These requirements are often difficult to satisfy, especially since much of our knowledge about the effects of low levels of radiation is subject to rather large uncertainties. Our participants have given their opinions about the risk of lung cancer induction resulting from exposure to radon in residences. Each agrees that exposures to high concentrations of radon are hazardous. However, as with low level exposures to other types of ionising radiation, quantifying relatively small risks is quite difficult. The national and international standards setting bodies have recommended a fairly conservative approach that may overestimate the 'true' magnitude of deleterious effects and their dependence upon exposure, but this is to be expected given the uncertainties in the data and the need to avoid underestimates. A conservative approach can have both positive and negative consequences, and it is also important to neither overstate nor understate these consequences.