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LETTER TO THE EDITOR

The assumption of radon-induced cancer risk

Krzysztof W. Fornalski[1,2] · Rod Adams[3] · Wade Allison[4] · Leslie E. Corrice[5] · Jerry M. Cuttler[6] · Chris Davey[7] · Ludwik Dobrzyński[8] · Vincent J. Esposito[9] · Ludwig E. Feinendegen[10] · Leo S. Gomez[11] · Patricia Lewis[12] · Jeffrey Mahn[13] · Mark L. Miller[13] · Charles W. Pennington[14] · Bill Sacks[15] · Shizuyo Sutou[16] · James S. Welsh[17]

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We read with interest the article by Axelsson et al. [1] about the potential risk of lung cancer due to inhalation of radioactive radon gas. Indeed, this has been the subject of many scientific papers around the world for years without clarification of whether there is risk or there is no risk when the radon concentration is low.

The paper by Axelsson et al. [1] states that "residential exposure to radon is considered to be the second cause of lung cancer after smoking." The authors cite the publications of many well-known radon experts, especially the analysis of 13 European case–control studies by Darby et al. [2]. They underscore their basic argument that in Sweden, there is a 16 % increase in the risk of radon-induced lung cancer per 100 Bq/m^3. However, there appear to be logical mistakes in their reasoning, which are presented below.

The first mistake concerns their chosen dose–response model. They determine the 16 % increase in risk per 100 Bq/m^3 by using the linear no-threshold (LNT) hypothesis. According to this hypothesis, the excess risk increases linearly versus Bq/m^3 (or vs. mSv effective dose) from zero to the maximum. There are no data that support the validity of this hypothesis over the whole range of doses. All existing studies are subject to a number of limitations. Moreover, there is a huge scatter in the results, so it is impossible to reach a coherent conclusion [3]. In fact, the LNT hypothesis has been criticized fundamentally in many independent studies [4].

The second fallacy—the "zero radon environment"—is related to the previous one. The authors [1] widely invoke the value "0 Bq/m^3," which makes no sense from both the

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Jeffrey Mahn has retired from Sandia National Laboratories, Albuquerque, NM, USA and Bill Sacks has retired from FDA, Rockville, MD, USA.

✉ Krzysztof W. Fornalski
krzysztof.fornalski@gmail.com

1 PGE EJ 1 Sp. z o.o., ul. Mokotowska 49, 00-542 Warsaw, Poland

2 Polish Nuclear Society (PTN), Warsaw, Poland

3 Atomic Insights LLC, New York, NY, USA

4 University of Oxford, Oxford, UK

5 New York, NY, USA

6 Cuttler & Associates, Vaughan, ON, Canada

7 King Abdullah University of Science and Technology, Thuwal, Saudi Arabia

8 National Centre for Nuclear Research (NCBJ), Otwock-Swierk, Poland

9 University of Pittsburgh, Pittsburgh, PA, USA

10 Heinrich-Heine University, Düsseldorf, Germany

11 Leo S. Gomez Consulting, Albuquerque, NM, USA

12 Free Enterprise Radon Health Mine, Boulder, MT, USA

13 Sandia National Laboratories, Albuquerque, NM, USA

14 Executive Nuclear Energy Consultant, Alpharetta, Georgia, USA

15 Green Valley, AZ, USA

16 Shujitsu University, Okayama, Japan

17 Loyola University Chicago Stritch School of Medicine, Maywood, IL, USA

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physical and the epidemiological point of view. There is no place on earth without a concentration of radon, and epidemiological data with "zero" dose from radon do not exist. That prohibits empirical confirmation of any extrapolation from high doses or concentrations down to zero radon level. All assumptions based on 0 Bq/m^3 make no sense.

The third mistake also pertains to the use of the LNT model. The authors of the analysis of the 13 European case–control studies [2] state at the beginning of their paper, "Firstly, a model was fitted in which the risk of lung cancer was proportional to $(1 + \beta x)$, where x is measured radon concentration and β the proportionate increase in risk per unit increase in measured radon." Finally, they reported their results as "the risk of lung cancer increased by 8.4 % (…) per 100 Bq/m^3 increase in measured radon concentration" [2]. The authors selected a linear model to process the data, which are very uncertain, and then showed that the data fit the linear model that they assumed. There are also other models that would equally well fit the widely scattered data.

Finally, the results presented by Axelsson et al. [1] are just statistical calculations based on the LNT model. They calculate the number of hypothetical, prevented cancer cases based on selected assumptions. However, the authors did not consider many studies that show no correlation or even a negative correlation between lung cancer and low radon concentration [3, 5–8]. Moreover, the radon levels in Sweden are rather low, and there is no evidence of any statistically, let alone clinically, significant increase in cancer risk that could be related to Swedish conditions.

The examples of effects opposite to those claimed in the Axelsson et al. paper [1] can be divided into various types, e.g., the case–control study by Thompson et al. [5] or the well-known ecological US study by Cohen [6]. Also, investigations on animals that serve as good models for humans, like beagle dogs, following inhalation of alpha-radioactive $^{239}PuO_2$ [9]—analogous to radon inhalation—fail to show a linear increase in risk versus cumulative dose, but rather demonstrate the presence of a threshold effect. Additionally, Cuttler and Feinendegen [10] estimated that a radon concentration of 1000 Bq/m^3 gives a cumulative lung dose after 91 years of 100 cGy in humans, the apparent threshold for increased risk.

All of the existing radon studies can be easily analyzed together as one meta-study, leading to the conclusion that there is no evidence for excess lung cancer risk below 800 Bq/m^3 [3]. The conclusion remains the same even when the pro-hormesis studies are excluded from the analysis [11].

The final and general conclusion of this letter is that excess risk of lung cancer due to low concentrations of radon has been neither empirically detected nor theoretically demonstrated, while the opposite has, in fact, been supported by voluminous evidence. The putative increase in lung cancer risk due to low concentrations of radon is not a real effect; it is an assumption only.

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