



Exposure to Radon as a Public Health Issue- A Review

A. M. Asere^{1*} and I. R. Ajayi¹

¹Department of Physics and Electronics, Adekunle Ajasin University, Akungba Akoko, Ondo State, Nigeria.

Authors' contributions

This work was carried out in collaboration between both authors. Author AMA designed the study, wrote the concept of the article, manage the literature searches and wrote the first draft of the manuscript. Author IRA vetted and provided finishing touch to this manuscript. Both authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JSRR/2015/18779

Editor(s):

(1) Viroj Wiwanitkit, Department of Laboratory Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand.

Reviewers:

- (1) Anonymous, Akdeniz University, Turkey.
(2) Sainudeen Pattazhy, University of Kerala, India.
(3) Anonymous, Kemerovo State University, Russia.

Complete Peer review History: <http://sciencedomain.org/review-history/10020>

Mini-review Article

Received 9th May 2015
Accepted 15th June 2015
Published 2nd July 2015

ABSTRACT

Radon (^{222}Rn) is a noble gas formed from the decay of radium (^{226}Ra), which is a decay product of Uranium (^{238}U). Radon is an inert noble gas, having a half-life of 3.8 days emanating from rocks and soils and tending to concentrate in enclosed spaces like underground mines or houses. Radon was classified as a human carcinogen by the International Agency for Research on Cancer in 1988, a review of the major studies of underground miners exposed to radon that were available in the 1990s and case-control study of residential radon exposure confirmed radon as a carcinogen. This paper reviews the publications of the World Health Organization, the International Atomic Energy Agency and other relevant publications specifically on sources and historical perspectives of radon, the association between lung cancer and radon, health effects and radon risk communication.

Keywords: Radon; lung cancer; health effect; radon policy; radon risk communication.

*Corresponding author: Email: aseream@gmail.com;

1. INTRODUCTION

One of the major characteristics of the radioactive series is the existence of a radioactive gas common to all which is a different isotope of element radon. In the Uranium series the gas $^{222}_{86}\text{Rn}$ is called Radon; in the Thorium series, the gas $^{220}_{86}\text{Rn}$ is called Thoron while the gas $^{219}_{83}\text{Rn}$ is called Actinon in the Actinium series. Radon (^{222}Rn) is a direct decay product of ^{226}Ra in the Uranium series of element, the adverse contributions of Thoron and Actinon to exposure is of little importance because of their very short half life. Thoron has much shorter half life (55s) than radon (3.83 days), the distance it can travel before undergoing decay is shorter than the distance radon can travel in the same medium. Therefore, its expression in the environment is quite different from that of radon [1].

UNSCEAR results published in 2000 reported the annual dose average over the world population to be about 2.8 mSv. Over 85% of this is from natural sources with about half coming from radon decay products in the home. The worldwide average annual effective dose from the decay products of radon is estimated to be about 1.2 mSv [2]. The ICRP and IAEA have recommended the use of action levels in the range 200-600 Bqm⁻³ above which house holders are advised to reduce radon levels in their homes. Several studies have confirmed ^{222}Rn to be the cause of lung cancer in the underground miners and indoor environment and that it is a major contributor to the ionizing radiation dose received by the general population [3].

Uranium occurs naturally in varying levels in all rocks and soils. Radon produced in the rocks and soils escapes to the air thereby making it present everywhere. The air we breathe in contains radon. Exposure to radon-222 is unavoidable in public places, workplaces and dwellings. The IAEA and the International Labour Office acknowledges the importance of controlling radon exposure in workplaces other than mines. The rise in the indoor air concentration was recognized as a radiation health hazard, potentially causing an increase in the incidence of lung cancer. Radon has thus become a concern for underground miners and buildings constructed with materials containing elevated amount of radium [4].

While it is the radon progeny rather than radon gas itself that presents the greater risk, the word 'radon' is also used generally as a convenient shorthand for both the gas and its progeny. Much of the discussion in the scientific literature on indoor radon is expressed in terms of radon concentrations rather than concentrations of radon decay products, for two principal reasons. Firstly it is much easier to measure concentrations of radon gas than concentrations of its progeny, especially for long term measurements. The second reason is that, owing to the higher dose conversion factor of the unattached fraction of radon progeny in lung dosimetry models and the inverse relationship between the unattached fraction and the equilibrium factor in indoor air, the effective dose relates more to the radon gas concentration than to the equilibrium equivalent radon concentration [2].

The short lived radon decay products are radioactive because they are alpha particle emitters, ^{218}Po and ^{214}Po are electrically charged and can attach themselves to tiny dust particles, water vapours, trace gases in indoor air and other solid surfaces and can be easily inhaled [5]. In homes, underground mines and workplaces, which may be poorly ventilated, the level of these radionuclides and its decay products can accumulate to high levels. Most of our time is spent indoor either at home or in workplaces, there is every tendency of exposure to cancerous effects of radon.

2. RADON HISTORICAL PERSPECTIVE

High mortality rate from respiratory diseases among miners in Central Europe had been identified before the seventeen century. In the nineteenth century, it was confirmed that the disease was lung cancer. Radon was first suspected as the primary cause of these cancers. As early as in the 1950s, domestic and drinking water from drilled wells were observed to have high concentrations of radon. By the mid-1970s, building materials was recognized to emanate radon in some areas due to the use of alum shale with enhanced levels of radium. In 1978, soil gas infiltration became recognized as the most important source of radon as indoor radon concentration were found not to be associated with well water transport or emanation from building materials [3]. Radon has been classified as a human carcinogen [6,7] based on the epidemiological studies of

underground miners exposed to high radon concentrations in their workplaces.

A large number of studies have examined the relationship between indoor radon and lung cancer since then. The three pooled-analyses from the major studies in Europe, North America and China present very similar pictures of the risks of lung cancer from residential exposure to radon. Together, they provide overwhelming evidence that radon is causing a substantial number of lung cancers in the general population and they provided a direct estimate of the magnitude of the risk. They also suggested that an increased risk of lung cancer cannot be excluded even below 200 Bq/m³, which is the radon concentration at which action is currently advocated in many countries [3]. Radon is now recognized as the second most important cause of lung cancer after smoking in the general population [3].

3. SOURCES OF RADON

Radon emanates naturally from the decay of uranium and radium which occurs from rocks and soils everywhere. It is an inert noble gas with half life of 3.82 days. When it decays, the resulting products remain in the rock at the place of its production and it is capable of moving through pore spaces next to fracture or a discontinuity in the rock. The radon in the pore spaces is mainly transported by diffusion, with the transport rate depending on the porosity and permeability of the soil or by convection, dependent on the presence of cracks and faults. Radon is soluble in water; groundwater that passes through uranium bearing soils and rocks contains radon. Radon levels may be high in wells, borehole, bottled and sachets waters produced for commercial purposes and in underground workplaces.

When radon – rich groundwater is used as drinking water people are exposed both through the water consumption and by radon being released from the water to air and being inhaled [1]. Radon levels may be high in workplaces such as laundries and restaurant kitchens as a result of the use of such waters. Many countries do not have recommendations on radon concentrations in drinking water. Buildings may have high radon levels depending on the composition of the under soil, presence of cracks in the floors and foundation of the building, the area of building with contact with the ground, ventilation habit, lifestyles, meteorological and

seasonal parameters, pressure driven flow of the gas. This flow arises because buildings are normally at a slight under pressure with their surroundings. Some buildings materials may have elevated levels of ²²⁶Ra and a high porosity that allows the radon gas to escape, thereby act as a significant sources of indoor radon [4].

4. ASSOCIATION BETWEEN LUNG CANCER AND RADON EXPOSURE

Efforts to directly investigate the association between indoor radon and lung cancer have provided convincing evidence of increased lung cancer risk causally associated with radon even at levels commonly found in buildings. Risk assessment for radon both in mines and residential settings have provided clear insights into the health risk due to radon. It is not possible to present review results of radon studies from every country, some are particularly noteworthy.

4.1 Biological Effect of Ionizing Radiation VI Reports

A review on the lung cancer risks in radon exposed miners studies carried out by the BEIR VI,1999 where eleven cohort studies were considered including a total of 60,000 miners in Europe, North America, Asia and Australia, among whom 2,600 deaths from lung cancer had occurred. Eight of these studies were of uranium miners, and the remainders were of miners of tin, flourspar or iron. Lung cancer rates generally increased with increasing cumulative radon exposure [3].

4.2 German Cohort Study

A total of 59,001 men were studied; a total of 2 388 lung cancer deaths was reported to had occurred at the time of the first mortality follow-up. The miners were reported to come from the same geographical area and had the same social background, and the entire cohort was subjected to the same follow-up procedure and the same system of exposure assessment [3].

4.3 The IOWA Lung Cancer Study

The subjects were female IOWA residents who had occupied their home for at least 20 years. The study was an epidemiological study involving 413 cases of women who had developed lung cancer and 614 controls that did not have lung cancer. IOWA was known to have the highest

average radon concentrations in the U.S. 28% of the living areas for the controls and 33% of the living areas for the cases exceeded the EPA's action level of 4 pCi/l [8]. The risk estimates suggested that cumulative radon exposure in the residential environment is significantly associated with lung cancer risk.

4.4 Scotland Study

In examining the relationship between lung cancer and radon in small areas across Scotland, for population aged over 54, there was no significant relationship between radon exposure and lung cancer incidence. For aged less than 55 lung cancer rates were significantly higher in places expected to have the highest levels of radon [9].

4.5 US Cohort Study

11 cohort studies of radon in exposed underground miners including 65,000 men and more than 2,700 lung cancer deaths. In the study, conclusion were made that in mines, about 40% of all lung cancer deaths may be due to radon progeny exposure, 70% of lung cancer deaths in never smokers and 39% of lung cancer deaths in smokers. In the U.S. 10% of all cancer deaths might be due to indoor radon exposure, 11% of lung cancer deaths in smokers and 30% of lung deaths in never smokers [10].

4.6 The European Pooling Study

Over 7,000 lung cancer cases and more than 14,000 controls from all thirteen European studies were entered into pooled analysis. The risk of lung cancer increases by 8% per 100 Bq/m³ increase in measured radon concentration. Significant association between radon concentration and lung cancer was found even when the analysis was restricted to people in homes with measured radon concentrations below 200 Bq/m³. When the random year-to-year variability in radon concentration was taken into account the risk coefficient was 16% per 100 Bq/m³ [3].

4.7 The North American Pooling Study

The North American pooling study involved 3,662 lung cancer cases and 4 966 control from seven studies in USA and Canada. The risk of lung

cancer increases by 11% per 100 Bq/m³ in measured radon concentration [3].

4.8 The Chinese Pooling Study

The Chinese pooling study, involved 1,050 cases of lung cancer and 1,996 controls from two studies. The risk of lung cancer increases with 13% per 100 Bq/m³ [3].

4.9 Overall Evidence on the Risk of Lung Cancer

From the three pooled radon studies, it is evident that radon is a cause of lung cancer in the general population even at low concentrations in homes. In all the three pooling studies there was no evidence that the proportionate increase in risk per unit increase in radon concentration varied with the age, sex or smoking habits of the study subjects more than would be expected by chance. In addition, the dose-response relationship appeared to be linear, with no evidence of a threshold, and there was substantial evidence of a risk increase even below 200 Bq/m³, the concentration at which action is currently advocated in many countries. Today, many studies are going on in different countries to determine radon concentration levels in their respective dwellings and workplaces.

5. SMOKING AND LUNG CANCER

Individual that smoke have every tendency to have lung cancer since the majority of lung cancer induced by radon are caused jointly by radon and smoking in the miners study for which smoking information is available and in the European studies. At an individual level, the risk of radon-induced lung cancer following exposure to a given radon concentration is much higher among current cigarette smokers than among lifelong non-smokers. Lung cancer would not have occurred if either the individual had not smoked cigarettes or had not been exposed to radon. For lifelong non-smokers, it was estimated that living in a home with an indoor radon concentration of 0, 100 or 800 Bq/m³ was associated with a risk of lung cancer death (at the age of 75) of 4, 5 or 10 in a 1,000, respectively. However, for a cigarette smoker, each of these risks would be substantially greater, namely 100, 120 and 220 in 1000. For those having stopped smoking, the radon-related risks are substantially lower than for those who continue to smoke [3].

6. HEALTH EFFECT OF RADON

^{222}Rn is the principal source of internal radiation exposure and has the potential to cause lung cancer. When an individual spends time in an atmosphere that contains radon and its decay products, the part of the body that receives the highest dose of ionizing radiation is the bronchial epithelium, although the extra thoracic airways and the skin may also receive appreciable doses. In addition, other organs, including the kidney and the bone marrow, may receive low doses [11]. If an individual drinks water in which radon is dissolved, the stomach will also be exposed. When inhaled the short-lived radon progeny can deposit within the respiratory tract at locations dependent on the diffusion properties of the particles, predominantly the size distribution of the aerosols. The decay products present in the air deposit in the nasal cavities, on the walls of the bronchial tubes and in the deep lung. Because of their relatively short half-lives, the radon progeny decay mainly in the lung before biological clearance can take place.

Two of these short-lived progeny, polonium-218 and polonium-214, emit alpha particles whose deposited energy dominates the dose to the lung. It is believed that the irradiation of the sensitive basal cells of these organs by the alpha particles emitted by polonium-218 and polonium-214 may lead to health effects, principally lung cancer [12]. There is no known threshold concentration below which radon exposure presents no risk. Even low concentrations of radon can result in a small increase in the risk of lung cancer. The majority of radon-induced lung cancers are caused by low and moderate radon concentrations rather than by high radon concentrations, because in general less people are exposed to high indoor radon concentrations.

The radioactive daughters of radon gas disintegrate with the emission of alpha particle, beta particles and gamma-rays. Alpha particles is the most damaging out of the three radiations, it has high linear energy transfer and short range but gamma rays and beta particles have longer range and lower biological effectiveness so their dose equivalent to the lung is negligible. The nucleus of the cell is the most sensitive part to radiation and can undergo severe changes upon interaction with ionizing radiation. The DNA molecule contains the genetic code of each species and is the inherited compound that controls the structure and function of the cell.

Radiation damage to the cell may cause the chromosome or DNA molecule to break either by direct action or indirect action. The passage of alpha particles produced by radon and its daughter products may damage the DNA directly by displacement of electrons from the DNA molecule, most of the indirect action of radiation is on water resulting in the production of highly reactive free radicals that are chemically toxic which may produce new chemical compounds unhealthy for the organism. A damage cell may recover dies or grow abnormally if the radiation has damage the RNA and DNA of the molecules. The interaction of alpha particle with the fluids that lines the lungs damages the DNA of nearby cells. There is no threshold limit exposure to alpha radiation, even at the lowest dose may produce the reactive chemical species within cells and their production increases with alpha particle dose. If an embryo is exposed to radiation at the early stages, developmental defects such as mental retardation may occur.

7. RADON RISK COMMUNICATION

The primary objective of a radon risk communication programme should be to persuade policy makers at national and local government level that exposure to radon is an important public health issue that requires action. In presenting awareness to the public, several communication techniques ranging from passive to active communication can be used such as direct mailing through the use of facts sheets or brochures, road signs and billboards, news paper advertisements, press release, phone-in sessions on local radio, holding briefing at community group meetings, radio or television interviews, meeting with construction industry and several other methods not mention here.

An important criterion in effective radon communication campaigns is to identify the target audiences one wishes to inform. These audiences can be grouped into direct and indirect categories. These direct categories are individuals whose actions could directly result in reducing lung cancer risk. People in this category are householders, tenants, smokers, architect, engineers, builders, construction companies, real estate companies, and financial institutions. The indirect category are individuals whose actions either by decision making or by highlighting radon problem would help to increase public awareness and perception thereby help to encourage radon prevention and reduction in the communities. People in this category are

governmental and political decision makers, local authorities, legal advisers, lawyers, media, professional associations and financial institutions.

It's important to let the public realize that radon is a radioactive gas present in homes; easy to measure, prevent and reduce and the lower the radon concentrations in homes the lower the risk. Exposure to radon in homes is one of the most important causes of lung cancer deaths worldwide. Absolute risk to smokers at any level of radon exposure is much greater than that of never-smokers or former smokers and that the majority of radon related lung cancer deaths occur in current and former smokers. The numbers of people that die from radon related lung cancer cases are much higher than that from other several common cancers from other sources.

It's equally important to assess and evaluate the public's knowledge and perception of radon after a radon communication campaigns. This will allow the government, policy makers and governmental agencies to focus on, improve, and establish the core messages of the campaigns. To crown it all, it's necessary to motivate individuals to test for radon and fix problems if arises. Encouraging the public to take action on reducing radon is necessary otherwise the objective of risk communication may not be achievable [3].

8. NATIONAL RADON POLICY

Countries need to comprehensively plan and implement national radon programmes aiming at protecting the public against indoor radon exposure. This will require input from national agencies, Public Health Agencies, media, press, building research scientists, construction codes agencies, geological institutions, mitigation code experts, measurement laboratories, training providers and professional associations. Countries need to develop radon prevention and mitigation programmes reflecting elements that are unique to their regions (e.g. radon sources, transport mechanisms, building regulations, building codes and construction characteristics). Construction industry and building professional need to consistently plan to integrate a radon reduction strategy to achieve the best outcome in building construction.

People are exposed to radon as members of the public in dwellings or as workers in workplaces.

They also are exposed to radon in public or private places open to the public either as members of the public, as patients or as workers. Since an individual can move between many places during the same day, a radon policy should ideally provide consistency in the management of the different locations in a given area and should also provide an integrated approach even though the time of occupancy varies from a location to another. The radon policy should consider reducing population radon exposure and related health risk, set a national reference level for radon in homes, and have a framework to keep radon concentrations as low as reasonably achievable and creating radon awareness among the public.

9. CONCLUSION

The historical review of emergence of radon, epidemiological studies of underground miners and case - control studies of indoor radon and other various studies performed so far have shown a relationship between radon and lung cancer. Despite the fact that many researches are going on all over the world to know radon levels both in dwellings and workplaces, there still exist countries where the knowledge about radon or its effects is still sparse. This confers on various governmental bodies, stakeholders, researchers the need to initiate radon sensitization and monitoring programme; set guidelines and reference levels in order to save the people from ignorant exposure to radon in homes and workplaces. If possible the WHO, IAEA, ICRP, UNSCEAR and other international recognized bodies should make it mandatory for each country to start initiative programmes on radiation exposure and set radiological control policy.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. United Nations Scientific Committee on the Effects of Atomic Radiation Reports, Annex E: Source-to-effects assessment for radon in homes and workplaces. New York: United Nations, UNSCEAR; 2006.
2. International Atomic Energy Agency, Radiation, People and the environment; 2004.

3. World Health Organization (WHO). Handbook on Indoor radon. A public health perspective. WHO Press, Geneva; 2009.
4. International Atomic Energy Agency. Radiation protection against radon in workplaces other than mines. Safety Report Series; 2003.
5. Kant K, Charkavarti SK. Radiological impact of airborne radon and its progeny in dwellings. Indian Journal of Pure and Applied Physics. 2004;42:157-161.
6. International Agency for Research on Cancer. Monographs on the evaluation of carcinogenic risk to humans: Man made fibers and radon. IARC. 1988;43.
7. World Health Organization, 1986 Indoor air quality research: A report on WHO meeting, Stockholm, 1984. WHO; 1986.
8. Field RW, Lynch CF, Steck DJ, Smith DJ, Brus CP, Neuberger, et al. The IOWA radon lung cancer study. Heartland Radon Research and Education Program (HRREP); 2000. Available:www.cheec.uiowa.edu/misc/radon.html
9. Pearse J, Boyle P. Examining the relationship between lung cancer and radon in small areas across Scotland. Health Place. 2005;11(3):275-82.
10. Lubin JH, Boice JD, Jr Edling C, Hornung RW, Howe GR, Kunze, et al. Lung cancer in radon exposed miners and estimation of risk from indoor exposure. J. Natl. Cancer Insti. 1995;87(11):817-27.
11. Kendall GM, Smith TJ, Doses to organs and tissues from radon and its decay products. J Radiol Prot. 2002;22(4):389-406.
12. International Commission on Radiological Protection. Lung cancer risk from radon and progeny. ICRP Publication. 2011;115.

© 2015 Asere and Ajayi; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
<http://sciencedomain.org/review-history/10020>