Environtropica, Jan 2008 – December 2010, Vols.5, 6 & 7, 1 – 12. ISSN: 1597-815X

Environmental radon and its potential health effects

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Accepted on March 10, 2008.

Abstract

Radon is a naturally occurring radioactive gas that is formed from uranium present throughout the earth crust. It is released into various homes from radon polluted outdoor air and movement of gases in the soil beneath homes and can become highly concentrated indoors when it is unable to disperse. Inhaled radon is mostly exhaled by humans, but prior to exhalation, its progeny, which are electrically charged radioactive elements can deposit into the lining of the lung, where they also emit alpha radiation. Alpha radiation has the potential to disrupt the DNA structure of the lung cells thereby initiating a cancerous condition. Radon has been conclusively shown in epidemiologic studies of underground miners to cause lung cancer and this is also supported by evidence from experimental studies of animals. Molecular and cellular studies also provide an understanding of the mechanisms by which radon causes lung cancer. In order to provide more understanding of the phenomenon of radon exposure, a review on the production of radon, its mode of release into the home and environment, exposure route and potential health effects are presented. Also presented were the various methods of measuring radon and the need to conduct nationwide radon survey in order to generate detailed data bank on radon exposure.

Key words: *Radioactivity, radon, diffusion, aerosol, alpha radiation, dosimetry, DNA, reactive oxygen species, lung cancer, epidemiology.*

Introduction

Radon is an odourless, tasteless and invisible radioactive noble gas produced from radioactive decay of uranium and radium, that are commonly present throughout the earth crust especially in soil, rock and underground water. Some types of rocks such as granite, phosphates, shale and pitchblende are characterized with relatively high uranium concentration and so store natural deposits of radon. Consequently, there is elevated radon concentration in soils and weathered bedrock where they are located (Gall *et al.*, 1995; Otton, 1989). Areas which receive sediments, surface or ground water from rock units with high concentration of uranium also have increased chances of elevated radon concentration. When formed, it is released into tiny air or water-containing pores between soil and rock particles. It may also be released into ground water and near surface water of oceans, tailings from mines (particularly uranium and phosphate mines), building materials, coal residues and natural gas.

As a result of the long half-lives of uranium and radium and their abundance in the earth's surface, this gas is continually being formed in soil and released to air. Typically it diffuses from the ground to the air before decaying by emission of an alpha particle into a series of short lived radioactive progeny such as ^{218}Po and ^{214}Po, which also decay by emitting alpha particles. Atmospheric radon does not pose major hazard because radon emitted into the atmosphere is rapidly diluted to low levels by continuous circulation through the immense volume of outdoor air. However, in closed or poorly ventilated spaces such as caves and mines it can become highly concentrated. Its concentration can also build up in homes, where exposure of the general population mostly occurs (Yu *et al.*, 1992; Cohen *et al.*, 1984), especially in areas with poor ventilation. This is because it may diffuse through cracks and pores in the foundation of a house built on radon-contaminated soil and can then become trapped inside, especially if the home is well-insulated so that it may not be able to escape. Besides, radon is present in many building materials such as granite, shale, insulation materials, Gypsum in cement etc. that are used to construct houses and so radon is found in homes all over the world.

It is important to note that the phenomenon of radon production in the natural radioactivity chain, the behaviour of its daughter nuclei in air, measurement of radon and radon transport and radon radiation dosimetry are all based on principles of physics. However, some other aspects such as radon emanation studies can be explained in terms of geology of the area while those aspects involving health effects can be explained based on results of epidemiological studies.

Most of the studies on radiation measurements carried out in Nigeria were based on measurement of radioactivity using portable radiation monitors (Okunade *et al.*, 2007; Nwankwo and Akoshile, 2005; Sanni, 1973), measurement of activities of radio-nuclides in fertilizers and Nigerian phosphate rocks using gamma spectrometer with Ge(Li) and NaI(Tl) detectors (Jonah *et al.*, 2002; Ogunleye *et al.*, 2001), and radon emanation study of uranium ore samples using NaI(Tl) based gamma spectrometer (Funtua *et al.*, 1997), to mention a few. However extensive nationwide survey of environmental radon and radon exposure in the home has not yet been carried out in Nigeria and many other African countries. This article is therefore aimed at providing detailed information about radon production, radon measurement, its potential to human exposure and its health effects on humans and to sensitize African governments and other stakeholders on the imperatives of conducting nationwide radon survey.

Production of radon in the ground

Radon is produced from the radioactive decay of uranium and thorium. ^{222}Rn commonly known as radon is produced from the decay of ^{226}Ra in the ^{238}U decay chain and ^{220}Rn commonly known as thoron is produced from the decay of ^{224}Ra in the ^{232}Th decay chain. ^{238}U and ^{232}Th have almost equal abundances in the ground and are also approximately in activity equilibrium with their daughter nuclei down to the stable ^{208}Pb. At a given location, the levels of uranium and thorium in the ground depend on local geology (Larson and Gotfried, 1961). For example granitic rock often contains uranium in concentrations higher than crustal average

and consequently are a potential source of elevated radon levels in soil and underground water (Nero and Nazaroff, 1984).

In soil, radon is transported primarily by alpha recoil and mechanical flow of air and water (Gundersen, 1992; Sogaard-Hansen and Damkjaer, 1987; Schroeder, 1965) resulting in release of radon into the pore spaces between soil particles. In groundwater, radon transportation is determined primarily by diffusion patterns and the direction of mechanical flow of water. The solubility of radon in water is relatively low resulting in its decay (because of its short half live) before it is released from ground water. If such radon-containing groundwater migrates close to the surface, the radon will inevitably be released into the atmosphere. Although the majority of radon present in groundwater will decay prior to its arrival at the surface, groundwater is nevertheless considered the second most prominent source of environmental radon. Radon is also minimally released from water located at or slightly beneath the ocean's surface. Municipal water is sufficiently aerated and as a result, there is diminished level of radon concentration. Well water may have potentially high levels of radon contamination but its concentration in deep aquifers varies depending on uranium content of the underlying rock, distribution of the aquifer relative to the rock and groundwater flow patterns. Atmospheric concentration of radon is a function of the soil porosity and meteorological factors, such as precipitation and atmospheric pressure. Its dispersion in ambient air is thus determined by atmospheric stability, including vertical temperature gradients and effects of wind.

Mode of entry into the home

Outdoors, radon generally disperses into the atmosphere, resulting in generally low level of radon immediately above the ground. Even in areas with large uranium deposits and permeable soil, environmental radon does not often build up to hazardous levels because of the mixing capability of the atmosphere through its immense volume. But inside homes that are poorly ventilated and have cracks in their foundations or floor drains, radon levels may gradually build up to hazardous levels.

Generally, radon gas enters a building by diffusion, mainly by pressure-driven flow mechanism (Rogers and Nielson, 1991; Tanner, 1980). The flow is caused by the typically negative indoor pressure compared with that in the soil and the outdoor atmosphere. Negative pressure in the home relative to the soil is caused by exhaust fans and by rising warm air created by fireplaces, clothes dryers, and furnaces. In addition to pressure differences, the type of building foundation can affect radon entry. Cracks in basements for example, readily allow soil gas entry.

Increasing attention is now directed towards radon generated in the concrete floor and radon diffusion from the soil through the concrete floor (Lively and Goldberg, 1999). Radon is released from the concrete floors by diffusion, but the amount released is much less than the advective flux through cracks in the floor. Thus, it is desirable to examine the diffusive properties of concretes and also characterize the relative importance of radon generated within the concrete to determine whether aggregates or other concrete components may contribute significantly to indoor radon. In some cases building materials, such as concrete block, brick,

granite, and sheet rock, contain some radium and are sources of indoor radon. Normally, these construction materials do not contribute significantly to elevated indoor radon levels. In rare cases, however, building materials themselves are the main source of radon.

Radon may also enter into homes via the water supply. In the case of municipal water or surface reservoirs, most of the radon volatilizes to air or decays before the water reaches homes, leaving only a small amount from decay of uranium and radium. It is instructive to note that groundwater that passes over rock rich in uranium and radium, might dissolve some of the radon gas produced from radium decay. The domestic use of water from such sources releases radon into the indoor air through splashing.

Once in the atmosphere, radon daughter ions attach themselves to the natural aerosol particles in air. This behaviour of radon daughter has profound influence on their airborne abundance and of their removal by plate-out on surfaces. For example, when an atom of ^{218}Po is formed from the decay of ^{222}Rn, it appears as a free ion. Molecules of water vapour coalesce almost immediately around the ion, forming a molecular cluster (George and Breslin 1967). Unattached ^{218}Po is highly mobile and may easily plate out on indoor surfaces or be transported outdoors by ventilation. Alternatively, it may simply decay into an unattached ^{214}Pb. If on the other hand, the air is rich in natural aerosol particles, it may attach itself to such particles and persist in room air until it decays (Porstendorfer, 1994).

Radon measurement

Atmospheric radon measurement

Atmospheric concentration of radon is generally in the range of 200-700 pCi/m^3 depending on soil type and meteorological conditions (Price *et al.*, 1996). Its concentrations in air can be measured using the scintillation counter (Cothern and Smith, 1987). The measurement involves introducing the gas into a counting cell, whose inside walls are coated with zinc sulfide (ZnS), except one end which is covered with a transparent window for coupling to a photomultiplier tube. When an alpha particle emitted by the gas strikes the wall of the cell, a flash of light is emitted from the ZnS coating. The light is then detected by the photomultiplier tube and converted into an electrical signal. Another method is to count the alpha particles from the decay of radon and its daughters in an ionization chamber, which measure electrical pulses from individual decay events or currents resulting from the integrated effect of all decays.

Alternatively, radon daughters can be collected by drawing air through a filter since they deposit readily on dust particles or other surfaces (Porstendorfer, 1994). In order to determine the concentrations, counting must begin shortly after the sample is collected because of the short half-lives of radon daughters. For measurement of both radon and radon daughter concentrations, the two-filter or several-filter-method can be used (Wallner, 1997). In this method the radon and its progeny are differentially collected in separate filters. The filter samples containing either the radon or its daughters can then be measured using alpha particle scintillation counter with a ZnS phosphor. The filter containing the daughters is usually covered with a thin sheet of ZnS phosphor and then placed on the photomultiplier tube for counting. An alternative and faster method is to use combined α-β spectroscopy (Cothern and Smith, 1987).

Measurement of radon in water

Measurement of radon in water usually requires correction for its decay during the decay between sampling and analysis. Liquid scintillation counters are usually used in cases where its concentrations in water are sufficient high (i.e >1000 pCi/L), as is often the case with well water (Wallner and Irlweck, 1997; Hair and Baldwin, 1995). Alternatively, its concentration can be determined by direct counting of gamma rays from its daughter decay using standard gamma-ray spectroscopic techniques with a Ge(Li) detector. The original concentration can be distinguished from the radium-226 concentration by repeating the count after 30 days, a period long enough for the original radon to have virtually decayed and so is in secular equilibrium with radium-226. A more sensitive method of detection suitable at lower concentrations is provided by the gas extraction technique. In this method, radon is extracted as a gas and the emitted alpha particles are counted in a ZnS scintillation cell (Cothern and Smith, 1987).

Measurement of indoor radon

Residential radon monitoring and testing devices include Charcoal canister, consisting of a small can covered by a screen, with the charcoal contained in the space below the screen. Radon and its decay products are absorbed onto the charcoal and are measured by counting with a sodium iodide detector or a liquid scintillation counter. Another detector that can be used is the alpha track detector, which contains a small sheet of plastic that is exposed for a period of between one and twelve months. Alpha particles etch the plastic as they strike it. These marks are then chemically treated so that the tracks are enlarged. They are then counted in the laboratory to determine the radon concentration. Other detectors used for measuring indoor radon include Electret ion detectors containing an electrostatically charged teflon disk. Ions generated by the decay of radon strike and reduce the surface voltage of the disk. By measuring the voltage reduction, the radon concentration can be determined. Continuous radon monitors are active devices that continuously measure and record the amount of radon in the home and thus require uninterrupted power supply.

Residential radon measurement can be short termed lasting between two days and three months or long termed lasting more than three months. For short-term measurement, any of the above devices can be used. Prior to radon measurement, doors and windows should be closed for at least twelve hours and measurement taken at least 50cm above the floor in the lowest level of dwelling. It is instructive to note that short termed radon measurement is usually characterized with low precision and high uncertainty because of daily variations (Darby *et al.*, 2001; Debaje *et al.*, 1996; Price and Nero *et al.*, 1996), but the result is rapid. In the case of long termed indoor radon survey, lasting more than three months, alpha track detector, electret ion detector or continuous monitor are recommended. Even though the result may not be available until the end of the survey the result obtained usually indicates accurate average radiation level and exposure.

Human exposure to radon

Although radon is chemically inert and electrically uncharged, the radon progeny formed via the radioactive decay series are in fact electrically charged and readily attach themselves to microscopic dust particles. These dust particles are frequently inhaled into the lungs or

.ingested (from drinking water) into the gastrointestinal tract. If inhaled, radon itself is mostly exhaled immediately before it can decay and deposit a significant radiation dose to the lung tissue. Its short lived progeny (which are soluble solid substances) are however released from the dust particles after they undergo solvation. This is subsequently followed by deposition on the bronchial epithelium by impaction, sedimentation or diffusion (ICRP, 1994).

In spite of the fact that the radon daughters are eventually cleared from the respiratory tract through mucociliary action or engulfment by macrophages, they readily undergo subsequent radioactive decay processes by emitting alpha radiation which slowly penetrates the inner lung surface before they are removed. Interaction of alpha radiations with lung cells has the effect of disrupting the DNA structure of these cells (Prise 1994; Cornforth and Goodwin 1991; Brooks *et al.*, 1990; Aghamohammadi *et al.*, 1988) thereby potentially initiating the first step in a chain of events that can lead to cancer. However, these alpha particles can rarely reach cells in other organs because of their short travel distance and for this reason lung cancer is the most potentially important cancer hazard posed by radon.

The carcinogenic property of radon was in fact confirmed through epidemiologic studies of miners of uranium and other ores (Samet *et al.*, 1989). Many populations of underground miners exposed to radon and its progeny have been shown to be at increased risk of lung cancer (Lubin and Boice 1997) which is related roughly linearly to exposure. For this reason, among others, risk models for exposures received by the general population were based on extrapolation of dose received by miners (by about three orders of magnitude) to the lower dose exposure in the home (Lubin *et al.*, 2004; Darby *et al.*, 1992) Exposure of animals to radon has also provided confirmatory evidence of its carcinogenicity (Cooper *et al.*, 1982).

Dissolved radon in water gain entry into the body through oral ingestion, although a substantial portion of the total dissolved radon may be lost by aeration and would be available for inhalation. Ingestion of water with dissolved radon gas releases charged radon daughters which attach themselves to the stomach lining (Khursheed, 2000). Uncharged radon gas are absorbed into the blood stream through the stomach or intestinal walls and distributed among the organs according to the blood flow and the relative solubility of radon in the organs as compared to the blood (Nussbaum and Hursh, 1957). Common organs of destinations are the liver, lungs, kidney and other adipose tissue stores. Radon dissolved in blood that enters the lung will equilibrate with air in the gas-exchange region, after which the bulk of it is eliminated by exhalation. Once the dissolved gas decays and becomes charged, it can bind and decay further within various body tissues, and emit harmful, mutagenic and cytotoxic alpha particles. Long-lived radon progeny had been detected in excreted urine of miners. (Schramel *et al.*, 1997; Dang *et al.*, 1992).

Health effects of radon

Genetic damage caused by radon

The increased risk of lung cancer from radon primarily results from alpha particles emitted by the short lived radon daughters, which penetrates the cell nucleus. The passage of these

energetic alpha particles may damage the DNA, the inherited compound that controls the structure and function of cells. The alpha particles initiate the DNA damage by breaking the electron bonds that hold molecules together. The DNA damage may occur directly by displacement of electrons from the DNA molecule, or indirectly by changing the structure of other molecules in the cell, which may then interact with the DNA. When one of these events occurs, a cell can be destroyed quickly or its growth or function may be altered, thus causing genomic changes typically in the form of point mutations and transformations. Fortunately cells have the ability to repair the damage done to DNA by radiation, chemicals, or physical trauma. The effectiveness of these cellular repair mechanisms depends on the kind of cell, the type and dose of radiation, the individual and other biological factors.

An alpha particle emitted from radon daughter often decays is in the form of a high energy helium ion, $He^{2}+$. These helium ions penetrate the cell nucleus in a linear pattern and deposit energy known as Linear Energy Transfer (LET). High LET α-particles are fundamentally different from low LET radiation such as β-particles and γ-rays in terms of the physical damage to tissues and the DNA. Therefore the biological effects would also be fundamentally different especially at different environmental dose level. At the annual equivalent dose from natural background of low LET radiation of about 1 mSv, each cell nucleus is traversed approximately by one low LET particle per year, whereas for α-LET only one in approx 5000 cell nuclei is traversed per year. Thus majority of the cells are not traversed by α-particles, but for the few that are traversed, the radiation dose is very large. It is instructive to know that the deposition pattern of low LET radiation is such that energy deposition is spread throughout the cell, whereas in the case of α particles, energy deposition is confined to a much smaller number of narrow tracks, with large parts of the cell receiving no energy at all (Wilson and Paretzke, 1980). As a result of such concentration of physical damage, α-particles can readily induce DNA irreparable double strands breaks, whereas low dose low LET radiation may be capable of inducing only DNA single strand breaks, which are readily repairable (Wolff, 1996). Thus the passage of a single alpha particle has the potential to preserve the irreparable DNA damage and incorporate it into the genetic structure of transcribed DNA. Since DNA mutations induced by alpha particles may continue through several cellular generations, the mutational insult may later become too cytotoxic for the cell to continue replication. The inevitable response is tumor progression thus providing an indication that cancers often originate from damage to a single cell (Day, 1999). These observations provide a mechanistic basis for a linear relationship between alpha-particle dose and cancer risk at very low exposure levels. These considerations were the basis for the adoption of a linear-non-threshold model for the relationship between radon exposure and lung cancer risk.

Health effects caused by reactive oxygen species resulting from radon exposure

Radon exposure may also cause toxic health effects through production of oxygen radicals otherwise known as reactive oxygen species (ROS). ROS consists of chemically reactive super-oxide (pair of oxygen atoms) and hydrogen peroxide, the later being electrically charged. These intermediate molecules damage nucleotide bases and form DNA lesions as secondary by-products of formation (Emerit, 1994; Evans 1992). Therefore, these molecules could be the

source of damage within a cell that sparks the onset of cancer, since they can persist and continue to cause mutations in DNA. This negates the widely held assumption that alpha particles cause genetic changes only through direct traversals of cell nuclei. The interaction of alpha particles with the fluids that line the lungs damages the DNA of nearby cells. Exposure even to the lowest doses of alpha emission produces the very reactive chemicals within cells and their production increases with the alpha-particle dose. This confirms that radon is dangerous even at very low exposure levels i.e. no safe threshold. In some cases, radon may be disproportionately more harmful at lower concentrations. A study by Day (1999) provides clear evidence that a single alpha particle can induce mutations and chromosome aberrations in cells that received no direct radiation exposure to their DNA. These results indicate the need to re-assess the potential genotoxic effect of low dose radiation and suggest that the adoption of direct proportionality in radiation may significantly underestimate the risk of low-dose radiation.

Radon gas is soluble in lipids and accumulates in lipid tissue throughout the body with the highest concentration in the brain, bone marrow, and nervous system. But none of its heavy metal daughters are soluble in the lipids and consequently, remain trapped in the brain and bones, where they continue to emit gamma radiation and alpha particles. This may result in development of leukemia, which is a cancer of the blood and other types of cancers. Some Researchers have also discovered that the presence of radioactive radon daughters in the brains of non-smoking persons with Alzheimer's and Parkinson's disease was ten times greater than it was in the brains of persons with no previous evidence of neurological disorders (Momčilović *et al.*, 2006). This is confirmed by the geographic distribution of Parkinson's disease mortality, which is considerably higher in areas with a greater radon potential. In addition, animals exposed to high concentrations of radon progeny display beside lung carcinoma other diseases such as emphysema, pulmonary fibrosis, and a shortened life span. Furthermore deposition of radon gas carried by aerosol particles on basal layers of the skin may result in development skin cancer. Nevertheless, the risk of other cancers or diseases is much lower than that of lung cancer. ICRP estimated the cancer risk to other organs at about 2% of the lung cancer risk (ICRP, 1993)

Teratogenic effects caused by radon exposure

Exposure to alpha particle during early stages of a fetus development may have profound effects on the health and survival of the fetus. This is because dissolved radon in the blood stream of the mother can pass through the placenta into the developing child. During the embryo phase of a fetus, formation of DNA lesions arising from deposition of radon progeny may result in death of the fetus. At such an early developmental stage, the presence of inheritable DNA lesions causes too much genomic instability and result in negative growth response of the fetus. On the other hand, if the developing child is in the fetal stages, a radon particle passing into the fetus would likely move to lipid portions of the unborn child, namely the brain and other organs. At this stage, exposure to alpha particle may not result in death of the fetus but results in severe inhibition in brain development leading to mental retardation. After birth, exposure follows the same pathways as for adults.

Need for Nationwide radon survey

The phenomenon of radon production and its mode of release into various homes suggest that radon can be found in every home, offices and other enclosures. Epidemiologic studies conducted on uranium miners exposed to high concentrations of radon and radon daughter products suggest a dose-response relationship in favour of lung cancer risk (Steinhausler, *et al.*, 1983; Whittmore and McMillan 1983). Based largely on extrapolations of these findings to lower doses, recent estimates suggest that inhalation exposure to indoor radon and its progeny may be responsible for as many as 24,000 new cases of lung cancer in the United States yearly (Puskin and Boyce, 1989). Experimental evidence in support of non-threshold low dose conducted by Bruce and Goodwin (1997) also confirmed that a relatively low dose of alpha particles results in the generation of extra cellular factors, which, upon transfer to unexposed normal human cells containing free radicals, causes alterations in DNA. In spite of the limitations of the data available, risk assessments based on the studies of miners suggest a need for concern regarding a positive association between the level of indoor radon and the occurrence of lung cancer. It is instructive to acknowledge the fact that ionizing radiation causes random damage to the chromosomes and DNA molecules contained in the nucleus of the cell, including genetic mutations that may affect future generations. Damage to the DNA of reproductive cell, e.g. gamma irradiation of sperm, can lead to genetic deficiencies in the offspring, and if an embryonic cell is damaged, the normal development of the fetus can be disrupted. For example, in areas of high natural background radiation, an increased frequency of chromosome aberrations has been observed. In this regard indoor radon should be considered a potential public-health problem. The scope of the exposed population in recent studies and the large number of lung-cancer cases attributed to radon also provides a strong impetus for nationwide radon survey in every country including Nigeria and other African Countries. It is noteworthy that WHO recently launched the International Radon Project (IRP) initiative aimed at reducing lung cancer risk all over the world. This is an aftermath of the observed consistency of the findings from the latest pooled analyses of case-control studies from Europe, North America and China, which provides a strong argument for an international initiative to reduce indoor radon risks.

Conclusion

The processes of radon production and transport in the environment indicated that radon can be found in all homes worldwide, particularly in areas having soil and rocks that are very rich in uranium and radium. Radon has been conclusively implicated as a carcinogen as a result of alpha radiation associated with its daughter nuclei and the effects of genomic changes caused by interaction of these alpha particles with oxygen radicals present in lung cells. Epidemiologic studies of underground miners, which indicated a markedly increased risk of lung cancer also provides additional evidence. The risk of lung cancer is further increased by cigarette smoking since cigarette smoking and exposure to radon are complimentary agents of lung cancer risk. On the basis of these findings, exposure to radon in homes is expected to be a cause of lung cancer risks in the general population. This therefore confers on various governments the responsibility of setting action guideline for radon exposure and putting in place a well articulated environmental radon monitoring initiative aimed at reducing radon exposure levels in the general population.

References

Aghamohammadi, S.Z., D.T. Goodhead and J.R. Savage (1988). Induction of sister chromatid exchanges (SCE) in G_0 lymphocytes by plutonium-238 α-particles. Int J Radiat Biol 53:909-915.

Brooks, A.L., G.J. Newton, L.J. Shyr, F.A. Seiler and B.R. Scott (1990). The combined effects of α-particles and X-rays on cell killing and micronuclei induction in lung epithelial cells. Int J Radiat Biol 58:799-811.

Bruce E.L. and E.H. Goodwin (1997). A new mechanism for DNA alterations induced by alpha particles such as those emitted by radon and radon progeny, Environmental Health Perspectives 105 (Suppl 5):1095-1101.

Cohen, B.L., D.R. Kulwicki, K.R. Warner, and C.L. Grassi (1984). Radon concentrations inside public and commercial buildings in the Pittsburgh area, Health Physics 47:399-405.

Cornforth, M.N. and E.H. Goodwin (1991). The dose-dependent fragmentation of chromatin in human fibroblasts by 3.5 MeV α -particles from 238Pu: experimental and theoretical considerations pertaining to single-track effects. Radiat Res 127:64-74.

Cooper, J.R., G.N. Stradling, H. Smith and G.E Ham (1982). The behavior of uranium-233 oxide and uranyl-233 nitrate in rats. Int. J. Radiat. Biol. 41: 421-433.

Cothern, J.C. and R.E. Smith Jr (1987). Environmental Radon. Plenum Press, New York, 378pp

Dang, H.S., V.R. Pullat, K.C. Pillai (1992). Determining the normal concentration of uranium in urine and application of the data to its biokinetics. Health Phys. 62: 562-566.

Darby, S.C., D. Hill and R. Doll (2001). Radon: a likely carcinogen at all exposures Ann. Oncol. 12:1341–51.

Day, C. (1999). Alpha radiation can damage DNA even when it misses the cell nucleus, Phys Today 52:19-20.

Debaje, S.B., T.V. Ramachandran and K.G. Vernekar (1996). Study of atmospheric radon-222 concentrations at Pune, Ind. J. Environ. Prot. 16: 755-760.

Emerit, I. (1994). Reactive oxygen species, chromosome mutation, and cancer: possible role of clastogenic factors in carcinogenesis. Free Radic Biol Med 16:99-109.

Evans, J.H. (1992). Alpha particle after effects. Nature 355:674-675.

Funtua, I.I., A. Onojah, S.A. Jonah, B.W. Jimba and I.M. Umar (1997). Radon emanation study of uranium ore samples from N. E. Nigeria. Appl. Radiat. Isot. 48(6):867-869

Gall, I. K., R.W. Ritzi, Jr., A.D. Baldwin, P.D. Pushkar, C.K. Carney and J.F. Talnigijr (1995). The correlation between bedrock uranium and dissolved radon in ground water of a fractured carbonate aquifer in southwestern Ohio, Ground Water 33: 197-206.

George, A.C. and A.J. Breslin (1967). Deposition of natural radon daughters in human subjects, Health Physics 13: 375 – 378.

Gundersen, L.C.S. (1992). The effect of rock type, grain size, sorting, permeability, and moisture on measurements of radon in soil gas, a comparison of two measurement techniques, Journal of Radioanalytical and Nuclear Chemistry, 161(2): 325-338.

Hair, T.L. and A.D. Baldwin, Jr. (1995). The determination of radon activities in ground water from Wisconsin tills in Southwestern Ohio and Southeastern Indiana Ohio, J. Sci. 95 (3): 245-253.

International Commission on Radiation Protection (ICRP) (1994). Human respiratory tract model for radiological protection ICRP Publication 66 Ann. ICRP 24: 1-3.

International Commission on Radiation Protection (ICRP) (1993). ICRP 1993 Protection against radon-222 at home and at work ICRP Publication 65 Ann. ICRP 23 2.

Jonah, S. A. Okunade, I. O. Ibeanu, I. G. E. and Jimba B. W. 2002. Natural radio-nuclides and elemental composition of chemical fertilizers used in Nigeria. Algerian Review Nucl. Sci. 4 (1): 49-52

Khursheed, A. (2000). Doses to systemic tissues from radon gas Radiat. Prot. Dosim. 88:171-81

Larson, E.S. and D. Gotfried (1961). Distribution of uranium in rocks and minerals of Mesozoic batholiths in Western United States, US Geological Survey Bulletin 1070-C: 63-103.

Lively, R.S. and L.F. Goldberg (1999) diffusion of radon through concrete block walls-a significant source or indoor radon, Radiation Protection Dosimetry 82:31-42.

Lubin, J.H. and J.D. Boice (1997). Lung cancer risk from residential radon: meta-analysis of eight epidemiological studies J. Natl Cancer Inst. 89: 49-57

Lubin, J.H., L. TomáBek, C. Edling, R.W. Hornung, G. Howe and E. Kunz (1997) Estimating lung cancer mortality from residential radon using data for low exposures of miners. Radiat Res 147:126-134.

Lubin, J.H., Z.Y. Wang, J.D. Boice Jr, Z.Y. Xu, W.J. Blot and L.D. Wang (2004). Risk of lung cancer and residential radon in China: pooled results of two studies. Int J 109:132-137.

Momčilović, B., G.I. Lykken and M. Cooley (2006). Natural distribution of environmental radon daughters in the different brain areas of an Alzheimer Disease victim. Molecular Neurodegeneration 1:11-15.

Nero, A.V. and W.W. Nazaroff (1984). Characterising the source of radon indoors, Radiation Protection Dosimetry 7:23-39.

Nielson, K.K., V.C. Rogers and G.W. Gee (1984). Diffusion of radon through soils: a pore distribution model, Soil Sci. Soc. Am. J. 48:482-487.

Nussbaum E and J.B. Hursh (1957) Radon solubility in rat tissues, Science 125: 552-553.

Nwankwo, L.I. and C.O. Akoshile (2005). Background radiation study of Offa industrial area of Kwara State, Nigeria. J. Appl. Sci. Environ. Mgt. 9 (3): 95-98

Ogunleye, P. O. Maiyaki, M. C. Mapu, I. Y. 2002. Radioactivity and heavy metal composition of Nigerian phosphate rocks: possible environmental implications. Journal of Environmental radioactivity 60: 39-48.

Okunade, I.O., A.K. Okunade, S.D. Barau, E.A. Edalere and D.J. Adeyemo (2007). Background radiation study of a local quarry in Zaria. Journal of Nigerian Environmental Society in Press.

Otton, J.K. (1989). Mapping the radon potential of rocks and soils, The Professional Geologist, 26 (5):8-9.

Porstendorfer, J. (1994). Properties and behaviour of radon and thoron and their decay products in the air, J.Aero. Sci. 25: 219-263.

Price, P.N., Gelman, A. and A.V. Nero (1996). Bayesian prediction of mean indoor radon concentrations for Minnesota counties, Health Physics 71:922–936.

Price, P. N. and A.V. Nero (1996). Joint analysis of long- and short-term radon monitoring data from the Northern U.S., Environment International 22: S699–S714.

Rogers, V.C. and K.K. Nielson (1991). Correlations for predicting air permeabilities and 222Rn diffusion coefficients of soils, Health Physics 61(2): 225-230.

Prise, K.M. (1994). Use of radiation as a probe for DNA lesion complexity. Int J Radiat Biol 65:43-48.

Puskin J.S., and J.D. Boice (1989). EPA's perspective on risks from residential radon exposure. J. Air Pollut Control Assoc 39:915-920.

Samet, J.M., D.R. Pathak M.V. Morgan, M.C. Marbury C.R. Key and A.A. Valdivia (1989). Radon progeny exposure and lung cancer risk in New Mexico U miners. Health Phys 56:415- 421.

Sanni, A.O. (1973). Seasonal variation of atmospheric radioactivity at Ibadan. Tellus 25:80-85.

Schramel, P., I. Wendler P. Roth and E. Werner (1997). A method for the determination of thorium and uranium in urine samples by ICP-MS. Mikrochim. Acta 126: 263-266.

Schroeder, G.L. (1965). Diffusion of radon in several naturally occurring soil types, J. Geophysical Research 70: 471-474.

Sogaard-Hansen, J. and A. Damkjaer (1987). Determining 222Rn diffusion lengths in soils and sediments, Health Physics 53(5): 455-459.

Steinhausler, F., W. Hoffmann, E. Pohl and J. Pohl-Ruling (1983). Radiation exposure of the respiratory tract and associated carcinogenic risk due to inhaled radon daughters. Health Phys 45:331-337.

Tanner, A.B. (1980). Radon Migration in the Ground: A supplementary review. In: Gessel, T.F. and W.M. Lowder (Eds). The Natural Radiation Environment III: : U.S. Dept. Energy Rept. CONF-780422(1): 5-56.

Wallner, G. (1997). Simultaneous determination of 210Pb and 212Pb progenies by liquid scintillation counting, Appl Radiat Isot. 48:511–514.

Wallner G. and K. Irlweck (997). Determination of lead-210 and its progenies in aerosol fractions of different particles sizes, Radiochim Acta 78:173–176.

Whittmore A.S., and A. McMillan (1983). Lung cancer mortality among US uranium miners: a reappraisal, J. Natl Cancer Inst 71:489-493.

Wilson W.E., and H.G. Paretzke (1980). Spatial dependence of frequency distributions in ionization and energy imparted in nanometer volumes by 0.25 to 3 MeV protons. Proc 7th Symp on Microdosimetry, pp 423–434.

Wolff, S. (1996). Aspects of the adaptive response to very low doses of radiation and other agents, Mutat Res 358:135-142.

Yu, K.N., E.C.M. Young, M.J. Stokes, D.L. Luo and C.X. Zhang (1992). Indoor radon and environmental gamma radiation in Hong Kong, Radiation Protection Dosimetry 40(4): 259-263.