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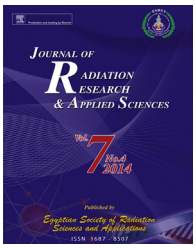

Radon levels and the expected population mortality in dwellings of Al-Kharj, Saudi Arabia

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ABSTRACT

Internal exposure from inhalation of radon and its progeny is one of the most significant sources of natural radiation exposure of the population. Radon levels and radon equilibrium factor were measured in the dwellings of Al-Kharj, Saudi Arabia using passive technique. Calibrated CR-39 diffusion type radon detectors were used for radon measurements and the method of can and bare is adapted for the measurement of radon equilibrium factor. Passive measurements enable the accumulation of the result over a long period and cover a wide area. The probability of cancer induction and then the expected mortality was calculated based on different approaches. The results show that the overall weighted mean of annual effective dose for Al-Kharj resident is equal to 1.51 ± 0.8 mSv and The average expected mortality for residents in dwellings of Al-Kharj city is ranged from 0.596 ± 0.25 to 0.369 ± 0.15 death per 10,000 persons of ages from 40 to 70 years respectively. Also, the lifetime excess absolute risk (LEAR) of the residents of the Al-Kharj city is equal to $(2.06 \pm 0.8) \times 10^{-4}$. The effect of dwelling types, ventilation and construction materials on the expected mortality is discussed.

1. Introduction

In 1988, the International Agency for Research on Cancer classified radon as a human lung carcinogen, based on a review of evidence from experimental data on animals and from epidemiological studies of underground miners (ICRP, 2010). Epidemiological studies confirm that radon in homes increases the risk of lung cancer in the general population.

Radon contributes more than half of all non-medical exposure to ionizing radiation dose received by general population (UNSCEAR, 1994). Due to its short half-life ($T_{1/2}$ = 3.82 d) it diffuses through soil and into the air. When inhaled, its short lived progeny, ^{218}Po and ^{214}Po, which are solid, tend to be deposited on the bronchial epithelium, thus exposing cells to irradiation. The alpha particles emitted by these short-lived decay products of radon can damage cellular

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DNA. Recent studies on indoor radon and lung cancer in Europe, North America and Asia provide strong evidence that radon causes a substantial number of lung cancers in the general population.

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 (WHO, 2009).

ICRP, 2012 currently considers the lifetime excess absolute risk (LEAR) of 5×10^{-4} per WLM to be the best estimate of lung cancer risk related to occupational exposure of 1 WLM. This value is updated from 2.8×10^{-4} per WLM in Publication 65 (ICRP, 1993) which close to half the value in Publication 115 (ICRP, 2010). ICRP recommendations on the risk of lung cancer at low annual exposure rates are in line with those from other international committees (UNSCEAR, 2009; WHO, 2009). ICRP has recently published a statement on radon, and the previous action limits have been divided by a factor of two (ICRP, 2010).

The aim of present study is to assess the expected mortality and the lifetime excess absolute risk (LEAR) due to indoor radon concentration in 100 dwellings in Al-Kharj city, Saudi Arabia. Radon level and equilibrium factor were measured by CR-39 detectors in schools, homes, hospitals and working places. The sampling districts of Al-Kharj city lies at 24°8′54″ North Latitude and 47°18′18″ East longitudes. The annual effective dose received by populations and their lifetime fatality risk or lifetime excess absolute risk estimates have been assessed in the light of guidelines given by International Commission on Radiological Protection (ICRP, 2010).

2. Experimental set up and theoretical approach

2.1. Radon and equilibrium factor measurement

The passive device used for measuring radon concentration is a diffusion cup (5.5 cm diameter and 8 cm high) fitted with CR-39 detector at its bottom while the open end was covered with filter paper of 170 μm thickness. Moreover, an external (bare) CR-39 detector was fixed outside the cup. For getting on good statistics, the diffusion cups with their external detectors were distributed in different positions inside each dwelling. The exposure time of the used detectors is extended to more than 100 days. At the end of exposure period, the detectors are collected and etched together under their optimum condition (6.25 N NaOH solution at 70 °C for 6 h) and counted manually under an optical microscope of 400X magnification power.

The radon concentration C_o inside each dwelling was calculated by the following equation (Nikezić, 1994).

$$C_o = \frac{D_o}{K_T\ t} \tag{1}$$

Where D_o is the obtained track density of filtered detector and t is the exposure time in days. K_T is the detector response of the used diffusion cup, it equals 0.20 ± 0.02 track cm^{-2} per Bqm^{-3} d which measured in the National Institute for Standard, Radiation Measurements Department, Egypt (Mansy, Sharaf, Eissa, El-Kamees, & Abo-Elmagd, 2006).

The obtained track density ration D/D_o between open (D) and filtered (D_o) detectors is used to determine the equilibrium factor F using the following equation (Abo-Elmagd & Sadek, 2014):

$$F = \left(0.21\frac{V}{A} + 0.49\right)\left(\frac{D}{D_o}\right)^{0.7} - 0.48 \tag{2}$$

Where V/A (in cm) is the used cup volume to its internal area A.

2.2. Annual effective dose

The dose in term of working level month (WLM) was defined as (Abo-Elmagd, Metwally, Elmongy, Salama, & El-Fiki, 2006):

$$\mathrm{WLM} = \mathrm{WL}\ \frac{t}{170} = \frac{F\ C_o}{3700} \times \frac{t}{170} \tag{3}$$

where WL is the number of working level and t is the exposure time (hour) which assumed that the exposure of 1 WL for 170 h produce 1 WLM (UNSCEAR, 2000).

Based on the dosemetric approach adapted by UNSCEAR, 1993, 2000, the effective dose rate can be calculated using the following equation:

$$\mathrm{Dose}\ (\mathrm{nSv/h}) = C_o(\varepsilon_r + \varepsilon_d F) \tag{4}$$

Where C_o is the radon concentration (Bqm^{-3}) and ε_r (0.17 nSv/h per Bqm^{-3}), ε_d (9 nSv/h per Bqm^{-3}) are the dose conversion factors for radon and its short-lived progeny respectively. F is the equilibrium factor between radon and its short-lived progeny. This equation is equivalent to use a dose conversion factor of 6 mSv WLM^{-1}.

As an alternative to a dosimetric approach, ICRP has derived a conversion convention for radon exposures based on the equality of detriments from epidemiological determinations. The rounded values of the conversion convention adopted by ICRP are 5 and 4 mSv WLM^{-1} for worker and for members of the public, respectively (ICRP, 1993, 1994), which is different from the value of 9 nSv/h per Bqm^{-3}derived using the dosimetric approach. This is not a big discrepancy, considering the complex physical and biological issues involved. The established value of 9 nSv/h per Bqm^{-3}, used in past UNSCEAR, 1993 calculations is still considered appropriate for average effective dose calculations (UNSCEAR, 2000).

2.3. The expected mortality

The probability of cancer induction for a person between the ages of 40 y and 85 y can be calculated by Harley (1981),

$$A(t, t_0) = 14 \times 10^{-6}\ N(t_0)P(t, t_0)\ \ \exp\left(-T\ \ln\frac{2}{20}\right) \tag{5}$$

Where $A(t,t_0)$ is the probability of getting cancer at age t, due to exposure to N WLM occurring at age t_0. $P(t,t_0)$ is the probability that a person alive at age t ($P(t,t_0)$ is taken to be 1). T is the time interval since the exposure ($T = t - t_0$).

The expected mortality of lung cancer among a population of 10 000 persons with an average age of Y (in years) is equal to