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A STUDY ON THE RADON CONCENTRATIONS IN TOBACCO IN JEDDAH (SAUDI ARABIA) AND THE ASSOCIATED HEALTH EFFECTS

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SUMMARY: Radon and cigarette smoking have synergistic, multiplicative effect on lung cancer rates. Smokers and nonsmoking residents of smoking households are at increased risk for lung cancer even when radon levels are relatively low. People who inhale tobacco smoke are exposed to higher concentrations of radioactivity. Ever since studies on the relation of smoking to cancer—particularly the lung cancer—has been established, there had been a great interest in studies concerned with the monitoring of the alpha radioactivity in tobacco. Radium-226 (^{226}Ra) is a significant source of radon-222 (^{222}Rn), which enters buildings through soil, construction materials, or water supply. When cigarette smoke is present, the radon daughters attach to smoke particles. Thus, the alpha radiation dose to a smoker's lungs from the natural radon daughters is increased because of smoking. To investigate whether the cigarette tobacco itself is a potential source of indoor radon, the levels of radon and thoron from radioactive decay were measured in tobacco samples of 15 different brands using CR-39 solid state nuclear track detectors (SSNTDs). The results showed that the ^{222}Rn and ^{220}Rn concentrations in cigarette tobacco samples ranged from 97 to 204 Bqm^{-3} and 38 to 104 Bqm^{-3}, respectively. The radon concentrations emerged from all investigated samples were significantly higher than the background level. The annual equivalent doses from use of these tobaccos were determined. The measurement of the average indoor radon concentrations in 30 café rooms was, significantly, higher than 30 smoke-free residential houses. The result refers to the dual (chemical and radioactive) effect of smoking as a risk factor for lung cancer.

Key words: ^{220}Rn, ^{222}Rn, tobacco, effective dose, lung cancer, CR-39, nuclear track detectors.

INTRODUCTION

The cigarette is increasingly becoming a crutch for many in this pressure-laden world, and they opt for this easy way out despite the hard facts of it being hazardous. It is not only them but also the people near them who sometimes pay dearly for this habit. Studies after studies have confirmed that this is a dangerous habit. Tobacco smoke has toxic, genotoxic, and carcinogenic properties and has been linked to fatal pregnancy outcomes (1). The consumption of tobacco products and the number of smokers have been increasing steadily throughout the world, and Saudi Arabia is no exception to this. Tobacco in Saudi Arabia continued to display high growth in 2010, rising significantly in both volume and value terms (2). Diabetes mellitus is a major public health challenge in both developed and developing countries. In Saudi Arabia, almost one Saudi in four beyond the age of 30 has diabetes mellitus. Some estimate that it will be 40%–50%

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in 2020 (3,4). The results of different authors indicate that smoking is associated with a substantial increase in the incidence of type 2 diabetes mellitus (5,6). Tobacco smoking will also help more in deteriorating the diabetes situation in this country.

Tobacco smoke contains more than 4000 different chemicals, most of which are generated during the combustion process. More than 40 compounds are carcinogenic, which include some radionuclides such as polonium (^{210}Po) and lead (^{210}Pb) (7). Radioactivity in cigarette smoke was measured by several authors, and it was suggested that ionizing radiation from cigarette smoke could originate a meaningful exposure of lung tissues. Smokers are 10 times at greater risk of developing lung cancer than that of nonsmokers (8-10).

Radon and its progeny are the greatest sources of natural radioactivity. It has been estimated that inhalation of short-lived radon progeny accounts for more than half of the effective dose from natural sources (11-15). Numerous cohort, case-controlled, and experimental studies have established the carcinogenic potential of radon (16-18). Prolonged exposure to radon may cause a negative effect on human health, causing lung cancer and bronchial tissue damage. Indoor radon and its decay products usually come from soil, building materials, and water supply. Because the decay products carry high electric charges, they readily attach themselves to indoor dust particles (19,20). Subsequent inhalation of radon and its short-lived decay products is considered an etiological factor for lung cancer (19,21,22).

Lung cancer is the leading cause of cancer-related deaths worldwide. Lung cancer kills thousands of Americans every year. Smoking, radon, and secondhand smoke are the leading causes of lung cancer. Smoking is the leading cause of lung cancer. Smoking causes an estimated 160,000 cancer deaths in the United States every year. And the rate among women is rising. A smoker who is also exposed to radon has a much higher risk of lung cancer (23).

Radon (^{222}Rn) in air is ubiquitous. Radon is a form of ionizing radiation and proven carcinogen (20). Lung cancer is the only known effect on human exposure to radon in air (24). Lubin *et al.* (25) reported that, in the United States, exposure to radon progeny may account for 10% of all lung cancer deaths and 30% of lung cancer deaths in nonsmokers, while an estimate from the National Academy of Sciences BEIR VI committee suggests 21,800 lung cancer cases annually resulting from radon exposure with uncertain bounds from 3,000 to 33,000, making this the second leading cause of lung cancer in the United States (24,25). Radon can damage the respiratory epithelium (the cells that line the lung) through the alpha-particle emissions. The damage to epithelial cells of the lung occurs when radiation interacts either directly with DNA in the cell nucleus or indirectly through the effect of free radicals (16,17).

Radon is the number one cause of lung cancer among nonsmokers, according to EPA estimates (26). Overall, radon is the second leading cause of lung cancer. Radon is responsible for about 21,000 lung cancer deaths every year. About 3,000 of these deaths occur among people who never smoked. Exposed to 1.3 pCi/L (the average indoor radon level) never-smokers have a 2 in 1,000 chance of dying from lung cancer, while smokers exposed to same level have a 20 in 1,000 chance. The World Health Organization (WHO) says radon causes up to 15% of lung cancers worldwide (27).

Secondhand smoke (referred to as environmental tobacco smoke) is the third leading cause of lung cancer and responsible for an estimated 3,000 lung cancer deaths every year. Smoking affects nonsmokers by exposing them to secondhand smoke. The lung cancer risk from secondhand smoke exposure is 20%–30% higher for those living with a smoker (17). The epidemiological and biochemical evidence on exposure to environmental tobacco smoke (ETS) provides compelling confirmation that breathing other people's tobacco smoke is a cause of lung cancer. When evidence from various studies is combined, they indicate that exposure to ETS increases the number of lung cancers detected in nonsmokers. Nonsmoking coworkers of smokers have a relative risk of approximately 1.39 (16,17).

The U.S. Environmental Protection Agency (EPA) states that exposure to tobacco smoke, especially directly from smoking, but also from secondhand smoke, when coupled with exposure to radon gas, can significantly increase the risk of lung cancer, when compared to either smoking or radon exposure alone. In fact, most radon-related lung cancer cases occur in individuals who also smoke, demonstrating a synergistic effect between tobacco smoke and radon. The synergistic effects of radon gas and smoking have been well documented through years of research and scientific studies (26).

Indoor cigarette smoking enhances the air concentration of submicron particles, which trap radon decay products. It has been reported that radon decay products

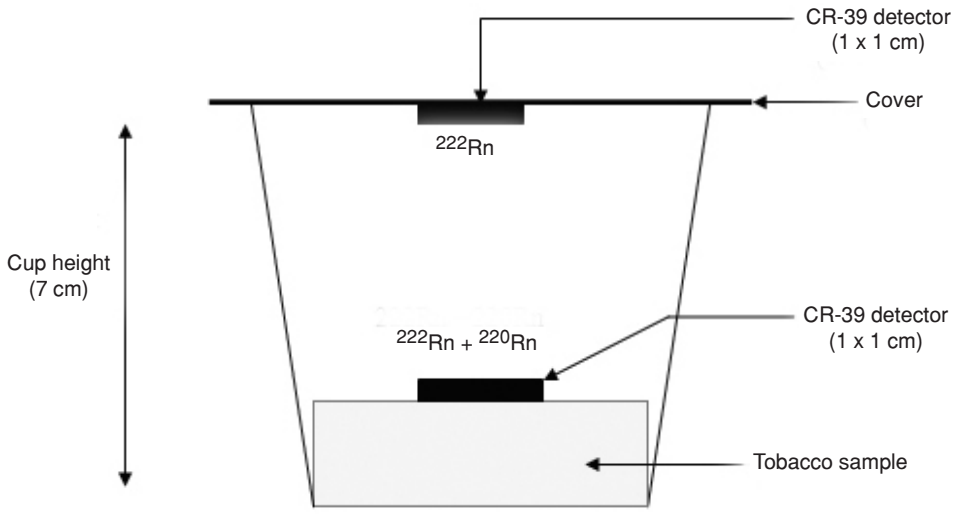

Figure 1: A schematic diagram of the sealed-cup technique.

that pass from room air through burning cigarettes into mainstream smoke are present in large, insoluble smoke particles that selectively deposited at bronchial bifurcation of the inhabitant (19-21) where the attached radon progeny undergo substantial radioactive decay before clearance. Consequently, in addition to the traditional implication of smoking cigarette in lung cancer, the high incidence of lung cancer in cigarette smokers and non-smokers may be attributed to the cumulative effect of alpha radiation dose from indoor radon and thoron progenies generated and/or trapped by tobacco and its smoke (20,22).

It has been known for over 20 years that all types of tobacco contain radioactive ^{210}Po ($t_{1/2}$ = 138.38 d)—which emits alpha particles—and radioactive ^{210}Pb ($t_{1/2}$ = 22.3 y)—which emits beta particles and is a precursor of ^{210}Po. There is a degree of consensus about how tobacco becomes radioactive (9). Most soils contain radioactive elements such as radium, which decays into ^{210}Pb and ^{210}Po. In addition, phosphate ore used as fertilizer in tobacco fields may contain such isotopes in relatively high concentrations. Thus it was anticipated that tobacco plants can absorb ^{210}Pb and ^{210}Po through their roots (9,28). During tobacco processing, the radiation is not completely removed.

The work presented here aims at shedding more light on the radiological health hazards due to cigarette tobacco smoking in Saudi Arabia. The concentrations of radon, thoron, and their daughters in cigarette tobacco have been determined using CR-39 solid state nuclear track detectors (SSNTDs). The annual effective dose equivalent from use of these tobaccos is computed. The exhalation rates of both ^{222}Rn and ^{220}Rn in cigarette tobacco samples have been determined using the integrated radon exposure. In addition, the difference between indoor radon in smoke-free and smoke-rich environments are also investigated.

MATERIALS AND METHODS

The use of SSNTDs is a convenient technique for low activity measurements since it is of low cost, is a simple operation, has high registration sensitivity, and has the possibility of use for long period exposures without any fading. CR-39 is very useful in the detection of alpha particles from disintegration of radon and radon daughters (12,20,22,29). The concentration and exhalation rate of radon can be measured using CR-39 detectors because of their capability to register tracks at different levels of registration sensitivity. The CR-39 detectors used in this work were supplied by Pershore Mouldings, UK, in the form of large sheets that were cut into 1 x 1 cm^2. Cigarettes of 15 different brands were purchased from local market. Measurements were made in 15 different brands' tobacco cigarette samples coded T1–T15. A fixed amount of tobacco sample was placed in plastic containers. The container was 7 cm in height and 5.2 cm in diameter. A piece of CR-39 detector with area 1 x 1 cm^2 was embedded in the sample in each container. At the same time a second piece of CR-39 detector was held at the top of the container (Figure 1). Measurements were carried out four times for each tobacco sample.

The cups were left at room temperature for two months exposure time. During this time alpha particles from the decay of radon, thoron, and their daughters bombarded the CR-39 nuclear track detectors in the air volume of the cup. After exposure the detectors were etched chemically in 6N NaOH solution at 70°C for 6 h to reveal the tracks. The tracks were counted using an optical microscope.

This experimental setup ensures that the lower detector recorded alpha particles from radon, thoron, and their daughter products present in tobacco samples. The upper detector, however, recorded only the ^{222}Rn component. Consequently the difference in the track densities of the two detectors represented the content of ^{220}Rn and their daughters in the samples. The density of tracks counted was assumed to be proportional to the $^{220, 222}Rn$ exposure (20,22,30). The track density (ρ) recorded on the detector, attenuation factor of ^{222}Rn (k), calibration coefficient of measuring system in terms of cm^2 d-1 Bqm^{-3} (η), and the exposure time (t) were applied to determine the ^{222}Rn concentration (C) from the relation (20,22,31):

$$C = \frac{\rho}{k\eta t} \quad \text{..... (1)}$$

The potential alpha energy concentration (PAEC) of ^{222}Rn and ^{220}Rn daughters in terms of working level (WL) units is calculated using the following formula (20,22,30,32):

$$F = \frac{WL \times 3700}{CRn} \quad \text{..... (2)}$$

where, F is the equilibrium factor of 0.4 and CRn is the radon concentration measured (Bqm^{-3}).

The annual effective dose equivalent, D (in units of $mSv.y^{-1}$) is computed from the integrated ^{222}Rn concentration using the following formula (20,22,29,31,33):

$$D = \frac{0.4\ R\ (3.88)\ 7000}{(3700)\ 170} \quad \text{..... (3)}$$

where, R is the integrated ^{222}Rn concentration in Bqm^{-3}, 0.4 is the equilibrium factor, 3.88 $mSv.WLM^{-1}$ is the ICRP conversion factor. The other factors are to take account of the house occupancy factor (33).

The exhalation rates of radon and thoron are calculated using the expression available in the literature (8,10,13,15,34).

Radon and thoron were measured in 30 residential houses and 30 café rooms using CR-39 plastic track detectors. The selected houses were occupied with nonsmoking inhabitants and the detectors were placed in totally smoke-free areas. We distributed closed diffusion chambers [(closed with sponge as a filter with 0.5 cm thick compressed): the filter slows down the diffusion of noble gases into the chamber and discriminates in favor of radon vs thoron] together with open (without filter) can technique containing CR-39 detectors inside each house or café room. The structure of these dosimeters had been described in previous works (31,32,35-37). The detectors were exposed for two months and, after retrieval, were etched and scanned as described above.

RESULTS AND DISCUSSION

Tobacco smoking is a risk factor for six out of eight main death causes all over the world; with lung cancer being one of the six causes, tobacco represents the most important one (38). Each year 1.35 million new cases are diagnosed, which represents more than 12% of all the new cancer cases (38,39). Furthermore, smoking is responsible for 1.18 million deaths from cancer (17.6% of the world total), of which 21,400 are lung cancers from secondhand smoking (38). Radon is a class A carcinogen and the second leading cause of lung cancer. Radon comes from the radioactive breakdown of naturally occurring radium found in most soils. As a gas in the soil, it enters buildings through small openings in the foundation. Since the building can hold the radon similarly to smoke trapped under a glass, indoor radon concentrations can increase to many times that of outdoor levels. Thousands of preventable lung cancer deaths annually in the world are attributable to indoor residential exposure to radon. Either smoking or radon exposure can independently increase the risk of lung cancer. However, exposure to both greatly enhances that risk. Darby *et al.* (40) provided compelling evidence that indoor ^{222}Rn is an important contributor to the risk of lung cancer. However, the derived estimates of ^{222}Rn-attributable lung cancers may have a low bias. The authors estimated an increase in the lung cancer risk of 16% for each incremental 100 Bqm^{-3} of ^{222}Rn from a pooling of the European residential case-control studies. They estimated that ^{222}Rn may contribute to 9% of all lung cancers in those countries on the basis of an estimated average ^{222}Rn concentration of 59 Bqm^{-3} for 29 European countries. Although a huge amount of data is available about the biological effect of tobacco smoking (38), here we investigate the possible involvement of ^{222}Rn derived from tobacco as a risk factor of lung cancer. This study has investigated the ^{222}Rn and ^{220}Rn content of tobacco samples of 15 different brands (coded T1–T15) used in cigarette manufacture. The data obtained revealed that sample T4 recorded the highest level of ^{222}Rn, whereas

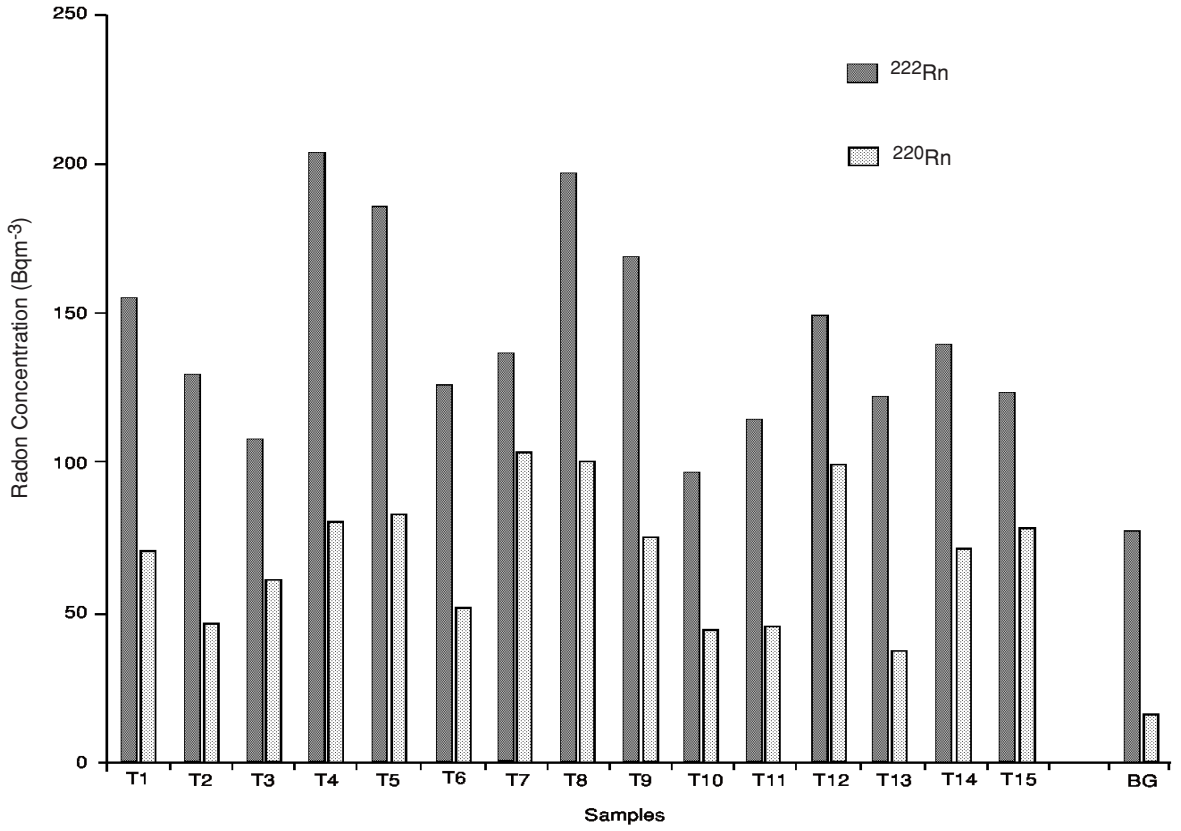

Figure 2: Radon and thoron concentrations in 15 tobacco samples. BG is the background value.

T7 contained the highest level of ^{220}Rn. Compared to the background levels (77 ± 3 Bqm^{-3} and 16 Bqm^{-3}) all samples had significantly higher ^{222}Rn and ^{220}Rn values (Figure 2).

In descending order, the ^{222}Rn concentrations among the investigated samples were those of T4, T8, T5, T9, T1, T12, T14, T7, T2, T6, T15, T13, T11, T3, and T10. In terms of 220Rn concentrations the order was T7, T8, T12, T5, T4, T15, T9, T14, T1, T3, T6, T2, T11, T10, and T13.

The PAECs of ^{222}Rn and ^{220}Rn were calculated. The alpha activities due to the ^{222}Rn were observed to be higher than those due to the ^{220}Rn series for different investigated tobacco samples.

It is anticipated from the results that smokers consuming tobaccos T1, T2, T4, T5, T7, T8, T9, T12, and T14 samples are exposed to higher alpha doses. Previous studies (41,42) have indicated that in a smoker's lungs the ciliary action to clear the lungs is reduced to about half the normal. The average length of time during which the insoluble forms of ^{210}Pb and ^{210}Po remain at the bronchial bifurcations is 3-5 months. Coincidentally, the surface tissue of smoker's bronchi at the bifurcations is replaced by damaged abnormal tissue.

The exhalation rates of both ^{222}Rn and ^{220}Rn in different tobacco samples have been determined. The values of radon and thoron exhalation rates vary from 6.79 mBq kg^{-1} h^{-1} to 14.02 mBq kg^{-1} h^{-1} and from 2.62 mBq kg^{-1} h^{-1} to 7.82 mBq kg^{-1} h^{-1}, respectively.

The concentrations of ^{222}Rn and ^{220}Rn progenies measured are shown in Figure 3. The values of ^{222}Rn progeny concentration were lower in T10 (39 Bqm^{-3}) and higher in T4 (82 Bqm^{-3}). Also, the values of 220Rn progeny were lower in the T13 (15 Bqm^{-3}) and higher in T7 (42 Bqm^{-3}) (14,20,22).

Indoor air quality is a contributing factor of lung cancer, although the attributable lung cancer risk from ^{222}Rn in homes may be low. Due to the presence of dust, the ^{222}Rn and ^{220}Rn daughters (from building materials, soil, or underground water supply) mainly attach to room surfaces, but indoor smoking allows ^{222}Rn daughters to

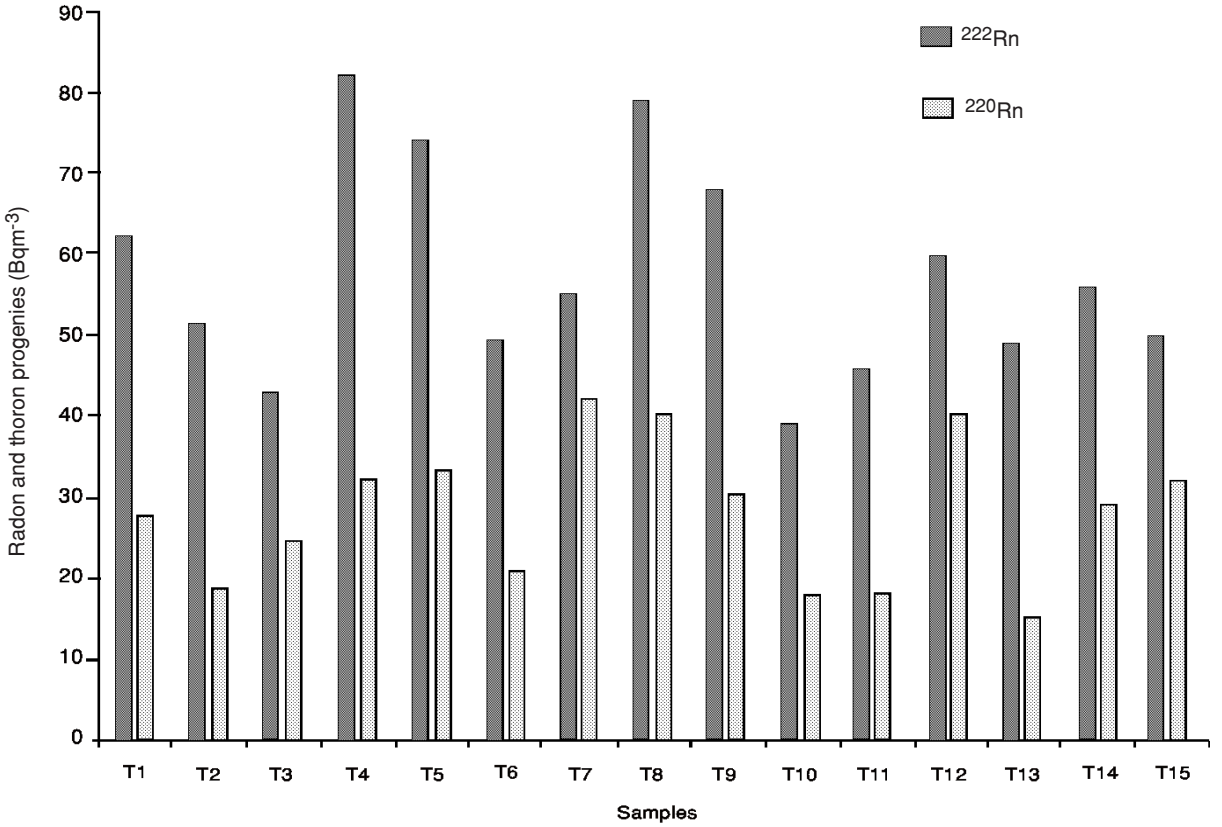

Figure 3: Distribution of the concentrations of radon and thoron progenies in 15 tobacco samples.

attach to smoke particles. Thus, the alpha radiation to a smoker's lungs from the natural ^{222}Rn daughters is increased because of smoking. When cigarette smoke is present (after lighting five cigarettes), the radioactivity attached to airborne smoke particles and the radioactivity concentration in the room increase up to 30 times compared to a smoke-free room. This is because the tobacco smoke works as a kind of 'magnet' for airborne radioactive particles. It remains suspended and available until inhaled as 'secondhand' smoke by anyone in the room. Thus, smoking indoors greatly increases the lung cancer risk to all inhabitants (42).

The resulting estimates of dose due to the presence of ^{222}Rn, ^{220}Rn, and their daughters are presented in Figure 4. The values of radon and thoron were, respectively, 1.67–3.52 ($mSv.y^{-1}$) with high values in T4 samples and low in T10, and 0.66–1.80 ($mSv.y^{-1}$) with high values in T7 samples and low in T13. These values are comparable with those reported by Ghany (20,22). However, the fact that these higher doses of radiation are delivered to vulnerable tissues at the location where malignancy is most frequently observed argues strongly for alpha radiation playing the most important role in causing lung cancer. These values correspond to 2/3 of a pack of cigarette, which means these values will increase by 25% when a complete pack is used (19). According to Martell (43), the cumulative dose of alpha radiation in bronchial bifurcations of smokers that die of lung cancer is approximately 16 Sv (80 rad). This dose is sufficient to induce a malignant transformation caused by alpha-particle interactions with basal cells. The risk for lung cancer among cigarette smokers increases with the duration of smoking and the number of cigarettes smoked per day. This observation has been made repeatedly in cohort and case-control studies (44). Prominent experts have shown that the chemical patterns and smoke components vary tremendously on a cigarette puff to puff basis (45).

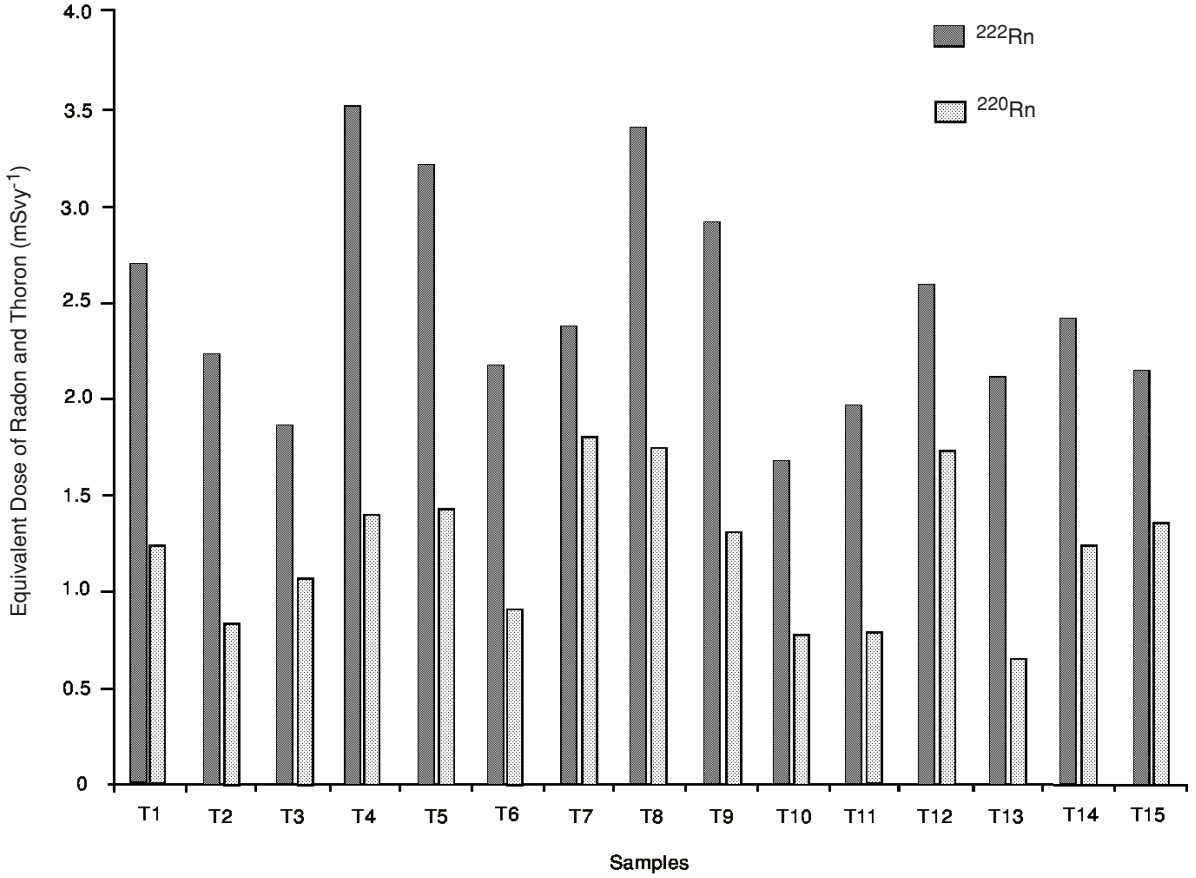

Figure 4: Resulting dose due to radon and thoron in 15 tobacco samples.

The measured values of both of radon and thoron in residential houses and café rooms (n = 30 each) are shown in Table 1. Radon and thoron concentrations, in most cases, were found to be higher in café rooms than residential houses. It is probable that the smoke-rich air of the café room enhances the presence of such elements compared to the relatively smoke-free environment. Smokers exposed to the higher indoor radon and thoron levels should experience the highest risk and the earliest incidence of lung cancer. This possibility was investigated cytogenetically by different researchers (46,47), who showed that chromosome aberrations in cultured peripheral blood lymphocytes are a sensitive measure of cumulative exposure to radon progeny. If most smokers who develop bronchial cancer are those with the highest cumulative radon progeny exposure, they should exhibit the highest prevalence of the indicator aberrations. Cigarette smokers exposed occupationally to inhalation of fibrous aerosols or toxic chemical agents that damage the bronchial epithelium and impair clearance may experience bronchial cancer at lower cumulative radon progeny exposures.

Lung cancer is a serious chronic health effect of cigarette smoking, and indoor radon progeny may be a factor in the etiology of some of the other cancers, in particular, of the larynx, pharynx, and esophagus (22).

When people stop smoking, the risk of lung cancer starts decreasing. Ten years after quitting, the risk of lung cancer is about one-third to one-half of that of a smoker (48). People who quit, even at middle age, avoid much of the future risk associated with smoking. The earlier someone quits, the greater the long-term benefit (49). Quitting is more effective than other measures to avoid the development of lung cancer and other smoking-

Table 1: Radon and thoron concentrations in residential houses and café rooms.

Location no.	Residential houses		Café rooms	
	^{222}Rn (Bqm^{-3})	^{220}Rn (Bqm^{-3})	^{222}Rn (Bqm^{-3})	^{220}Rn (Bqm^{-3})
1	38 ± 3.12	13 ± 0.81	82 ± 6.01	29 ± 1.54
2	36 ± 3.01	10 ± 0.68	76 ± 5.42	25 ± 1.41
3	39 ± 3.35	12 ± 0.75	94 ± 6.12	26 ± 1.52
4	30 ± 2.68	10 ± 0.87	93 ± 6.19	24 ± 1.47
5	41 ± 2.56	11 ± 0.87	80 ± 5.98	23 ± 1.46
6	40 ± 3.25	10 ± 0.62	79 ± 5.39	18 ± 1.33
7	34 ± 2.60	12 ± 0.71	64 ± 5.11	24 ± 1.50
8	29 ± 2.56	10 ± 0.51	69 ± 5.09	15 ± 1.18
9	36 ± 2.51	8 ± 0.64	85 ± 6.08	18 ± 1.09
10	42 ± 3.62	9 ± 0.71	79 ± 5.93	19 ± 1.09
11	43 ± 3.44	8 ± 0.59	70 ± 6.52	28 ± 1.81
12	28 ± 2.81	11 ± 0.63	72 ± 6.46	16 ± 1.16
13	34 ± 2.75	7 ± 0.62	67 ± 5.43	14 ± 1.10
14	33 ± 3.31	9 ± 0.73	90 ± 6.16	30 ± 1.87
15	43 ± 3.94	14 ± 0.92	57 ± 5.02	24 ± 1.20
16	38 ± 2.81	9 ± 0.66	79 ± 5.22	16 ± 1.28
17	41 ± 2.75	7 ± 0.60	84 ± 5.82	26 ± 1.39
18	34 ± 3.00	10 ± 0.75	86 ± 5.96	19 ± 1.32
19	44 ± 3.18	11 ± 0.53	111 ± 6.24	17 ± 1.26
20	45 ± 3.18	9 ± 0.56	99 ± 6.22	16 ± 1.24
21	48 ± 4.01	14 ± 0.93	55 ± 4.99	35 ± 1.56
22	54 ± 4.56	16 ± 0.95	90 ± 5.89	30 ± 1.81
23	28 ± 2.05	12 ± 0.75	78 ± 4.65	26 ± 1.68
24	44 ± 2.93	10 ± 0.61	94 ± 5.77	19 ± 1.93
25	47 ± 3.42	14 ± 0.81	89 ± 5.13	18 ± 1.91
26	48 ± 4.56	11 ± 0.56	68 ± 4.43	16 ± 1.21
27	34 ± 3.17	6 ± 0.56	87 ± 5.27	14 ± 1.17
28	29 ± 2.11	10 ± 0.76	73 ± 5.89	21 ± 1.38
29	42 ± 3.27	11 ± 0.80	83 ± 6.04	25 ± 1.56
30	35 ± 3.31	13 ± 0.81	98 ± 6.13	27 ± 1.62
Average ± SD	38.56 ± 3.13*	10.56 ± 0.71**	81.03 ± 5.68	21.93 ± 1.44

*222Rn and **220Rn were significantly and highly significantly lower (respectively) in residential houses compared with café rooms (P <0.0001 and <0.0001, respectively) (estimated by unpaired t test).

related diseases (50). Given that lung cancer continues to be the leading cause of cancer-related deaths globally, efforts for radon mitigation should go hand-in-hand with implementation of smoking cessation strategies.

A diet rich in vegetables has been associated with a reduced risk of lung cancer, but the protective effect may be limited to smokers (51,52); however, evidence remains mixed (53).

Higher fruit consumption has also been associated with a small reduction in risk (54,55). A study published in December, 2011, estimated that, in 2010, around 9% of lung cancers in the UK were linked to people eating less than approximately two to three portions a day (one portion=80 g) of fruit (56). However, that the link between diet and lung cancer is less certain than that of, for example, smoking (56).

CONCLUSION

Tobacco smoking is fatal in many ways and has severe health, economic, and social consequences. Although natural radioactivity in tobacco could be one of the main reasons for the health impacts of tobacco smoking, there are very limited publications on natural radionuclides concentration in tobacco and tobacco products except for ^{210}Po in cigarettes. The aim of this study was to shed more light on the concentrations of radon, thoron,

and their progenies in cigarette tobacco that is consumed in Saudi Arabia. The results of this study indicate the existence of a wide range of variations in ^{222}Rn and ^{220}Rn contents in tobacco consumed in Saudi Arabia. Based on the results obtained from this study the concentrations of ^{222}Rn and ^{220}Rn in 15 tobacco samples showed that the highest concentrations were observed in T4 and T7 samples. Annual equivalent doses due to radon, thoron, and its short-lived daughters from the inhalation of various cigarette smokes have been evaluated.

We wish to emphasize on the urgent needs for more research on the activity concentration of natural radionuclides in tobacco and tobacco products, their behavior during smoking, and on their concentration in smoke and smoker's intake. A public health priority should be essential to develop countermeasures for the banning of all forms of smoking (cigarettes, pipes, cigars, and narghiles) whenever and wherever possible in public area. In parallel, ways must be identified for an efficient policy of harm reduction related to the internal intake of different radionuclides and chemical compounds. Radon mitigation should accompany smoking cessation measures in lung cancer prevention efforts. People should be encouraged to include lots of fresh vegetables and fruits in their diets.

Finally, since people fear everything that is radioactive, the proper authority should take immediate steps for the placement of a clear indication about the radioactivity content on cigarette packets.

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