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Estimates of Cancer Mortality Due to the Ingestion of Mineral Spring Waters from a Highly Natural Radioactive Region of Brazil

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ABSTRACT

This study was performed in order to evaluate the lifetime risk of radiation-induced cancer due to the ingestion of ^{226}Ra, ^{228}Ra and ^{222}Rn in mineral spring waters from a highly natural radioactive region of Brazil. Water samples were randomly collected at 10 spring sites located in the Águas da Prata region. Concentrations ranging from $< 2{\cdot}2$ to $1{\cdot}80 \times 10^3$ mBq l^{-1} for ^{226}Ra, $< 3{\cdot}7$ to $2{\cdot}3 \times 10^1$ mBq l^{-1} for ^{228}Ra and 8×10^{-1} to $2{\cdot}16 \times 10^2$ Bq l^{-1} for ^{222}Rn were observed.

A total of eight radium-induced cancers (four head carcinomas plus four bone sarcomas) per 10^6 exposed persons were predicted. For radon, a total of 23 cases of fatal stomach cancers per 10^6 exposed persons were estimated. These predictions suggest that chronic ingestion of radium and radon at the levels observed at these springs will result in incremental increases of fatal cancers of 2 and 0.5%, respectively, above the background incidence rate. The uncertainties in evaluating the carcinogenic effects due to radium and radon ingestion were analyzed. These investigations suggest that the true risk due to radium ingestion is likely to be within an order of magnitude in either direction of the risk estimated in the present paper. Copyright © 1996 Elsevier Science Limited

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INTRODUCTION

Radium is a naturally occurring radioactive element that arises from the decay of primordial uranium and thorium in the Earth's crust. The occurrence of radium in drinking water supplies is governed by the physical and chemical behaviour of uranium, thorium and radium in aquifers and surface deposits (Hess *et al.*, 1985). Radium-226 and ^{228}Ra are the isotopes that, due to their mobility, are of primary environmental concern. Radon is an inert noble gas formed by radioactive decay of ^{226}Ra. Small quantities of radon can be found in all groundwater from natural sources as a result of decay of radium in the water, rock and soil matrix surrounding the water. The concentration of radon in groundwater may far exceed that of radium in the water because gaseous radon can migrate through the soil matrix into underground aquifers. Measurements of radon in water from selected water sources, including thermal springs, were recorded as early as 1905 (Boltwood, 1905). These measurements showed a large variation in radon concentration between different thermal springs, depending upon the characteristics of the soil, the geochemistry of the parent nuclide and the pH of the water at the source.

Epidemiological methods have been used to infer the cancer risk associated with a high exposure of radium and radon (NAS-BEIR IV, 1988). However, few epidemiological studies have investigated the consequences of the exposure to environmental levels of radon and radium (NAS-BEIR V, 1990), and the findings cannot yet be used to characterize the risks due to low radiation exposures. Consequently, the cancer hazard posed by environmental exposures to radium and radon has been addressed through risk estimation procedures. The quantitative risks have been estimated using risk projection models incorporating exposure–response relationships derived from the epidemiological investigations of underground miners and radium dial painters (NAS-BEIR IV, 1988).

The present study was carried out in order to estimate the lifetime risk of radiation-induced cancer due to the ingestion of mineral spring waters from Águas da Prata, which is one of the highest natural radioactive regions of Brazil. Although there are several studies about the determination of the levels of radioactivity in this region (Cullen, 1977; Eisenbud, 1987; Oliveira, 1993), there have been no estimates up until now of the biological detriment due to the ingestion of these mineral spring waters.

The quantification of the carcinogenic effects of ingestion of ^{226}Ra and ^{228}Ra in drinking water was done based on the methodology proposed by Mays *et al.* (1985), who fitted dose–response models to epidemiological data concerning the incidence of bone and head cancers as a function of

estimated intake of ^{226}Ra and ^{228}Ra among radium dial painters. These exposures were extrapolated to low (environmental) levels. For the estimation of risk due to the ingestion of ^{222}Rn in drinking water the biokinetic model derived from Crawford-Brown (1990), was used and according to this methodology fatal stomach cancer is the primary effect of concern.

A similar study, examining the concentrations of U and Th isotopes is in progress but not yet complete therefore, these elements will not be discussed here.

Area of study

The Águas da Prata region is located on the Poços de Caldas Plateau. This Plateau is a deeply weathered alkaline igneous intrusion of the Cretaceous period (Adams, 1975), located about 270 km north of São Paulo city. At present, the remnant of the intrusion takes the form of a circular caldera, about 35 km in diameter, covering an area of approximately 800 km^2. In this plateau many health resorts are found, based on sources of thermal and mineral waters. The Águas da Prata spring waters are among the most visited of them by tourists and patients from Brazil.

In this Plateau can be found different radioactive anomalies. The region is abundant in radionuclides of the uranium and thorium series, with uranium associated with zirconium and molybdenum, and thorium with iron and manganese oxides.

MATERIAL AND METHODS

For this investigation, water samples were collected quarterly at 10 spring sites over a period of 1 year and were analyzed for the determination of ^{228}Ra, ^{226}Ra and ^{222}Rn concentration. For ^{226}Ra and ^{228}Ra determination, 5 litre samples of water were collected at each spring site. The pH was adjusted by addition of nitric acid to prevent losses by sorption in the vessels. The ^{226}Ra and ^{228}Ra were determined by co-precipitation with barium sulfate at pH 4·5–5·0 in the presence of ethylenediamine tetraacetic acid, after separation from its decay products by complexion with nitrile triacetic acid (titriplex I) at pH 12·5–13·0 (Godoy, 1990).

The ^{226}Ra was determined by gross alpha counting of $Ba(Ra)SO_4$ precipitate, after decay of ^{224}Ra and of ^{223}Ra; that is, after 25 days. The determination of ^{228}Ra was done by measuring the gross beta activity of the same precipitate. This measurement was carried out by gross beta counting of its decay product ^{228}Ac, because it emits beta rays of higher

energy (1·2 MeV) in contrast to the lower energy of ^{228}Ra beta particles (40 keV). Both measurements were carried out in a low background gas flow proportional counter. The overall chemical yield achieved was around 90%.

The ^{222}Rn concentration was determined by the liquid scintillation method (Sampa, 1979). For this analysis, the samples were collected directly in the counting vessels, in which the scintillation solution Aquasol was previously added.

Typical lower limits of detection (LLD) for these methods were 2·2 mBq l^{-1} for ^{226}Ra, 3·7 mBq l^{-1} for ^{228}Ra and $1{\cdot}9 \times 10^{-1}$ Bq l^{-1} for ^{222}Rn, at a 95% confidence level. The overall uncertainty in the radium concentration determination varied from 6 to 50%, including the uncertainties in the measurement technique and in the seasonal variability in the data. For radon concentration determination, the overall corresponding uncertainty varied from 4 to 87%.

Risk estimates

Radium

When humans ingest radium, about 20% is absorbed into the circulation (Maletskos *et al.*, 1966). Radium is initially distributed to soft tissues and bone, but accumulates preferentially in growing bone. Normal processes of bone remodeling release radium, with an estimated biological half-life of 10 years (Wrenn *et al.*, 1985). Excretion is primarily through the feces (Maletskos *et al.*, 1966). Two basic approaches may be used to quantify the carcinogenic effects of ingestion of ^{226}Ra and ^{228}Ra in drinking water (EPA, 1991*a*). The first is to fit dose–response models to the incidence of bone sarcomas and head carcinomas among radium dial painters exposed to ^{226}Ra and/or ^{228}Ra (Mays *et al.*, 1985). The second is implemented in the RADRISK model (EPA, 1991*a*), which predicts the dose of ionizing radiation delivered to radiosensitive organs by intake of radium and calculates cancer risk based on a synthesis of human epidemiological data on the carcinogenic potency of radiation. However, the second approach takes into account the leukemia risk for ^{226}Ra based on associations reported for ^{224}Ra and for thorotrast. Speculation on the occurrence of leukemia and other types of cancers as a result of ^{226}Ra ingestion is inconsistent with the observation of radium dial painters and can result in an overestimation of the radiation-induced risk (BEIR IV, 1988).

Based on this consideration, the risk assessment in the present study was carried out using the method proposed by Mays *et al.* (1985). They

analyzed the predicted incidence of bone sarcomas and head carcinomas among low-dose dial painters following the studies performed by Rowland *et al.* (1983). These authors have performed several analyses of the epidemiological data with dose calculated as the total intake of radium to blood, weighing ^{228}Ra 2.5 times higher than ^{226}Ra. According to Mays, the relationships that provide acceptable fits to the epidemiological data at low intake of ^{226}Ra and ^{228}Ra are as follows:

(a) For the bone-sarcoma data:

$$I = 2{\cdot}7 \times 10^{-10} D_i$$

where:

I: Yearly excess incidence (bone sarcomas per person-year),
D_i: Total lifetime intake to the blood (^{226}Ra plus 2.5 times the intake of ^{228}Ra (Bq)).

(b) For the head carcinoma data:

$$I = 4{\cdot}3 \times 10^{-10} D_i$$

where:

I: Yearly excess incidence (head carcinomas per person-year),
D_i: Total ^{226}Ra lifetime intake (Bq).

In case (b), just the ^{226}Ra was considered since these investigators demonstrated that the incidence of head carcinomas was clearly associated with accumulation and decay of ^{222}Rn in air spaces in the head. The radon decay product of ^{228}Ra is too short-lived for substantial accumulation in the head cavities (NAS/BEIR IV, 1988).

In the present study, the lifetime risk was estimated taking into account that the life expectancy in Brazil is 65 years. For the incidence of head carcinoma, only the intake during the first 55 years was considered effective since the minimal latency period for the occurrence of this type of cancer is 10 years (Rowland *et al.*, 1978). For the incidence of bone sarcoma, it was assumed that intake occurred during the first 60 years, since in this case the latency period is 5 years (Rowland *et al.*, 1978). Ingestion was converted to intake into the blood using a gastrointestinal absorption factor of 20% (Maletskos *et al.*, 1969). The daily water consumption at all springs was assumed to be 1.2 litres per person (ICRP, 1975). No adjustment was made for age at exposure since the analysis performed by Chmelevsky (1986), established that there was not a significant difference between the risk due to the intake of radium by children and adults.

Radon

There are two main routes of exposure to radon entering a structure through water supplies. The first is emanation of radon into the air, followed by decay to charged radioactive progeny. These particles are then inhaled and deposited in the passageways of the lung, thus irradiating adjacent tissues. The primary concern with inhalation is with the induction of lung cancer within the bronchial epithelium (NAS-BEIR IV, 1988). The second route of exposure is direct ingestion of radon into the stomach, followed by translation and/or decay. Stomach cancer is the primary effect of concern here following ingestion, although other organs can contribute significantly to the number of fatalities (Crawford-Brown, 1990). Ingestion of short-lived radon decay products in water is not expected to be of significant concern compared to the ingestion of radon (Cross *et al.*, 1985).

The present study only explored the health effects due to the ingestion of radon. The risk from ingestion of radon was evaluated assuming lifetime ingestion of 1·2 litres/day of mineral water being consumed directly from the spring, which loses 20% of its initial radon content during the processes of filling a glass and drinking the water (EPA 1991*b*). Lifetime organ-specific radiation doses following radon ingestion were calculated using the biokinetic model of Crawford-Brown and organ-specific lifetime risk coefficients were derived from quantitative evaluation of epidemiological data on human cancer risk following exposure to several types of ionizing radiation (Crawford-Brown, 1990). For comparison to background fatal cancer rate, stomach cancer data collected in Brazil have been used, since this cancer type corresponds most closely with that induced by radon ingestion. The estimated total lifetime (65-year) probability of fatal stomach cancer was $2{\cdot}3 \times 10^{-6}$ per $Bq\,l^{-1}$ of ^{222}Rn in water, assuming a daily consumption of 1·2 litres.

RESULTS AND CONCLUSIONS

The radionuclide concentration data are presented in Table 1. Geometric means ranging from $<2{\cdot}2$ to $1{\cdot}80 \times 10^{3}\,mBq\,l^{-1}$ and from $<3{\cdot}7$ to $2{\cdot}3 \times 10^{1}\,mBq\,l^{-1}$ were observed for ^{226}Ra and ^{228}Ra, respectively. For ^{222}Rn the geometric means varied from 8×10^{-1} to $2{\cdot}16 \times 10^{2}\,Bq\,l^{-1}$. Measurable concentrations of ^{228}Ra were observed only in seven springs. Such results are in good agreement with those of Lauria *et al.* (1988) and follow from the geological characteristics of the area, which is more abundant in uranium than in thorium mineral deposits. Waters having a

TABLE 1
Concentration of ^{226}Ra, ^{228}Ra and ^{222}Rn in the Spring Waters of Águas da Prata

Spring	*^{226}Ra Concentration ($mBq\,l^{-1}$)*		*^{228}Ra Concentration ($mBq\,l^{-1}$)*		*^{222}Rn Concentration ($mBq\,l^{-1}$)*	
	Geometric mean	*Concentration range*	*Geometric mean*	*Concentration range*	*Geometric mean*	*Concentration range*
Vilela Bosque	$1{\cdot}80 \times 10^3$	$1{\cdot}37 \times 10^3$–$2{\cdot}42 \times 10^3$	< LLD	—	$1{\cdot}24 \times 10^2$	$1{\cdot}04 \times 10^2$–$1{\cdot}46 \times 10^2$
São Bento	$1{\cdot}36 \times 10^3$	$1{\cdot}23 \times 10^3$–$1{\cdot}63 \times 10^3$	< LLD	—	$2{\cdot}16 \times 10^2$	$2{\cdot}10 \times 10^2$–$2{\cdot}23 \times 10^2$
Padre	5	3–8	$2{\cdot}0 \times 10^1$	$1{\cdot}5 \times 10^1$–$3{\cdot}3 \times 10^1$	8×10^{-1}	2×10^{-1}–2·3
Vitória	$2{\cdot}49 \times 10^2$	$1{\cdot}59 \times 10^2$–$3{\cdot}19 \times 10^2$	< LLD	—	6·8	$4{\cdot}3$–$1{\cdot}9 \times 10^1$
Prata Nova	$5{\cdot}1 \times 10^1$	$3{\cdot}4 \times 10^1$–$6{\cdot}7 \times 10^1$	$2{\cdot}0 \times 10^1$	9–$3{\cdot}9 \times 10^1$	$2{\cdot}7 \times 10^1$	$2{\cdot}2 \times 10^1$–$3{\cdot}0 \times 10^1$
Prata Radioativa	$4{\cdot}4 \times 10^1$	$2{\cdot}9 \times 10^1$–$5{\cdot}9 \times 10^1$	$2{\cdot}3 \times 10^1$	9–$4{\cdot}7 \times 10^1$	$1{\cdot}2 \times 10^1$	$7{\cdot}7$–$1{\cdot}6 \times 10^1$
Prata Antiga	$1{\cdot}62 \times 10^2$	$1{\cdot}19 \times 10^2$–$1{\cdot}82 \times 10^2$	$1{\cdot}0 \times 10^1$	< LLD–$6{\cdot}2 \times 10^1$	3·6	2·6–5·6
Paiol	4	< LLD–7	$1{\cdot}2 \times 10^1$	6–$1{\cdot}7 \times 10^1$	5·8	5·3–6·3
Platina	< LLD	< LLD–4	$1{\cdot}6 \times 10^1$	$1{\cdot}3 \times 10^1$–$2{\cdot}9 \times 10^1$	2·5	9×10^1–7·2
Hotel Balneário	$5{\cdot}3 \times 10^1$	$2{\cdot}2 \times 10^1$–98×10^1	$2{\cdot}2 \times 10^1$	$1{\cdot}3 \times 10^1$–$3{\cdot}7 \times 10^1$	4·8	$1{\cdot}2$–$1{\cdot}0 \times 10^1$
Geometric means of all springs	$5{\cdot}9 \times 10^1$		$1{\cdot}1 \times 10^1$		$1{\cdot}0 \times 10^1$	
Geometric standard deviation	2		1		1·7	

high concentration of ^{226}Ra also present a high concentration of ^{222}Rn, although the two radionuclides are not in equilibrium. This finding was expected since radon concentration is due not only to radium decay but also to the emanation from the host rock. The measured concentrations of these natural radionuclides in the spring waters considered here are slightly higher than those observed in other countries (Bettencourt *et al.*, 1988), where the ^{226}Ra concentrations (geometric means) vary from 6·8 to 44 mBq l^{-1} and the ^{222}Rn concentrations vary from 7·7 to 16·7 Bq l^{-1}. In Brazil, the springs known to have the highest concentrations of these natural radionuclides are respectively, São Bento and Vilela (Mourão, 1992).

Based upon the measured concentrations, the lifetime risk due to the ingestion of radium and radon was estimated by using the geometric mean considering all the springs together. Using the methodology proposed by Mays, a total of eight radium-induced cancers (four head carcinomas and four bone sarcomas) were predicted per 10^6 exposed persons. The background incidence of these types of cancer in the Southeast region of Brazil is $7{\cdot}0 \times 10^{-6}$/year (Brumini *et al.*, 1982), which means 455 cases per million of persons assuming a life expectancy of 65 years. Therefore, one can predict an excess of 2% above the background incidence rate due to ingestion of mineral spring waters. For radon the cumulative risk estimation was carried out according to Crawford-Brown's method considering the occurrence of fatal stomach cancer. Based on the risk value presented in that work, the predicted incidence of fatal cancers is 23 cases per million of exposed persons. The background incidence rate of stomach cancer in the Southeast region of Brazil is $6{\cdot}8 \times 10^{-5}$/year, which means 4437 cases per million of person assuming a life expectancy of 65 years. Consequently, an excess of 0.5% in the background incidence rate due to ingestion of these spring waters can be expected.

Uncertainties in the risk assessment

The main source of uncertainty in the assessment of cancer risk due to the radium ingestion is the shape of the dose–response curve for bone cancer induction. If the true response were quadratic (dose-squared), due to a two-hit model of cancer induction (NAS-BEIR IV, 1988), true risks could be more than an order of magnitude lower than those calculated. Another source of contribution to the uncertainty to be considered is the variability of the parameters. The key parameters used in evaluating the cancer risk of radium ingestion are the gastrointestinal absorption fraction and the drinking water ingestion rate. A gastrointestinal absorption fraction of 0·2

was used, and, according to the work done by Maletskos *et al.* (1966), this value is likely to be correct within a factor of 2 or 3. A drinking water ingestion rate of 1·2 litres/day was used, although members of the assumed critical group comprising area residents may indeed consume more than this quantity on a daily basis. The uncertainty in this value may be approximately a factor of 2. Yet another source of uncertainty is the variability among the springs, which contributes a factor of 2. These considerations suggest that the true risk is likely to be within an order of magnitude higher, or two orders of magnitude lower, than those estimated here.

The main sources of uncertainty in the quantification of the carcinogenic effects due to radon ingestion are the shape of the dose–response curve, the derivation of organ-specific doses per unit of radon ingestion and the variability of the parameters. The assumption was made in assessing ingestion risk that the dose–response curve is linear. A linear response to α-particle exposure by ingestion is consistent with the human and animal data (NAS-BEIR IV, 1988). Although no direct data are available for ingested radon, the uncertainty contributed by possible nonlinearities in the dose–response curve is less than a factor of 2 (EPA, 1991*b*). Crawford-Brown (1991) evaluated the uncertainty in calculated dose per unit of intake as a factor of 3. The key parameters that are used in evaluating the cancer risk of radon ingestion, and that contribute for parameter variability, are the amount of radon ingested as a function of drinking-water concentration and the value of organ-specific risk coefficients. In the case of the drinking water ingestion rate, the same considerations are valid as for radium ingestion. Therefore, the uncertainty in this value is a factor of 2. EPA (1991*b*) estimated that the uncertainty in the organ-specific risk coefficients used to evaluate the ingestion risk of radon from low dose extrapolation was a factor of 2 or less. Another source of uncertainty is the variability in concentration among springs, which contributes with a factor of 2. Based on these considerations the true risk is likely to be within an other of magnitude in either direction of the risk calculated here.

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