Malcolm S. Field – Risks to cavers and cave workers from exposures to low-level ionizing  $\alpha$  radiation from <sup>222</sup>Rn decay in caves. *Journal of Cave and Karst Studies*, v. 69, no. 1, p. 207–228.

# RISKS TO CAVERS AND CAVE WORKERS FROM EXPOSURES TO LOW-LEVEL IONIZING α RADIATION FROM <sup>222</sup>RN DECAY IN CAVES

MALCOLM S. FIELD

Office of Research and Development, U.S. Environmental Protection Agency, Washington, D.C. 20460, field.malcolm@epa.gov

**Abstract:** Human health risks posed by exposure to elevated levels of <sup>222</sup>Rn in caves are not well documented. Various studies throughout the world have detailed the often very high <sup>222</sup>Rn gas concentrations in caves and exposures to cavers and commercial tour guides and other employees, but without a consequent assessment of the overall impact on human health. Although <sup>222</sup>Rn concentrations in caves are considered high relative to most above ground dwellings, the levels identified are also considered to be low for ionizing α radiation. Low-level ionizing radiation impacts on human health are deduced by application of the linear no-threshold theory (LNT) of radiation carcinogenesis. Comprehensive reviews of the published literature and an understanding of exposure time suggests that commercial cave workers (e.g., tour guides) and commercial <sup>238</sup>U-mine workers are both exposed for the same number of hours per month ( $\sim$ 170 h), but cave workers are exposed to much lower <sup>222</sup>Rn concentrations than are mine workers. Cavers will generally be exposed for a smaller number of hours per month. Risk estimates suggest that cavers will likely be subject to insignificant risks, but that cave workers may be subject to low-level risks of developing lung cancers from elevated levels of <sup>222</sup>Rn gas concentrations in caves.

#### Introduction

This paper was developed to provide the National Speleological Society reader with an intensive investigation of the potential health effects posed by exposure to elevated levels of radon in caves. To the author's knowledge, no other publication on radon in caves has delved into the risks to cavers from exposure to radon in caves to the extent that this paper does.

Radon-222 is generally regarded as a naturally occurring inert radioactive gas with a half life of 3.824 days and is produced within the <sup>238</sup>U decay series (Fig. 1), the process of which is described in detail in Field (1994, p. 52–60) and where the phenomenon of radioactivity is described in detail in Ivanovich (1992, p. 1–33). In fact, <sup>222</sup>Rn is only partly inert. Radon-222 may also be regarded as a metalloid<sup>1</sup>, an element that lies on the diagonal of the Periodic Table between the true metals and nonmetals (Fig. 2). Because <sup>222</sup>Rn is a metalloid, it exhibits some characteristics of both metals and nonmetals, such as forming a series of clathrate compounds<sup>2</sup> (inclusion compounds), and reacts readily with fluorine and fluorides (Stein, 1987; Cigna, 2005).

Radon-222 poses a substantial threat to human health when build-up occurs in confined spaces such as homes, mines, and caves (ICRP, 1994a, p. 1) and when exposure time is sufficiently long. The average annual per person radiation dose from exposure to <sup>222</sup>Rn from caves is estimated to be 1 nSv (0.1 μrem)<sup>3</sup>, although cavers and cave workers are expected to receive much higher doses (ATSDR, 1997, p. 217). Show caves are a recognized hazard in terms

of <sup>222</sup>Rn exposure to cave workers (tour guides, maintenance personnel, employees working in shops built over cave entrances, etc.) (IAEA, 2003, p. 5-6 and 46), but because of the sensitive nature of cave environments, high <sup>222</sup>Rn gas concentrations cannot easily be remediated (IAEA, 2003, p. 60). Forced air ventilation in caves is regarded as unthinkable because of the likely deleterious effects on the microclimates and biota (Yarborough and Meyers, 1978, p. 28 and 73). The U.S. Environmental Protection Agency (EPA) believes that the risks posed to human health by low levels of <sup>222</sup>Rn gas in single-family residences to be more significant than the risks to uranium miners exposed to very high levels of <sup>222</sup>Rn gas because the miners are only exposed for 170 h per month in the mine (1 Working Level Month) while homeowners spend more time in their dwellings and receive a greater overall exposure (Abelson, 1991). The principal threat is by the formation of lifespan shortening lung cancer, pulmonary emphysema, and pulmonary fibrosis through damage to the respiratory epithelium (Samet, 1997; Cross, 1987, p. 215-216).

The existence of elevated concentrations of <sup>222</sup>Rn in caves is well established in the literature (Table 1). Table 1 is a sampling of the literature that contains extensive <sup>222</sup>Rn concentration values, but does not list all of the basic literature on caves and <sup>222</sup>Rn (see for example, Cigna, 2005; Gunn, 2003, p. 617–618). However, the risks to

**Disclaimer:** The views expressed in this paper are solely those of the author and do not necessarily reflect the views or policies of the U.S. Environmental Protection Agency.

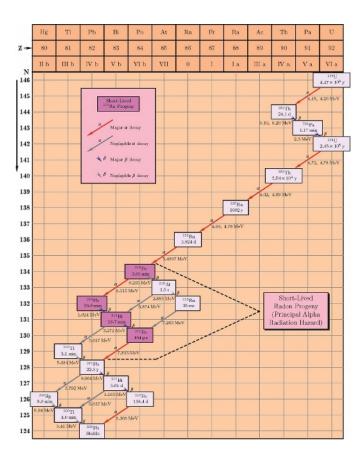


Figure 1. Uranium-238 decay series. The horizontal scale, Z, is the number of protons in the nucleus, and the vertical scale, N, is the number of neutrons in the nucleus. Also shown are the half-lives, type of decay (either by  $\alpha$ - or  $\beta$ -particles), and the major radiation energies (in MeV) of  $^{238}\mathrm{U}$  and its progeny. Modified from Cothern (1987, p. 7) and NRC (1988, p. 26). Negligible percentage decays included even though they are not a human health concern. See Table 12 in the Appendix for the historic names of the  $^{222}\mathrm{Rn}$  progeny.

cavers and cave workers from exposure to the relatively high <sup>222</sup>Rn concentrations in caves are poorly understood and rarely reported in the literature (Kobal et al., 1986, 1987; Vaupotič et al., 1998, 2001).

The purpose of this paper is to explore the risks posed to the health of cavers and tour guides as a result of exposure to low-level ionizing radiation from  $^{222}$ Rn gas and its progeny. Radon-219 and  $^{220}$ Rn (commonly known as thoron) do not represent as serious a concern as does  $^{222}$ Rn because (1)  $^{219}$ Rn is relatively rare and has a very short half life ( $T_{\frac{1}{2}} = 4$  s) and (2)  $^{220}$ Rn has a very short half life ( $T_{\frac{1}{2}} = 56$  s) (ATSDR, 1990, p. 11). Radon-219 has only rarely been found at elevated levels in caves (Yarborough and Meyers, 1978, p. 42).

Particular attention will be directed at the linear nothreshold theory (LNT) of radiation carcinogenesis because this is the accepted model for estimating risks posed by exposure to <sup>222</sup>Rn gas and its progeny (Dicus, 2001;

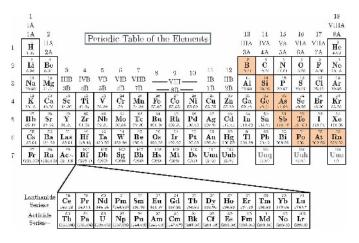


Figure 2. Arrangement of the metalloid elements (dark shading) in the Periodic Table (modified from Stein (1987).

Kellerer and Nekolla, 2000; NCRP, 2001; NAS, 2005; NRC, 1999, p. 69) even though there is some dispute as to its appropriateness (e.g., Bond et al., 1996; Cohen, 2000). There is, however, good reason to continue relying on the LNT even with all its attendant problems because it is a reasonable risk model (Oughton, 2006).

#### Threats Posed by <sup>222</sup>Rn and its Progeny

Radon-222 is the heaviest of the noble gases and because it is a relatively nonreactive gas that exists everywhere in the environment, it tends to migrate to and concentrate in enclosed spaces (e.g., basements, caves, tunnels, etc.). However, <sup>222</sup>Rn is not a major health risk by itself.

High concentrations of <sup>222</sup>Rn progeny (<sup>218</sup>Po, <sup>214</sup>Pb, <sup>214</sup>Bi, and <sup>214</sup>Po) are widely recognized as a source of lung cancer and possibly other cancers (Henshaw et al., 1990; Bridges et al., 1991) through the emission of  $\alpha$ - and  $\beta$ particles with dense ionization along their tracks although it is the  $\alpha$ -particles that are most responsible for the resulting high-linear energy transfer (LET)<sup>4</sup> because of its large +2 charge and relatively large mass (~8,000 times that of an electron). Emission of β-particles (high-energy electrons) results in low-LET because of its ±1 charge and much smaller mass than  $\alpha$ -particles. In either case, both low and high LET interactions can cause significant DNA damage (ATSDR, 1997, p. 30-31), but it is the densely ionizing radiation produced by α-particle decay that causes many double strand DNA breaks that are most difficult for cell repair and are most likely to give rise to cancer formation (Craven and Smit, 2006). According to Craven and Smit, sparsely ionizing radiation typically results in single strand DNA breaks which are much easier for cells to repair.

Cothern (1989) states that "between 4,000 and 30,000 fatal lung cancers occur each year due to exposure to radon in indoor air," but does not offer any supporting data or references for such a contention. The EPA had projected

Table 1. Summary of <sup>222</sup>Rn literature (modified from Hyland and Gunn (1994). Note that many of the references include <sup>222</sup>Rn measurements from several sources<sup>a</sup>.

	Mean <sup>222</sup> Rn Concentration	Number of <sup>222</sup> Rn	Max. <sup>222</sup> Rn Concentration	Min. <sup>222</sup> Rn Concentration	D. C
Country	$(Bq m^{-3})$	Measurements	$(Bq m^{-3})$	$(Bq m^{-3})$	Reference
Australia	610	274	4,045	9	Solomon et al. (1996)
China <sup>b</sup>	141	32	278	38	Wiegand et al. (1995)
Czech Republic	1,235	60	21,000	200	Burian and Stelcl (1990)
Great Britain	2,907	820	46,080	10	Hyland and Gunn (1994)
Great Britain		2,000	155,000	100	Hyland and Gunn (1994)
Great Britain	35,890	34	155,000	7,400	Gunn et al. (1991)
Great Britain	9,306	13	12,552	68	Gillmore et al. (2000)
Great Britain	365	42	3,187	26	Gillmore et al. (2002)
Great Britain	315	28	3,047	34	Gillmore et al. (2002)
Greece	25,179	6	88,060	185	Papastefanou et al. (1986)
Hungary	3,300	25	14,000	500	Somogyi et al. (1989)
Hungary	2,468	8	13,200	200	Lenart et al. (1990)
Ireland	4,127	26	7,940	200	Duffy et al. (1996)
Japan	11	5	20	< 1	Miki and Iauthora (1980)
Malaysia	596	39	1,978	100	Gillmore et al. (2005)
Poland	1,166	279	4,180	60	Przylibski (1999)
Russia	2,390	14	8,550	373	Gunn (1991)
Slovenia	1,412	101	7,220	15	Kobal et al. (1986)
Slovenia	965	66	5,920	60	Kobal et al. (1987)
Spain	108	301	488	5	Dueñas et al. (1998)
Spain	3,564	8,587	7,120	186	Lario et al. (2005)
South Africa	267	63	2,319	3	Gamble (1981)
Switzerland	25,000	6	40,000	2,000	Surbeck (1990)
United States	1,927	60	9,350	37	Yarborough (1976)
United States	2,589	11	9,460	370	Eheman et al. (1991)
United States	1,475	•••	2,350	740	Ahlstrand (1980)
United States		860	1,850	333	Ahlstrand and Fry (1976)
United States	11,678	37	82,177	11	Bashor (undated)

<sup>&</sup>lt;sup>a</sup> Data quality control likely varies for each study conducted for each country which should be regarded as problematic.

14,000 lung-cancer deaths per year from residential <sup>222</sup>Rn exposure with an uncertainty range of 7,000 to 30,000 (Page, 1993), but now estimates 21,000 lung-cancer deaths per year from residential <sup>222</sup>Rn exposure with an uncertainty range of 8,000 to 45,000 (U.S. EPA, 2006). In fact, the expected lung cancers and other adverse health effects may be more a result of smoking than of inhalation of <sup>222</sup>Rn gas (Pisa et al., 2001). In addition, the expected lung cancers in cavers and cave workers appear to be conspicuously missing from the published literature, perhaps because no one has yet linked lung cancers in cavers and cave workers to long-term exposure to high <sup>222</sup>Rn concentrations (Halliday, 2003).

Risks to cavers and cave workers by exposures to high levels of <sup>222</sup>Rn may not be as serious as is often presented. The potential overestimation of the risks posed by elevated levels of <sup>222</sup>Rn and the belief by many individuals that overestimation may actually be the case, have caused some

consternation among some researchers (Cothern, 1989, 1990; Little, 1997). The possibility that <sup>222</sup>Rn and its progeny may be responsible for some cancers other than lung cancer also is not strongly supported in the literature (Tomasek et al., 1993). For example, Law et al. (2000) were unable to establish an association between household exposure to <sup>222</sup>Rn and the development of leukemia in adults in Great Britain. Lauier et al. (2001) obtained similar results.

A significant reason why <sup>222</sup>Rn and its progeny may not be as serious a threat to cavers and cave workers may be because although <sup>222</sup>Rn concentrations in caves are considered to be elevated, these levels are also considered to be relatively low in terms of ionizing radiation. For example, employees exposed to radiation in the work place in Great Britain (e.g., cave tour guides) are not allowed to receive annual effective radiation doses<sup>5</sup> above 50 mSv yr<sup>-1</sup> (10 WLM yr<sup>-1</sup>) with an action level of 15 mSv

b Measurements taken in Chinese cave dwellings built in the Chinese Loess Plateau which is mainly composed of Mesozoic sandstones overlain by Tertiary red clays that are covered by Quaternary loess tens to over one hundred meters thick (Wiegand et al., 1995) where the number of inhabitants exceed three million (Yanada, 2003)

Table 2. Inhalation exposure studies of <sup>222</sup>Rn (modified from ATSDR, 1990, pp. 13-15). Superscript numbers next to each entry correspond to data point numbers in Figure 3.

Properties   Pro	0						
Species         Trequency/ Trequency         Hibert         (Rog m²²)         Less Serious (Bq m²²)         Serious (Bq m²²)           Mouse*         1 d         Hemato         Beath         8.14 × 10° (30 d LD <sub>w</sub> )           Mouse*         2 d wk²¹         Beath         1.11 × 10°         8.14 × 10° (30 d LD <sub>w</sub> )           Rad*         Lifespan         Death         1.11 × 10°         1.78 × 10° (dec lifespan)           Mouse*         Lifespan         Death         1.78 × 10° (dec lifespan)         1.78 × 10° (dec lifespan)           Rad*         Lifespan         Death         1.78 × 10° (dec lifespan)         1.78 × 10° (dec lifespan)           Rad*         Lifespan         Death         1.78 × 10° (dec lifespan)         1.78 × 10° (dec lifespan)           Rad*         Lifespan         Resp         1.78 × 10° (dec lifespan)         1.78 × 10° (metaplasia)           Mouse*         Lifespan         Resp         1.78 × 10° (dec lifespan)         1.78 × 10° (metaplasia)           Mouse*         Lifespan         Resp         1.78 × 10° (dec lifespan)         1.78 × 10° (metaplasia)           Mouse*         Lifespan         Resp         1.78 × 10° (dec lifespan)           Mouse*         Lifespan         Resp         1.78 × 10° (dec lifespan)           Mouse* <t< td=""><td>7</td><td>Exposure</td><td></td><td></td><td>LOA</td><td>EL (Effect)</td><td></td></t<>	7	Exposure			LOA	EL (Effect)	
Aceute Exposure Mouse*         1 d 1 d 1 d 1 d 1 d 1 d 1 d 1 d 1 d 1 d	Species	rrequency/ Duration	Effect	$(Bq m^{-3})$	Less Serious (Bq m <sup>-3</sup>		Reference
Mouse of Mouse o							
Mouse*         1 d         Hemato         8.14 × 10° (anemia)           Rat*         2 d wk-1         Death         1.11 × 10°         8.14 × 10° (anemia)           Rat*         Lifespan         Death         1.11 × 10°         1.78 × 10° (dec lifespan)           Mouse*         Lifespan         Death         1.78 × 10° (dec lifespan)         1.78 × 10° (dec lifespan)           Rat*         Lifespan         Resp         1.78 × 10° (dec bw)         1.78 × 10° (metaplasia)           Mouse*         Lifespan         Resp         1.78 × 10° (dec bw)         1.78 × 10° (metaplasia)           Mouse*         Lifespan         Resp         1.78 × 10° (dec bw)         1.78 × 10° (metaplasia)           Mouse*         Lifespan         Resp         1.78 × 10° (dec bw)         1.78 × 10° (metaplasia)           Mouse*         Lifespan         Resp         1.78 × 10° (dec bw)         1.78 × 10° (metaplasia)           Mouse*         Lifespan         Resp         1.78 × 10° (dec bw)         1.78 × 10° (metaplasia)           Mouse*         Lifespan         Resp         1.78 × 10° (dec bw)         1.78 × 10° (metaplasia)           A wk-1         Other         1.78 × 10° (dec bw)         1.78 × 10° (metaplasia)           Dog 1         Licspan         Resp         2.04 × 10		1 d	Death			$8.14 \times 10^9 \ (30 \ d \ \text{LD}_{50})$	Morken (1955)
Part		1 d	Hemato			$8.14 \times 10^{9}$ (anemia)	Morken (1955)
Rat <sup>2</sup>							
Rat*         4-6 mo         Death         1111 × 10*           2 d wk - 1         2 d wk - 1         1.16 d - 1           2 d wk - 2         2 d wk - 1         1.78 × 10° (dec lifespan)           90 h wk - 1         1.50 h wk - 1         1.55 × 10° (dec lifespan)           150 h wk - 1         1.50 h wk - 1         1.78 × 10° (dec lifespan)           150 h wk - 1         1.65 m w - 1         1.78 × 10° (dec lifespan)           150 h wk - 1         1.50 h wk - 1         1.50 × 10° (dec lifespan)           150 h wk - 1         1.50 h wk - 1         1.50 × 10° (dec lifespan)           150 h wk - 1         1.50 h wk - 1         1.50 × 10° (dec lifespan)           150 h wk - 1         1.50 × 10° (dec lifespan)         1.50 × 10° (metaplasia)           150 h wk - 1         1.50 × 10° (dec lifespan)         1.50 × 10° (metaplasia)           150 h wk - 1         1.50 × 10° (dec lifespan)         1.78 × 10° (metaplasia)           150 h wk - 1         1.50 × 10° (dec lifespan)         1.78 × 10° (metaplasia)           150 h wk - 1         1.50 × 10° (dec lifespan)         1.78 × 10° (metaplasia)           150 h wk - 1         1.50 × 10° (dec lifespan)         1.78 × 10° (metaplasia)           150 h wk - 1         1.50 × 10° (dec lifespan)         1.78 × 10° (metaplasia)           150 h wk - 1			7	50.			-
Lifespan   Lifespan   Lifespan		4-6 mo	Death	$1.11 \times 10^{-}$			Chameaud et al. (1984)
Rat*         Lifespan         Death         1.78 × 10° (dec lifespan)           Mouse*         2 d wk <sup>-1</sup> /2 d wk <sup>-1</sup> Death         1.55 × 10° (dec lifespan)           Hamster*         Lifespan         Death         1.78 × 10° (dec lifespan)           Hamster*         Lifespan         Resp         1.78 × 10° (dec lifespan)           Rat*         Lifespan         Resp         1.78 × 10° (dec lifespan)           Mouse*         Lifespan         Resp         1.55 × 10° (dec lifespan)           Mouse*         Lifespan         Resp         1.55 × 10° (dec lifespan)           Mouse*         Lifespan         Resp         1.78 × 10° (dec lifespan)           Hamster*         Other         1.78 × 10° (dec bw)         1.78 × 10° (fibrosis)           Dog**         Lifespan         Resp         1.78 × 10° (dec bw)         1.78 × 10° (fibrosis)           Dog**         Lifespan         Resp         1.78 × 10° (dec bw)         1.78 × 10° (fibrosis)           Rat**         Lifespan         Resp         2.04 × 10° (fibrosis)           Rat**         Lifespan         Resp         2.04 × 10° (fibrosis)           Rat**         Lifespan         Resp         2.78 × 10° (fibrosis)           Rat**         Lifespan         Resp         2		2 d wK -					
Kart         Lifespan         Death         Lifespan           2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> 1.55 × 107 (dec lifespan)           150 h wk <sup>-1</sup> Death         1.78 × 108 (dec lifespan)           Hamster <sup>6</sup> Lifespan         1.78 × 108 (dec lifespan)           Lifespan         Resp         1.78 × 108 (dec lifespan)           Rat <sup>7</sup> Other         1.55 × 107 (dec lifespan)           Mouse <sup>8</sup> Lifespan         Resp           Lifespan         Resp         1.55 × 107 (dec lifespan)           Mouse <sup>8</sup> Lifespan         Resp           Lifespan         Resp         1.78 × 108 (dec bw)           Loher         1.78 × 108 (dec bw)         1.78 × 108 (fibrosis)           Lifespan         Resp         1.78 × 108 (dec bw)           Dog <sup>11</sup> 1.50 d wk <sup>-1</sup> Other           Dog wk <sup>-1</sup> Other         1.78 × 108 (dec bw)           Lifespan         Resp         2.04 × 107 (fibrosis)           S d wk <sup>-1</sup> Other         2.04 × 107 (fibrosis)           Rat <sup>12</sup> 2.5-8 wk         Cancer           Life dec bw)         1.11 × 107 (CEL-lung)		1 n d				3.1 5.7 80 1 91 1	70000
Mouse <sup>5</sup> Lifespan         1.55 × 107 (dec lifespan)           Hamster <sup>6</sup> Lifespan         1.50 h wk <sup>-1</sup> Hamster <sup>6</sup> Lifespan         1.78 × 108 (dec lifespan)           Lifespan         Resp         1.78 × 108 (dec lifespan)           Lifespan         Resp         1.55 × 107 (dec lifespan)           Mouse <sup>8</sup> Lifespan         Resp           Lifespan         Resp         1.55 × 107 (dec bw)           Lifespan         Resp         1.55 × 107 (dec lifespan)           Mouse <sup>9</sup> Lifespan         Resp           Lifespan         Resp         1.78 × 108 (dec bw)           Lifespan         Resp         1.78 × 108 (dec bw)           Hamster <sup>10</sup> Lifespan         Resp           Lifespan         Resp         1.78 × 108 (dec bw)           Dog <sup>11</sup> 1-50 d         Resp           Loh wk <sup>-1</sup> Other         1.78 × 108 (dec bw)           S d wk <sup>-1</sup> Resp         2.04 × 107 (fibrosis)           S d wk <sup>-1</sup> Resp         2.04 × 107 (fibrosis)           S d wk <sup>-1</sup> 2.5-8 wk         2.5-8 wk           A d wk <sup>-1</sup> 2.5-8 wk         2.5-8 wk           A d wk <sup>-1</sup> 2.5-8 wk		Lirespan	Death			$1.78 \times 10^{-}$ (dec mespan)	Falmer et al. (1973)
Mouse*         Lifespan         Death         1.55 × 107 (dec lifespan)           Hamster*         Lifespan         Death         1.78 × 108 (dec lifespan)           Rat7         Lifespan         Resp         1.78 × 108 (dec lifespan)           Rat7         Lifespan         Resp         1.78 × 108 (dec lifespan)           Mouse*         Lifespan         Resp         1.55 × 107 (dec lymph)           Mouse*         Lifespan         Resp         1.78 × 108 (dec bw)           Mouse*         Lifespan         Resp         1.78 × 108 (fibrosis)           Hamster¹o         Lifespan         Resp         1.78 × 108 (fibrosis)           Hamster¹o         Lifespan         Resp         1.78 × 108 (dec bw)           Dog¹¹         Lifespan         Resp         1.78 × 108 (dec bw)           Hamster¹o         Lifespan         Resp         1.78 × 108 (dec bw)           Dog¹¹         Loh wk⁻¹         Other         1.78 × 108 (dec bw)           S d wk⁻¹         Cancer         2.04 × 107 (fibrosis)           Rat¹²         2.5-8 wk         Cancer         2.78 × 107 (CEL-lung)           Rat¹⁴         6-6 mo         Cancer         1.11 × 107 (CEL-lung)           Lod wk⁻¹¹         2 d wk⁻¹²         1.11 d d a and and and and		2  d wK 90 b $\text{w/c}^{-1}$					
150 h wk		I ifesnan	Death			$1.55 \times 10^7$ (dec lifespan)	Morken and Scott (1966)
Lifespan Death Resp 1.78 × 10 <sup>8</sup> (dec lifespan) 1.78 × 10 <sup>8</sup> (metaplasia) 2 d wk <sup>-1</sup> Other 1.78 × 10 <sup>8</sup> (dec bw) 1.78 × 10 <sup>8</sup> (metaplasia) 1.50 h wk <sup>-1</sup> Hemato 1.55 × 10 <sup>7</sup> (dec bw) 1.55 × 10 <sup>8</sup> (dec bw) 1.78 × 10 <sup>8</sup> (de		$150 \text{ h wk}^{-1}$				(	
Lifespan Resp 1.78 × 108 (dec bw) 2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> Lifespan Resp 1.55 × 10 <sup>7</sup> (dec bw) 1.50 h wk <sup>-1</sup> Henato Other 1.55 × 10 <sup>7</sup> (dec bw) 1.78 × 108 (dec bw) 2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> 1.50 d Resp 1.78 × 108 (dec bw) 2 d wk <sup>-1</sup> 1.50 d Resp 1.78 × 108 (dec bw) 2 d wk <sup>-1</sup> 1.50 d Resp 1.78 × 108 (dec bw) 2 d wk <sup>-1</sup> 3 d wk <sup>-1</sup> 2 d wk <sup>-1</sup> 3 d wk <sup>-1</sup> 3 d wk <sup>-1</sup> 4 d wk <sup>-1</sup> 5 d wk <sup>-1</sup> 5 d wk <sup>-1</sup> 6 d wc	Hamster <sup>6</sup>	Lifespan	Death			$1.78 \times 10^8$ (dec lifespan)	Palmer et al. (1973)
2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> Lifespan 150 h wk <sup>-1</sup> 2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> 10 Lifespan 150 h wk <sup>-1</sup> 2 d wk <sup>-1</sup> 10 Lifespan 10 Lifesp	$Rat^7$	Lifespan	Resp			$1.78 \times 10^8$ (metaplasia)	Palmer et al. (1973)
Lifespan		$2 \text{ d wk}^{-1}$	Other		$1.78 \times 10^8$ (dec bw)		
Lifespan  2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> 2 d wk <sup>-1</sup> 1-50 d  Resp  Lifespan  2 d wk <sup>-1</sup> 2 d wk <sup>-1</sup> 1-50 d  Lifespan  2 d wk <sup>-1</sup> 2 d wk <sup>-1</sup> 2 d wk <sup>-1</sup> 2 d wk <sup>-1</sup> Lifespan  2 d wk <sup>-1</sup> Lili x 10 <sup>7</sup> (CEL-lung)  2 d wk <sup>-1</sup> Lili x 10 <sup>7</sup> (CEL-lung)  2 d wk <sup>-1</sup> Lili x 10 <sup>7</sup> (CEL-lung)	c	$90 \text{ h wk}^{-1}$				ţ	
150 h wk <sup>-1</sup>   Hemato   1.55 × 10' (dec lymph)     2 d wk <sup>-1</sup>   Other   1.55 × 10' (dec lymph)     2 d wk <sup>-1</sup>   Other   1.78 × 10' (dec bw)     2 d wk <sup>-1</sup>   Other   1.78 × 10' (dec bw)     2 d wk <sup>-1</sup>   Other   1.78 × 10' (dec bw)     2 d wk <sup>-1</sup>   Other   1.78 × 10' (dec bw)     2 d wk <sup>-1</sup>   Cancer   2.5-8 wk   Cancer   2.5-8 wk     3-6 h d <sup>-1</sup>   Cancer   2.5-115 d     4-5 h d <sup>-1</sup>   Cancer   2.78 × 10' (CEL-lung)     6-6 mo   Cancer   1.11 × 10' (CEL-lung)     2 d wk <sup>-1</sup>   Cancer   1.11 × 10' (CEL-lung)     4 d wc <sup>-1</sup>   Cancer   1.11 × 10' (CEL-lung)     4 d wc <sup>-1</sup>   Cancer   1.11 × 10' (CEL-lung)     5 d wk <sup>-1</sup>   Cancer   1.11 × 10' (CEL-lung)     6 d mo   Cancer   1.11 × 10' (CEL-lung)     1 h d <sup>-1</sup>   1 h d <sup>-1</sup>   1 h d <sup>-1</sup>	$Mouse^8$	Lifespan	Resp		1		Morken and Scott (1966)
Lifespan Resp 1.78 × 10° (dec bw) 1.78 × 10° (fibrosis) 2 d wk <sup>-1</sup> Other 2 d wk <sup>-1</sup> Other 1.78 × 10° (dec bw) 2 d wk <sup>-1</sup> Other 1.78 × 10° (dec bw) 2 d wk <sup>-1</sup> Other 1.78 × 10° (dec bw) 2 d wk <sup>-1</sup> Resp 2.5-8 wk Cancer 2.5-8 wk <sup>-1</sup> Cancer 2.5-115 d Cancer 2.5-8 wk <sup>-1</sup> Cancer 2.5-8 wk <sup>-1</sup> Cancer 2.5-8 wk <sup>-1</sup> 1.11 × 10° (CEL-lung) 2.5-8 wk <sup>-1</sup> 1.11 × 10° (CEL-lung) 2.5-8 wk <sup>-1</sup> 1.11 × 10° (CEL-lung) 3-6 h d <sup>-1</sup> 2.5-115 d Cancer 1.11 × 10° (CEL-lung) 2.5-8 wk <sup>-1</sup> 1.11 × 10° (CEL-lung) 3-6 wc <sup>-1</sup> 1.11 × 10° (CEL-lung) 2.5-8 wk <sup>-1</sup> 1.11 × 10° (CEL-lung) 3-6 wc <sup>-1</sup> 1.11 × 10° (CEL-lung)		$150 \text{ h wk}^{-1}$	Hemato		$1.55 \times 10^7$ (dec lymph		
Lifespan  Lifespan  2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> 10 Lifespan  2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> 10 Lifespan  2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> 1-50 d 5 d wk <sup>-1</sup> 2 d wk <sup>-1</sup> 3 - 6 h d <sup>-1</sup> 2 - 115 d 6 - 6 mo  2 d wk <sup>-1</sup> 2 d wk <sup>-1</sup> 3 - 6 h d <sup>-1</sup> 2 d wk <sup>-1</sup> 1 h d <sup>-1</sup> 1 h d <sup>-1</sup>	c		Other		$1.55 \times 10'$ (dec bw)	c	
2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> 1.78 × 10° (dec bw) 90 h wk <sup>-1</sup> 2 d wk <sup>-1</sup> 2 d wk <sup>-1</sup> 90 h wk <sup>-1</sup> 1-50 d 5 d wk <sup>-1</sup> 2.5-8 wk 2.5-8 wk 2.5-8 wk 4-5 h d <sup>-1</sup> 2-5 d wk <sup>-1</sup> 3-6 h d <sup>-1</sup> 2-5 d wk <sup>-1</sup> 3-6 h d <sup>-1</sup> 2.5 wk <sup>-1</sup> 3-6 h d <sup>-1</sup> 3-78 × 10 <sup>7</sup> (CEL-lung) 2 d wk <sup>-1</sup> 1 h d <sup>-1</sup>	Mouse	Lifespan	Resp		c	$1.78 \times 10^8$ (fibrosis)	Palmer et al. (1973)
90 h wk       1.78 × 108       (metaplasia)         2 d wk <sup>-1</sup> Other       1.78 × 108       (dec bw)         90 h wk <sup>-1</sup> 2.04 × 107       (fibrosis)         5 d wk <sup>-1</sup> 2.04 × 107       (fibrosis)         2.5-8 wk       Cancer       1.11 × 107       (CEL-lung)         4 d wk <sup>-1</sup> 3-6 h d <sup>-1</sup> 2.78 × 107       (CEL-lung)         4-5 h d <sup>-1</sup> 6-6 mo       Cancer       1.11 × 107       (CEL-lung)         2 d wk <sup>-1</sup> 1 h d <sup>-1</sup>		$2 \text{ d wk}^{-1}$	Other		$1.78 \times 10^{\circ}$ (dec bw)		
ter'' Lifespan Resp 1.78 × 108 (dec bw) 2 d wk <sup>-1</sup> Other 2.5 d wk <sup>-1</sup> Other 2.5 d wk <sup>-1</sup> Resp 2.5 d wk <sup>-1</sup> Cancer 2.5 d wk <sup>-1</sup> Cancer 2.5 d wk <sup>-1</sup> 3-6 h d <sup>-1</sup> Cancer 6-6 mo Cancer 2.5 d wk <sup>-1</sup> 1.11 × 107 (CEL-lung) 2.11 × 107 (CEL-lung) 2.11 × 107 (CEL-lung) 2.5 d wk <sup>-1</sup> 1.11 × 107 (CEL-lung) 3.5 d wk <sup>-1</sup> 1.11 × 107 (CEL-lung) 3.	Ç	90 h wk				0	
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Hamster	Lifespan	Resp		0	$1.78 \times 10^{\circ}$ (metaplasia)	Palmer et al. (1973)
90 h wk $^{-1}$ Resp $^{-1}$ S d wk $^{-1}$ $^{-1}$ 2.04 × 10 $^{7}$ (fibrosis) $^{-1}$ 2.0 h wk $^{-1}$ $^{-1}$ Cancer $^{-1}$ 4 d wk $^{-1}$ $^{-1}$ 3-6 h d $^{-1}$ $^{-1}$ Cancer $^{-1}$ 2.78 × 10 $^{7}$ (CEL-lung) $^{-1}$ 4.5 h d $^{-1}$ $^{-1}$ 6-6 mo $^{-1}$ Cancer $^{-1}$ 1.11 × 10 $^{7}$ (CEL-lung) $^{-1}$ 1 h d $^{-1}$		$2 \text{ d wk}^{-1}$	Other		$1.78 \times 10^{\circ} (\text{dec bw})$		
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	=	90 h wk	,				
$5 \text{ d wk}^{-1}$ $20 \text{ h wk}^{-1}$ $2.5-8 \text{ wk}$ $4 \text{ d wk}^{-1}$ $3-6 \text{ h d}^{-1}$ $25-115 \text{ d}$ $4-5 \text{ h d}^{-1}$ $2-78 \times 10^7 \text{ (CEL-lung)}$ $4-5 \text{ h d}^{-1}$ $6-6 \text{ mo}$ $2 \text{ d wk}^{-1}$ $1 \text{ h d}^{-1}$	Dogʻʻ	I-50 d	Kesp			$2.04 \times 10^{\circ}$ (fibrosis)	Morken (1973)
$20 \text{ h wk}$ 2.5-8 wk $4 \text{ d wk}^{-1}$ $1.11 \times 10^7 \text{ (CEL-lung)}$ $4 \text{ d wk}^{-1}$ $3-6 \text{ h d}^{-1}$ $2.5-115 \text{ d}$ Cancer $4-5 \text{ h d}^{-1}$ $2.78 \times 10^7 \text{ (CEL-lung)}$ $6-6 \text{ mo}$ $2 \text{ d wk}^{-1}$ $1 \text{ h d}^{-1}$		$5 \text{ d wk}^{-1}$					
$2.5-8 \text{ wk}$ Cancer $4 \text{ d wk}^{-1}$ $4 \text{ d wk}^{-1}$ $3-6 \text{ h d}^{-1}$ $2.78 \times 10^7 \text{ (CEL-lung)}$ $4-5 \text{ h d}^{-1}$ $6-6 \text{ mo}$ Cancer $2 \text{ d wk}^{-1}$ $1.11 \times 10^7 \text{ (CEL-lung)}$		$20 \text{ h wk}^{-1}$				t	
$4 \text{ d wk}^{-1}$ $3-6 \text{ h d}^{-1}$ $25-115 \text{ d}$ Cancer $2-115 \text{ d}$ Cancer $6-6 \text{ mo}$ Cancer $2 \text{ d wk}^{-1}$ $1 \text{ h d}^{-1}$	$Rat^{12}$	2.5-8  wk	Cancer			$1.11 \times 10^7$ (CEL-lung)	Chameaud et al. (1982)
$3-6 \text{ h d}^{-1}$ $25-115 \text{ d}$ $4-5 \text{ h d}^{-1}$ $6-6 \text{ mo}$ $2 \text{ d wk}^{-1}$ $1 \text{ h d}^{-1}$		$4 \text{ d wk}^{-1}$					
$\begin{array}{cccccccccccccccccccccccccccccccccccc$		$3-6 \text{ h d}^{-1}$					
$4-5~\mathrm{h}~\mathrm{d}^{-1}$ $6-6~\mathrm{mo}$ Cancer $2~\mathrm{d}~\mathrm{wk}^{-1}$ $1~\mathrm{h}~\mathrm{d}^{-1}$	$Rat^{13}$	25–115 d	Cancer			$2.78 \times 10^7$ (CEL-lung)	Chameaud et al. (1974)
6-6 mo Cancer 1.11 $\times$ 10 <sup>7</sup> (CEL-lung) 2 d wk <sup>-1</sup> 1 h d <sup>-1</sup>		$4-5 \text{ h d}^{-1}$				1	
$2 \text{ d wk}^{-1}$ $1 \text{ h d}^{-1}$	$\mathrm{Rat}^{14}$	om 9–9	Cancer			$1.11 \times 10^7 \text{ (CEL-lung)}$	Chameaud et al. (1984)
$1\mathrm{h}\mathrm{d}^{-1}$		$2 \mathrm{d} \mathrm{wk}^{-1}$					
		$1 \text{ h d}^{-1}$					

Table 2. Continued.

	Exposure			LOAEL (Effect)		
Species	Frequency/ Duration	Effect	NOAEL $(Bq m^{-3})$	Less Serious (Bq m <sup>-3</sup> ) Serio	Serious (Bq m <sup>-3</sup> )	Reference
Chronic Exposure Hamster <sup>15</sup>	Lifespan 5 d wk <sup>-1</sup>	Death	$1.15\times10^7$			Cross et al. (1978)
Hamster <sup>16</sup>	$6 \text{ h d}^{-1}$ >1 mo - 18 yr	Resp		>3.70 × 1	10 <sup>3</sup> (tuberculosis)	$>3.70 \times 10^3$ (tuberculosis) Waxweiler et al. (1981)
Hamster <sup>17</sup>	$({ m occup})$ Lifespan $5 { m d} { m wk}^{-1}$	Resp Hemato	$1.15\times10^7$	$9.62  imes 10^6$	$9.62 \times 10^6$ (hyperplasia)	Cross et al. (1978)
Human <sup>18</sup>	6 h d <sup>-1</sup> Other 0.5–23 yr (occup) Cancer	Other ()		$9.62 \times 10^6 \text{ (dec bw)}$ $1.26 \times 10^4$	$1.26 \times 10^4 \text{ (CEL-lung)}$	Gottlieb and Husen
Human <sup>19</sup> Human <sup>20</sup> Human <sup>21</sup>	(occup) 0–14 yr (occup) >29 yr (occup)	Cancer Cancer Cancer		$7.40 \times 10^{3}$ $3.70 \times 10^{3}$ $2.22 \times 10^{3}$	$7.40 \times 10^3$ (CEL-lung) $3.70 \times 10^3$ (CEL-lung) $2.22 \times 10^3$ (CEL-lung)	(1982) Morrison et al. (1981) Solli et al. (1985) Edling and Axelson
Human <sup>22</sup>	>1->20  yr (occup)	Cancer		$1.85\times10^3$	$1.85 \times 10^3$ (CEL-lung)	(1983) Damber and Larsson (1985)
Human <sup>23</sup>	$48 \text{ wk yr}^{-1}$ $48 \text{ wk}^{-1} (\text{occur})$	Cancer		$1.85\times10^3$	$1.85 \times 10^3 \text{ (CEL-lung)}$	Howe et al. (1987)
Human <sup>24</sup>	>10 yr (occup)			$1.11 \times 10^3$	<sup>3</sup> (CEL-lung)	Snihs (1973)
$\operatorname{Human}^{26}$ $\operatorname{Human}^{27}$	>2-30 yr (res) (occup) >1 mon-18 vr	Cancer Cancer Cancer		$\begin{array}{c} 5.55 \times 10 \\ 8.88 \times 10^3 \\ 3.70 \times 10^3 \end{array}$	$5.53 \times 10^{\circ}$ (CEL-1ung) $8.88 \times 10^{3}$ (CEL-1ung) $3.70 \times 10^{3}$ (CEL-1ung)	Svensson et al. (1989)  Fox et al. (1981)  Waxweiler et al. (1981)
Human <sup>28</sup>	(occup) >1 mon-30 yr	Cancer		$1.48 \times 10^4$	$1.48 \times 10^4 \text{ (CEL-lung)}$	Roscoe et al. (1989)
	(occnb)					
CEL = Cancer Effect Level						

CEL = Cancer Effect Level hemato = hematological resp = respiratory occup = occupational dec = decreased bw = body weight res = residential

(1.5 rem) (Hyland and Gunn, 1994). This total annual effective dose of 50 mSv yr<sup>-1</sup> is also applicable in some instances in the United States (U.S. Navy, 2001, p. 4-1 and NRC, 2005, p. 5), although 20 mSv  $yr^{-1}$  (4 WLM  $yr^{-1}$ ) is generally the accepted level (OSHA, 1988, 41 CFR §57.5038) while the NCRP (1993, pp. 34–35) has suggested more flexibility to control worker exposure. However, lowradiation doses are considered to range from near 0 to 100 mGy<sup>6</sup> (0 to 10 rad), medium doses from 100 mGy to 1 Gy (10 to 100 rad), and high doses from 1 Gy to 20-60 Gy (100 to 2,000–6,000 rad) (NRC, 2005, p. 374).

By this definition it would appear that an action level based on an effective dose of 15 mSv may be overly protective. The human equivalent dose  $H_T$  is estimated using Equation (1)

$$H_T = \sum_R W_R D_{T,R} \tag{1}$$

and the effective dose  $E_D$  is estimated from

$$E_D = \sum_{T} W_{T_i} \sum_{R} W_R D_{T,R}$$
 (2)

Using  $W_R = 20$  for  $\alpha$ -particles (ICRP, 1980, p. 94) results in an absorbed dose  $D_{T,R}$  of 0.75 mGy, which is at the lower spectrum of a low-radiation dose. Using  $W_{T_L} = 0.12$ for the lung (0.24 for lungs) (ICRP, 1991, p. 68) results in 6.25 mGy which is still at the lower spectrum of a lowradiation dose.

### <sup>222</sup>Rn Progeny

Although it is true that <sup>222</sup>Rn represents a risk to cavers in terms of lung cancer, its relatively long half life (3.824 d) will more often result in the exhalation of <sup>222</sup>Rn prior to emanation of an α-particle that could penetrate the epithelium of the lung to cause a cancerous growth. So even though the energy associated with the emission of an  $\alpha$ -particle from <sup>222</sup>Rn is relatively high (5.49 MeV) and it is possible that α-particle emission from inhaled <sup>222</sup>Rn gas may have an adverse affect on human health, it is not likely that α emission will actually occur during the time that the <sup>222</sup>Rn gas resides in the lung. This situation is considerably

different for <sup>222</sup>Rn progeny.

The four <sup>222</sup>Rn progeny (<sup>218</sup>Po, <sup>214</sup>Pb, <sup>214</sup>Bi, and <sup>214</sup>Po) are either metals (<sup>214</sup>Pb and <sup>214</sup>Bi) or metalloids (<sup>218</sup>Po and <sup>214</sup>Po) that are relatively short-lived and emit  $\alpha$ -particles with relatively high energy and β-particles with relatively low energy (Fig. 1). It is these features, principally the  $\alpha$ particles, that represent the main risk posed by <sup>222</sup>Rn. Each of the four principal <sup>222</sup>Rn progeny are quite reactive, which causes them to plate-out<sup>7</sup> in the lung as well as enhancing their tendency to adsorb to smoke and dust particles. The risk of lung cancer occurrence is exacerbated by smoke and dust particles because these metals and metalloids readily react with and adsorb to the particles which are easily inhaled.

#### Threats from <sup>218</sup>Po

Polonium-218 has a half life of 3.05 min and is the immediate progeny resulting from the decay of <sup>222</sup>Rn. When  $^{218}$ Po decays, it emits an  $\alpha$ -particle with a relatively high energy of 6.12 MeV. With a half life of 3.05 min it is possible that <sup>218</sup>Po will emit an α-particle during the time that it resides in the lung. However, its relatively short half life tends to prevent its being easily distributed throughout the body from the lungs.

Threats from <sup>214</sup>Pb and <sup>214</sup>Bi Lead-214 and <sup>214</sup>Bi are metals with half lives of 26.8 min and 19.7 min, respectively. These two radioisotopes decay by low energy \beta emission, but are still a threat to human health, although less so than the other short-lived <sup>222</sup>Rn progeny that decay by α emission (Fig. 1). Their relatively longer half lives and low energy relegate <sup>214</sup>Pb and <sup>214</sup>Bi to a slightly lesser threat status. In addition, their half lives are still too short to allow for substantial distribution throughout the body.

#### Threats from <sup>214</sup>Po

Polonium-214 has a very short half life (164 µs). It emits an α-particle with a high energy of 7.69 MeV. With a half life of just 164  $\mu$ s it is highly likely that <sup>214</sup>Po will emit an  $\alpha$ particle during the time that it resides in the lung. Its very short half life and high energy makes <sup>214</sup>Po a significant threat to human health. Although the very short half life of <sup>214</sup>Po prevents its distribution throughout the body, the relatively long half life of its immediate progeny <sup>210</sup>Pb (22.3 vr) can result in serious harm in parts of the body other than the lungs from the decay of <sup>210</sup>Pb.

Polonium radionuclides have many of the characteristics of rare-earth elements, are amphoteric, and tend to form hydroxides and radiocolloids in vitro<sup>8</sup> and in vivo<sup>9</sup>. The latter tends to cause polonium to become phagocytized<sup>10</sup> by cells of the reticuloendothelial system<sup>11</sup> for eventual deposition in the spleen, lymph nodes, bone marrow, liver, and kidneys after parenteral administration<sup>12</sup> (NRC, 1988, p. 161). Fortunately, the half lives of the polonium radionuclides in the immediate <sup>222</sup>Rn-decay series are of such a short duration ( $T_{1/2}$  for  $^{218}$ Po = 3.05 min and  $T_{1/2}$  for  $^{214}$ Po = 164  $\mu$ s) these problems are generally not a major concern. However,  $^{210}$ Pb with its much longer half life ( $T_{\frac{1}{2}} = 22.3 \text{ yr}$ ) is of concern.

#### HEALTH EFFECT ESTIMATES FROM EXPOSURES TO <sup>222</sup>RN AND ITS PROGENY

Inhalation exposure to significant levels of <sup>222</sup>Rn and its progeny (assumed to be in equilibrium) have been shown to cause acute and chronic effects on laboratory animals and humans (Table 2 and Fig. 3) (ATSDR, 1990, p. 12-27). However, the processes linking inhalation of <sup>222</sup>Rn and its progeny to increased lung cancer risk are complex (ICRP, 1994a, p. 2) primarily because of the numerous confound-

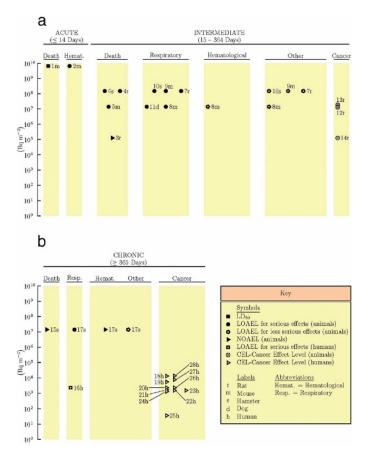


Figure 3. Levels of significant inhalation exposure to  $^{222}$ Rn (modified from ATSDR, 1990, pp. 16–17). Numbers next to each data point correspond to superscripts for each entry in Table 2. Acute and Intermediate effects = a and chronic effects = b.

ing factors<sup>13</sup> (ICRP, 1994a, p. 7) (e.g., smoking). For this reason, many of the epidemiological studies on miners and animals are inadequate so that health research continues.

#### REVIEW OF SELECTED ANIMAL AND HUMAN STUDIES

The  $^{222}$ Rn concentrations used in the studies cited by ATSDR (1990, p. 12–27) ranged from a low of 56 Bq m<sup>-3</sup> (human studies) to a high of  $8.14 \times 10^9$  Bq m<sup>-3</sup> (animal studies). In the human studies, a cancer effect level in lungs was identified (actual exposure frequency/duration was from >2–30 yr). In the animal studies, mouse mortality and development of hematological (anemia) symptoms occurred after a 30 d Median Lethal Dose (LD<sub>50</sub>) study (actual exposure frequency/duration was from 5–40 h).

#### Animal Studies

The mouse studies obviously involved much higher doses of <sup>222</sup>Rn than would typically be experienced by a caver (see Table 1), but the exposure time would be comparable. The human study and other similar studies cited by ATSDR (1990) include <sup>222</sup>Rn concentrations that

a typical caver may be exposed to, but the examined exposure times are generally longer than would be typical for a caver (an exception can be made for tour guides, maintenance workers, etc.).

The animal studies listed in Table 2 resulted in few lung cancers (21% in dogs, zero in mice, and 1.3% in Syrian hamsters) even though the  $^{222}\rm{Rn}$  doses to which the animals were exposed were extremely high (NRC, 1999, p. 43). Syrian hamsters did not develop any tumors at exposures below 3.89  $\times$  10 $^5$  J s m $^{-3}$  (3.0  $\times$  10 $^4$  WLM) whereas rats showed a high incidence of respiratory-tract tumors after exposure to  $^{222}\rm{Rn}$ . However, according to NRC (1999, p. 43–44) the mechanistic bases of these interspecies differences are such that species-to-species extrapolations of absolute risk cannot be used. As a result, direct extrapolation of animal data to humans cannot be used to predict absolute risk.

#### Human Studies

Epidemiological studies on the effects of <sup>222</sup>Rn gas and its progeny on human health consist primarily of studies on <sup>238</sup>U and phosphate miners (<sup>238</sup>U is associated with phosphate deposits). The human studies, except for the Svensson et al. (1989) study (number 25 in Table 2 and Figure 3), mostly tend to cluster in the cancer region for <sup>222</sup>Rn concentrations around 1,000 to 10,000 Bq m<sup>-3</sup> (Figure 3). These epidemiological studies of cohorts of miners confirm that long-term exposure to high levels of <sup>222</sup>Rn gas and its progeny represent a very serious threat to human health.

One human study (Svensson et al., 1989) while suggesting a clear link between <sup>222</sup>Rn and small cell carcinoma in the lung, also notes that cancers were less prevalent in the rural cohort over the urbanized cohort where ambient air pollution was a positive confounder. This discrepancy is regarded by the authors as a serious flaw in the study. Additionally, according to Snihs (1973) no conclusions regarding dose and effect below 50 mSv (5 rem) may be drawn because of the large uncertainties and statistical errors. This suggests that the risks to cavers and cave workers from exposure to <sup>222</sup>Rn in caves may not be overly significant.

## Exposure of Cavers and Cave Workers to $^{222}$ Rn and its Progeny

The formation of <sup>222</sup>Rn and its progeny is shown by the decay sequence in Figure 1. Radon-222 readily migrates to areas with a negative air space, such as caves and tunnels. It is also soluble in water and will reside in cave waters and atmospheres in equilibrium (Fig. 4). In addition, the <sup>222</sup>Rn parent, <sup>226</sup>Ra, will react with and precipitate on cave walls as RaCO<sub>3</sub> and thus provides a continuous source of <sup>222</sup>Rn. The net result is that <sup>222</sup>Rn concentrations in caves are considerably higher than typically occur in above ground residences, but are significantly less than those found in <sup>238</sup>U mines.

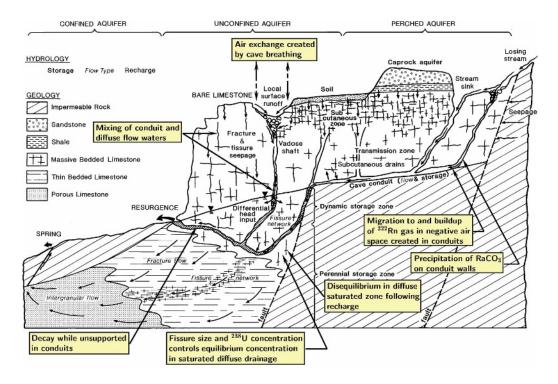


Figure 4. Factors controlling the ingrowth and decay of <sup>222</sup>Rn equilibrium activities in a cave system (modified from Smart and Friederich, 1986; Smart, 1991). Radon-222 ingrowth and decay processes in karst aquifer waters provided by Peter Smart (*pers. comm.*). Other important factors include dilution and volatilization. Cave breathing effects on <sup>222</sup>Rn concentrations are described in Cunningham and LaRock (1991); Yarborough and Meyers (1978, pp. 22–42. Precipitation of RaCO<sub>3</sub> on conduit walls is described in Field (1994, pp. 59–61). Radon-222 secular equilibrium is established after 26 days.

#### CUMULATIVE EXPOSURE

The decay of  $^{222}$ Rn to its progeny results in secular equilibrium, provided none of its progeny plate out (i.e., adsorb to cave walls). Cumulative exposure  $C_E$  has historically been calculated in terms of working levels (WL) with 170 h for a working level month (WLM) and is calculated (in SI units of J h m $^{-3}$ ) using (NRC, 1999, p. 178)

$$C_E = \sum_{i=1}^n \left( \overline{C}_{Rn} \right)_i \frac{t_i}{170} \tag{3}$$

where  $(\overline{C}R_n)_i$  is the average concentration of <sup>222</sup>Rn decay products during an exposure interval expressed in J m<sup>-3</sup> and  $t_i$  is the number of hours of the exposure.

The significance to cavers of Equation (3) is the hours of exposure. According to NRC (1999, p. 178), the cumulative exposure for individuals who continuously occupy a residence (commonly known as shut-ins) at a given decay product concentration is greater than four times that for an occupational exposure (8,766 h compared to 2,000 h worked on an annual basis). This means that for cave tour guides who work no more than 170 hours per month will be exposed to one quarter that of individuals who do not leave their dwelling. For recreational caving, exposure will generally be considerably less.

The net result is that individuals living in above ground dwellings, but are not necessarily shut-ins, are annually exposed to 4.8 mSv of <sup>222</sup>Rn, as compared to coal and metal miners who are annually exposed to just 0.7 and 2.7 mSv of <sup>222</sup>Rn, respectively (Wrixon et al., 2004, p. 40). For cavers and cave workers, radiation doses are likely to be much less because, although <sup>222</sup>Rn concentrations in caves are likely to be similar to that of coal mines, they will be lower than in <sup>238</sup>U mines while exposure times will typically be much less than that of an occupant of a dwelling.

#### Comparative Dosimetry

The activity of the  $^{222}$ Rn decay products is described by the Potential Alpha-Energy Concentration (PAEC) which is a non-equilibrium mixture. The PAEC is obtained from the potential alpha energy per unit of activity (Bq) of the considered radionuclide according to  $\varepsilon_p/\lambda_r = (\varepsilon_p T_{1/2}/\ln 2)$  (ICRP, 1994a, p. 3). The total airborne PAEC may be obtained from (ICRP, 1994a, p. 4)

$$C_p = \sum_{i} C_i \left( \frac{\varepsilon_{p,i}}{\lambda_{r,i}} \right) \tag{4}$$

where values for  $\varepsilon_p$  and  $\lambda_r$  are listed in Table 3. The half life of <sup>214</sup>Po is so short ( $T_{\frac{1}{2}} = 164 \,\mu s$ ; see Fig. 1) that for all practical purposes it is always in equilibrium with its parent

		Potential α-Energy							
	Half-Life	per A	Atom	per Unit of	f Activity, $\varepsilon_p$				
Radionuclide	(min)	(MeV)	$(10^{-12} \text{ J})$	(MeV Bq <sup>-1</sup> )	$(10^{-10} \text{ J Bq}^{-1})$				
<sup>222</sup> Rn Progeny									
<sup>218</sup> Po	3.05	13.69	2.19	3,615	5.79				
<sup>214</sup> Pb	26.8	7.69	1.23	17,840	28.6				
<sup>214</sup> Bi	19.9	7.69	1.23	13,250	21.2				
<sup>214</sup> Po	$2.73 \times 10^{-6}$	7.69	1.23	$2.0 \times 10^{-3}$	$3.0 \times 10^{-6}$				
Total (at equilibrium), per Bq or <sup>222</sup> Rn				34,710	55.6				

Table 3. Potential α-energy per atom and per unit activity (modified from ICRP, 1994a, p. 3).

<sup>214</sup>Bi and is not needed in the decay chain calculations (NRC, 1999, p. 137). The Potential Alpha-Energy Exposure (PAEE) may then be calculated from (ICRP, 1994a, p. 4)

$$P_p(t) = \int_0^t C_p(t) \tag{5}$$

where time *t* is expressed as the amount of time an individual is exposed (e.g., one week, one month, etc.).

The equilibrium factor F is defined as the ratio of  $^{222}$ Rn decay product concentration to that of  $^{222}$ Rn and is given by (Hopke et al., 1995)

$$F = \frac{1.18 \times 10^8 \ C_p}{C_{R_R}} \tag{6}$$

where the value of F ranges from 0.2 to 0.8, but typically ranges from 0.35 to 0.40. A default indoor value of 0.4 is recommended by ICRP (1994a, p. 20). However, because of the difficulty of estimating <sup>222</sup>Rn decay product concentrations in caves which range from 0.04 to 0.95, a mean value of 0.5 is usually assigned to F for cave studies (see for example, Hyland and Gunn, 1994) although a strong basis for this contention has not been reported. Aley et al. (2006) suggested that, for some notable exceptions, F for most show caves probably ranges between 0.5 and 1.0, although a strong basis for this contention was not supported in this instance either.

Using Equation (6) it is possible to calculate the actual Equilibrium Equivalent Exposure (EEQ) from (ICRP, 1994, P. 4)

$$P_{eq}(t) = \int_0^t C_{eq}(t) \tag{7}$$

where

$$C_{ea} = F C_{Rn} \tag{8}$$

The EEQ is a measure of the exposure to <sup>222</sup>Rn and its progeny that an individual receives for a given <sup>222</sup>Rn concentration. It is the EEQ that determines how seriously an individual has been exposed to a given concentration of <sup>222</sup>Rn and its progeny for a given period of time.

#### RECOMMENDED <sup>222</sup>RN EXPOSURES

Allowable exposures to cave workers to PAEC have varied over the years as cancer risks have become better understood. Initial U.S. Government regulations were first set in 1976, but were later revised.

#### 1976 Recommendations

In 1976, the National Institute of Occupational Safety and Health (NIOSH) recognized that <sup>222</sup>Rn progeny at several caves managed by the National park Service (NPS) were near the occupational limits as set forth in Occupational Safety and Health Administration (OSHA) standards for <sup>238</sup>U miners. NPS caves in which the PAEC exceeded 6.24 µJ m<sup>-3</sup> (0.30 WL) include Carlsbad Caverns National Park, N.M., Lehman Caves National Monument, Nev., Mammoth Cave National Park, Ky., Oregon Caves National Park, Ore., and Round Spring Cave in Ozark National Scenic Riverways, Mo. Additionally, the PAEC inside the caves and above ground buildings cooled by cave air at Mammoth Cave were 12.48 µJ m<sup>-3</sup> (0.60 WL) (Baier, 1976). Specific recommendations by NIOSH are shown in Table 4.

#### Current Recommendations

Current recommended regulations regarding exposures of workers to  $^{222}$ Rn are listed in Tables 5 and 6. The recommendations listed in Table 5 are intended to be conservatively protective. These levels are applicable to cave workers (e.g., tour guides), but are overly restrictive for infrequent cave explorers. According to Strom et al., (1996, p. 5) (citing NCRP, 1993, p. 49) effective dose in the workplace should not exceed 5 cSv (5 rem) in any one year with  $A_{ge} \times 1$  cSv as a lifetime limit. If the ICRP (1994a) recommendations are applied, then the the NCRP recommendations convert to 5 cSv (5 rem) in any one year with  $A_{ge} \times 7.08$  mJ h m $^{-3}$  or  $A_{ge} \times 1$  cSv as a lifetime limit.

Regulations specific to cave workers (and miners) are shown in Table 6. The cave worker regulations were developed and published by OSHA (OSHA, 1988) and Mine Safety and Health Administration (MSHA) (MSHA, 1989). OSHA sets an individual exposure limit equal to  $14.0 \text{ mJ h m}^{-3} = 20.0 \text{ mSy yr}^{-1} (4.0 \text{ WLM yr}^{-1}) (\text{OSHA},$ 

Table 4. Recommended regulations by NIOSH for exposure of cavers to <sup>222</sup>Rn decay progeny in 1976 (modified from Baier, 1976).

	PAEC Level	
$(\mu J m^{-3})$	(WL)	Recommended Regulation
>2.08	>0.1	All-underground smoking stopped
2.08-4.16	0.1 - 0.2	Monitor workspace at least quarterly
4.16 - 6.24	0.2-0.3	Monitor workspace quarterly
>6.24	>0.3	Monitor workspace weekly and maintain exposure records on all exposed employees
20.80-41.60	1.0-2.0	Immediate corrective action to lower PAEC below 20.80 μJ m <sup>-3</sup> (1.0 WL)
>41.60	>2.0	Withdraw all workers not necessary to lower PAEC below 20.80 $\mu J \ m^{-3}$ (1.0 WL)

Cumulative individual exposure shall not exceed 14.0 mJ h m<sup>-3</sup> yr<sup>-1</sup> (4.0 WLM yr<sup>-1</sup>).

1988, 41 CFR \$57.5038). MSHA also sets a maximum cumulative dose equal to 14.0 mJ h m<sup>-3</sup> = 20.0 mSv yr<sup>-1</sup>  $(4.0 \text{ WLM yr}^{-1})$  (MSHA, 1989, 30 CFR Part 57). However, ICRP65 was a little more specific in that it set a recommended effective dose at  $14.0 \text{ mJ} \text{ h} \text{ m}^{-3} =$  $20.0 \text{ mSv yr}^{-1}$  (4.0 WLM yr<sup>-1</sup>) averaged over five years and 35.0 mJ h m<sup>-3</sup> = 50.0 mSv yr<sup>-1</sup> (10.0 WLM yr<sup>-1</sup>) in any single year (ICRP, 1994a, p. 21).

Aley et al. (2006) lays out a strategy for reducing total α radiation exposures of show-cave employees to As Low As Reasonably Achievable (ALARA<sup>14</sup>) levels. Although not yet approved in 2006, it is likely that some form of the strategy will be approved by the National Caves Association in which each member will be required to develop a Cave Radiation Management Plan following guidelines developed by OSHA.

#### DETERMINING <sup>222</sup>RN RISKS TO CAVERS

The risks posed by exposure to elevated levels of <sup>222</sup>Rn gas have not adequately addressed exposures to recreational cavers and cave workers. Regulations not specific to

caves have been promulgated (e.g., MSHA, 2005, \$57.5037–\$57.5046) while regulations specific to caves have been developed (NPS, 1980) and are being updated (NPS, 2005). These regulations generally specify acceptable Working Levels for individuals, but not exposure rates, absorbed doses, or effective doses which are necessary for determining risks. However, because human health effects caused by elevated <sup>222</sup>Rn concentrations are based on epidemiological studies of miners subjected to much higher <sup>222</sup>Rn exposures (concentrations and times) as well as confounding factors (smoking, dust, etc.), risk estimates for cavers and cave workers need to be established using the linear no-threshold theory (LNT) even though the associated uncertainty in the cancer risk per unit dose at low dose and dose rate is difficult to quantify (Eckerman et al., 1999, p. 11–12).

#### Application of the LNT to <sup>222</sup>Rn and its Progeny

The LNT for radiation carcinogenesis is based on the concept that all radiation doses, no matter how small, can cause cancer (i.e., there is no acceptable radiation threshold at which cancers will not be initiated). According to this

Table 5. Recommended regulations by DOE for exposure to <sup>222</sup>Rn-decay progeny in 1996 (modified from Strom et al., 1996, p. 6).

	PAE	EC		PAEE	
Country <sup>a</sup>	$(\mu \ J \ m^{-3})$	(WL)	$(mJ m^{-3} yr^{-1})$	(mSv yr <sup>-1</sup> )	(WLM yr <sup>-1</sup> )
United States	6.93	1/3	14.0	20.0	4.0
Canada, France, Great Britain	8.32	0.4	16.8	24.0	4.8
			17.5	25.0	5.0 <sup>b</sup>
			35.1	50.0	$10.0^{\rm c}$
			35.1	50.0	10.0 <sup>d</sup>

a The United States values are based on U.S. Environmental Protection Agency (EPA), U.S. Department of Energy (DOE), and U.S. Nuclear Regulatory Commission (NRC)

Values are for any single year —  $A_{ge} \times 3.54$  mJ h m<sup>-3</sup> ( $A_{ge} \times 1$  WLM).

<sup>&</sup>lt;sup>c</sup> Values are for any single year —  $A_{ge} \times 7.08$  mJ h m<sup>-3</sup> ( $A_{ge} \times 2$  WLM). <sup>d</sup> Values are for any single year —14.0 mJ h m<sup>-3</sup> yr<sup>-1</sup> (4.0 WLM yr<sup>-1</sup>) averaged over 5 yr.

Table 6. Published regulations by OSHA and MSHA for exposure of cavers to <sup>222</sup>Rn-decay progeny in 1976 (modified from ATSDR, 1990, p. 93–94).

PAEC	Level	PAEE	Level		
$(\mu J m^{-3})$	(WL)	(mSv yr <sup>-1</sup> )	(WLM yr <sup>-1</sup> )	Published Regulations	Reference
•••	•••	20.0	4.0	Individual exposure limit	OSHA <sup>a</sup>
2.08	0.1	•••	•••	Monitor workspace at least once yearly	$OSHA^b$
2.08-6.24	0.1 - 0.3	•••	•••	Monitor workspace quarterly	$OSHA^{c}$
>6.24	>0.3	•••	•••	Monitor workspace weekly and maintain exposure records on all exposed employees	OSHA <sup>d</sup>
20.80	1.0	•••		Immediate corrective action to lower PAEC	OSHA <sup>e</sup>
20.80	1.0	•••		Instantaneous maximum	$MSHA^{\mathrm{f}}$
		20.0	4.0	Maximal cumulative dose	$MSHA^f$

a (OSHA, 1988, 41 CFR §57.5038).

theory then, if exposure to 1 Gy (100 rad) causes a cancer risk R, the risk from exposure to  $10^{-2}$  Gy (1 rad) is R/100, the risk from exposure to  $10^{-5}$  Gy (1 mrad) is  $R/10^{5}$ , and so on, which means that only a zero radiation dose will result in a zero risk of cancer (Cohen, 2002).

## CANCER RISK MODELING FOR EXPOSURE TO <sup>222</sup>RN AND ITS PROGENY

Models intended to address cancer risks from exposure to <sup>222</sup>Rn and its progeny have evolved over the years, although all have followed the LNT (Yu et al., 2006). The most current model was developed and published in ICRP66 (ICRP, 1994b).

## Human Respiratory Tract Model for Effective Dose Estimation

Using the program Lungdose.F90 (Nikezic and Yu, 2001) which was developed according to the Human Respiratory Tract Model (HRTM), an estimated equilibrium factor F equal to 0.366 was obtained, which closely matches the ICRP65 recommended value (F = 0.4) (ICRP, 1994a, p. 5) and the BEIR VI arithmetic average value of 0.408 (James et al., 2003). An average inhalation rate  $I_h =$  $2.16 \times 10^4 \,\mathrm{m}^3 \,\mathrm{s}^{-1}$  in a residence resulted in an estimated thoracic dose  $D_T$  of 79.20 nSv (Bq h m<sup>-3</sup>)<sup>-1</sup> (126.112 mSv WLM<sup>-1</sup>). Most interestingly, Lungdose.F90 resulted in an estimate for a Dose Conversion Factor (DCF) equal to 9.50 nSv (Bq h m<sup>-3</sup>)<sup>-1</sup> (15.13 mSv WLM<sup>-1</sup>) which is considerably larger than the epidemiological estimate for a DCF equal to 3.18 nSv (Bq h m<sup>-3</sup>)<sup>-1</sup> (5.06 mSv WLM<sup>-1</sup>) for workers and 2.44 nSv (Bq h  $m^{-3}$ )<sup>-1</sup> (3.88 mSv WLM<sup>-1</sup>) for the public (ICRP, 1994a, p. 13). This discrepancy emphasizes the complexities and uncertainties when calculating risks posed by exposure to PAEC (Gourmelon et al., 2005, p. 19).

The difference between the Lungdose.F90 estimate for a DCF equal to  $9.50 \text{ nSv} (\text{Bq h m}^{-3})^{-1} (15.13 \text{ mSv})$ WLM<sup>-1</sup>) and the ICRP65 estimate for a DCF equal to  $3.18 \text{ nSv (Bq h m}^{-3})$  (5.06 mSv WLM<sup>-1</sup>) is not regarded as significantly large because of the complex physical and biological issues involved and reasonably matches a previously epidemiologically-estimated value of 9.0 nSv (Bq h m<sup>-3</sup>) <sup>-1</sup> (14.33 mSv WLM<sup>-1</sup>) (UNSCEAR, 2000, p. 107). An evaluation of the discrepancy has resulted in the suggestion that the epidemiologically-based estimate for DCF will need to be increased and that for now, the larger DCF value estimated dosimetrically using the HRTM is recommended for use in risk calculations (UNSCEAR, 2000, p. 107). However, others feel that the epidemiologically-based estimates are more scientifically sound (Neal Nelson, pers. comm.).

Using the values estimated from Lungdose.F90 developed by Nikezic and Yu (2001) with  $I_h = 3.33 \times 10^4$  m<sup>3</sup> s<sup>-1</sup> to account for a combination of resting, light and heavy exercise (James et al., 2003) typical of caving, the <sup>222</sup>Rn concentrations listed in Table 1 and Equation (9) (Wiegand et al., 1995)

$$E_{DA} = C_{Rn} F T_i D_{CF} (9)$$

produced the annual effective doses  $E_{DA}$  for <sup>222</sup>Rn exposures to recreational cavers (50 h yr<sup>-1</sup>), professional cavers (600 h yr<sup>-1</sup>), part-time cave workers (1,760 h yr<sup>-1</sup>), and full-time cave workers (2,000 h yr<sup>-1</sup>) (Table 7). Realistically, there is no reliable way to estimate the average number of hours experienced by recreational cavers, professional cavers, and part-time cave workers. The number of caving hours per year for cavers (50 h yr<sup>-1</sup> and 600 h yr<sup>-1</sup>) are considered reasonable estimates. Part-time cave worker hours equal to 1,760 h yr<sup>-1</sup> was used as an estimate because this value is recommended for

<sup>&</sup>lt;sup>b</sup> (OSHA, 1988, 41 CFR \$57.5087).

c (OSHA, 1988, 41 CFR §57.5037).

d (OSHA, 1988, 41 CFR \$57.5037).

e (OSHA, 1988, 41 CFR §57.5041).

f (MSHA, 1988, 30 CFR Part 57).

Table 7. Estimated annual effective doses using the Lungdose.F% program<sup>a</sup> for exposures to cavers and cave workers for mean <sup>222</sup>Rn concentrations listed in Table 1. Superscript numbers next to each entry correspond to the x-axis on Figures 5–8. Entries without a superscript were not plotted.

	Recreation	nal Caver <sup>b</sup>	Profession	nal Caver <sup>c</sup>	Part-Time C	ave Worker <sup>d</sup>	Full-Time (	Cave Worker <sup>e</sup>
Country	(mSv yr <sup>-1</sup> )	(WLM yr <sup>-1</sup> )	(mSv yr <sup>-1</sup> )	(WLM yr <sup>-1</sup> )	(mSv yr <sup>-1</sup> )	(WLM yr <sup>-1</sup> )	(mSv yr <sup>-1</sup> )	(WLM yr <sup>-1</sup> )
<sup>1</sup> Australia	0.14	0.03	1.73	0.35	5.07	1.01	5.77	1.15
<sup>2</sup> China <sup>f</sup>	0.03	0.01	0.40	0.08	1.17	0.23	1.33	0.27
<sup>3</sup> Czech Republic	0.29	0.06	3.50	0.70	10.27	2.05	11.67	2.33
<sup>4</sup> Great Britain	0.69	0.14	8.24	1.65	24.18	4.84	27.48	5.50
Great Britain								
<sup>5</sup> Great Britain	8.48	1.70	101.77	20.35	298.53	59.71	339.24	67.85
<sup>6</sup> Great Britain	2.20	0.44	26.39	5.28	77.41	15.48	87.96	17.59
<sup>7</sup> Great Britain	0.09	0.02	1.04	0.21	3.04	0.61	3.45	0.69
<sup>8</sup> Great Britain	0.07	0.01	0.89	0.18	2.62	0.52	2.98	0.60
<sup>9</sup> Greece	5.95	1.19	71.40	14.28	209.44	41.89	238.00	47.60
<sup>10</sup> Hungary	0.78	0.16	9.36	1.87	27.45	5.49	31.19	6.24
<sup>11</sup> Hungary	0.58	0.12	7.00	1.40	20.53	4.11	23.33	4.67
<sup>12</sup> Ireland	0.98	0.20	11.70	2.34	34.33	6.87	39.01	7.80
<sup>13</sup> Japan	< 0.01	< 0.01	0.03	0.01	0.09	0.02	0.10	0.02
<sup>14</sup> Malaysia	0.14	0.03	1.69	0.34	4.96	0.99	5.63	1.13
15Poland	0.28	0.06	3.31	0.66	9.70	1.94	11.02	2.20
<sup>16</sup> Russia	0.56	0.11	6.78	1.36	19.88	3.98	22.59	4.52
<sup>17</sup> Slovenia	0.33	0.07	4.00	0.80	11.75	2.35	13.35	2.67
<sup>18</sup> Slovenia	0.23	0.05	2.74	0.55	8.03	1.61	9.12	1.82
<sup>19</sup> Spain	0.03	0.01	0.31	0.06	0.90	0.18	1.02	0.20
<sup>20</sup> Spain	0.84	0.17	10.11	2.02	29.65	5.93	33.69	6.74
<sup>21</sup> South Africa	0.06	0.01	076	0.15	2.22	0.44	2.52	0.50
<sup>22</sup> Switzerland	5.91	1.18	70.89	14.18	207.95	41.59	236.31	47.26
<sup>23</sup> United States	0.46	0.09	5.46	1.09	16.03	3.21	18.21	3.64
<sup>24</sup> United States	0.61	0.12	7.34	1.47	21.54	4.31	24.47	4.89
<sup>25</sup> United States	0.35	0.07	4.18	0.84	12.27	2.45	13.94	2.79
United States								
<sup>26</sup> United States	2.76	0.55	33.12	6.62	97.14	19.43	110.38	22.08

a Lungdose. F90 program (Nikezik and Yu, 2001) estimate for DCF = 12.92 nSv (Bq h m<sup>-3</sup>)<sup>-1</sup> (20.75 mSv WLM<sup>-1</sup>) and  $D_T$  = 107.63 nSv (Bq h m<sup>-3</sup>)<sup>-1</sup> (171.38 mSv WLM<sup>-1</sup>) for  $I_h$  = 3.33 × 10<sup>-4</sup> m<sup>3</sup> s<sup>-1</sup>.

outside exposure by UNSCEAR (2000, p. 107), even though UNSCEAR recommended a larger equilibrium factor of (0.6) for external exposures, but which was not used in the calculations because it is not appropriate for caves.

An  $I_h = 3.33 \times 10^4 \text{ m}^3 \text{ s}^{-1}$  resulted in a greater  $D_T$  and a greater overall  $E_{DA}$  for cavers and cave workers than when an  $I_h = 2.16 \times 10^4 \text{ m}^3 \text{ s}^{-1}$  was used because of the much greater breathing activity. Mean annual effective doses  $E_{DA}$  listed in Table 7 typically ranged from much less  $(0.03 \text{ mSy yr}^{-1})$  to much greater  $(339.27 \text{ mSv yr}^{-1})$  than the recommended maximums of 20 to 50 mSv yr<sup>-1</sup>. Unfortunately, the great range of data and variability

evidenced make it very difficult to realistically estimate the risks posed to cavers and cave workers from the estimated  $E_{DA}$ . However, it appears from Table 7 that recreational cavers and, for the most part, professional cavers are likely to be only minimally exposed to excess <sup>222</sup>Rn concentrations whereas cave workers should be more concerned about exposure to excess <sup>222</sup>Rn concentrations for the five countries with high mean <sup>222</sup>Rn concentrations (Great Britain, Greece, Japan, Switzerland, and the United States) (Table 7).

Figures 5–8 illustrate the threat to cavers and cave workers from exposure to elevated levels of <sup>222</sup>Rn gas in caves relative to acceptable limits. From Figures 5–8 it is

<sup>&</sup>lt;sup>b</sup> Recreational cavers = 50 h yr<sup>-1</sup> of caving.

<sup>&</sup>lt;sup>c</sup> Professional cavers = 600 h yr<sup>-1</sup> of caving.

<sup>&</sup>lt;sup>d</sup> Part-time cave worker = 1,760 h yr<sup>-1</sup> of cave work.

<sup>&</sup>lt;sup>e</sup> Full-time cave worker = 2,000 h yr<sup>-1</sup> of cave work.

<sup>&</sup>lt;sup>f</sup> The measured <sup>222</sup>Rn concentrations for the China data listed in Table 1 are better represented by 7,000 h yr<sup>-1</sup> exposure with an  $I_h = 2.16 \times 10^{-4}$  m<sup>3</sup> s<sup>-1</sup> because these data are from cave dwellings resulting in an  $E_{DA} = 3.43$  mSv yr<sup>-1</sup> (0.69 WLM yr<sup>-1</sup>).

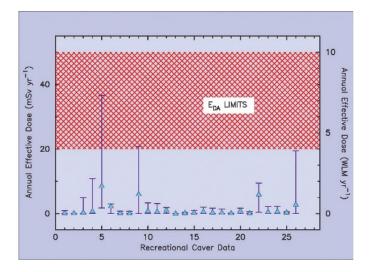


Figure 5. Plot of mean, minimum, and maximum annual effective dose values for recreational cavers from Table 7 relative to published acceptable limits of  $20-50~\text{mSv yr}^{-1}$  ( $4-10~\text{WLM yr}^{-1}$ ). The x-axis numerical values correspond to the superscript labels in Table 7. (Note that data sets listed in Table 7 missing mean values [Great Britain and United States] are not plotted).

apparent that cavers are generally not at risk while cave workers appear to be minimally at risk.

A series of notched boxplots (see Chambers et al., 1983, for a description of notched boxplots) using the data listed in Table 7 and shown in Figure 9 further demonstrate that only cave workers will be minimally impacted at the lower

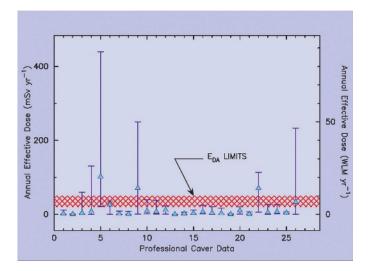


Figure 6. Plot of mean, minimum, and maximum annual effective dose values for professional cavers from Table 7 relative to published acceptable limits of 20–50 mSv yr-1 (4–10 WLM yr<sup>-1</sup>). The x-axis numerical values correspond to the superscript labels in Table 7. (Note that data sets listed in Table 7 missing mean values [Great Britain and United States] are not plotted).

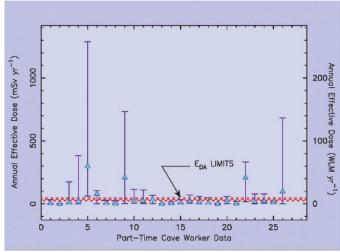


Figure 7. Plot of mean, minimum, and maximum annual effective dose values for part-time cave workers from Table 7 relative to published acceptable limits of 20–50 mSv yr<sup>-1</sup> (4–10 WLM yr<sup>-1</sup>). The x-axis numerical values correspond to the superscript labels in Table 7. (Note that data sets listed in Table 7 missing mean values [Great Britain and United States] are not plotted).

 $E_{DA}$  limit of 20 mSv yr<sup>-1</sup> (4 WLM yr<sup>-1</sup>). However, the median line of each notched boxplot for the part-time and full-time cave workers are also below the minimum acceptable limit for exposure, suggesting that neither the part-time nor the full-time cave workers are impacted at the lower  $E_{DA}$  limit, and only the the more extreme values

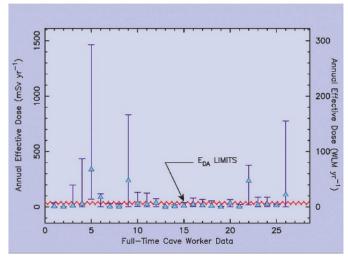


Figure 8. Plot of mean, minimum, and maximum annual effective dose values for full-time cave workers from Table 7 relative to published acceptable limits of 20–50 mSv yr<sup>-1</sup> (4–10 WLM yr<sup>-1</sup>). The x-axis numerical values correspond to the superscript labels in Table 7. (Note that data sets listed in Table 7 missing mean values [Great Britain and United States] are not plotted).

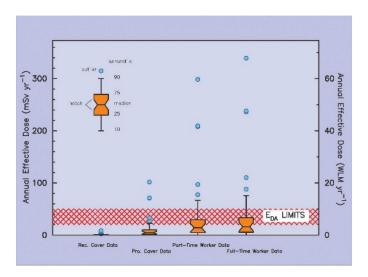


Figure 9. Notched boxplots of estimated annual effective doses relative to acceptable limits. (Note that data sets listed in Table 7 missing mean values [Great Britain and United States] are not plotted).

(i.e., >90th percentile) exceed the 50 mSv yr<sup>-1</sup> (10 WLM yr<sup>-1</sup>) limit.

**Exposure of Cavers to <sup>222</sup>Rn and its Progeny.** Exposure of cave workers and cavers to <sup>222</sup>Rn and its progeny is obtained from (Eckerman et al., 1999, p. F4)

$$P_{eq}(t) = C_i \int_0^t e^{(-\ln 2t)/T_{1/2}}$$
 (10)

which considers the decay series of <sup>222</sup>Rn through its progeny, of which only the principal progeny identified in Figure 1 are used in the calculations. Each progeny concentration is estimated from the concentration of its respective parent. According to the nuclear decay data listed in Eckerman et al. (1999, p. G19–G20), each radionuclide for the <sup>222</sup>Rn decay series decays to between 99.98% and 100% to its principal progeny (e.g., 99.98% of <sup>218</sup>Po decay results in <sup>214</sup>Pb) (Table 8), which renders insignificant the other progeny shown in Figure 1 (e.g., <sup>218</sup>AT and <sup>218</sup>Rn).

Applying Equation (10) allows for exposures to cavers from <sup>222</sup>Rn and its progeny to be estimated from

the  $^{222}$ Rn concentrations listed in Table 1. Estimated exposures are listed in Table 9, where it may be noted that the smallest estimated exposure is produced by  $^{214}$ Po. This appears contrary to the notion that the very short half life of  $^{214}$ Po ( $T_{\frac{1}{2}} = 164 \,\mu s$ ) will result in the greatest lung doses because  $^{214}$ Po will likely emit an  $\alpha$ -particle before it can be exhaled. Polonium-214 decays only once, but is in secular equilibrium with its parent radionuclide  $^{214}$ Bi. However,  $^{214}$ Po has the greatest number of decays per unit intake of  $^{222}$ Rn in equilibrium with its progeny. Its half life, therefore, is not a factor (N. Nelson, pers. comm.).

Risks to Cavers from Exposure to <sup>222</sup>Rn and its Progeny. Risks to cave workers and cavers need to be estimated from the exposures listed in Table 9 and risk coefficients determined from epidemiological studies when available. Risks include both mortality and morbidity.

The risks of mortality (cancer death) and morbidity (cancer with or without death) from exposure to <sup>222</sup>Rn and its progeny are estimated from (Eckerman et al., 1999, p. F-8)

$$R_{M_i} = P_{eq_i} S_C I_h M_{R_i} \tag{11}$$

where a scaling coefficient  $S_C$  of 1.11 for inhalation was considered appropriate (Eckerman et al., 1999, p. E-5) and the values for the mortality<sup>15</sup> and morbidity<sup>16</sup> risk coefficients  $M_{R_c}$  are listed in Table 10.

The EPA estimates mortality risks from exposure to  $^{222}$ Rn gas as a function of WLM; specifically, EPA expects  $5.4 \times 10^{-4}$  lung cancer deaths per WLM. This methodology was not used here because the WLM does not account for such factors as breathing rates, tidal volumes, or the fraction of progeny unattached to aerosols, which modify the relationship between exposure and risk (Cothern, 1987, p. 26).

Morbidity risks to cavers and cave workers can not be directly calculated because the relevant morbidity risk coefficients exist only for <sup>214</sup>Pb and <sup>214</sup>Bi (Table 10). The difference between the mortality and morbidity coefficients for <sup>214</sup>Pb and <sup>214</sup>Bi are relatively insignificant, but the same cannot be said for the other radionuclides listed in Table 10. In general, it is reasonable to expect that the risk of morbidity to cavers and cave workers will be somewhat greater than is the estimated risk for mortality.

In order to develop a rough estimate for the risk of morbidity for cavers and cave workers, the values for the

Table 8. Nuclear decay products and fractions for <sup>222</sup>Rn and its progeny (modified from Eckerman et al., 1999, p. G19-G20).

		Decay	Radioact	tive Decay Pro	oducts and Fraction	al Yield
Radionuclude	$T_{1\!/_{\!2}}$	Mode	Radionuclide	Fraction	Radionuclide	Fraction
<sup>222</sup> Rn <sup>218</sup> Po <sup>214</sup> Pb <sup>214</sup> Bi	3.8325 d 3.05 min 26.8 min 19.9 min	α α β- β- α β-	<sup>218</sup> Po <sup>214</sup> Pb <sup>214</sup> Bi <sup>214</sup> Po	1.0000 0.9998 1.0000 0.9998	 <sup>218</sup> At  <sup>210</sup> Tl	0.0002  0.0002
<sup>214</sup> Po	164.3 µs	αρ	<sup>210</sup> Pb	1.0000		

Table 9. Estimated time-integrated concentration exposures to <sup>222</sup>Rn and its progeny for cavers for the mean <sup>222</sup>Rn concentrations listed in Table 1<sup>a</sup>.

		In	halation Exposure		
	<sup>222</sup> Rn	<sup>218</sup> Po	<sup>214</sup> Pb	$Z^{14}$ Bi	<sup>214</sup> Po
Country	$(Bq s^{-1} m^{-3})$				
Australia	$4.82 \times 10^{7}$	$3.35 \times 10^4$	$3.35 \times 10^4$	$3.35 \times 10^4$	$5.58 \times 10^{-4}$
China	$1.11 \times 10^{7}$	$7.74 \times 10^{3}$	$7.74 \times 10^{3}$	$7.74 \times 10^{3}$	$1.29 \times 10^{-4}$
Czech Republic	$9.76 \times 10^{7}$	$6.78 \times 10^4$	$6.78 \times 10^4$	$6.78 \times 10^4$	$1.13 \times 10^{-3}$
Great Britain	$2.30 \times 10^{8}$	$1.60 \times 10^{5}$	$1.59 \times 10^{5}$	$1.59 \times 10^{5}$	$2.66 \times 10^{-3}$
Great Britain	•••	•••	•••		•••
Great Britain	$2.84 \times 10^{9}$	$1.97 \times 10^{6}$	$1.97 \times 10^{6}$	$1.97 \times 10^{6}$	$3.28 \times 10^{-2}$
Great Britain	$7.35 \times 10^{8}$	$5.11 \times 10^{5}$	$5.11 \times 10^{5}$	$5.11 \times 10^{5}$	$8.51 \times 10^{-3}$
Great Britain	$2.88 \times 10^{7}$	$2.00 \times 10^{4}$	$2.00 \times 10^4$	$2.00 \times 10^4$	$3.34 \times 10^{-4}$
Great Britain	$2.49 \times 10^{7}$	$1.73 \times 10^{4}$	$1.73 \times 10^{4}$	$1.73 \times 10^4$	$2.88 \times 10^{-4}$
Greece	$1.99 \times 10^{9}$	$1.38 \times 10^{6}$	$1.38 \times 10^{6}$	$1.38 \times 10^{6}$	$2.30 \times 10^{-2}$
Hungary	$2.61 \times 10^{8}$	$1.81 \times 10^{5}$	$1.81 \times 10^{5}$	$1.81 \times 10^{5}$	$3.02 \times 10^{-3}$
Hungary	$1.95 \times 10^{8}$	$1.35 \times 10^{5}$	$1.35 \times 10^{5}$	$1.35 \times 10^{5}$	$2.26 \times 10^{-3}$
Ireland	$3.26 \times 10^{8}$	$2.26 \times 10^{5}$	$2.26 \times 10^{5}$	$2.26 \times 10^{5}$	$3.77 \times 10^{-3}$
Japan	$8.69 \times 10^{5}$	$6.04 \times 10^{2}$	$6.04 \times 10^{2}$	$6.04 \times 10^{2}$	$1.01 \times 10^{-5}$
Malaysia	$4.71 \times 10^{7}$	$3.27 \times 10^{4}$	$3.27 \times 10^4$	$3.27 \times 10^4$	$5.45 \times 10^{-4}$
Poland	$9.21 \times 10^{7}$	$6.40 \times 10^4$	$6.40 \times 10^4$	$6.40 \times 10^4$	$1.07 \times 10^{-3}$
Russia	$1.89 \times 10^{8}$	$1.31 \times 10^{5}$	$1.31 \times 10^{5}$	$1.31 \times 10^{5}$	$2.19 \times 10^{-3}$
Slovenia	$1.12 \times 10^{8}$	$7.75 \times 10^4$	$7.75 \times 10^4$	$7.75 \times 10^4$	$1.29 \times 10^{-3}$
Slovenia	$7.63 \times 10^{7}$	$5.30 \times 10^4$	$5.29 \times 10^4$	$5.29 \times 10^4$	$8.82 \times 10^{-4}$
Spain	$8.53 \times 10^{6}$	$5.93 \times 10^{3}$	$5.93 \times 10^{3}$	$5.93 \times 10^{3}$	$9.87 \times 10^{-5}$
Spain	$2.82 \times 10^{8}$	$1.96 \times 10^{5}$	$1.96 \times 10^{5}$	$1.96 \times 10^{5}$	$3.26 \times 10^{-3}$
South Africa	$2.11 \times 10^{7}$	$1.47 \times 10^{4}$	$1.46 \times 10^{4}$	$1.46 \times 10^{4}$	$2.44 \times 10^{-4}$
Switzerland	$1.98 \times 10^{9}$	$1.37 \times 10^{6}$	$1.37 \times 10^{6}$	$1.37 \times 10^{6}$	$2.29 \times 10^{-2}$
United States	$1.52 \times 10^{8}$	$1.06 \times 10^{5}$	$1.06 \times 10^{5}$	$1.06 \times 10^{5}$	$1.76 \times 10^{-3}$
United States	$2.05 \times 10^{8}$	$1.42 \times 10^{5}$	$1.42 \times 10^{5}$	$1.42 \times 10^{5}$	$2.37 \times 10^{-3}$
United States	$1.17 \times 10^{8}$	$8.09 \times 10^{4}$	$8.09 \times 10^{4}$	$8.09 \times 10^4$	$1.35 \times 10^{-3}$
United States					
United States	$9.23 \times 10^{8}$	$6.41 \times 10^5$	$6.41 \times 10^5$	$6.41 \times 10^5$	$1.07 \times 10^{-2}$

<sup>&</sup>lt;sup>a</sup> Only the mean time-integrated concentration exposures are shown here. Maximum and minimum exposure values were calculated from the maximum and minimum <sup>222</sup>Rn concentrations listed in Table 1 but are not shown here due to space limitations.

morbidity risk coefficient  $M_{R_B}$  for  $^{214}{\rm Pb}$  and  $^{214}{\rm Bi}$  were taken from Table 10. For the other radionuclides, the mortality risk coefficients  $M_{R_T}$  listed in Table 10 were increased by a factor of 1.5 on the assumption that such an

Table 10. Mortality and morbidity risk coefficients for <sup>222</sup>Rn and its progeny.

ty Morbidity ) (Bq <sup>-1</sup> )
$0^{-11}$ $0^{-11}$ $0^{-10}$ $9.81 \times 10^{-10}$ $0^{-10}$ $7.84 \times 10^{-10}$
(

<sup>&</sup>lt;sup>a</sup> Risk coefficients source: Puskin and Nelson (1994, p. 53).

increase will reasonably represent a morbidity risk coefficient  $M_{R_B}$  for those radionuclides for which morbidity risk coefficients are not yet available.

Mortality and morbidity risks (Table 11) were averaged over all ages and both genders for a population with specified mortality and morbidity for the mean exposures listed in Table 9. The mean risks of mortality and morbidity ranged from  $10^{-5}$  (1 in 100,000) to  $10^{-7}$  (1 in 10,000,000) where  $10^{-6}$  (1 in 1,000,000) is usually considered an acceptable risk.

The significance of the mortality and morbidity inhalation risks posed to cavers and cave workers is shown in Figures 10 and 11. It will be noted from Figures 10 and 11 that the majority of the mean values are very close to the  $10^{-6}$  acceptable risk level, but the maximum measured risks appear considerably greater than the  $10^{-6}$  acceptable risk level. Only data sets represented by numbers 5 (Great Britain), 6 (Great Britain), 9 (Greece), 22 (Switzerland), and 26 (United States) exhibited mean values substantially greater than the  $10^{-6}$  acceptable risk level.

<sup>&</sup>lt;sup>b</sup> Risk coefficients source: Eckerman et al. (1999, p. 71).

<sup>&</sup>lt;sup>c</sup> Risk coefficients source: Eckerman et al. (1999, p. 72).

Table 11. Inhalation risks from <sup>222</sup>Rn and its progeny for exposures to cavers and cave workers for the <sup>222</sup>Rn concentrations listed in Table 1. Superscript numbers next to each entry corresponds to the x-axis on Figures 10 and 11. Entries without a superscript were not plotted.

	Inhal	ation Motality	Risk	Inhala	ation Morbidity	Risk
Country	Mean	Maximum	Minimum	Mean	Maximum	Minimum
<sup>1</sup> Australia	$5.9 \times 10^{-7}$	$3.9 \times 10^{-6}$	$8.8 \times 10^{-9}$	$8.8 \times 10^{-7}$	$5.9 \times 10^{-6}$	$1.3 \times 10^{-8}$
<sup>2</sup> China	$1.4 \times 10^{-7}$	$2.7 \times 10^{-7}$	$3.7 \times 10^{-8}$	$2.0 \times 10^{-7}$	$4.0 \times 10^{-7}$	$5.5 \times 10^{-8}$
<sup>3</sup> Czech Republic	$1.2 \times 10^{-6}$	$2.0 \times 10^{-5}$	$1.9 \times 10^{-7}$	$1.8 \times 10^{-6}$	$3.0 \times 10^{-5}$	$2.9 \times 10^{-7}$
<sup>4</sup> Great Britain	$2.8 \times 10^{-6}$	$4.5 \times 10^{-5}$	$9.7 \times 10^{-9}$	$4.2 \times 10^{-6}$	$6.7 \times 10^{-5}$	$1.4 \times 10^{-8}$
Great Britain	•••	$1.5 \times 10^{-4}$	$9.7 \times 10^{-8}$		$2.2 \times 10^{-4}$	$1.4 \times 10^{-7}$
<sup>5</sup> Great Britain	$3.5 \times 10^{-5}$	$1.5 \times 10^{-4}$	$7.2 \times 10^{-6}$	$5.2 \times 10^{-5}$	$2.2 \times 10^{-4}$	$1.1 \times 10^{-5}$
<sup>6</sup> Great Britain	$9.1 \times 10^{-6}$	$1.2 \times 10^{-5}$	$6.6 \times 10^{-8}$	$1.3 \times 10^{-5}$	$1.8 \times 10^{-5}$	$9.8 \times 10^{-8}$
<sup>7</sup> Great Britain	$3.6 \times 10^{-7}$	$3.1 \times 10^{-6}$	$2.5 \times 10^{-8}$	$5.3 \times 10^{-7}$	$4.6 \times 10^{-6}$	$3.8 \times 10^{-8}$
<sup>8</sup> Great Britain	$3.1 \times 10^{-7}$	$3.0 \times 10^{-6}$	$3.3 \times 10^{-8}$	$4.6 \times 10^{-7}$	$4.4 \times 10^{-6}$	$4.9 \times 10^{-8}$
<sup>9</sup> Greece	$2.5 \times 10^{-5}$	$8.6 \times 10^{-5}$	$1.8 \times 10^{-7}$	$3.6 \times 10^{-5}$	$1.3 \times 10^{-4}$	$2.7 \times 10^{-7}$
<sup>10</sup> Hungary	$3.2 \times 10^{-6}$	$1.4 \times 10^{-5}$	$4.9 \times 10^{-7}$	$4.8 \times 10^{-6}$	$2.0 \times 10^{-5}$	$7.2 \times 10^{-7}$
<sup>11</sup> Hungary	$2.4 \times 10^{-6}$	$1.3 \times 10^{-5}$	$1.9 \times 10^{-7}$	$3.6 \times 10^{-6}$	$1.9 \times 10^{-5}$	$2.9 \times 10^{-7}$
<sup>12</sup> Ireland	$4.0 \times 10^{-6}$	$7.7 \times 10^{-6}$	$1.9 \times 10^{-7}$	$6.0 \times 10^{-6}$	$1.1 \times 10^{-5}$	$2.9 \times 10^{-7}$
<sup>13</sup> Japan	$1.1 \times 10^{-5}$	$1.9 \times 10^{-8}$	$7.2 \times 10^{-10}$	$1.6 \times 10^{-8}$	$2.9 \times 10^{-8}$	$1.1 \times 10^{-9}$
<sup>14</sup> Malaysia	$5.8 \times 10^{-7}$	$1.9 \times 10^{-6}$	$9.7 \times 10^{-8}$	$8.6 \times 10^{-7}$	$2.9 \times 10^{-6}$	$1.4 \times 10^{-7}$
<sup>15</sup> Poland	$1.1 \times 10^{-6}$	$4.1 \times 10^{-6}$	$5.8 \times 10^{-8}$	$1.7 \times 10^{-6}$	$6.0 \times 10^{-6}$	$8.7 \times 10^{-8}$
<sup>16</sup> Russia	$2.3 \times 10^{-6}$	$8.3 \times 10^{-6}$	$3.6 \times 10^{-7}$	$3.5 \times 10^{-6}$	$1.2 \times 10^{-5}$	$5.4 \times 10^{-7}$
<sup>17</sup> Slovenia	$1.4 \times 10^{-6}$	$7.0 \times 10^{-6}$	$1.5 \times 10^{-8}$	$2.0 \times 10^{-6}$	$1.0 \times 10^{-5}$	$2.2 \times 10^{-8}$
<sup>18</sup> Slovenia	$9.4 \times 10^{-7}$	$5.8 \times 10^{-6}$	$5.8 \times 10^{-8}$	$1.4 \times 10^{-6}$	$8.6 \times 10^{-6}$	$8.7 \times 10^{-8}$
<sup>19</sup> Spain	$1.1 \times 10^{-7}$	$4.8 \times 10^{-7}$	$4.9 \times 10^{-9}$	$1.6 \times 10^{-7}$	$7.1 \times 10^{-7}$	$7.2 \times 10^{-9}$
<sup>20</sup> Spain	$3.5 \times 10^{-6}$	$6.9 \times 10^{-6}$	$1.8 \times 10^{-7}$	$5.2 \times 10^{-6}$	$1.0 \times 10^{-5}$	$2.7 \times 10^{-7}$
<sup>21</sup> South Africa	$2.6 \times 10^{-7}$	$2.3 \times 10^{-6}$	$2.9 \times 10^{-9}$	$3.9 \times 10^{-7}$	$3.4 \times 10^{-6}$	$4.3 \times 10^{-9}$
<sup>22</sup> Switzerland	$2.4 \times 10^{-5}$	$3.9 \times 10^{-5}$	$1.9 \times 10^{-6}$	$3.6 \times 10^{-5}$	$5.8 \times 10^{-5}$	$2.9 \times 10^{-6}$
<sup>23</sup> United States	$1.9 \times 10^{-6}$	$9.1 \times 10^{-6}$	$3.6 \times 10^{-8}$	$2.8 \times 10^{-6}$	$1.4 \times 10^{-5}$	$5.4 \times 10^{-8}$
<sup>24</sup> United States	$2.5 \times 10^{-6}$	$9.2 \times 10^{-6}$	$3.6 \times 10^{-7}$	$3.7 \times 10^{-6}$	$1.4 \times 10^{-5}$	$5.4 \times 10^{-7}$
<sup>25</sup> United States	$1.4 \times 10^{-6}$	$2.3 \times 10^{-6}$	$7.3 \times 10^{-7}$	$2.1 \times 10^{-6}$	$3.4 \times 10^{-6}$	$1.1 \times 10^{-6}$
United States		$1.8 \times 10^{-6}$	$3.2 \times 10^{-7}$		$2.7 \times 10^{-6}$	$4.8 \times 10^{-7}$
<sup>26</sup> United States	$1.1 \times 10^{-5}$	$8.0 \times 10^{-5}$	$1.1 \times 10^{-8}$	$1.7 \times 10^{-5}$	$1.2 \times 10^{-4}$	$1.6 \times 10^{-8}$

Risks were estimated for the mean, maximum, and minimum <sup>222</sup>Rn concentrations listed in Table 1 even though exposures for the maximum and minimum <sup>222</sup>Rn concentrations are not shown in Table 9.

Label numbers refer to data position in Figures 10 and 11.

Figure 12 shows a set of notched boxplots for the mortality and morbidity inhalation risks. From Figure 12 it can be seen that the median measure of the mean risk values is only slightly greater than the  $10^{-6}$  acceptable risk level, but the notches extend to the  $10^{-6}$  acceptable risk level, suggesting that the overall risks may be acceptable. However, in individual caves and locations within certain caves, risks may be significant, as evidenced by the fact that the 75th percentile, the 90th percentile, and various outliers extend well beyond the  $10^{-6}$  acceptable risk level.

#### DISCUSSION AND CONCLUSIONS

Attempts have been made to regulate exposures to cavers and cave workers to excess levels of <sup>222</sup>Rn gas in caves ever since high levels of <sup>222</sup>Rn gas were discovered in some caves administered by the NPS (Yarborough and

Meyers, 1978, p. 19). Protection levels for cave workers were implemented at the earliest possible time (Baier, 1976) and have continued to evolve as more is learned. Unfortunately, little is still known about the effects of low-level ionizing  $\alpha$  radiation from  $^{222}$ Rn and its progeny. Still, it is widely recognized that the development of lung cancers may be expected based on numerous animal studies and epidemiological studies of miners.

Measuring <sup>222</sup>Rn is of little value unless these concentrations are converted to risk estimates. Calculating annual effective doses (mSv yr<sup>-1</sup> or WLM yr<sup>-1</sup>) is the generally accepted method for determining human-health threats. Using appropriate limits (20 mSv yr<sup>-1</sup> to 50 mSv yr<sup>-1</sup>) helps to put the calculated values in a health-risk context.

In general, it would seem that recreational and professional cavers are minimally at risk of developing lung cancers from exposure to <sup>222</sup>Rn, part-time cave workers are

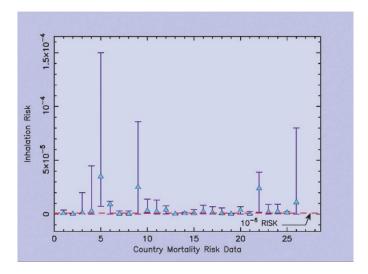


Figure 10. Plot of mean, minimum, and maximum mortality risk values from Table 11 relative to a  $10^{-6}$  acceptable risk. The x-axis numerical values correspond to the superscript labels in Table 11. (Note that data sets listed in Table 11 missing mean values [Great Britain and United States] are not plotted).

somewhat more at risk in some caves, and full-time cave workers more so (Table 7). This conclusion was similarly obtained by Craven and Smit (2006) for non-smokers. Unfortunately, the large degree of uncertainty associated with the calculations and potential discrepancies in the <sup>222</sup>Rn measurements, necessitate that the calculations listed in Table 7 be viewed with some degree of skepticism (it is not possible to determine if the calculated annual effective

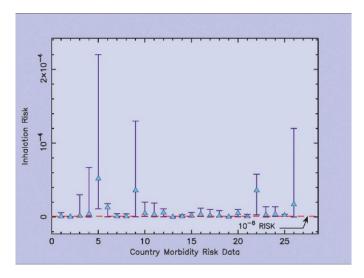


Figure 11. Plot of mean, minimum, and maximum morbidity risk values from Table 11 relative to a  $10^{-6}$  acceptable risk. The x-axis numerical values correspond to the superscript labels in Table 11. (Note that data sets listed in Table 11 missing mean values [Great Britain and United States] are not plotted).

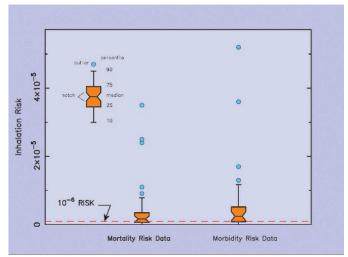


Figure 12. Notched boxplots of estimated mortality and morbidity risks relative to a  $10^{-6}$  acceptable risk. (Note that data sets listed in Table 11 missing mean values [Great Britain and United States] are not plotted).

doses in Table 7 are too high or too low or by how much). However, the annual effective doses listed in Table 7 should still be viewed as representative of the potential risks cavers and cave workers might be subject to when spending any significant amount of time underground.

A less common method of calculating risks posed by low-level ionizing  $\alpha$  radiation from  $^{222}$ Rn and its progeny, but which is a more typical method of calculating risks in general, is to use cancer slope factors (mortality and morbidity risk coefficients  $M_{R_i}$ ) to produce dimensionless risk estimates. The mortality and morbidity risk estimates listed in Table 11 for the  $^{222}$ Rn concentrations listed in Table 1 are of negligible concern.

For short-term exposures, typical of recreational cavers, the risk levels listed in Table 11 for the mean <sup>222</sup>Rn concentrations are probably of little concern. The same is probably true for professional cavers. However, for long-term exposures, typical of cave workers, these risk levels warrant some degree of minor concern especially in areas of poor ventilation (Kobal et al., 1988). If the maximum <sup>222</sup>Rn concentrations listed in Table 1 are considered, the risks will increase slightly, which may warrant a greater concern.

Overall, it appears that risks to cavers and cave workers are generally low, but in selected caves risks to cave workers may be significant. However, proper cave worker precautions for caves with elevated <sup>222</sup>Rn concentrations will minimize the risks. In addition, given the uncertainties associated with use of the LNT, concerns over risks to cave workers may need to depend on the eventual improvements or abandonment of the LNT. Changes to the LNT may result in a reduction or increase in the estimated risks to cavers and cave workers from exposure to elevated levels of <sup>222</sup>Rn. Other uncertainties, such as extreme seasonal

Table 12.	<sup>222</sup> Rn	progeny	current	and	historic	names.
-----------	-------------------	---------	---------	-----	----------	--------

Cu	rrent	Historic		
Symbol	Name	Symbol	Name	
<sup>218</sup> Po	Polonium-218	RaA	Radium A	
<sup>214</sup> Pb	Lead-214	RaB	Radium B	
<sup>214</sup> Bi	Bismouth-214	RaC	Radium C	
<sup>214</sup> Po	Polonium-214	RaC'	Radium C'	
<sup>210</sup> Tl	Thallium-210	RaC"	Radium C"	
<sup>210</sup> Pb	Lead-210	RaD	Radium D	
$^{210}\mathrm{Bi}$	Bismuth-210	RaE	Radium E	
<sup>210</sup> Po	Polonium-210	RaF	Radium F	

variations in measured <sup>222</sup>Rn concentrations (Yarborough and Meyers, 1978, p. 22) [e.g. 740 versus 22,165 Bq m<sup>-3</sup> in Magic Garden, Postojna Cave (Kobal et al., 1988)], further complicate risk calculations.

#### **APPENDIX**

#### RADON-222 PROGENY AND HISTORIC NAMES

When first discovered, the current <sup>222</sup>Rn progeny were known as decay products of <sup>226</sup>Ra and were formerly designated as Radium A – Radium F. The <sup>222</sup>Rn progeny are now known to be the isotopes listed in Table 12.

RADIATION SI UNITS AND CONVERSION TO TRADITIONAL UNITS

Radiation units have evolved over the years. As a result, radiation units can be quite confusing. To alleviate some of the confusion, selected radiation parameters are identified in Table 13 along with the SI special name, symbol, SI derived units, and traditional units.

#### ACKNOWLEDGMENTS

The author would like to thank Dr. Dragoslav Nikesic for providing me with his program Lungdose.F90 after convert-

Table 13. International System (SI) units and equivalents for traditional units (modified from Taylor, 2001, 1995; Nero, 1988, p. 39).

	SI Derived Unit				
Parameter	Special Name	Special Symbol	Expressed in Terms of Other SI Units	Expressed in Terms of SI Base Units	Conversion for traditional Unit
Activity	becquerel	Bq		$s^{-1}$	$1 \text{ Ci} = 3.7 \times 10^{10} \text{ Bq (1 pCi} =$
Concentration PAEC <sup>a</sup>		$\begin{array}{c} \text{Bq m}^{-3} \\ \text{J m}^{-3} \end{array}$			0.037 Bq) 1 pCi L <sup>-1</sup> = 37 Bq m <sup>-3</sup> 1 WL = $1.3 \times 10^8$ MeV m <sup>-3</sup> = $2.08 \times 10^{-5}$ J m <sup>-3</sup>
EEDC <sup>b</sup> Exposure		$\begin{array}{c} Bq\ m^{-3} \\ J\ s\ m^{-3} \end{array}$			$2.08 \times 10^{-3} \text{ m}$ 1 WL PAEC = 3740 Bq m <sup>-3</sup> 1 WLM = 12.97 J s m <sup>-3</sup> = 3.60 × $10^{-3}$ J h m <sup>-3</sup>
Exposure Exposure Rate Exposure Rate		Bq s m-3 $J m-3$ $Bq m-3$			1 WLM = 73.9 Bq yr m <sup>-3</sup> 1 WLM yr <sup>-1</sup> = 4.11 × 10 <sup>-7</sup> J m <sup>-3</sup> 1 WLM yr <sup>-1</sup> = 73.9 Bq m <sup>-3</sup>
Absorbed Dose	gray	Gy	$J kg^{-1}$	$m^2 s^{-2}$	1 rad = 1 cGy = $10^{-2}$ Gy 1 rad s <sup>-1</sup> = $10^{-2}$ Gy s <sup>-1</sup>
Absorbed Dose Rate Dose Equivalent <sup>c</sup>	sievert	Gy s <sup>-1</sup> Sv	$\rm J~kg^{-1}$	$m^2$ s $m^2$ s $m^2$ s $m^2$ s	1 rad s $^{-}$ = 10 $^{-}$ Gy s $^{-}$ 1 rem = 1 cSv = $10^{-2}$ Sv = $10^{-2}$ J kg <sup>-1</sup>
Effective Dose		$J\ s\ m^{-3}$			$1 \text{ WLM yr}^{-1} = 5 \text{ mSv yr}^{-1}$

<sup>&</sup>lt;sup>a</sup> Potential Alpha-Energy Concentration (PAEC).

<sup>&</sup>lt;sup>b</sup> Equilibrium-Equivalent Decay-Product Concentration (EEDC).

<sup>&</sup>lt;sup>c</sup> Also known as Biologically Effective Dose.

ing relevant portions to English. This program has been essential to the understanding of <sup>222</sup>Rn risks to cavers. The author would also like to thank Ms. Lindsey Bender and Dr. Neal Nelson of the U.S. Environmental Protection Agency's Office of Radiation and Indoor Air for their support and guidance and for providing specific radiation risk guidance materials. Lastly, the author thanks Dr. Neal Nelson and Dr. Ivan Kobal of the Jožef Stefan Institute (Ljubljana, Slovenia) for their critical reading of the manuscript.

#### NOTATION

$A_{ge}$	age of an exposed individual (dimen.)
$C_{eq}^{ge}$	EEDC — <sup>222</sup> Rn concentration that would result
eq	if $F = 1$
$C_E$	cumulative exposure (J h m <sup>-3</sup> )
$C_i$	activity concentrations for <sup>222</sup> Rn and its prog-
- 1	eny (Bq $m^{-3}$ )
$C_p$	PAEC — total α-particle energy potentially
- <i>p</i>	emitted by any mixture of <sup>222</sup> Rn per unit volume
	of air $(J m^{-3})$
$(\overline{C}R_n)_i$	average concentration of <sup>222</sup> Rn decay products
(- 11)1	during exposure interval (J m <sup>-3</sup> )
$C_{Rn}$	<sup>222</sup> Rn concentration (Bq m <sup>-3</sup> )
$D_{CF}$	Dose Conversion Factor
$D_{T,R}$	mean absorbed radiation dose to tissue T from
1,10	radiation $R$ (Gy)
$E_{DA}$	annual effective radiation dose to organs and
DA	tissues (Sv yr <sup>-1</sup> )
$E_D$	effective radiation dose to organs and tissues
D	(Sv)
F	equilibrium factor (dimen.)
$H_T$	human equivalent radiation dose to tissue $T(Sv)$
$I_h$	inhalation rate (m <sup>3</sup> s <sup>-1</sup> )
$M_{R_B}$	morbidity-risk coefficient for <sup>222</sup> Rn and its
Б	progeny (Bq <sup>-1</sup> )
$M_{R_T}$	mortality-risk coefficient for <sup>222</sup> Rn and its
	progeny (Bq <sup>-1</sup> )
$P_{eq}$	EEQ — time-integrated exposure to EEDC (Bq
	$s^{-1} m^{-3}$ )
$P_p$	PAEE — time-integrated exposure to PAEC (J s
	$\mathrm{m}^{-3}$ )
R	risk
$R_{M_B}$	risk of morbidity (dimen.)
$R_{M_{_T}}$	risk of mortality (dimen.)
$S_C$	scaling coefficient for a current (mobile) popu-
	lation to a stationary population (dimen.)
t	time (T)
$T_i$	exposure time — subscript <i>i</i> refers to exposure

for part-time cavers (50 h yr<sup>-1</sup>), full-time cavers

(600 h yr<sup>-1</sup>), part-time cave workers (1,760 h

yr<sup>-1</sup>), and full-time cave workers (2,000 h yr<sup>-1</sup>)

radioactive half-life of considered radionuclide

 $(h yr^{-1})$ 

(T)

 $T_{\frac{1}{2}}$ 

$W_R$	radiation weighting factor for various types of
	radiation (dimen.)
$W_{T_i}$	tissue weighting factor for differing sensitivities
•	of various human tissues to radiations (dimen.)
$\varepsilon_p$	potential alpha energy per unit of activity (Bq)
$\lambda_r$	decay constant for considered radionuclide (dimen.)
	,

#### ACRONYMS

DCF	Dose Conversion Factor				
DNA	Deoxyribonucleic Acid				
DOE	U.S. Department of Energy				
EEDC	Equilibrium-Equivalent Decay-Product Con-				
	centration				
EEQ	Equilibrium Equivalent Exposure				
EPA	U.S. Environmental Protection Agency				
HRTM	Human Respiratory Tract Model				
LD50	Median Lethal Dose				
LET	linear energy transfer				
LNT	linear no-threshold theory				
LOAEL	Lowest Observed Adverse Effect Level				
<b>MSHA</b>	Mine Safety and Health Administration				
NIOSH	National Institute of Occupational Safety and				
	Health				
NOAEL	No Observed Adverse Effect Level				
NPS	National Park Service				
NRC	U.S. Nuclear Regulatory Commission				
OSHA	Occupational Safety and Health Administration				
PAEC	Potential Alpha-Energy Concentration				
PAEE	Potential Alpha-Energy Exposure				
	· ·				

#### Notes

<sup>1</sup>A metalloid is an element with properties intermediate between those of metals and nonmetals.

<sup>2</sup>Clathrate compounds are formed by trapping the <sup>222</sup>Rn in the lattice of surrounding atoms rather than forming chemical bonds.

<sup>3</sup>See Table 13 in the Appendix for a brief overview of radiation SI units and conversion to traditional units.

<sup>4</sup>The linear energy transfer (LET) of radiation is a measure of the spatial energy distribution stated in terms of the amount of energy deposited per unit length of particle track, dE/dx, with typical units of keV μm (NRC, 1990, p. 11). It is the energy lost by charged particles in electronic collisions per unit track length where a low-LET is taken as <10 keV  $\mu m^{-1}$ and a high-LET is taken as >10 keV  $\mu m^{-1}$  (NRC, 2005, p. 375).

<sup>5</sup>In radiation biology, dose specifically pertains to the amount of energy ionizing radiation deposits in an organ tissue (ATSDR, 1997, p. 35).

<sup>6</sup>Effective dose (Sv) converts to absorbed dose (Gy) according to 1 Sv  $= 1 \text{ Gy} \times W^R.$ 

The term "plate-out" refers to the attraction of the negatively charged ion to surfaces such as a cave wall or the epithelium of the lung. <sup>8</sup>In vitro refers to the technique of performing experiments in a test

tube or in a living organism.

<sup>9</sup>In vivo refers to experiments conducted on living tissue of a whole living organism as opposed to a partial or dead organism.

<sup>10</sup>Phagocytized refers to the ingestion of particles or organisms by phagocytosis.

The reticuloendothelial system consists of a group of cells capable of phagocytosis.

12Parenteral administration refers to the route of particle administra-

tion (transport) through the body.

<sup>13</sup>Confounding factors are associated with the finding of an association for the wrong reason. It is associated with both the risk and the disease being studied, but need not be a risk factor for the disease under study. The confounding variable can either inflate or deflate the true relative risk (Wartenberg et al., 2000).

<sup>14</sup>ALARA means making every reasonable effort to maintain exposures to radiation as far below the dose limits in this part as is practical consistent with the purpose for which the licensed activity is undertaken, taking into account the state of technology, the economics of improvements in relation to state of technology, the economics of improvements in relation to benefits to the public health and safety, and other societal and socioeconomic considerations, and in relation to utilization of nuclear energy and licensed materials in the public interest (U.S.NRC, 2006, 10 CER, 2006,10 CFR § 20.1003).

15.4 A mortality risk coefficient is an estimate of the risk to an average member of the U.S. population, per unit activity inhaled or ingested for internal exposures or per unit time-integrated activity concentration in air or soil for external exposures, of dying from cancer as a result of intake of the radionuclide or external exposure to its emitted radiations" (Eckerman et al., 1999, p. 1).

<sup>16</sup>"A morbidity risk coefficient is a comparable estimate [mortality estimate] of the average total risk of experiencing a radiogenic cancer, whether or not the cancer is fatal" (Eckerman et al., 1999, p. 1).

#### REFERENCES

- Abelson, P.H., 1991, Mineral dusts and radon in uranium mines: Science, v. 254, no. 5033, p. 27–36.
- Ahlstrand, G., 1980, Alpha radiation levels in two caves related to external air temperatures and atmospheric pressure: Bulletin of the National Speleological Society, v. 42, no. 3, p. 39–41.
- Ahlstrand, G., and Fry, P., 1976, Alpha radiation monitoring in Carlsbad Caverns, *in* Proceedings of the 1st International Conference on Scientific Research in National Parks, p. 691–693.
- Aley, T., Castillon, K., and Sagendorf, J., 2006, Strategy for managing alpha radiation in show caves to protect caves, cave employees, and cave businesses, *in* Proceedings of the 2005 National Cave and Karst Management Symposium, Greyhound Press, p. 62–71.
- ATSDR, 1990, Toxicological Profile for Radon: Agency for Toxic Substances and Disease Registry TP-90-23, U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, 159 p.
- ATSDR, 1997, Toxicological Profile for Ionizing Radiation: Agency for Toxic Substances and Disease Registry (Draft for Public Comment), U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, 357 p.
- Baier, E.J., 1976, Radon daughters: Current Intelligence Bulletin 10, URL http://www.cdc.gov/niosh/78127\_10.html, [accessed February 8, 2006], DHHS (NIOSH) Publication No. 78-127.
- Bashor, B., undated, Big Bone Cave State Natural Area, Van Buren County, Tennessee: Health Consultation, U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry (ATSDR), URL www.atsdr.cdc.gov/ HAC/PHA/BigBoneCaveHC091304\_TN/BigBoneCaveHC091304-TN. pdf, [accessed January 19, 2006], 7 p.
- Bond, V.P., Wielopolski, L., and Shani, G., 1996, Current misinterpretations of the linear no-threshold hypothesis: Health Physics, v. 70, no. 6, p. 877–882.
- Bridges, B.A., Cole, J., Arlett, C.F., Green, M.H.L., Waugh, A.P.W., Beare, D., Henshaw, D.L., and Last, R.D., 1991, Possible association between mutant frequency in peripheral lymphocytes and domestic radon concentrations: The Lancet, v. 337, no. 8751, p. 1187–1189.
- Burian, I., and Stelcl, O., 1990, Radon and its daughters in the touristic caves of the Moravian Karst, *in* Blasko, C., ed., International Conference of Anthropogenic Impact and Environmental Changes in Karst: Czechoslovakia, Institute of Geographers, Czechoslovak Academy of Science, p. 27–32.
- Chambers, J.M., Cleveland, W.S., Kleiner, B., and Tukey, P.A., 1983, Graphical Methods for Data Analysis: Boston, Dunbury Press, 395 p.
- Chameaud, J., Masse, R., and Lafuma, J., 1984, Influence of radon daughter exposure at low doses on occurrence of lung cancer in rats: Radiation Protection Dosimetry, v. 7, p. 385–388.

- Chameaud, J., Perraud, R., Lafuma, J., Masse, R., and Pradel, J., 1974, Lesions and lung cancers induced in rats by inhaled radon-222 at various equilibriums with radon daughters, *in* Karbe, E., and Park, J., eds., Experimental Lung Cancer: Carcinogenesis and Biological Assays: Berlin, Springer Verlag, p. 410–421.
- Chameaud, J., Perraud, R., and Lafuma, J., 1982, Cancers induced by radon-222 in the rat, *in* Proceedings Specialist Meeting on the Assessment of Radon and Daughter Exposure and Related Biological Effects: Salt Lake City, Utah, RD Press, p. 189–209.
- Cigna, A.A., 2005, Radon in caves: International Journal of Speleology, v. 34, no. 1–2, p. 1–18.
- Cohen, B.L., 2000, Test of the Validity of the Linear-No Threshold Theory of Radiation Carcinogenesis with a Survey of Radon Levels in U.S. Homes, *in* Lehr, J.H., and Lehr, J.K., eds., Standard Handbook of Environmental Science, Health, and Technology: New York, The McGraw-Hill Companies, p. 20.12–20.20.
- Cohen, B.L., 2002, Cancer risk from low-level radiation: American Journal of Roentgenology, v. 179, p. 1137–1143.
- Cothern, C.R., 1987, Properties, *in* Environmental Radon:, Cothern, C.R., and Smith, J.E. Jr., eds., New York, Plenum Publ. Corp., Environmental Science Research, v. 35, p. 1–29.
- Cothern, C.R., 1989, Health effects of inhaled radon progeny: Journal of Environmental Science and Health — Part C: Environmental Carcinogenesis Reviews, v. C7, no. 1, p. 75–108.
- Cothern, C.R., 1990, Widespread apathy and the public's reaction to information concerning the health effects of indoor air radon concentrations: Cell Biology and Toxicology, v. 6, no. 3, p. 315–322.
- Craven, S.A., and Smit, B.J., 2006, Radon in Caves: Clinical Aspects: International Journal of Speleology, v. 35, no. 2, p. 93–101.
- Cross, F.T., 1987, Health effects, *in* Cothern, C.R., and Smith, Jr., J.E., eds., Environmental Radon: New York, Plenum Publ. Corp., Environmental Science Research, v. 35, p. 215–248.
- Cross, F.T., Palmer, R.F., Filipy, R.E., Busch, R.H., and Stuart, B.O., 1978, Study of the Combined Effects of Smoking and Inhalation of Uranium Ore Dust, Radon Daughters and Diesel Oil Exhaust Fumes in Hamsters and Dogs: Tech. Rep. PNL-2744, U.S. Research Triangle Park, N.C., Department of Health, Education and Welfare, National Institute of Environmental Health Sciences by Pacific Northwest Laboratory, 143 p.
- Cunningham, K.I., and LaRock, E.J., 1991, Recognition of microclimate zones through radon mapping, Lechuguilla Cave, Carlsbad Caverns National Park, New Mexico: Health Physics, v. 61, no. 4, p. 493–500.
- Damber, L., and Larsson, L.G., 1985, Underground mining, smoking, and lung cancer: A case-control study in the iron ore municipalities in Northern Sweden: Journal of the National Cancer Institute, v. 74, p. 1207–1213.
- Dicus, G.J., 2001, Why the NRC bases its regulation on the linear non-threshold theory, NRC News, U.S. Nuclear Regulatory Commission, No. S-01-006, Address to the 2001 Spring Joint Meetings of The Virginia Chapter of the Health Physics Society and The Virginia Section of the American Nuclear Society, Hampton, Va.
- Dueñas, C., Fernández, M.C., Cañete, S., Carretero, J., and Liger, E., 1998, <sup>222</sup>Rn concentrations, natural flow rate and the radiation exposure levels in the Nerja Cave: Atmospheric Environment, v. 33, p. 501–510.
- Duffy, J.T., Madden, J.S., Mackin, G.M., McGarry, A.T., and Colgan, P.A., 1996, A reconnaissance survey of radon in show caves in Ireland: Environmental International, v. 22, no. S1, p. S415–S423.
- Eckerman, K.F., Leggett, R.W., Nelson, C.B., Puskin, J.S., and Richardson, A.C.B., 1999, Cancer Risk Coefficients for Environmental Exposure to Radionuclides: Federal Guidance Report No. 13, EPA 402-R-99-001, Washington, D.C., U.S. Environmental Protection Agency.
- Edling, C., and Axelson, O., 1983, Quantitative aspects of radon daughter exposure and lung cancer in underground miners: British Journal of Industrial Medicine, v. 40, p. 182–187.
- Eheman, C., Carson, B., Rifenburg, J., and Hoffman, D., 1991, Occupational exposure to radon daughters in Mammoth Cave National Park: Health Physics, v. 60, no. 6, p. 831–835.
- Field, M.S., 1994, Radon Concentrations and their Use in Estimating Fissure Porosities and Recharge Areas in the Karst Aquifers of Walkersville, Maryland [Ph.D. dissertation]: Fairfax, Va., George Mason University.

- Fox, A., Goldblatt, P., and Kinlen, L., 1981, A study of the mortality of Cornish tin miners: British Journal of Industrial Medicine, v. 38, p. 378–380.
- Gamble, F.M., 1981, Alpha radiation in karst caves of the Transvaal, South Africa: Transactions of the British Cave Research Association, v. 8, no. 4, p. 254–260.
- Gillmore, G., Gilbertson, D., Grattan, J., Hunt, C., McLaren, S., Pyatt, B., mani Banda, R., Barker, G., Denman, A., Phillips, P., and Reynolds, T., 2005, The potential risk from <sup>222</sup>Rn posed to archaeologists and earth scientists: Reconnaissance study of radon concentrations, excavations, and archaeological shelters in the Great Cave of Niah, Sarawak, Malaysia: Ecotoxicology and Environmental Safety, v. 60, p. 213–227.
- Gillmore, G.K., Phillips, P.S., Denman, A.R., and Gilbertson, D.D., 2002, Radon in the Creswell Crags Permian limestone caves: Journal of Environmental Radioactivity, v. 62, p. 165–179.
- Gillmore, G.K., Sperrin, M., Phillips, P., and Denman, A., 2000, Radon hazards, geology, and exposure of cave users: A case study and some theoretical perspectives: Ecotoxicology and Environmental Safety, v. 46, p. 279–288.
- Gottlieb, L., and Husen, L., 1982, Lung cancer among Navajo uranium miners: Chest, v. 81, p. 449–452.
- Gourmelon, P., Barbey, P., Barescut, J.C., Bouville, A., Cancio, D., Harrison, J.D., Luccioni, C., Murtih, C., Nénot, J.C., Paquet, F., Rollinger, F., Sene, M., Smeesters, P., Sugier, A., and Tirmarche, M., 2005, Health Consequences of Chronic Internal Contaminations by Radionuclides: Comments on the ECRR Report "The Health Effects of Ionizing Radiation Exposure at Low Doses for Radiation Protection Purposes" and IRSN Recommendations, 2005-20, Clamart, France, Institut de Radioprotection et de Sûeté Nucléaire.
- Gunn, J., 1991, Radon concentrations in three Russian cave areas: Cave and Karst Science, v. 18, no. 2, p. 85–87.
- Gunn, J., 2003, Radon in caves, in Gunn, J., ed., Encyclopedia of Caves and Karst Science: London, G.B., Fitzroy Dearborn (an imprint of Taylor & Francis Books, Inc.), p. 617–619.
- Gunn, J., Fletcher, S., and Prime, D., 1991, Research on radon in British limestone caves and mines, 1970–1990: Cave and Karst Science, v. 18, no. 2, p. 63–65.
- Halliday, W.R., 2003, Disease, in Gunn, J., ed., Encyclopedia of Caves and Karst Science: London, G.B., Fitzroy Dearborn (an imprint of Taylor & Francis Books, Inc.), p. 293–295.
- Henshaw, D., Eatough, J.P., and Richardson, R.B., 1990, Radon: A causative factor in the induction of myeloid leukaemia and other cancers in adults and children?: The Lancet, v. 335, no. 8696, p. 1008–1112.
- Hopke, P.K., Jensen, B., Li, C.-S., Montassier, N., Wasiolek, P., Cavallo, A.J., Gatsby, K., Socolow, R.H., and James, A.C., 1995, Assessment of the exposure to the dose from radon decay products in normally occupied homes: Environmental Science and Technology, v. 29, no. 5, p. 1359–1364.
- Howe, G.R., Nair, R.C., Newcombe, H.B., Miller, A.B., Burch, J.D., and Abatt, J.D., 1987, Lung cancer mortality (1950–80) in relation to radon daughter exposure in a cohort of workers at the Eldorado Port Radium uranium mine: Possible modification of risk by exposure rate: Journal of the National Cancer Institute, v. 79, p. 1255–1260.
- Hyland, R., and Gunn, J., 1994, International comparison of cave radon concentrations identifying the potential alpha radiation risks to British cave users: Health Physics, v. 67, no. 2, p. 176–179.
- IAEA, 2003, Radiation Reports Series, International Atomic Energy Agency, Safety Reports Series No. 33, Vienna, Austria, International Atomic Energy Agency and International Labour Office.
- ICRP, 1980, Biological Effects of Inhaled Radionuclides, ICRP Publication 31, International Commission on Radiological Protection, Annals of the ICRP 4, no. 1/2, Oxford, Pergamon Press, 108 p.
- ICRP, 1991, 1990 Recommendations of the International Commission on Radiological Protection, ICRP Publication 60, International Commission on Radiological Protection, Annals of the ICRP 21, no. 1–3, Oxford, Pergamon Press, 201 p.
- ICRP, 1994a, Protection Against Radon-222 at Home and at Work, ICRP Publication 65, International Commission on Radiological Protection, Annals of the ICRP 23, no. 2, Oxford, Pergamon Press, 45 p.
- ICRP, 1994b, Human Respiratory Tract Model for Radiological Protection: A Report of a Task Group of the International Commission on Radiological Protection, ICRP Publication 66, International

- Commission on Radiological Protection, Annals of the ICRP 24, no. 14, Oxford, Pergamon Press.
- Ivanovich, M., 1992, The phenomenon of radioactivity, *in* Ivanovich, M., and Harmon, R.S., eds., Uranium-Series Disequilibrium: New York, Oxford University Press, p. 1–33.
- James, A.C., Birchall, A., and Akabani, G., 2003, Comparative dosimetry of BEIR VI revisited: Radiation Protection Dosimetry, v. 108, no. 1, p. 3–26.
- Kellerer, A.M., and Nekolla, E.A., 2000, The LNT-controversy and the concept of "controllable dose": Health Physics, v. 79, no. 4, p. 412–418.
- Kobal, I., Smodiš, B., and Škofljanec, M., 1986, Radon-222 air concentrations in the Slovenian karst caves of Yugoslavia: Health Physics, v. 50, no. 6, p. 830–834.
- Kobal, I., Smodiš, B., Burger, J., and Škofljanec, M., 1987, Atmospheric radon-222 in tourist caves of Slovenia, Yugoslavia: Health Physics, v. 52, no. 4, p. 473–479.
- Kobal, I., Ančik, M., and Škofljanec, M., 1988, Variations of <sup>222</sup>Rn air concentrations in Postojna Cave: Radiation Protection Dosimetry, v. 25, no. 3, p. 207–211.
- Lario, J., Sánchez-Moral, S., Cañaveras, J.C., Cuezva, S., and Soler, V., 2005, Radon continuous monitoring in Altamira Cave (northern Spain) to assess user's annual dose: Journal Environmental Radiation, v. 80, p. 161–174.
- Lauier, D., Valenty, M., and Tirmarche, 2001, Radon exposure and the risk of leukemia: A review of epidemiological studies: Health Physics, v. 81, no. 3, p. 272–288.
- Law, G.R., Kane, E.V., Roman, E., Smith, A., and Cartwright, R., 2000, Residual radon exposure and adult acute leukaemia: The Lancet, v. 355, no. 9218, p. 1888.
- Lenart, L., Somogyi, G., Hakl, J., and Hunyadi, I., 1990, Radon mapping in caves of eastern Bukk region, *in* Blasko, C., ed., Proceedings of the 10th International Congress of Speleology: Czechoslovakia, Institute of Geographers, Czechoslovak Academy of Science, p. 21–31.
- Little, J.B., 1997, What are the risks of low-level exposure to α radiation from radon?: Proceedings of the National Academy of Science, v. 94, p. 5996–5997.
- Miki, T., and Iauthora, M., 1980, Accumulation of atmospheric radon in calcite caves: Health Physics, v. 39, no. 2, p. 351–354.
- Morken, D.A., 1955, Acute toxicity of radon: American Medical Association Archives of Industrial Health, v. 12, p. 435–438.
- Morken, D.A., 1973, The Biological Effects of Radon on the Lung, in Stanley, R.E., and Moghissi, A.A., eds., Noble Gases, CONF-703915, U.S. Energy Development and Research Agency, National Environmental Research Center, p. 501–506.
- Morken, D.A., and Scott, J., 1966, The Effects on Mice of Continual Exposure to Radon and its Decay Products on Dust: UR-669, U.S. Atomic Energy Commission, University of Rochester.
- Morrison, H.I., Wigle, D.T., Stocker, H., and deVillers, A.J., 1981, Lung cancer mortality and radiation exposure among the Newfoundland fluorspar miners, *in* Gomez, M., ed., International Conference, Radiation Hazards in Mining: Control, Measurement, and Medical Aspects: New York, Society of Mining Engineers of the American Institute of Mining, Metallurgical and Petroleum Engineers, p. 372–376.
- MSHA, 1989, Mineral Resources, *in* Code of Federal Regulations: 30 CFR Part 57, Washington, D.C., U.S. Government.
- MSHA, 2005, Mineral Resources, *in* Code of Federal Regulations: 30 CFR Part 57, Washington, D.C., U.S. Government, p. 341–343.
- NAS, 2005, News: Low levels of ionizing radiation may cause harm: National Academy of Sciences, http://www.4nationalacademies.org/ news.nsf/isbn/030909156X?OpenDocument [accessed January 3, 2006].
- NCRP, 2001, Evaluation of the linear-nothreshold dose-response model for ionizing radiation: NCRP Report No. 136, Bethesda, Md., NCRP Publications.
- NCRP, 1993, Limitation of exposure to ionizing radiation, National council on radiation protection and measurements, NCRP Report No. 116, Bethesda, Md., NCRP Publications, 88 p.
- Nero, A.V. Jr., 1988, Radon and its decay products in indoor air: An overview, *in* Radon and its Decay Products in Indoor Air: New York, John Wiley & Sons, p. 1–53.
- Nikezic, D., and Yu, K.N., 2001, Alpha hit frequency due to radon products in human lung cells: International Journal of Radiation Biology, v. 77, no. 5, p. 559–565.

- NPS, 1980, Cave radiation safety and occupational health management guideline: National Park Service NPS-14, Washington, D.C., National Park Service
- NPS, 2005, §19 Reserved ("Radiation Safety" under development), in Director's Order 50B/Reference Manual 50B: Washington, D.C., National Park Service.
- NRC, 1988, Health effects of radon and other internally deposited alphaemitters: BEIR IV, National Research Council, Washington, D.C., National Academy Press, 602 p.
- NRC, 1990, Health effects of exposure to low levels of ionizing radiation: BEIR V, National Research Council, Washington, D.C., National Academy Press, 421 p.
- NRC, 1999, Health effects of exposure to radon: BEIR VI, National Research Council, Washington, D.C., National Academy Press, 500 p.
- NRC, 2005, Health risks from exposure to low levels of ionizing radiation: BEIR VII-Phase 2: Washington, D.C., National Research Council, National Academy Press, 750 p.
- OSHA, 1988, Health, *in* Code of Federal Regulations: 41 CFR Part 57, Washington, D.C., U.S. Government.
- Oughton, D., 2006, Hypothesis testing and the choice of the dose-response model: Toxicology letters, v. 162, no. 1, p. 98–110.
- Page, S., 1993, EPA's strategy to reduce risk of radon: Journal of Environmental Health, v. 56, p. 27–36.
- Palmer, R.F., Stuart, B.O., and Filipy, R.E., 1973, Biological Effects of Daily Inhalation of Radon and its Short-Lived Daughters in Experimental Animals, in Stanley, R.E., and Moghissi, A.A., eds., Noble Gases, CONF-703915, U.S. Energy Development and Research Agency, National Environmental Research Center, p. 507–519.
- Papastefanou, C., Manolopoulou, M., Savvides, E., and Charalambous, S., 1986, Natural radiation dose in Petralona Cave: Health Physics, v. 50, no. 2, p. 281–286.
- Pisa, F.E., Barbone, F., Betta, A., Bonomi, M., Alessandrini, B., and Bovenzi, M., 2001, Residential radon and risk of lung cancer in an Italian alpine area: Archives of Environmental Health, v. 56, no. 3, p. 208–215.
- Przylibski, T.A., 1999, Radon concentration changes in the air of two caves in Poland: Journal of Environmental Radiation, v. 45, p. 81–94.
- Puskin, J.S., and Nelson, C.B., 1994, Estimating Radiogenic Cancer Risks, EPA 402-R-93-076, Washington, D.C., U.S. Environmental Protection Agency.
- Roscoe, R.J., Steenland, K., and Halperin, W., 1989, Lung cancer mortality among nonsmoking uranium miners exposed to radon daughters: Journal of the American Medical Association, v. 262, p. 629–633.
- Samet, J.M., 1997, Indoor radon exposure and lung cancer: Risky or not?—all over again: Journal of the National Cancer Institute, v. 89, p. 4–6. Smart, P.L., 1991, Hydrological implications of radon in Mendip
- Limestone groundwaters: Cave Science, v. 18, no. 2, p. 89–90.
- Smart, P.L., and Friederich, H., 1986, Water movement and storage in the unsaturated zone of a maturely karstified carbonate aquifer, Mendip Hills, England, *in* First Conference on Environmental Problems, Karst Terranes and Their Solutions (Bowling Green, Ky.), Proceedings, Dublin, Ohio, National Water Well Association, p. 59–87.
- Snihs, J.O., 1973, The approach to radon problems in non-uranium mines in Sweden, in Proceedings 3rd International Congress on International Radiation Protection Association (IRPA), USAEC Conf. 73097-P.2., U.S. Atomic Energy Commission, p. 900–911.
- Solli, H.M., Andersen, A., Stranden, E., and Langard, S., 1985, Cancer incidence among workers exposed to radon and thoron daughters in a niobium mine: Scandinavian Journal of Work and Environmental Health, v. 11, p. 7–13.
- Solomon, S.B., Langroo, R., Peggie, J.R., Lyons, R.G., and James, J.M., 1996, Occupational exposure to radon in Australian tourist caves, An Australia-wide study of radon Levels: Tech. Rep. 93/0436, Yallabie, Victory, Australia, Australian Radiation Laboratory, 16 p.
- Somogyi, G., Hunyadi, I., and Hakl, J., 1989, Historical review of one decade of radon measurements in Hungarian caves performed by solid state nuclear track detection technique, in Proceedings of the 10th International Congress of Speleology, Erscheinung Publishers, p. 3–13.
- Stein, L., 1987, Chemical properties of radon, *in* Hopke, P.K., ed., Radon and Its Decay Products: Occurrence, Properties, and Health Effects: Washington, D.C., American Chemical Society, p. 241–251.
- Strom, D.J., Reif, R.H., Andrews, D.A., George, A.C., George, J.L., James, A.C., Jones, C.R., Langner, Jr., G.H., Gavrilas-Guinn, M., Neton, J.W., Rabovsky, J.L., Runkle, G.E., Carlson, D.S., Dudney, C.S., Gammage, R.B., Maisler, J.A., Rose, S., and Wilson, D.L.,

- 1996, Occupational Exposure to Radon and Thoron, Tech. Rep. PNNL14108, Washington, D.C., U.S. Department of Energy, Radiological Control Coordinating Committee, Radon Subcommittee, URL www.pnl.gov/bayesian/strom/pdfs/Strom1996A\_PNNL\_14108\_Occ\_Exp\_to\_Radon\_Thoron\_DOE\_RCCC\_1996\_01.pdf, [accessed February 8, 2006], 45 p.
- Surbeck, H., 1990, Radon-222 transport from soil to karst caves by percolation water, *in* Proceedings of the 22nd Congress of the IAH, International Association of Hydrogeologists, p. 349–355.
- Svensson, C., Pershagen, G., and Klominek, J., 1989, Lung cancer in women and type of dwelling in relation to radon exposure: Cancer Research, v. 49, p. 1861–1865.
- Taylor, B.N., 1995, Guide for the use of the International System of Units (SI), 811, Gaithersburg, Md., National Institute of Standards and Technology, 74 p.
- Taylor, B.N., 2001, The International System of Units (SI), Tech. Rep. 330, Gaithersburg, Md., National Institute of Standards and Technology, 68 p.
- Tomasek, L., Darby, S.C., Swerdlow, A.J., Placek, V., and Kunz, E., 1993, Radon exposure and cancers other than lung cancer among uranium miners in West Bohemia: The Lancet, v. 341, no. 8850, p. 919–923.
- UNSCEAR, 2000, Sources and effects of ionizing radiation Annex B: Exposures from natural radiation sources, UNSCEAR 2000 Report Vol. I, Vienna, Austria, United Nations Scientific Committee on the Effects of Atomic Radiation, 156 p.
- U.S. EPA, 2006, Fact Sheet: Updated risk assessment for radon in indoor air, Tech. rep., Washington, D.C., U.S. Environmental Protection Agency, URL http://www.epa.gov/iaq/radon/risk\_assessment\_factsheet.html, [accessed March 31, 2006].
- U.S. Navy, 2001, Radiation health protection manual, U.S. Navy NAVMED P-5055, Washington, D.C., Bureau of Medicine and Surgery, 68 p.
- U.S. NRC, 2006, Standards for protection against radiation, in Code of Federal Regulations, 10 CFR Part 20, Washington, D.C., U.S. Government
- Vaupotič, J., Dujmovič, P., and Kobal, I., 1998, Radiation doses due to radon and progeny in Postojna Cave: Acta Carsologica, v. XXVII, no. 1, p. 395–406.
- Vaupotič, J., Csige, I., Radolić, V., Hunyadi, I., Planinić, J., and Kobal, I., 2001, Methodology of radon monitoring and dose estimates in Postojna Cave, Slovenia: Health Physics, v. 80, no. 2, p. 142–147.
- Wartenberg, D., Ramsey, D., Warner, J., and Ober, D., 2000, Problems in conducting epidemiological studies, Facsnet, URL http://www. facsnet.org/tools/ref\_tutor/epidem/problems.php3, [accessed March 14, 2006].
- Waxweiler, R.J., Roscoe, R.J., Archer, V.E., Thun, M.J., Wagoner, J.K., and Lundin, Jr., F.E., 1981, Mortality follow-up through 1977 of the white underground uranium miners cohort examined by the United States Public Health Service, in Gomez, M., ed., International Conference, Radiation Hazards in Mining: Control, Measurement, and Medical Aspects, New York, Society of Mining Engineers of the American Institute of Mining, Metallurgical and Petroleum Engineers, p. 823–830.
- Wiegand, J., Feige, S., Quingling, X., Schreiber, U., Wieditz, K., Wittmann, C., and Xiarong, L., 1995, Radon and thoron in cave dwellings (Yan'an, China): Environmental Science and Technology, v. 29, no. 5, p. 1359–1364.
- Wrixon, A.D., Barraclough, I., and Clark, M.J., 2004, Radiation, People and the Environment: Booklet No. IAEA/PI/A.75/04-00391, Vienna, Austria, International Atomic Energy Agency (IAEA), Division of Public Information, Ford, J., ed., 81 p.
- Yanada, Y., 2003, Radon exposure and its health effects: Journal of Health Science, v. 49, no. 6, p. 417–422.
- Yarborough, K., 1976, Investigation of radiation produced by radon and thoron in natural caves administered by the National Park Service, in Aley, T., and Rhodes, D., eds., Proceedings of the National Cave Management Symposium: Albuquerque, N.M., Speleobooks, p. 59–69.
- Yarborough, K., and Meyers, C., 1978, Sputum cytology and personnel exposures at National Park Service administered caves, *in* Conference/Workshop on Lung Cancer Epidemiology & Industrial Applications of Sputum Cytology, Golden, Colo., Colorado School of Mines, p. 17–82.
- Yu, K.N., Lau, B.M.F., and Nikezic, D., 2006, Assessment of environmental radon hazard using human respiratory tract models: Journal of Hazardous Materials, v. 132, p. 98–110.