

# **RADON AND HUMANS FROM ANOTHER PERSPECTIVE**

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## **ABSTRACT**

Radon-induced lung cancer can be traced back to 16<sup>th</sup> century miners in Europe, but recently there has been world-wide concern that elevated radon progeny levels in dwellings may be implicated in lung cancer; however, this has not been established unequivocally. Historical and experimental evidence is presented to document how inhaled radon is distributed throughout the body and stored in fats and lipids; background counts, in a steel room, of a whole body counter were observed to progressively decrease throughout the day, attributed to a lowering of radon as subjects accessed the steel room. Reported increases in potassium-40 ( $^{40}\text{K}$ ) counts in marathon runners were attributed to inhalation of environmental radon, and radon progeny were verified by measuring contributions to the  $^{40}\text{K}$  photopeak by  $^{214}\text{Bi}$  in cyclists and an untrained subject who exercised in a room with radon-laden air. Effective half-lives and regional  $^{214}\text{Bi}$  emissions were found to be highest from the cranium (brain) and abdominal (omentum) regions when filtered radon-laden air was inhaled. These observations prompted analyses for radon progeny ( $^{210}\text{Bi}$  and  $^{210}\text{Po}$ ) from brain tissues of persons who had developed Alzheimer's and Parkinson's Diseases (AD & PD). Protein in AD and lipids in PD were high in these progeny relative to control tissues. Whole body counts ( $^{214}\text{Bi}$  emissions) of subjects over a period of 24 years were analyzed for radon body content (Rn-conc). Statistically significant correlations were found among total body fat and Rn-conc in females, as well as between seasonal home radon concentrations and seasonal Rn-conc in subjects participating in community-based studies. It is concluded that environmental radon is indeed stored in the body and body concentration correlates with body fat in females, and that these reflect seasonal concentrations in their dwellings. Radon decay products include a number of alpha and beta particle emitters, emissions which produce a radiation risk and may play a role in multiple sclerosis and mammary cancer as well as cancers of other fat-rich tissues. This paper presents factual evidence that challenges current fallacies and misconceptions of radon uptake by the body and subsequent in vivo behavior.

## **Introduction**

With respect to health issues, radon may be considered an enigma. Since ancient Roman times, they and other civilizations visited radon spas (Clarke 2003); radon spas in Austria (Foisner 2004) and throughout Europe (Austria 2004) are still popular today. Old uranium mines in the United States have been converted into health spas (Free Enterprise Radon Health Mine 2002 and Merry Widow Health Mine 2004); even the United States

Army used a radon spa in Hot Springs AK as a treatment center in the early 1900s. Scully (1934) in a report on the effect of radioactive properties of natural spring waters at a meeting of the Arkansas Medical Society stated: "*Radium emanation can be taken into the body by drinking water in which it is held in solution, or it can be breathed in with the air or vapors arising from the radioactive spring waters.... The emanation has a special affinity for lipoids, and is therefore stored chiefly in the organs rich in lipoids, such as the nervous system and bone marrow.*"

In the early 1900s commercial devices were introduced to provide radon-charged waters intended to duplicate that found at mineral hot springs or health spas (Landa et al. 1988). In a promotional pamphlet the recommendation for the Revigator (Revigator 1928) was "*One should drink water from the Revigator at all times and at least eight full glasses per day.*"

Radon-induced lung cancer has captured attention in the media over the past 70 years, particularly in uranium miners (Kreuzer et al. 2002). However, "mountain sickness" a fatal disease of pitchblende miners, later identified as lung cancer, dates back to medieval mines of the Erz Mountains in Germany (Lafavore 1987). Beginning in the late 1980s, lung cancer in the general populace has been attributed to environmental radon exposure in dwellings (Van Pelt 2003). In spite of a report that exhalation of environmental Rn originally inhaled from the home environment and stored in body fluids and tissues increased after meal consumption (Rundo et al. 1978a); there were suggestions to the contrary. Common beliefs are: "*Chemically, radon is a noble gas. As such, it is similar, for example to helium and neon. These gases do not readily interact chemically with other elements and are relatively difficult, although not impossible, to trap. Like any other noble gas, radon is colorless and odorless. If it is in the air, it is inhaled along with all other gases. It is also exhaled promptly, and were one dealing with radon alone there would be little reason for concern. The radon hazards do not come primarily from radon itself, but rather from radioactive products formed in the decay of radon-222*" (Bodansky 1987).

The decay scheme of environmental radon-222 (ERn) can be seen in Fig. 1. Approximately 28 MeV of kinetic energy is released in the decay of one  $^{222}\text{Rn}$  atom to  $^{206}\text{Pb}$ ; therefore, it is of vital health concern to determine if an appreciable fraction of the rare gas radon is taken up through the lungs and stored in body fats and lipids as suggested by Scully 1934 and Rundo et al. 1978a. In the latter report, the  $^{214}\text{Bi}$  body content of a subject was measured as a function of time after leaving a house with a relatively high radon concentration ( $> 800 \text{ kBq/m}^3$ ). The data for these measurements are displayed in Fig. 2.

### **Observations in North Dakota**

In September 1978, we noticed erratic behavior in whole body counter (WBC) data at the USDA ARS Grand Forks Human Nutrition Research Center (USDA ARS GFHNRC). In July 1979, we attributed these variations to fluctuations in the background, and, in May 1980, we noted an 11% decrease in background counts between early morning and afternoon. We postulated the radon concentration in the whole body counter steel room

had built up during the evening when the chamber was closed and was gradually “diluted” as the door to the chamber was repeatedly opened and closed for successive whole body counts. Our WBC was programmed to monitor prominent Bi-214 photopeaks, including ones at 610, 1726 keV as well as those near the K-40 photo peak (1460 keV). At this time, three well-conditioned cyclists participating in other research were studied (Lykken et al. 1983). Each cyclist rode an average of 354 kilometers per week outdoors and pedaled the equivalent of 178 kilometers indoors. Before and after an outdoor ride, 10 minute collimated gamma ray  $^{40}\text{K}$  and  $^{214}\text{Bi}$  counts were measured with NaI (*Tl*) detectors from the thighs of the cyclists (Fig. 3). Potassium-40 counts had earlier been reported to have increased in marathon runners after extensive outdoor runs (Lane et al. 1978 and Londeree and Forkner 1978) with the excess counts decreasing over a 30 minute time interval. It was suggested that potassium redistribution may be the source of the excess  $^{40}\text{K}$  counts and we wanted to check if the redistribution occurred in the working muscle (Fig. 3). Interestingly, we found strong correlations between increased  $^{40}\text{K}$  counts, and  $^{214}\text{Bi}$  counts from both the 610, 1726 keV photopeaks, attributed to decay of environmental radon (ERn) and its progeny inhaled by the cyclists as they pedaled outdoors (Fig. 4). Lykken and Ong (1989) demonstrated that ERn was readily absorbed and stored in the body, and that stored ERn was “flushed out” by breathing ERn-free air after food consumption.

In a related experiment, Lykken et al. (1990) studied a subject who exercised on a Monark bicycle ergo meter (Quinton Instrument Company, Seattle, WA) in a room laden with radon (14-25kBq/m<sup>3</sup>). Regional  $^{214}\text{Bi}$  gamma emissions were obtained using lead collimators (Fig. 5), and effective half-lives and regional  $^{214}\text{Bi}$  emissions were found to be highest from the cranium (brain) and abdominal (omentum) regions when radon-laden air was inhaled, filtered by a mask designed to remove dust and radionuclide. (Fig. 6). Furthermore, a post-exposure Electroencephalogram (EEG) differed from a pre-exposure EEG in that the occipital lobe alpha (8-12 Hz) power decreased with time up to 30 min. after leaving the Rn-laden atmosphere and then increased over the next 12 min. approaching the pre-exposure values. Rn progeny including  $^{214}\text{Bi}$  and  $^{214}\text{Pb}$  activities were detected in the subject’s post-exposure urine.

In a pilot study, Lykken and Alkhatib (1993) measured  $^{210}\text{Po}$  alpha particle emissions from 14 persons including 7 cigarette smokers (S) and 7 nonsmokers (NS); subjects had been exposed to bedroom ERn concentrations of  $0.61 \pm 0.8$  kBq/m<sup>3</sup> (CS) and  $1.1 \pm 1.6$  kBq/m<sup>3</sup> (NS). They concluded the  $^{210}\text{Po}$  entered the hair through an internal pathway.

### **Brain Research**

These observations prompted Momčilović et al. (2001) to study the occurrence of ERn progeny,  $^{210}\text{Bi}$  (beta particle emitter) and  $^{210}\text{Po}$  (alpha particle emitter), in the protein and lipid fractions of cortical gray and subcortical white matter from frontal and temporal lobes of human brains of persons with Alzheimer’s Disease (AD), Parkinson’s Disease (PD), cigarette smokers (S) or persons with no known neurological disease (controls, C). A ten-fold increase in  $^{210}\text{Pb}$  and  $^{210}\text{Po}$  radioactivity in the protein fraction from both the

cortical gray and subcortical white matter in AD and S and a similar increase in the lipid fraction in PD was found (Fig. 6). The pathognomonic distribution of the radon progeny to the lipids in PD and the proteins in AD were inferred to reflect the increase in local chlorine availability to which radon daughters bound selectively.

We also found that radon distributes differently in the various anatomical structural compartments of the brain in an Alzheimer's disease victim (Fig 8). Evidently, these changes in the brain radon distribution are quite complex and remarkable. At this moment it suffices to say that radon notably accumulates in the hippocampus and amygdala, the two brain structures crucial for the human faculty of memory and emotional behavior, respectively. It should be noted that the level of radioactivity recorded in amygdala may kill all the cells of that brain anatomical structure within a short period of time of only a few years. Indeed, every high energy 5.3 to 7.69 MeV radon progeny alpha particles (Fig. 1) may kill at least three cells in a row in whatever direction it may choose to go. Recent evidence showed that for high energy alpha particle it is enough to pass through the cytoplasm without hitting the nucleus to kill the cell" (Day 1999).

### **Seasonal Variation of Radon Concentrations**

Indoor radon concentrations in dwellings vary with season from typical winter to summer ratios of approximately two (Papastefanou et al. 1994, and Huber et al. 2001). If, indeed, radon is stored in body fats and lipids, this storage should be reflected in  $^{214}\text{Bi}$  gamma emissions from subjects measured in a whole body counter. Residential radon concentrations in Grand Forks, ND homes have been measured over a sixteen year period beginning in 1988. Whole body counter data, steel room  $^{214}\text{Bi}$  background and subject  $^{214}\text{Bi}$  concentrations have also been measured in the USDA ARS GFHNRC whole body counter.

The seasonality of radon is shown in Fig. 9. Shown on this four-tier graph are: (1) home ambient air radon (bottom tier); (2) relative background radon activity in the USDA, ARS, GFHNRC Whole Body Counter (above bottom); (3) seasonal  $^{214}\text{Bi}$  in the men (below top), and (4) seasonal  $^{214}\text{Bi}$  in women (top). The radon activity data were collected over the several years and plotted along the days of a year. The central line represents the best fit for the average yearly function with the 95% prediction region between lines above and below that central tendency. The data indicated that radon in ambient air and in the bodies of men and women varies with the days of the year. Indeed, there is a cyclic summer drop and subsequent winter rise of radon in the home ambient air and radon accumulated in the whole body of men and women; the pattern was statistically significant ( $p < 0.05$ ). Evidently, the environmental radon does accumulate in the human body, especially so in women, presumably owing to much higher fat content than men. This accumulation showed a regular seasonality pattern indicating that human body radon accumulation follows the changes of the environmental radon concentration. However, that equilibration was not passive, since the human body accumulates more radon than if the equilibration were a passive one. This is, to our knowledge, the first such conclusive evidence on radon accumulation in the human body and its seasonal

pattern. Seasonal rhythms of human nutrient intake and meal patterns in agricultural societies as well as affluent societies have been reported to reflect seasonal changes in human body weight (de Castro 1991). Exceptionally high intake of carbohydrates was recorded in the fall. It would take some time for fat to deposit after the luxurious food intake so that fat stores would be expected to peak during the winter time. Precisely the time when the radon concentrations in the human body are the highest. Hence, our seasonal dependent changes in whole body radon retention may reflect the natural seasonal cycle of fat accumulation and depletion. Implying that the level of radon in the human body is higher than it is in the environment and that it would be constant if there were no seasonal changes in either fat stores or environmental radon concentrations. This synergism should be more pronounced in women than in men due to the greater fat stores in women. Interestingly, Lykken et al. (2000) reported correlation between body fat mass and total body radon to be statistically significant in women (n = 40) but not in men (n = 57).

### **Pilot Study of Cancerous Breast Tissues (Incomplete)**

In 1943, six weeks after an atomic bomb was dropped on Nagasaki (Japan), Dr. Horne was appointed chief medical officer in charge of civil population (Horne 1995). That event stimulated his life-long interest in radiation and its effects on humans. We collaborated with Dr. Horne and his colleagues in the Boston area in a study designed to measure  $^{210}\text{Po}$  emissions from breast tissues of women who had developed cancerous breasts. Data from this incomplete pilot study are displayed in Table I. The mean  $^{210}\text{Po}$  activity 600  $\mu\text{Bq/g}$  corresponds to 0.04 dis/min·g, a value that is in the lower range of that found in AD and PD tissues (see Fig. 7). Note the actual radiation dose to the tissue includes not only that due to  $^{210}\text{Po}$  alpha particles ( $\approx 5 \pm 4$  pGy, mean  $\pm$  standard deviation) but also from beta particles emitted by the parent nuclei,  $^{210}\text{Pb}$  and  $^{210}\text{Bi}$ . Furthermore, if radon is actually stored in breast tissue the total alpha particle energy deposited locally would be approximately five times greater (Fig. 1) not to mention the dose from beta particles. Furthermore, the radiation doses delivered at the cellular level warrant a thorough microdose analysis of data from a case-controlled study of both  $^{210}\text{Bi}$  beta emissions and  $^{210}\text{Po}$  alpha emissions from cancerous breast tissues.

### **Pain Relief**

Ward (1989), in a private communication suggested how radon may provide relief from arthritis pain to persons visiting health spas and old uranium mines. He used an action potential physics model that included both dose and dose-rate dependence. *“I suspect that it is the  $^{210}\text{Pb}/^{210}\text{Bi}/^{210}\text{Po}$ , which has the long lasting effect. The biological uptake of Pb through the lungs into the blood [brain] is about 10 hours. The neural network would see about  $1 \times \text{Exp}(+10)$  signals/second raising the [neuron pain]threshold more than enough to quench the pain signals. The activation threshold for the neural transmitters is increased above the pain threshold due to the ionizing radiation of  $^{210}\text{Pb}^{+2}$  ions preferentially attached to the neural network.”* These conjectures would explain the

altered EEG signals upon exposure to radon-laden air reported by Lykken et al. (1990) discussed above.

## Conclusions

This paper presented factual evidence to challenge the following fallacies and misconceptions about radon: (1) although radon is a noble gas, it is not chemically inert. Radon **does** form compounds by weak van der Waals bonds (Stein 1997). (2) Inhaled radon is not simply exhaled from the body so that only its progeny accumulate in the lungs, but radon **does** accumulate in the human body fats and lipids on its own. (3) Radon decay results in distinct chemical transmutations; each element has specific toxicological and radio toxicological properties. These decay products are all heavy metals which have a strong affinity to the underlying protein structures and bind strongly to them thus impeding protein turnover. This bonding may have long ranging metabolic consequences such as the already described change of pain threshold after low radon irradiation.(4) Not enough specific attention is paid to the speciation of the energy spectrum of the radioactive decay products. Consideration of the average radiation from a spectrum of radiation energies may only lead to confounding and misinterpreting of the biological effects of radiation.

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All investigations involving human subjects were approved by the University of North Dakota Radioactive Drug Research Committee (UND RDRC 0119) and Institutional Review Board and by the USDA Human Studies Committee after each subject had given written consent.

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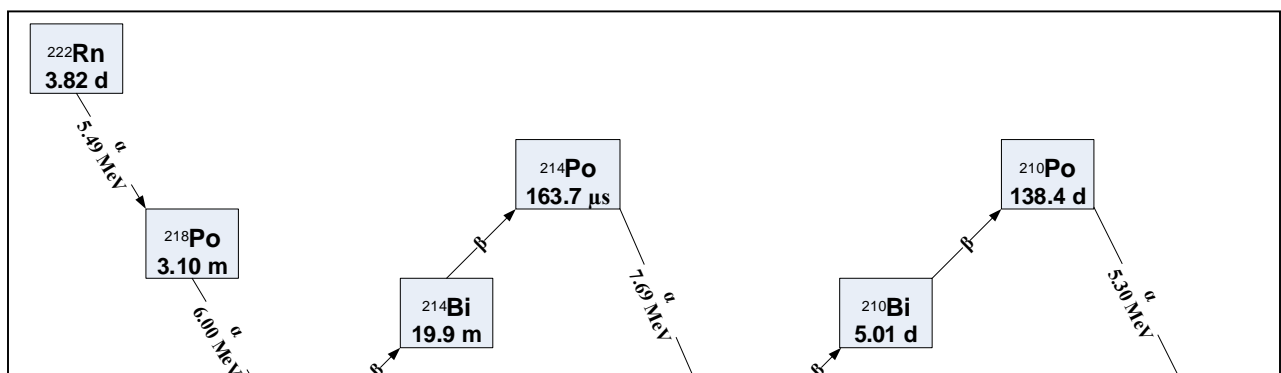


Figure 1

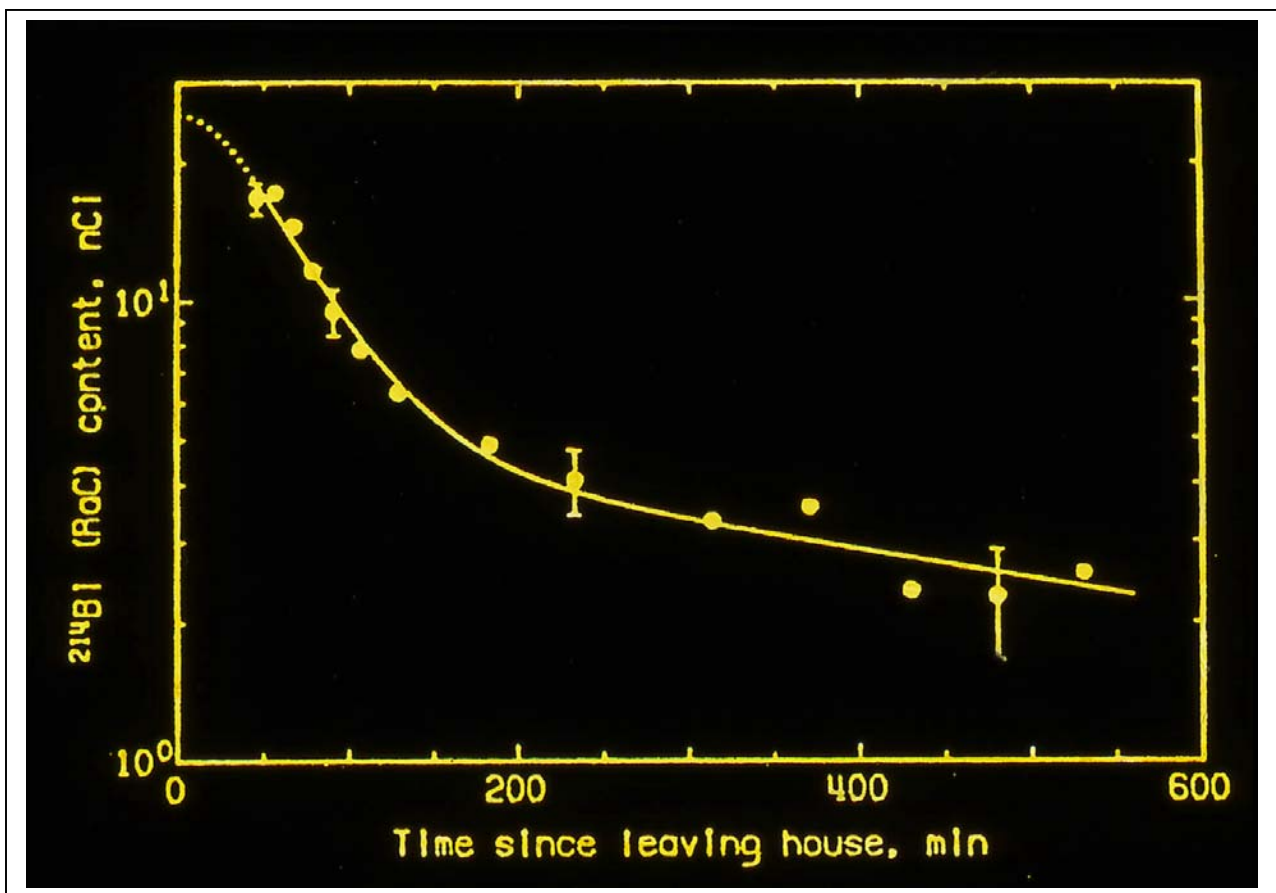


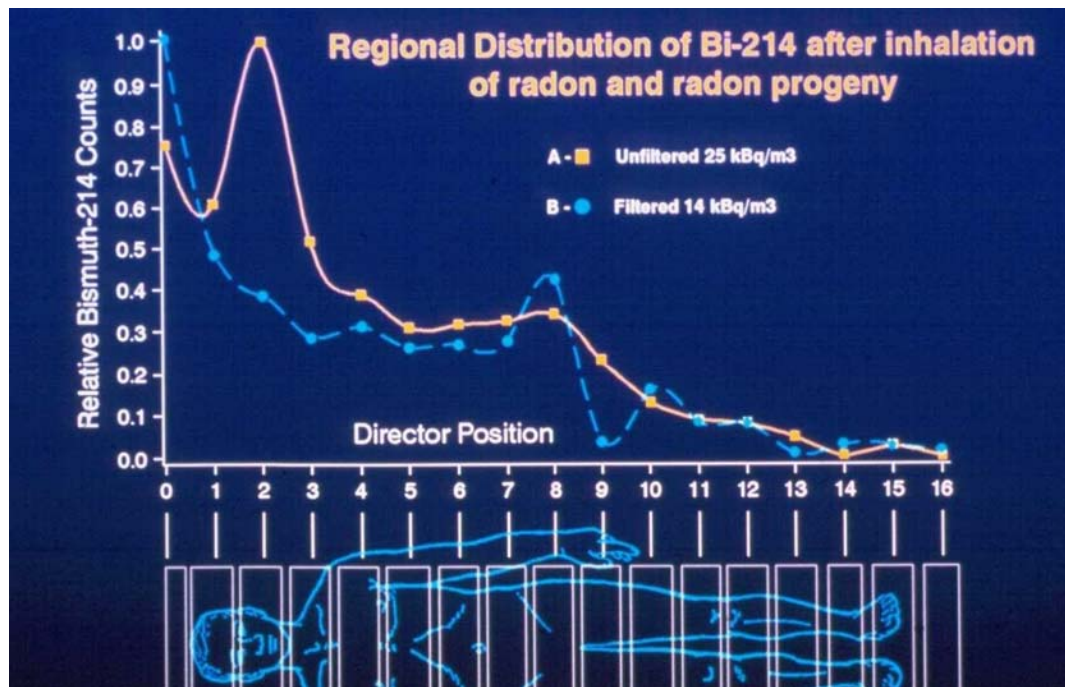
Figure 2







Figure 5



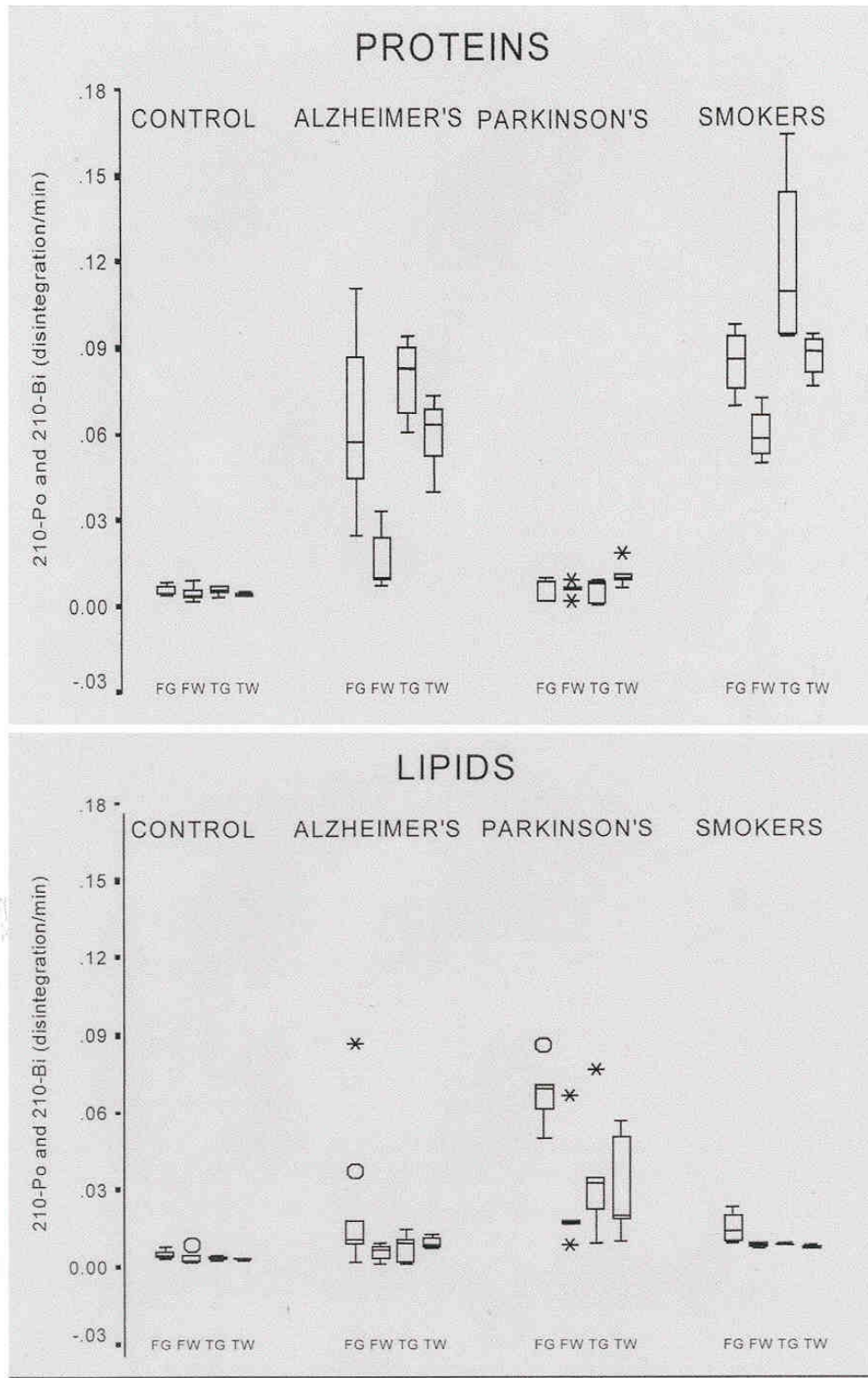


Figure 7

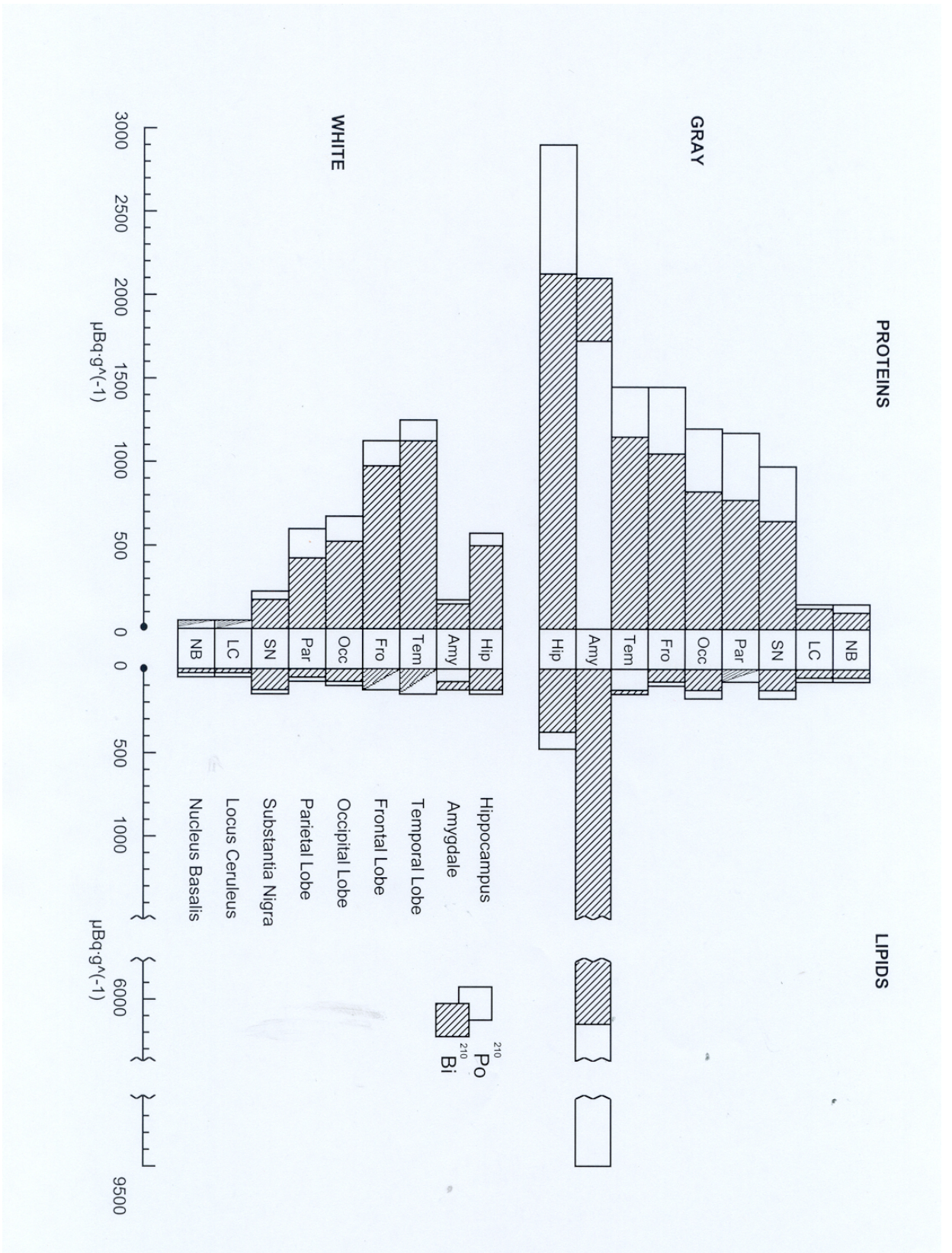


Figure 8

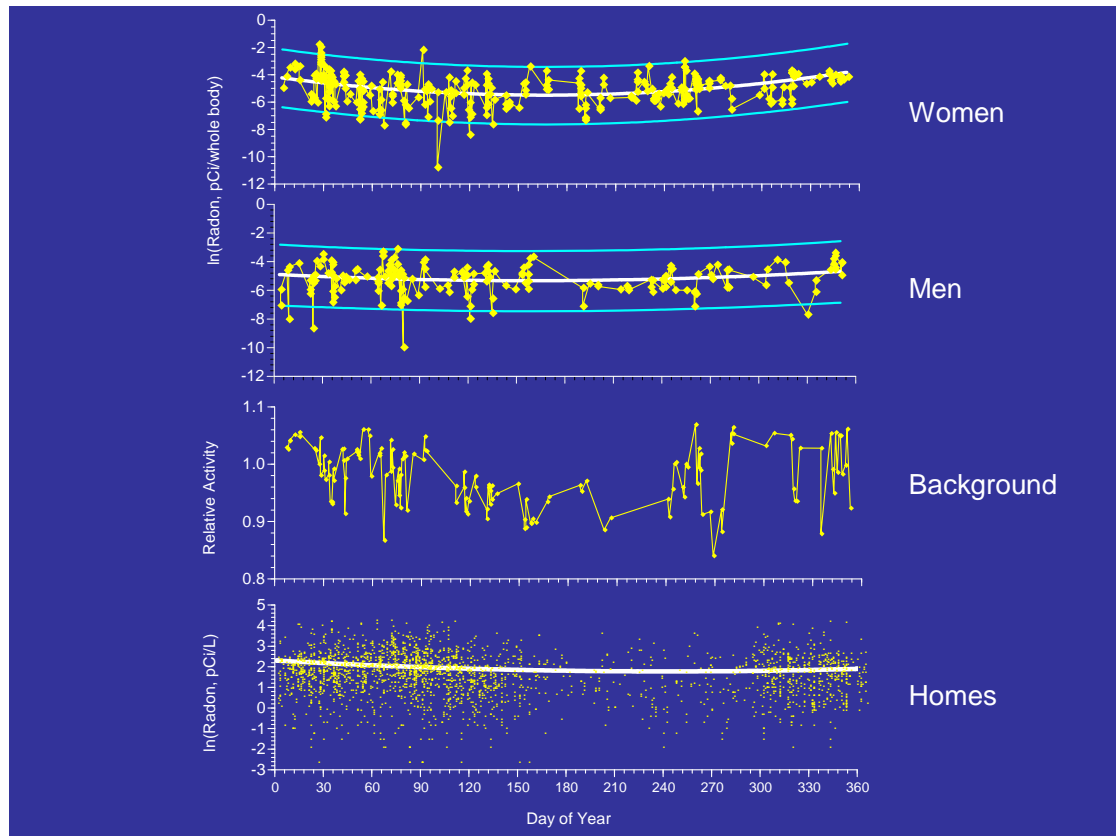


Figure 8

**Figure Captions-Lykken & Momcilovic**

Figure 1. Schematic representation of the major decay transitions in the decay of environmental radon ( $^{222}\text{Rn}$ ). Radioactive half-lives are shown with alpha particle decays indicated by downward sloping lines (energy in MeV) are illustrated by downward sloping lines. Beta particle emissions are indicated by upward sloping lines. Beta particle emissions are not shown because of multiple beta emissions from each beta emitter.

Figure 2. Bismuth-214 body content as a function of time after leaving the house for a subject with high dwelling radon concentration (see text and Rundo et al. 1978a).

Figure 3. Large cylindrical NaI(Tl) (28 cm x 10 cm) with lead collimation (shielding) used to collect  $^{40}\text{K}$  &  $^{214}\text{Bi}$  gamma emissions from the thighs of cyclists. (See text).

Figure 4. Illustration of cyclists riding outside and breathing in environmental radon emanating from the soil.

Figure 5. Subject in a whole body counter chamber with whole body counter equipped with lead collimators designed to measure regional gamma emissions. (See text and Fig. 5.)

Figure 6. Regional  $^{214}\text{Bi}$  net gamma counts, with effective half-lives, from a subject after a one-hour exposure to radon-laden atmospheres (RLA) under two measurement conditions. A ( )-Inhalation of an unfiltered RLA (25 kBq/m<sup>3</sup> Rn) and B ( )- RLA (14 kBq/m<sup>3</sup>) filtered through a mask designed to filter radioactive dusts and mists (3M 9925 Mask, 3M, St. Paul, MN 55144-1000<sup>1</sup>). Note the peak counts from the cranium and omentum regions in B.

Figure 7. Protein & Lipid  $^{210}\text{Po}$  and  $^{210}\text{Bi}$  in the protein (P) and lipid (L) fractions from the cortical gray (G) and subcortical white (W) matter from the frontal (F) and temporal (T) brain lobe in Alzheimer's disease (AD), Parkinson's disease (PD), cigarette smokers (S), and controls (C). Box-and whisker plots- The horizontal line inside the box represents the median. The lower boundary of the box is the 25th percentile and the upper boundary is the 75th percentile. The vertical lines (whiskers) show the largest and the smallest observed values that aren't outliers. Cases with values that are more than 3 box lengths from the upper or lower edge of the box are extreme values (\*). Cases with values that are between 1.5 and 3 box lengths from the upper or lower edge of the box are outliers (o). (SPSS for Windows 1993, SPSS Inc., Chicago, IL). In the protein fraction, AD-C differences and S-C differences were statically significant ( $\alpha=0.05$ ) for all four combinations of lobe and matter. In the lipid fraction, PD-C and S-C differences were significant for all combinations of lobe and matter (Momčilović et al. 2001).

Figure 8. Selective regional brain distribution of polonium-210 (□) and bismuth-210 (■) in the proteins and lipids of the gray and white brain matter in an Alzheimer's Disease victim ( $\mu\text{Bq g}^{-1}$  tissue).

Figure 9. Seasonal variation of mean radon concentrations (log transformations) measured over time periods indicated. Ninety-five percent (95%) prediction intervals shown.

(Women) Subjects (n=315) participating in community-based bioavailability studies over the period 1989-2004. (Men) Subjects (n=179) participating in community-based bioavailability studies over a period of 13 years. (Background) Daily average of daily whole body counter steel room background over the period 1995-2004. (Homes)

Individual home radon concentrations for Grand Forks, ND residents over the period 1988-2004.

Case #	Pathology #	Hospital	Pathology Report	Po-210 (cts/48 hrs/g)	Po-210 ( $\mu$ Bq/g)
2.	G-94-23192	B&W	yes	13	180
5.	33-528	FUH	no	94	1,300
8.	G-9444-20021	B&W	yes	18	250
10.	G-94-43312	B&W	yes	48	670
<b>Mean <math>\pm</math> SD</b>				<b>43 <math>\pm</math> 36</b>	<b>600 <math>\pm</math> 500</b>

**NOTE: Counts above 10 cts/48 hrs/g are considered to be significant.**

**B&W Brigham & Women's Hospital, Boston, MA**  
**FUH-Framingham Union Hospital, Framingham, MA**

Table 1. Preliminary results from a study of environmental radon-222 progeny in cancerous breast tissue. There were no control tissues as one of the participants died during the course of the study (Horne 1995).

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