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DETECTION OF ^{210}Pb IN THE LUNGS OF SMOKERS BY IN-VIVO GAMMA SPECTROMETRY

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Abstract

Since mainstream smoke is highly enriched in ^{210}Pb, alpha radiation from inhaled cigarette smoke particles has been proposed as a cancer-producing agent in cigarette smokers. ^{210}Po and ^{210}Pb have been observed in tobacco, cigarette smoke and in the lungs of smokers. Since ^{210}Pb is highly enriched in mainstream smoke, there have been estimates of yearly excesses of ^{210}Pb in the lungs of "one-pack-a-day" smokers of 3 - 10 pCi (0.11 - 0.37 Bq). The ORNL Whole Body Counter was used to verify this estimate by the methodology of high-resolution, in-vivo gamma spectrometry.

Measurements were made on 113 adult male non-radiation workers who have either smoked at least one pack of cigarettes per day for at least five years, or have never smoked cigarettes. An analysis-of-variance table was generated based on the Pb-ratio for each individual (described in this report) which revealed that there was no statistically significant increase in the amount of ^{210}Pb in the lungs of smokers over those of non-smokers. Sources of error are also discussed in this report.

Introduction

Since the reports from the Surgeon General's advisory committee on Smoking and Health were released in 1964, and the Surgeon General's Report on the Health Consequences of Smoking emerged in 1967, research into the carcinogenicity of tobacco smoking has intensified. In fact, epidemiological observations as far back as 1775 confirmed associations between smoking and cancer.[1]

It is difficult to identify the actual carcinogenic agents in tobacco and smoke since the mechanisms of cancer production are not well known. Alpha radiation from inhaled cigarette smoke particles has been proposed as a cancer-producing agent in cigarette smokers,[2] and exposure to alpha radiation from the short-lived radon daughters, ^{218}Po and ^{214}Po, has been considered to be the cause of bronchial cancer in uranium miners.[3] ^{210}Po and ^{210}Pb have been observed in tobacco, in cigarette smoke, and in the lungs of smokers,[4,5,6] therefore warranting serious consideration as possible agents of bronchial cancer among smokers.

Reports on the origin and distribution of ^{210}Pb and ^{210}Po in tobacco plants are contradictory. It has been concluded by some that the principle mechanism involves uptake into roots from soil and fertilizers;[7] however, others report deposition in rainfall.[8] Measurements made on 100 gm of raw tobacco leaves revealed approximately 1200 pCi (44.4 Bq) of alpha activity,[9] and published radiochemical data for inhaled mainstream smoke show about 0.036 pCi (1.33×10^{-3} Bq) of alpha per cigarette, or approximately 2.6 pCi (9.62×10^{-2} Bq) of alpha activity per gram of smoke.[2] From this information it has been proposed that a cigarette "habit" of one pack per day will give the smoker a yearly excess lung burden of 3 - 10 pCi (0.11 - 0.37 Bq) of ^{210}Po.[10]

When condensate samples of smoke are heated to drive off volatile constituents, the ^{210}Po is almost completely volatilized, whereas all the ^{210}Pb remains in the residue, thus indicating that ^{210}Po is inhaled in volatile form and ^{210}Pb is associated with the particulate fraction of mainstream smoke.[2] Since the ^{210}Pb is highly enriched in the insoluble particulate fraction, and the volatile smoke constituents are dispersed widely and removed rapidly from lung surfaces, one would expect a larger build-up of ^{210}Pb in the lungs of smokers than in the lungs of nonsmokers. (Trace amounts of ^{210}Pb are found in the lungs and bodies of most humans due to inhalation of atmospheric radon.) Based on this information, a task was undertaken to verify the above by the methodology of high resolution, in-vivo gamma spectrometry. Results obtained to date in this undertaking and a discussion thereof are presented in this report.

Background Information

^{210}Pb has a radiological half-life of 22.3 years, decaying to its granddaughter, ^{210}Po. The effective half-life of ^{210}Pb, total body, is 3.3 years.[11] Of the radiations produced by its decay, the only one detectable in the human body by the proposed methodology is a 46.5 keV (7.42×10^3 aJ) photon of relatively low yield. The whole body counter facility at Oak Ridge National Laboratory (ORNL) is equipped with a state-of-the-art system of low-energy photon detection--namely, a phoswich system--as well as a relatively new addition of an array of high-resolution, hyperpure germanium detectors. The operation, specifications and capabilities of the Ge array have been discussed in detail elsewhere,[12] but since this is the detector system that was used to obtain ^{210}Pb data on human subjects, a short description will be given.

Detection System

The system used for this project is a six-detector array of hyperpure germanium detectors totaling about 80 cm^2 active area (see Fig. 1). Each detector has approximately 7.5 mm active thickness. The signals from each detector are multiplexed together to function as a single counting unit. In the attempt to detect lung burdens of ^{210}Pb, the system is positioned over the right lung of each counting subject. The ADC (analog-to-digital converter) live-time for each count is one hour.

Since low limits of detection are required, emphasis is placed on reduction of competing background radiation, including measurable quantities of ^{210}Pb from atmospheric radon. In order to keep this contribution stable and to a minimum, the detector and subject are enclosed in a large, heavily shielded vault (36-cm-thick, pre-World War-II, naval armor plate, lined with lead and copper for degradation of characteristic X rays). The whole body counter facility is also equipped with a bottled air supply, that is stored for a number of weeks before use, and an airlock entrance system (which keeps the

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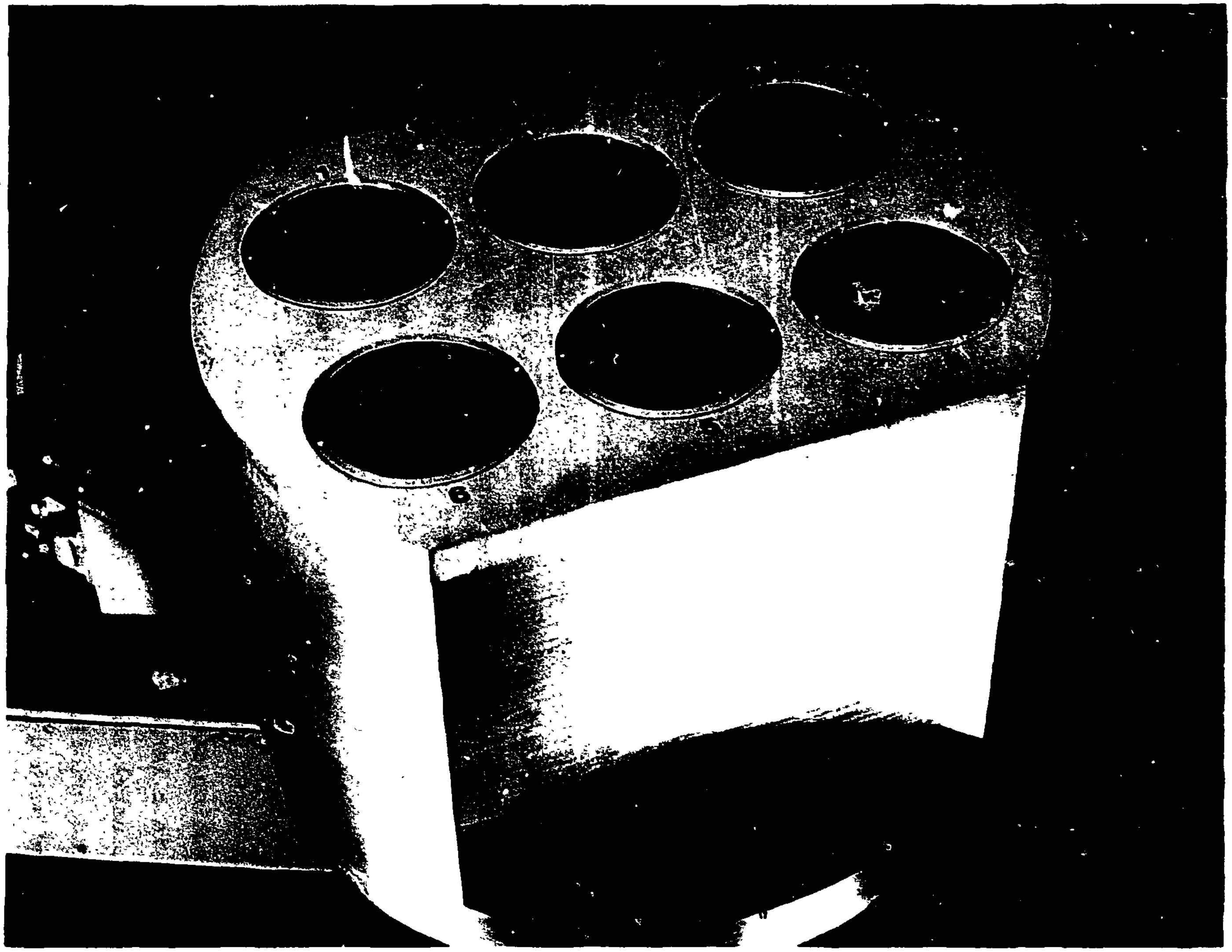

Figure 1: Bottom view of six-detector hyperpure germanium array.

inside of the vault under slightly positive pressure) so that fluctuations in atmospheric background are minimized. Room background counts in the 46.5 keV (7.42×10^3 aJ) region vary by less than 2% from day to day. The detector system itself becomes inefficient for collection of photons greater than 70 keV (1.12×10^4 aJ), therefore greatly reducing the interference from higher energy emitters incorporated within the body.

Analysis Procedure

Figure 2 shows typical human gross spectral data acquired by the germanium array. The small marker is positioned over the 46.5 keV (7.42×10^3 aJ) band, or the ^{210}Pb peak. As stated previously, most spectra show some ^{210}Pb, most of which disappears when detector background is subtracted. (Molecular sieve material in the detector contains a small amount of thorium and daughters.) One can see that the entire spectral region—from 0 to 130 keV (2.07×10^4 aJ)—is relatively flat. The majority of counts in this low-energy region are from Compton scattering of ^{40}K gamma rays in the subject's body. Rather than comparing total net counts in the ^{210}Pb region on the spectra of smokers and nonsmokers (which can fluctuate due to increases in body potassium and, therefore, increased low-energy scattering) we chose to compare what will be called the "Pb-ratio." This is merely the ratio of integrated counts in the ^{210}Pb region to integrated counts in a control region on the spectrum. In other words:

$$\text{Pb ratio} = \frac{\int_{\varepsilon_1}^{\varepsilon_2} (\text{counts-Pb region})\, d\varepsilon}{\int_{\varepsilon_3}^{\varepsilon_4} (\text{counts-control region})\, d\varepsilon} .$$

The control region was chosen arbitrarily to be from 115 to 125 keV (1.83×10^4 to 1.99×10^4 aJ), which is a relatively flat portion of the spectrum. The limits of the Pb region were chosen to be the

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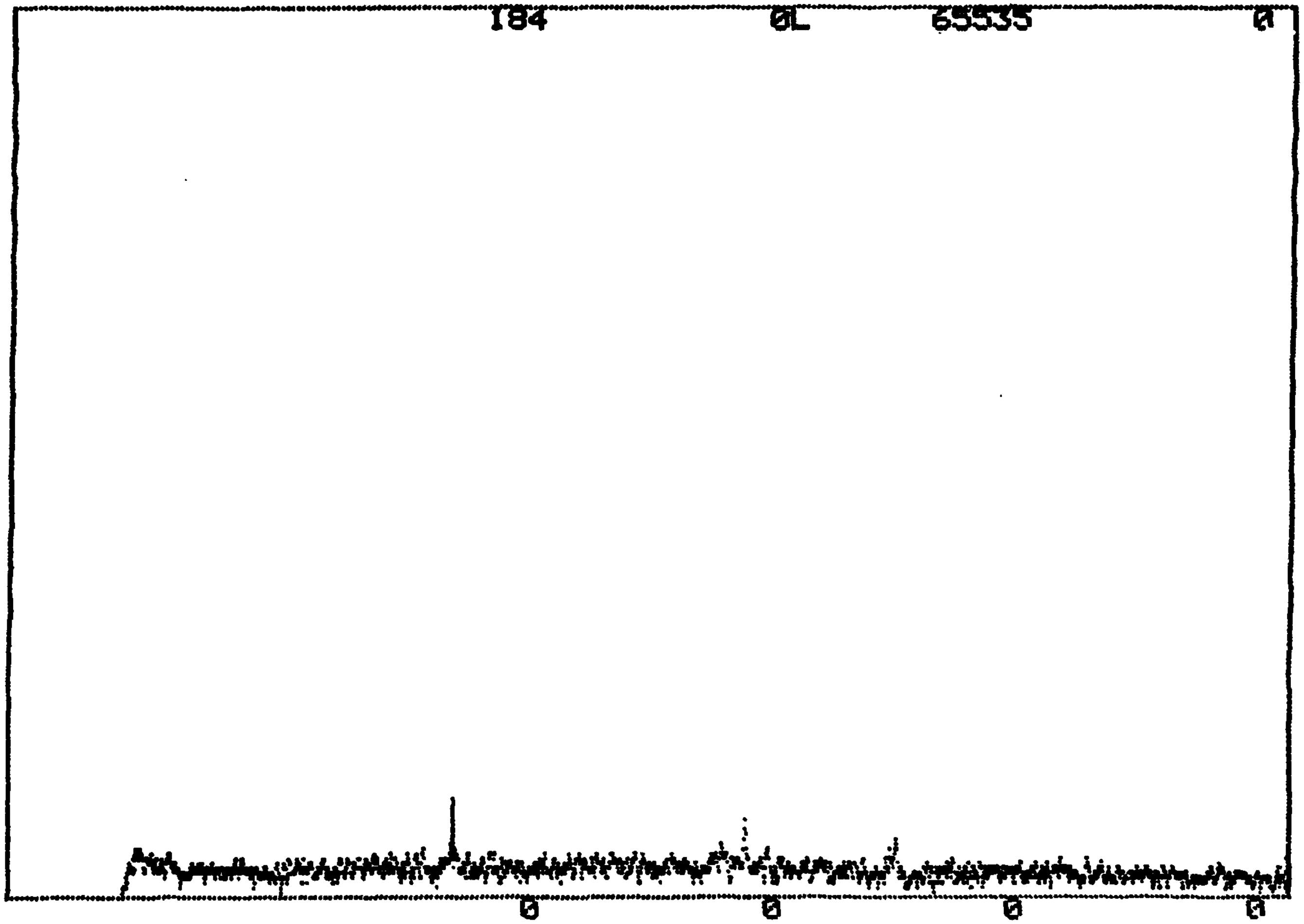

Figure 2: Gross human spectral data. Energy calibration is approximately 65 ev per channel for 2048 channels. *Marker is positioned over the 46.5 keV line.*

area around the 46.5 keV (7.42×10^3 aJ) peak at 1.18 of full width at half maximum (FWHM), which has been demonstrated to be the optimum region of integration for maximization of signal-to-noise.[12]

Subject Choice

Subjects used for this study were adult male nonradiation workers with undetectable amounts of internal contamination other than those occurring naturally in the body (^{40}K, ^{137}Cs). Smokers' Pb-ratios (sample size of 61) were obtained from subjects who have been smoking at least one pack of cigarettes per day for at least five years. The Pb-ratios for nonsmokers (sample size of 52) were obtained from subjects who have never smoked cigarettes, cigars or pipe. Before each count, subjects were instructed to shower, shampoo, and put on low-background, disposable jumpsuits.

Results and Conclusions

The Pb-ratios were determined for each of the subjects mentioned above and were compiled into two groups—smoker ratios and nonsmoker ratios. In order to test whether the differences in the mean of each sample can be attributed to chance or whether they are indicative of actual differences among the corresponding population mean due to increased ^{210}Pb in the lungs, a one-way analysis of variance test was used.[13] The mean of the observations in each sample was calculated by:

$$\bar{x}_i = \frac{1}{n_i} \sum_{j=1}^{n_i} x_{ij} \quad ;$$

the total sum of squares (TSS) by

$$TSS = \sum_{i=1}^{k} \sum_{j=1}^{n_i} x_{ij}^2 - \frac{\left[\sum_{i=1}^{k} \sum_{j=1}^{n_i} x_{ij} \right]^2}{\sum_{i=1}^{k} n_i} \quad ;$$

and the treatment sum of squares (TrSS) by

$$TrSS = \sum_{i=1}^{k} \frac{\left[\sum_{j=1}^{n_i} x_{ij}\right]^2}{n_i} - \frac{\left[\sum_{i=1}^{k} \sum_{j=1}^{n_i} x_{ij}\right]^2}{\sum_{i=1}^{k} n_i} ;$$

where x_{ij} = j^{th} observation from the i^{th} population, k = sample mean, i = the given sample, and n_i = the number of observations in that sample.

The null hypothesis to be tested is that the mean of the Pb-ratios (x_i) for the smoker and nonsmoker populations are equal. Based on the data obtained to date, the level of significance was chosen as $\alpha = 0.01$, and an ANOVA (analysis-of-variance) table was generated (see Table 1).

The F ratio for 1 and 73 degrees of freedom, for $\alpha = 0.01$, is 6.82. Since the F ratio shown in Table 1 is less than 6.82, the null hypothesis is accepted. In other words, there is no statistically significant increase in the Pb ratio among this population of smokers as compared to the population of nonsmokers.

The means and standard deviations for the two populations are shown in Table 2. It is important to note that the sample sizes are small. There are so many variables involved in a study such as this (e.g., brand of cigarettes smoked; filter or nonfilter cigarettes; smoking style; agricultural region where specific tobaccos are grown; type of fertilizer used; etc., as well as human variability) that it would be premature to disclaim a detectable increase in ^{210}Pb in the lungs of smokers by the above methodology. We are nonetheless continuing to collect Pb-ratio data in order to increase population sizes and develop improved confidence in results.

No attempt was made to arrive at a precise minimum detectable true activity (MDTA) of ^{210}Pb for the described counting system for various reasons. A "calibration" was performed using a point source

Table 1: ANOVA Table for ^{210}Pb Ratios

	SS	df	MS	F
Treatments	0.36	1	0.36	0.35
Error	115.42	111	1.04	
Total	115.79	112		

SS = sum of squares
df = degrees of freedom
MS = mean square
F = F-ratio ($F = \frac{\text{Treatment Mean Square}}{\text{Error Mean Square}}$)

Table 2: Pb-Ratio Sample Means and Standard Deviations

	Mean	Standard Deviation
Smokers (n=61)	1.38	±0.44
Nonsmokers (n=52)	1.27	±0.40

of ^{210}Pb placed in the lungs of a REMAB phantom, giving a MDTA value of 0.011 nCi (0.37 Bq) using:

$$\text{cal} * (k_\alpha + k_\beta)\sqrt{2C_B}$$

where cal = calibration factor (nCi/count), C_B = background counts taken from the population of non-smoking subjects, and k_α and k_β are constants given in tables for the normal distribution function. [α is the probability of concluding there is internally deposited activity when there is none, and β is the probability of concluding that there is no activity in the body when there actually is some.[14]] This value is not conclusive, as a distributed source within the lung would give an entirely different calibration value as well as a different MDTA. Since one of the unknown factors involved in this study is the distribution of ^{210}Pb in the lungs of each subject (contributing to a potentially large source of error in quantification) we have chosen to look at relative increases and decreases in the Pb-ratio without attempting to quantify the amount of ^{210}Pb in each subject.

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