Lead-210 and Polonium-210 in Human Tissues

It has recently been shown[1] that appreciable amounts of lead-210 and polonium-210 may occur in certain human foodstuffs as a result of deposition from the atmosphere of long-lived radon decay-products (Table 1). Absorption of these products may therefore occur by both inhalation and ingestion, but, although measurements of the activity of lead-210 in many human bone samples have been reported[3–6], the ratio of polonium-210 to lead-210 in bone and the activities of these nuclides in human soft tissues have been reported in only a few samples[1,3,7,8]. This communication describes the results of preliminary radiochemical estimations of polonium-210 and lead-210 in human bone and soft tissue samples.

Table 1. Radon Decay Products

	Nuclide	Half-life (ref. 2)	Decay
Radon	Rn-222 (Rn)	3·823 d	α
Short-lived daughters	Po-218 (RaA)	3·05 m	α
	Pb-214 (RaB)	26.8 m	β^-
	Bi-214 (RaC)	19·7 m	β^-
	Po-214 (RaC′)	160 μs	α
Long-lived daughters	Pb-210 (RaD)	20 y	β^-
	Bi-210 (RaE)	5·0 d	β^-
	Po-210 (RaF)	138·4 d	α
Stable lead	Pb-206	—	—

The method used was similar to that of Black[7]. Up to 80 g of wet tissue were digested with nitric and perchloric acids, care being taken to keep the temperature below 200° C because of the volatility of polonium. From a final solution, 0·5 N in hydrochloric acid, polonium was electrochemically deposited on silver foil and the α-activity was measured in a low background scintillation counter[9]. The specificity of the separation for polonium-210 was checked by α-spectroscopy of several of the foils, using the α-spectrometer which is described elsewhere[10]. By low-level β-counting for bismuth-210 in an aged deposit on silver from an equilibrated lead-210 solution, it was shown that less than 0·01 per cent of lead-210 was deposited under the plating conditions used in the tissue analyses. Hence, lead-210 was estimated in some of the tissue samples by deposition of the polonium-210 from the tissue solutions several months after the first deposition. Reproducibility was checked by several estimations of polonium-210 and lead-210 in samples from the same tissue. The overall yield was estimated by spiking one member of each of ten pairs of samples with an equilibrated lead-210/bismuth-210/polonium-210 solution and the measured overall yields for the first and second depositions were 90 $\pm$ 5 per cent and 90 $\pm$ 9 per cent respectively. The average activities of the tissues determined in this manner are as shown in Table 2.

Table 2. Po-210 and Pb-210 in Human Tissues

Tissue	Po-210 pc./100 g wet.		Po-210/Pb-210
Vertebra	1·7	(9)	0·82
			0·43
Liver	1·0	(4)	3·1
			1·8
Kidney	0·71	(2)	8·9
			4·1
Spleen	0·32	(3)	1·8
			0·4
Lung	0·30	(2)	—
Skeletal muscle	0·11	(5)	—
Testis	0·33	(4)	—

The figures in parentheses (Table 2) indicate the number of specimens of each type of tissue analysed. The ratios in column 3 are from individual determinations. Taking account of the uncertainties in the yields and the counting statistics, the probable errors of the individual determinations were less than 10 per cent for polonium-210 and 20 per cent for lead-210. The extreme values from separate determinations for a given type of tissue were within a factor of 2·5 of the mean value tabulated.

A few observations may be made in the light of these results. The average polonium-210 concentration in the bone samples is comparable with previously reported values and there are indications that the lead-210–polonium-210 series may not be in radioactive equilibrium in vertebræ. This is in keeping with the findings of Black[7] and Holtzman[3]. The latter author has suggested that the lead-210 concentration (per gram ash) in vertebræ is close to the average for the whole skeleton. On this basis, assuming an ash content in vertebræ of 21 per cent, a total skeletal ash of 2,800 g and, from the foregoing, a polonium-210/lead-210 ratio in vertebræ of 0·6, the skeletel burden of lead-210 is approximately 400 pc.

The results of tracer studies with animals and a few human beings, summarized in the I.C.R.P.[11] tables, have indicated that polonium-210 may be concentrated in the soft tissues, particularly liver and kidney, and the foregoing measurements suggest that this is also the case at lower levels, the polonium-210 being measurably in excess of lead-210. α-spectroscopic measurements on other tissue samples[1] have demonstrated that polonium-210 is in excess of other α-emitters. The measurements reported here give average values for polonium-210 activity in the major soft tissues higher than those spectroscopic determinations, probably because of volatilization of polonium in the dry-ashing procedure required for the spectroscopy.

If the concentration in those types of soft tissue not measured here is comparable with that measured in skeletal muscle, the foregoing results indicate an average body burden of polonium-210 in the range 330–450 pc. with at least 20 per cent of the activity in the soft tissues. The upper and lower limits correspond to the assumptions that the more highly mineralized cortical portion of the skeleton has a polonium-210/lead-210 ratio equal (*a*) to that in the trabecular region, taken here as 0·6, or (*b*) to unity, respectively.

The radiation dose rate from the concentrations of lead-210 (β-energy, 17 keV maximum) and polonium-210 (α-energy, 5·30 MeV) in tissues, reported here, is due predominantly to the polonium-210 α-particle. The *in vivo* activity of bismuth-210 (β-energy 1·16 MeV maximum) is difficult to measure because of its rather short half-life. However, since bismuth does not appear to be concentrated very markedly in any of the larger organs[11], it seems unlikely that redistribution of bismuth-210 following the decay of lead-210 would affect the validity of the foregoing statement. A polonium-210 concentration of 0·1 pc./100 g wet. tissue gives a dose rate of approximately 1 mrem/y, so estimates of the dose rate to the various soft tissues from the measured activities may readily be obtained from Table 2. Comparison of these dose rates with those from radium-226 plus short-lived daughters and the radium-228 series in human bone and soft tissues[12] indicates that the rem dose rate from polonium-210 in bone may be comparable with the rem dose rate from those nuclides and, in soft tissues, the rem dose rate from polonium-210 may be more than an order of magnitude greater than that from radium-226 plus short-lived daughters and the radium-228 series.

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[1] Hill, C. R., *Health Phys.*, **8**, 17 (1962).
[2] Stehn, J. F., *Nucleonics*, **18**, 11 (1960).
[3] Holtzman, R. B., *ANL*, 6199 (1960).
[4] Hursh, J. B., *Science*, **132**, 1666 (1960).
[5] Krause, D. P., *ANL*, 6398 (1961).
[6] Hill, C. R., and Jaworowski, Z. S., *Nature*, **190**, 353 (1961).
[7] Black, S. C., *Health Phys.*, **7**, 87 (1961).
[8] Holtzman, R. B., *ANL*, 6297 (1960).
[9] Turner, R. C., Radley, J. M., and Mayneord, W. V., *Brit. J. Radiol.*, **31**, 397 (1958).
[10] Osborne, R. V., *Nature*, **199**, 143 (1963).
[11] *Intern. Comm. Radiol. Protect.*, Comm. II Rep. (Pergamon Press, London, 1959).
[12] *United Nations, Rep. Sci. Comm. Effects of Atomic Radiation A/5216*, 215 (1962).