FACT SHEETS
1980

702522

FACT SHEETS

CENTER FOR HUMAN RADIOBIOLOGY

REPOSITORY ARGONNE / CHR
COLLECTION RECORDS RELATING TO INDUSTRIAL/MEDICAL EXP. TO RADIUM
BOX No. BOX 121 ASH BINDER
FOLDER #33 - CHR FACT SHEETS REVIEW COMMITTEES HISTORY OF CHR from ANL REPORTS

OCTOBER 1980

0019768

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Progress of Medical Studies

Radium cases (5 person-years)

Medical examinations are continuing as before; six are ordinarily scheduled each week, and the times of examination are coordinated with times of measuring radium burden. In addition to routine re-examinations this year, we have seen 30 new previously unmeasured radium workers. Frequency of re-examination of radium patients is determined by the radium burden and the age of the patient; however, all persons under follow-up are contacted at least annually by telephone or letter with questionnaire. Since a major thrust of the program is correlation of pathologic changes with radium burdens, ascertainment of radium content is of prime importance and efforts are made to obtain the best feasible medical profile of each person in the study.

1. A few individuals remain too anxious to be willing to visit the Center, and a small number have declared their active hostility. The mobile unit has enabled us to obtain measurements of some of these persons in several parts of the country. Several person have willed their bodies to the Center and the exhumation program has been opened up in the midwest.

2. In addition to an exhaustive medical history and physical examination, a full profile of biochemical analyses (SMA-24 and thyroid profile, plus others as indicated) and a full set of skeletal x rays including special views of mastoids and paranasal sinuses on at least one examination are secured. Careful audiograms are taken, including bone conduction, on account of the vulnerability of mastoids to radium damage.

The coding of pathologic states for computer retrieval, according to the standard nomenclature (SNOP) has been done on the 5400 cases on whom files exist in the Center, yielding over 150,000 entries. Most of the present work now consists in coding conditions found in hospital records that are being obtained, and in first examinations of new entries. About half of these files contain minimal information (this might arbitrarily be defined as less than 10 entries) and the remainder contain an average of 47, with apparently quite uniform recording of common conditions likely to be identified in a complete examination.

A file of tumor diagnoses has been compiled from the SNOP listings. This file includes 804 verified entries in 757 cases. By keeping this tumor diagnosis file up to date it will be possible to retrieve current data on any subgroup of patients in the radium study. The development of the SNOP tumor file makes it easier to investigate incidence as well as mortality. For example, an investigation of breast cancer incidence in measured radium workers before 1930 (numbering 779) has been published. This is of particular interest since no

Prepared by E. E. Adams, M.D. Argonne National Laboratory

0019769

human data on breast cancer association with internally administered radionuclides is currently available.

Our collection of slides and tissue blocks is being expanded to include as large a segment of our study population as possible. One result of this pathology review was the publication by Littman, Kirsh, and Keane in 1979 of a definitive paper on radiogenic tumors of the mastoid and paranasal sinuses. A similar effort is now being made in regard to bone sarcoma.

Former thorium workers (3 person-years)

Medical and radioactivity examinations have been done on 252 of the over 400 living persons (all males) who worked at a thorium refining plant for one year or longer. Measurements of body radioactivity and thoron exhalation show a considerable difference between individuals and reasonably good reproducibility between measurements on individuals made at different times.

The vivo measurements that are made are not yet translatable into body and organ thorium content, therefore each person at the time of examination is advised of the willed body program with a frank explanation of the scientific reasons for it. Several individuals have indicated willingness to participate.

The same medical and laboratory examinations are given as in the radium study, with the exception of detailed audiometry and the x-ray survey, which in thorium studies is limited to chest, abdomen, and any other films that are required for particular indications that become evident at examination. In addition, all of the participants in this study are given respiratory function testing including CO diffusion rates and total lung volume as well as the routine spirometric and voluntary ventilation measurements. It is assumed that the major mode of intake of thorium has been by inhalation.

October 1980

CENTER FOR HUMAN RADIOBIOLOGY
Fact Sheet on
Breast Cancer in Female Radium Dial Workers First Employed Before 1930

In 1979 all listings of malignancy in persons both living and dead were abstracted from our SNOP computer files and established as a separate file after chart verification that a true malignancy probably did exist. This provided a useful base for in-depth evaluation of the incidence and mortality rates of various malignancies in our study populations.

Using this file a study of breast cancer in radium dial painters was prepared. Female radium dial workers first employed before 1930 were analyzed for breast cancer mortality and incidence using method and rate tables described by Manson and the Mantel-Haenszel summary chi-square test for significance. Of 1180 located women, 736 were measured to estimate radium intake. This measured group was analyzed for breast cancer mortality and incidence according to four possible risk factors: radium intake doses, duration of employment, age at first exposure, and parity. The located women had a mortality ratio of 1.51 ($p < 002$). The measured women showed a significant excess of breast cancer incidence and mortality only among those women with a radium intake of 50 μCi or greater. Although not significant, incidence and mortality ratios were slightly higher for nulliparous women. See the following Table.

		Mortality †				Incidence †			
Risk Factor		Number of cases	Breast Cancer Deaths Observed	Breast Cancer Deaths Expected	Mortality Ratio	Number of cases	Breast Cancer Cases Observed	Breast Cancer Cases Expected	Incidence Ratio
Intake dose	≥ 50μCi	97	6	1.04	5.75*	97	9	2.72	3.31*
	< 50μCi	639	5	7.80	0.64	631	16	20.68	0.77
Age at first exposure	< 19 yrs	463	6	5.25	1.14	458	15	14.12	1.06
	≥19 yrs	273	5	3.60	1.39	270	10	9.29	1.08
Duration of exposure	≥50 wks	367	5	4.28	1.17	362	12	11.40	1.05
	< 50 wks	369	6	4.56	1.31	366	13	12.01	1.08
Live Births	0	248	6	2.99	2.01	245	13	7.75	1.68
	≥ 1	488	5	5.86	0.85	483	12	15.65	0.77
Total		736	11	8.84	1.24	728	25	23.40	1.07

† try into study at year of first measurement

* $p < 0.001$

Prepared by Evelyn E. Adams, M.D. Argonne National Laboratory

0019771

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Excretion Rates of ^{210}Pb and ^{210}Po for Workers at the Canon Industrial Park

We determined urinary excretion rates of ^{210}Pb and ^{210}Po, and also of ^{226}Ra, for 12 persons (10 men and 2 women) who were employed at the time of measurement at an industrial site formerly used for the processing of uranium ores and for the dumping of residues. A survey[1] of this site showed the presence in the buildings of high concentrations of radon (up to 200 pCi/L) and of radon daughters ranging from 0.06 to almost 2 WL (Working Levels*). High levels of uranium and ^{226}Ra were found in dust in the buildings.

Exposures to radon and its daughters were calculated from the urinary excretion rates of ^{210}Pb and from the properties of the nuclides of the radon series and those of ^{210}Pb as described in a metabolic model. It was assumed that the ^{210}Pb originated from the radioactive decay of short-lived radon daughters in the air, from ^{210}Pb itself present in the air, and from the radioactive decay of ^{222}Rn dissolved in body fluids. The latter source may contribute 50 to 80% of the ^{210}Pb excreted.

The geometric means and their geometric standard deviations (σ_g) of the urinary excretion rates of ^{210}Pb, ^{210}Po and ^{226}Ra were 0.86 pCi d^{-1} (σ_g 2.0), 1.93 pCi d^{-1} (σ_g 3.9) and 0.086 pCi d^{-1} (σ_g 6.4), respectively. The mean values were 4 to 9 times the normal rates of about 0.2, 0.2 and 0.02 pCi/day for the three nuclides respectively.

The exposures estimated from the urinary excretion rates of ^{210}Pb were based on a conversion factor of 6 pCi ^{210}Pb day^{-1} WL^{-1}. This factor was adjusted for the time elapsed between the last exposure of the subject to the high radon atmosphere and the collection of the urine sample.

The estimated exposures during an 8-hour working day ranged from 0.06 to 1.1 WL with a geometric mean of 0.14 WL (σ_g 2.3). The mean value was essentially identical to that expected from exposure to the radon daughter concentrations in the respective buildings measured two years prior to our study, namely 0.15 WL (σ_g 1.54).[1] Despite many uncertainties (in the details of the model, in the exposure patterns, and in the use of data on radon and its daughters obtained two years earlier), the individual exposures estimated by the two methods are in reasonable agreement, viz. to within a factor of 3 for 9 of the 12 subjects.

---

* Working Level is defined as the concentration of any combination of short-lived radon daughters (^{218}Po, ^{214}Pb and ^{214}Bi) in one liter of air that results in 1.3×10^5 MeV of potential alpha energy.

Prepared by R. B. Holtzman

Argonne National Laboratory

0019772

The ratios of the excretion rates of ^{210}Po to those of ^{210}Pb were used to estimate the biological half life of ^{210}Pb in the body. The value of about 1350 days is in agreement with that estimated from external counting of ^{210}Pb in uranium miners, although it is substantially less than values estimated from postmortem studies on bone containing ^{210}Pb produced from skeletal ^{226}Ra.

The use of ^{210}Pb excretion rates appears to be useful for estimating exposures to radon and its daughters. The method may be refined by comparison of excretion data on these and other subjects with more accurately determined exposures to radon and radon daughter concentrations.

Reference

1. Oak Ridge National Laboratory, Formerly Utilized MED/AEC Sites Remedial Action Program - Radiological Survey of the former Vitro Rare Metals Plant, Canonsburg, Pennsylvania. DOE/EV-0005/3 (1978).

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Relative Activities of ^{228}Ra and ^{226}Ra in Radium Dial Co. Workers

From 1918 to 1936, the Radium Dial Company operated a dial-painting plant in Illinois, and the former employees of this firm constitute a major cohort in the Center's studies of the health effects of radium. Both ^{228}Ra (half-life 5.75 yr) and ^{226}Ra (half-life 1600 yr) were used in luminous compounds in the early years of the dial-painting industry in the United States, and determination of the relative activities of these two radium isotopes in Radium Dial Company workers is important to the dosimetry of this study cohort.

Earlier Studies

Earlier studies of these workers obtained contradictory information on the use of ^{228}Ra by the Radium Dial Company, e.g., a) ^{228}Ra was used before 1926 (possibly in 1920) but not after 1926,[1] and b) ^{228}Ra was never used.[2] Daughter products of ^{228}Ra were detected by gamma-ray spectrometry in vivo in two workers[2,3] but the presence of ^{228}Ra in these two was ascribed to a probable iatrogenic origin.[3] Radiochemical determinations of ^{228}Ra and ^{226}Ra in bone samples of several of these workers had been made by earlier investigators and reports of the results were made available to the Center.

Current Studies

Through the Center's tissue-acquisition program additional bone samples have been collected and distributed by the Bone Microdosimetry Group and analysed for ^{228}Ra and ^{226}Ra by the Radioactivity Measurements Group. Because of the short half-life (5.75 yr) of ^{228}Ra, and the long times (55-60 yr) that have elapsed since the dial workers were exposed to it, the reduction in the activity by physical decay has been very large (factor of 750-1400). An exceptionally sensitive and highly specific method has to be used to determine the residual content of ^{228}Ra.[4]

Results

$^{228}Ra/^{226}Ra$ activity ratios were obtained in samples of bone from 15 workers first employed in the years 1920-1924. Measured activities were corrected for environmental levels of ^{228}Ra and ^{226}Ra in bone, and $^{228}Ra/^{226}Ra$ ratios at time of employment were calculated by correcting for radioactive decay.

In five persons whose work periods fell entirely within the years 1920-1923, the initial ratios ranged from 0.12 to 0.34 (median 0.17). In seven persons whose work periods began in 1920-1923 but extended into 1924 or beyond, the initial ratio ranged from 0.013 to 0.23 (median 0.09).

Prepared by A. T. Keane

Argonne National Laboratory

0019774

In one person employed in 1922 but whose length of employment is unknown, the initial ratio was <0.01. In two persons first employed in 1924, the median initial ratio was <0.01.

The results are consistent with an intake of 0.2 ± 0.1 (S.D.) μCi ^{228}Ra per μCi ^{226}Ra in the years 1920-1923 and negligible exposure to ^{228}Ra after the year 1923.

References

1. Aub, J. C., Evans, R. D., Hempelmann, L. H., and Martland, H. S., The late effects of internally-deposited radioactive materials in man. Medicine 31, 254,303 (1952)

2. Finkel, A. J., Miller, C. E., and Hasterlik, R. J., Radium-induced malignant tumors in man. In Delayed Effects of Bone-Seeking Radionuclides, C. W. Mays et al., Ed., University of Utah Press, Salt Lake City, 1969, p. 215

3. Miller, C. E., Finkel, A. J., and Hasterlik, R. J., Evaluation of clinical effect versus dose in radium dial painters. COO-2088-1, 1969, p. 8

4. Kuchta, D. R., Rundo, J., and Holtzman, R. B., Low-level determination of skeletal ^{228}Ra and ^{228}Th in the presence of gross amounts of ^{226}Ra, Proc. IAEA Symp. on Biological and Environmental Effects of Low-level Radiation, Vol. II, p. 183, Vienna, IAEA, 1975.

0019775

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Studies of the Gastro-intestinal Absorption of Plutonium in the Dog

In our investigation of the gastrointestinal absorption of plutonium in mice and rats, the values obtained for the fractions retained in vivo were about 2×10^{-3} and 3×10^{-3}, respectively, a factor of about 100 higher than the values obtained by other investigators.[1,2] These retentions were independent of the oxidation state of the plutonium administered (IV or VI), the medium in which it was administered (0.01 M sodium bicarbonate, 0.17 M citrate buffer, or 0.01 M nitric acid) and the feeding regimen (ad libitum or starved).

Additional studies of plutonium absorption have been made with the dog as the experimental animal. The plutonium administered was a mixture of ^{237}Pu (46 d) and ^{239}Pu (24,000 y), its concentration was 8×10^{-11} g/mL (the same as the MPC for ^{239}Pu), it was in the VI state, and the medium was 0.01 M sodium bicarbonate. (We had shown previously that the VI state is formed when drinking water is chlorinated,[3] and dilute bicarbonate simulates the composition of Lake Michigan water.) Determinations of the plutonium in tissue samples were made by counting Np K x-rays (emitted in the decay of ^{237}Pu) with a phoswich detector. The solution was administered in a gelatine capsule to six adult male beagles that were fasted for 21 hr prior and 3 hr subsequent to the administration. The dogs were sacrificed after 28 days. Tissues taken for analysis were the liver and gall bladder, and six groups of bones, viz. skull, cervical and thoracic vertebrae, lumbar and sacral vertebrae plus innominates, ribs and scapulae, femora and humeri, and remaining skeleton (primarily the distal appendicular bones).

The accuracy of the plutonium determinations was confirmed by comparing the uptake of ^{237}Pu in a particular bone sample with that of ^{236}Pu in the same sample. ^{236}Pu was an impurity in the ^{237}Pu. The method used to determine the ^{236}Pu was alpha spectrometric-isotope dilution. The fractions of administered ^{326}Pu and ^{237}Pu in the bone agreed to within the analytical errors of the determinations, ±10%.

The value obtained for the mean fractional absorption was $(6.5 \pm 1.4) \times 10^{-3}$, based on the data for five of the six dogs. (The value for the sixth dog was $(3.6 \pm 0.4) \times 10^{-3}$ and was clearly inconsistent with the other five.) The absorption in the dog is about a factor of four lower than those in the mouse and the rat and the difference is attributed to a lower value for the ratio of the length of the small intestine to body weight in the dog. (The values of this ratio in the mouse and the rat are more comparable to that of man than to that of the dog.) The significance of the value we obtained for absorption in the dog is that it is a factor of 20 higher than the value used to establish the MPC for plutonium in drinking water.

Prepared by R. P. Larsen

Argonne National Laboratory

0019776

The distribution of plutonium in the dog subsequent to gastrointestinal absorption is given in the table and compared with the distribution subsequent to intravenous injection.[4] The forms administered were Pu(VI) in 0.01 M bicarbonate and Pu(IV) in 0.1 M citrate buffer, respectively. The difference between the amount in the liver relative to that in the skeleton shows that the absorbed plutonium is metabolized differently from the injected plutonium. The particular importance of this observation is that the conclusions drawn from many studies of plutonium metabolism *in vivo* may be in error. In such studies it has been standard practice to incorporate plutonium in mammals by injecting Pu(IV) citrate intravenously. There is a possibility that plutonium in blood which entered a mammal as the result of G.I. absorption and/or inhalation may not be in the IV state, or if it is in this form, its behavior is altered by citrate complexation.

Table I. Plutonium Distribution in the Dog After Gastrointestinal Absorption of Pu(VI) Bicarbonate and Intravenous Injection of Pu(IV) Citrate, expressed as % of the total amount in the skeleton.

	Method of administration	
Tissue	G.I.	I.V.
Skull	6	12
Vertebrae & Innominates	48	40
Ribs & Scapulae	23	17
Femora & Humeri	13	19
Remaining skeleton	10	12
Liver	114	33

References

1. Katz, J. et al., Am. J. Roentgenol. Radium Therapy Nucl. Med., 73, 303, (1955)

2. Weeks, M. H. et al., Radiat. Res., 4 339, (1956)

3. Larsen, R. P. and Oldham, R. D., Science 201, 1008 (1978)

4. Stover, B. J. et al. in Radiobiology of Plutonium, B. J. Stover and W. S. S. Jee, eds. J. W. Press, Salt Lake City, pp 149-169 (1972)

Publications

Larsen, R. P. and Oldham, R. D., "Plutonium in Drinking Water: Effects of Chlorination on Its Maximum Permissible Concentration, Science 201, 1007, (1978).

Larsen, R. P., Oldham, R. D., Bhattacharyya, M. H., Moretti, E. S. and Austin, D. J., "Gastrointestinal Absorption of Plutonium," Radiat. Res., accepted.

Larsen, R. P., Oldham, R. D., Bhattacharyya, M. H., Moretti, E. S. and Austin, D. J., "On the Gastrointestinal Absorption of Plutonium," paper presented at Workshop on Measurements and Interpretation of Actinide Accumulation by Man, Snowbird, Utah, Oct 15-17, 1979. Proceedings to be published.

0016177

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet

Tumor Mechanisms

Rationale

Probably the most meaningful risk estimates of low doses of alpha emitters to large populations will come from human studies when precise measurements of the dose and damage can be correlated in the same individuals. The radium patients provide such a source of information for radioisotopes which deposit in bone, but additional knowledge of the mechanisms involved are necessary to extrapolate to low doses and to low dose rates. Basic mechanisms of bone tumor induction by alpha particles are, therefore, being studied by (1) direct alpha irradiation of mammalian cells in culture, (2) light and electron microscopy of the cells at risk in normal human adult bone, and (3) observation of changes in bones from radium patients at the sites where bone tumors most frequently develop.

Alpha Particle Irradiation of Mammalian Cells in Culture

Results to Date

We have shown for the first time (a) that mammalian cells can be transformed to become malignant with a dose dependent frequency when irradiated directly by alpha particles in culture,[1] (b) the mean lethal dose corresponds on average to the traversal of 14 alpha particles through a flattened cell nucleus and not to a single alpha particle as has commonly been supposed,[2] and (c) expression of the transformed phenotype can be inhibited by co-cultivation with untransformed cells.[3]

We have also shown that some human tumor cells (TE-85) are less sensitive than normal human diploid cells (NFS) to inactivation by alpha particle irradiation.[4] If this is a general finding, most of the earlier work on alpha particle inactivation needs to be re-evaluated because of the mistaken identity of T-1 cells, which have now been shown to be tumor cells (Hela) and not normal human kidney cells as supposed.[5]

Specific Aims and Long-Term Goals

To determine the shape of the dose responses (for cell transformation and cell killing) for alpha particles as a function of (a) cell geometry, (b) dose rate, (c) cell density and (d) LET. To determine the inactivation cross-section for different normal human diploid cells and different human tumor cell lines. To examine the mechanisms by which untransformed cells can suppress expression of the transformed phenotype in vitro.

Cells at Risk for the Induction of Bone Tumors in Man

Results to Date

For the first time, the geometry of the cells at risk for the production of bone tumors by alpha particles has been determined from electron micrographs of a normal human femur. Cells on the endosteal surface were found to be similarly flattened to those irradiated in culture. Electron micrographs of a femur from a high-level radium patient showed similarly flattened cells, but the cells were separated from the bone mineral by a fibrotic layer up to 50 μm thick. The intervening layer greatly reduces the effective dose to the endosteal cells, bringing the dose closer to that found to be

Prepared By: E. L. Lloyd Argonne National Laboratory

0019778

maximally effective for transforming cells in vitro (200-300 rads). This work suggests that the current practice of limiting the calculation of the effective carcinogenic dose to a layer 0-10 μm from bone mineral is unwarranted. In addition, this particular geometry probably means that more than one alpha particle, on average, is required to produce a carcinogenic event and may account for the steeper dose response in the radium workers when the incidence of osteosarcoma is compared with the more nearly linear dose relationship observed for carcinomas in the same workers - carcinomas arise from more rounded epithelial cells. A report of this work has been submitted for publication.[6]

Specific Aims and Long-Term Goals

The work, so far, has been confined to the examination of cortical bone from the femur; future work will include trabecular bone of the femur and the vertebrae in both control and radium bone. It is hoped that this will help us to understand why bone tumors in humans are mostly confined to the long bones, whereas the vertebrae in other species are frequently involved.

References

1. E. L. Lloyd, A. Gemmell, C. B. Henning, D. S. Gemmell, and B. J. Zabransky, Transformation of mammalian cells by alpha particles, Int. J. Radiat. Biol. 36, 467-478 (1979).

2. E. L. Lloyd, A. Gemmell, C. B. Henning, D. S. Gemmell, and B. J. Zabransky, Cell survival following multiple-track alpha particle irradiation, Int. J. Radiat. Biol. 35, 23-31 (1979).

3. E. L. Lloyd, M. A. Gemmell, and C. B. Henning, Inhibition of growth of human osteosarcoma cells in culture by normal human fibroblasts, American Society of Clinical Oncology, San Diego, California, May 26-27, 1980 and 71st Annual Meeting of the American Association for Cancer Research, San Diego, California, May 28-31, 1980. (Abstract)

4. E. L. Lloyd and M. A. Gemmell, Survival of human osteosarcoma cells and normal human fibroblasts following alpha particle irradiation, Radiological and Environmental Research Division Annual Report, July 1979-June 1980. (In Press)

5. Walter A. Nelson-Rees, Robert R. Flandermeyer, and David W. Daniels, T-1 cells are HeLa and not of normal human kidney origin, Science 209, 719-720 (1980).

6. E. L. Lloyd and C. B. Henning, Cells at risk for the production of bone tumors in radium-exposed individuals: An electron microscope study (1980). (Submitted for Publication)

0019779

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet

Theory of the Induction of Bone Cancer by Ionizing Radiation

In our attempt to find a relatively simple model of bone cancer induction which is consistent with known data for alpha and beta particles in man, animals, and cell culture, we have extended the Marshall-Groer model of 1977 (two initiating events in one cell) to the level of the DNA molecule in order to test its consistency with fundamental mechanisms of ionization and deletion of information.

The Marshall-Groer model gave us a linear-quadratic-plateau response of tumor rate versus endosteal dose which fits both the time dependence and the dose dependence of the osteosarcomas due to radium in man and in dog. The DNA model (2-4 ionizations per initiating event) promises to give us the linear-square-plateau dependence of the initiation cross section upon LET which yields the familiar peak in RBE at $\sim$ 100 Kev/μm. In particular, we calculate the ratio of the initiation cross section for alphas to that for betas, a ratio which can be checked experimentally using the data for Ra-226 versus Sr-90 in the Salt Lake beagle dogs.

Extension to the level of DNA required that we calculate the probability of clusters of ionizations within the molecule sufficient to disrupt it. Extending a bookkeeping technique developed by Wideroe and using the method developed by Kim for extrapolation from existing data for ionization cross section in water vapor, we have developed a complete electron delta ray tree down to fourth order delta rays. Last year's version has now been updated with better values for LET versus particle energy in such a way as to match the now available Combecher data for energy per ion pair exactly over the entire range of incident electron energy. From this new tree, we have derived a degradation spectrum for all energies of incident beta ray: the curve for 10 kev electrons agrees well with the curve from Paretzke's Monte Carlo analysis.

The alpha particle tree is more complicated, involving consideration of the changing charge state of the alpha due to pickup and loss of electrons as it slows down, the stripping of the electron from the He^+ state, the convoy electrons (electrons moving at the same speed and almost the same direction as the alpha after a collision with a water molecule), as well as the hard Rutherford collisions and the soft dipole collisions considered for the beta particle. Fortunately, a new set of ionization cross section data for He^{++} and He^+ ions incident on water vapor was given to us by Toburen, Wilson and Popovich (Battelle) before publication. We have combined available theory with these data to provide a new array for the number of deltas produced per unit energy per unit alpha track length for all energies of the incident alpha.

We are now generating a new and much more accurate alpha particle delta ray tree from these new data. (Last year's alpha tree had only the Rutherford term.) These corrections are important for both betas and alphas as they involve primarily the very low energy ($\leq$ 1 Kev) secondary and tertiary electrons which are (1) plentiful, (2) not

Prepared by John H. Marshall
and Antonio Pagnamenta

Argonne National Laboratory

0019780

included in the Rutherford or Moller formulas and (3) appear to be crucial to the calculation of the probability of clusters of ionization within distances of 20 Angstroms or less.

These tree structures for alpha and beta particles give us the number of ionizations per unit pathlength on the main track and on each of the delta ray tracks down to fourth order as a function of particle energy - they are stored as eight two-dimensional arrays in Speakeasy. From these arrays, we calculate the probability of finding 2, 3, or 4 ionizations within the core of stacked base pairs within the DNA molecule. We need at least 2 ionizations to explain the quadratic dependence of cross section upon LET which leads to the peak in RBE the order of 100 KeV/μm. We need more than two ionizations to deliver enough Coulomb repulsion between positive charges to disrupt DNA, but perhaps two are sufficient considering other mechanisms. We may not be able to postulate more than two or three ionizations without making initiation by beta rays practically impossible. Thus, the constraints are tight: a good sign for modelling.

We would like to acknowledge the continuing help provided us by Mitio Inokuti and Yong-Ki Kim of the Fundamental Mechanisms section of RER.

0019781

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Inhalation Exposures at Lindsay Chemical Company

The Center for Human Radiobiology is currently engaged in an assessment of possible health effects of employment in the thorium refining industry. An important part of this study is the determination of thorium and rare earth exposure conditions at a thorium refinery located in West Chicago, Illinois.

The West Chicago plant was operated by the Lindsay Light Company, and its corporate successors Lindsay Chemical Company and Kerr-McGee Chemical Corporation from 1932 until 1973. It is presently an empty shell being stripped and decontaminated for eventual demolition. Fortunately we have obtained approximately 10,000 air filter samples which were collected by Lindsay from 1962-1973. The sampling date and location (i.e. floor of a building) of each filter are known. We have also obtained the alpha counting records and airborne thorium calculations corresponding to each filter. In theory these records provide a survey of the time and location distribution of airborne thorium and in some cases thoron as well. However, some unjustified assumptions gave calculated values of thorium and especially thoron which are much too low.

From 1962-1964 approximately 300 filters were alpha counted 4 times each: at 1 hour after filtration, at 10.6 hours, 106 hours and 465 hours. To date we have concentrated our effort on these filters because the four measurements enable the levels of thorium, thoron, lead-212, and bismuth-212 all to be calculated. We use a more realistic model of radioactive growth and decay than Lindsay, making no assumptions about the filter collection efficiency, the thoron retention fraction and the state of equilibrium of retained thoron and its daughters. We have measured the filter collection efficiency to be typically 90% for standardized aerosols. The thoron retention was determined with our standard emanation system using a special emanation chamber for the filter. The mean thoron retention of 16 filters measured was 0.65 ± 0.2 ($\pm$ S.D.).

Lindsay Chemical reported the concentrations of lead-212 as thoron on the assumption that thoron and lead-212 in the air were in equilibrium. Because of ventilation it is very likely that the thoron levels were much larger. Using the bismuth-212 to lead-212 ratios we calculated an "effective age" of the air and then calculated the proper thoron levels. These were typically 20 times higher than previously reported. In addition our values for bismuth-212 and for thorium are typically 15% and 35% higher, respectively.

The airborne radioactivity levels were particularly high on the third floor of building 9. For example in 1964 the geometric means of airborne radioactivity concentrations (32 filters) for thorium, thoron, lead-212, and bismuth-212 were 5.8×10^{-2} pCi/L, 4077 pCi/L, 203 pCi/L, and 60 pCi/L respectively. These values can be compared to the maximum permissible concentrations[1] in air of

Prepared by L. F. Mausner

Argonne National Laboratory

0019782

these isotopes of 3×10^{-2} pCi/L, 300 pCi/L, 20 pCi/L and 100 pCi/L respectively. Thus the workers involved here were exposed to concentrations of thorium, thoron and lead-212 well in excess of present limits. Although high thorium levels were also reached in other years we do not have enough information to determine the thoron and daughter concentrations. We plan to determine thorium on some filters directly by energy dispersive x-ray fluorescence spectrometry to check our calculations.

Since the plant also produced rare earth chemicals (in fact almost exclusively in its last years), we have begun to measure the airborne rare earth concentrations. We have utilized energy dispersive x-ray fluorescence spectrometry to determine simultaneously lanthanum, cerium, praseodymium, and neodymium directly on the air filter samples. To date we have analyzed a total of 14 filters from 1967 and 1970. The relative amounts were usually in the order La > Ce > Nd > Pr. The amounts ranged from 7-54 μg, 4-108 μg, 4-12 μg, and 5-52 μg for La, Ce, Pr, and Nd respectively. This corresponds to airborne concentrations of 44-340 ng/L, 25-680 ng/L, 25-76 ng/L and 32-328 ng/L. Typical concentrations of these elements in urban aerosols are 0.005 ng/L, 0.01 ng/L, 0.003 ng/L and 0.006 ng/L.[2] The rare earth content of the filters did not correlate with the radioactivity measurements. Thus we are probably looking at different types of dusts.

The particle size distribution is also an important exposure variable. Therefore we have started to determine the size distribution of the filtered dusts and settled dust recently collected at the plant. This work is being done in collaboration with researchers from the Lovelace Inhalation Toxicology Research Institute in Albuquerque, N.M.

References

1. U.S. Nuclear Regulatory Commission, Rules and regulations, Title-10-Chapter 1, Code of Federal Regulations, Part 20, Standards for protection against radiation, U.S. Government Printing Office, Washington, D.C. (codified and reissued September 1978).

2. J. A. Cooper, Battelle Northwest Laboratories Report BNWL-SA-4690.

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Radon in Houses

A study is underway of the levels of naturally-occurring radon (the decay product of radium) and its short-lived radioactive daughter-products in houses in the Chicago area. The daughter-products are responsible for the largest dose of radiation from natural sources to any part of the body, i.e. lung. The most reasonable calculations seem to indicate about 10^4 fatalities per year in the United States due to lung cancer induced by radon daughters. Reduction of ventilation rates of houses with outside air to conserve energy can only increase this number. Thus, there is an urgent need for extensive data on the actual levels in houses of radon and its daughter-products, their variation with time, the sources of radon and the fate of its daughter-products under normal conditions in ordinary houses.

Methods of Measurement

Spot determinations of radon in the air of houses are made by taking "grab" samples of air into previously evacuated radon counting bottles. With background counting rates of about 0.1 min^{-1}, these have a limit of detection of approximately 0.1 pCi L^{-1} for a counting time of 400 minutes. Continuous determinations of radon are made with a detector of the Spitz-Wrenn design; counts are integrated over a period of 30-60 minutes. The limit of detection is about 0.3 pCi L^{-1} for a 60-minute observation.

The short-lived radon daughter-products can be determined serially with an "Environmental Working Level Monitor" (EWLM). The activity in about 100 litres of air is collected on a filter and simultaneous alpha-particle spectrometry and scintillation beta-particle counting permit the calculation in an internal computer of the concentrations of ^{218}Po, ^{214}Pb and ^{214}Bi. The results are printed out as pCi L^{-1}, and also in units of Working Level (WL; 1 WL is that concentration of short-lived daughter-products of radon that will ultimately yield 1.3×10^5 MeV of alpha-particle energy. The daughter-products in equilibrium with 100 pCi L^{-1} of radon are at a concentration of 1 WL.) The EWLM has a limit of detection of about 10^{-3} WL, corresponding to 0.1 pCi L^{-1} for each of the daughter-products.

Results

A total of 73 houses has now been investigated and a summary of the results is set out in the table overleaf. These data include results for 25 houses in a group of about 40 in an area of about 0.2 km^2 (50 acres) which are being investigated systematically. At least one sample of air was taken from each level in the house, during the months of August to October 1980. Some houses had windows open when the samples were taken, while in others ventilation with outside air was at a minimum, so that the results are not strictly comparable. However the data suggest that the distributions of the concentrations for each level are approximately log-normal with

Prepared by J. Rundo

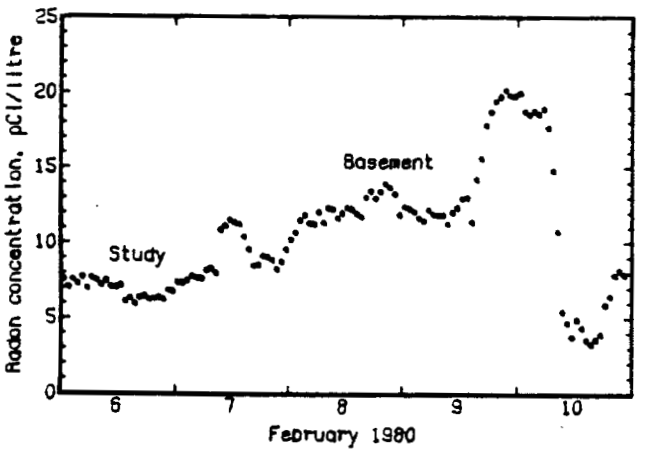
Argonne National Laboratory

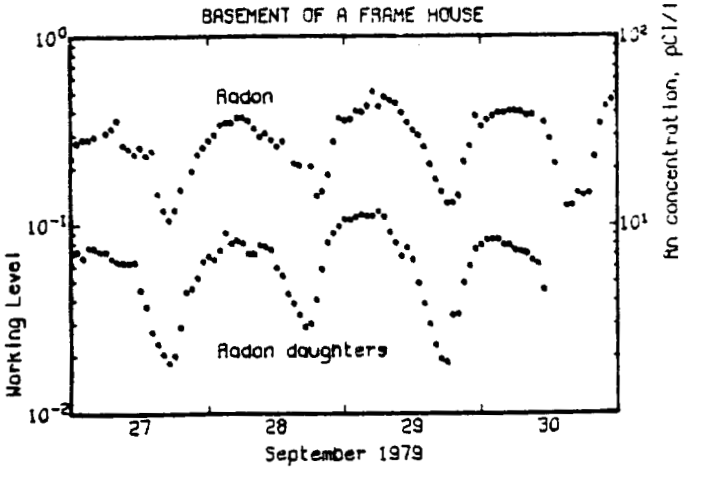
0019784

	No. of houses	Level	Range of Concentration of ^{222}Rn, pCi L^{-1}
All houses	65	Lower	0.19 - 51.9
	44	1	0.07 - 25.9
	26	2	0.11 - 54.5
Houses with unpaved crawl spaces	26	Lower	0.34 - 35.5
	8	1	0.29 - 19.2
	2	2	0.16 - 54.5
Earth-shielded houses	6	1	1.2 - 16.1

geometric mean values of 1.6 pCi L^{-1}, 0.5 pCi L^{-1} and 0.7 pCi L^{-1} for lower level (usually basement or crawl space), first floor (entrance level) and second floor respectively. The corresponding geometric standard deviations were 2.4, 3.3 and 2.4 respectively. The limited data on radon in earth-shielded houses suggest that the average concentration may be higher than in this group of conventional houses.

Data on the concentrations of the daughter-products have been collected in a few of the houses showing relatively high concentrations of radon, and in some cases the radon was determined simultaneously. Two sets of results from one house are shown in the diagrams below. From these results, and those in the table above, it is obvious that determination of radon in a single sample of air can not give a representative value for the concentration in a house. Much more data are needed to determine the reasons for the apparent seasonal differences in the results. From the data collected in September 1979 the value for the equilibrium fraction, F (ratio of observed WL to maximum possible WL), is seen to be about 0.1. This is at variance with the value of 0.5 observed by others and the reason for this is still being investigated.





Publication

Rundo, J., Markun, F. and Plondke, J. "Observation of High Concentrations of Radon in Certain Houses," Health Phys., 36, 729-730 (1979).

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Dosimetry of Sinus and Mastoid Epithelia

Carcinomas arising from the mucous membranes of the paranasal sinuses and mastoid air cells are a well-known late effect of radium deposition in humans. The tumors are produced by alpha particle bombardment from two locations on opposite sides of the epithelium: bone, the site of radium deposition, and the air spaces, where radon gas accumulates. The dose to epithelial cell nuclei is determined by several variables: tissue dimensions, the size of the air cavity, the specific activity of bone, and the rate of gas exchange between the airspace and the atmosphere.

Tissue Dimensions

Histological studies have revealed mucosa with rather thick connective tissue layers and somewhat thinner epithelial membranes. The lamina propria tends to be thicker in the sinuses than in the mastoids. The same is true of the epithelium.

Spot measurements of mucosal dimensions have been made in 13 subjects with little or no internal exposure above background. The thickness of the lamina propria is found to vary widely and often to exceed the ranges of alpha particles emitted by members of the ^{226}Ra and ^{228}Ra decay series. Therefore, in many locations, the epithelium is completely shielded from irradiation by bone.

Shielding from airspace irradiation also occurs in the sinuses because of the structure of the epithelium. There, it is usually columnar, with the target nuclei concentrated toward the lower ends of the cells, away from the airspace border. The thickness of cytoplasm interposed between the nuclei and the upper end of the cell is often a substantial fraction of the alpha particle range, thus providing an effective shield against radiation from the airspace. In healthy persons, a mucus layer of varying thickness covers the epithelium. We have no measurements of this but assume that its thickness would be comparable to the length of cilia observed on the epithelial border. These can be several microns long, making the mucus layer an additional factor of importance to shielding.

Quantitatively, the lamina propria thicknesses so far measured have fallen within the approximate range, 5 to 550 microns. Because of limited and somewhat biased sampling, we have no true measure of the average thickness. We have found, however, that the percentage of the lamina propria which is sufficiently thick to completely shield the epithelium, is greater than 75% in the sinuses and less than 25% in the mastoids.

The thickness of cell cytoplasm shielding the epithelial nuclei has, so far, been observed to lie in the range of about 1 to 150 microns. Thirty-five microns is typical for the sinuses and in the mastoids, the cytoplasm is typically less than 2 microns thick.

When dose calculations are performed using plausible assumptions about the radioactivity levels in bone and the airspaces, the dose from the airspace exceeds that from

Prepared By: R. A. Schlenker 0019786 Argonne National Laboratory

bone, in agreement with a hypothesis of long standing, that radon gas is the principal determinant of sinus and mastoid carcinomas in radium-exposed persons.

Air Cavity Size

The importance of size lies in the fact that radon alphas have a range of several centimeters in air and, therefore, cannot be stopped by air before reaching the epithelium. As a result, the larger the airspace, the greater will be the epithelial cell dose.

Good data exist in the literature on the linear dimensions and on the volumes of the paranasal sinuses. However, with the exception of a few qualitative statements, the literature contains no information on the sizes of the mastoid air cells. Dimensional data have been collected in 4 persons with radium dosage in the range known to produce carcinomas. The maximum diameters range from a few tenths of a millimeter to several centimeters. The size distribution is skewed, however, yielding a modal value of about one millimeter. This is in contrast to the ethmoid air cells, where the typical diameter is about 9mm. In the frontals and sphenoids, the diameter is 20mm and in the maxillaries, it is 30mm.

Bone Specific Activity

The radioactivity in bone adjacent to the airspaces determines the epithelial dose from bone. In order to quantify the specific activity, autoradiographic measurements have been made on 5 subjects with high ^{226}Ra body burdens. An interesting result is that the average radium level is generally lower adjacent to the air cells than in the skeleton as a whole. Four of the cases showed average specific activities for the air cells which lay between 14% and 92% of the average skeletal specific activity. In one case, the average specific activities were equal and, in no case, did the average activity near the air cells exceed the skeletal average. These results indicate that the dose from bone is lower than expected.

The factors which control the accumulation of radon in the sinuses have not yet been fully identified. One thing is clear, however: the radon concentrations which have been observed in the sinuses could only have been produced by radon generation in the surrounding bone. Radon in the arterial blood is at too low a level to explain them, and radon from more distant parts of the skeleton cannot reach the airspaces before being swept away by the circulatory system. Thus, not only does the adjacent bone directly bombard the epithelium, it is also, indirectly, the source of the epithelial dose delivered from the airspaces.

Gas Exchange

The sinus and mastoid airspaces are ventilated through ducts which communicate with the respiratory system; gas exchange with the atmosphere also occurs via the circulatory system. In this way, high concentrations of radon which accumulate in the airspaces are diluted, and the average radon concentration is established as a balance between the influx from bone and the efflux to the atmosphere.

Noble gas clearance by the ventilation and by the circulation both have a single exponential time dependence. For clearance by the circulation, the observed half-times lie in the range, 24-117 min; for ventilation, they run from 1 to 7 min, except in the mastoid where the half-time is several thousand minutes, due to the normal closure of the Eustachian tube. For comparison, the radioactive half-lives of ^{220}Rn and ^{222}Rn are 0.9 and 5500 min, respectively. This leads to the conclusion that almost all ^{222}Rn is cleared from the airspaces before it can decay and most ^{220}Rn decays in the airspaces before it can be cleared. In cases where equal amounts of ^{228}Ra and ^{226}Ra have been taken into the body, this fact appears to guarantee that roughly equal epithelial doses will be delivered by the ^{228}Ra and ^{226}Ra decay series. This contrasts with assumptions, made by others, that ^{228}Ra is unimportant to the dosimetry of sinus and mastoid carcinomas, regardless of the amount present in the body.

0019181

October 1980

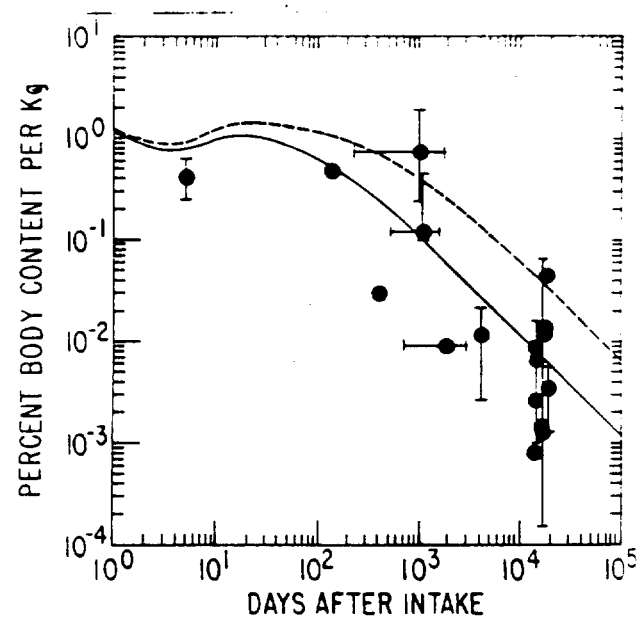
CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Soft Tissue Retentions and Dosimetry of Radium

A significant excess of breast cancer has been observed among female radium dial workers. The tumor incidence appears to vary with total radium intake to blood but not with the duration of exposure. This suggests that internal rather than external radiation is the principal carcinogen.

To lay a foundation for internal dosimetry, the data on radium retention in humans have been collected from widely scattered sources in the literature, reviewed and compared with predictions of the ICRP model of alkaline earth metabolism. Radium-226 specific activity data were found for a total of 87 samples of 40 organs from 17 subjects at 5 to 19000 days after first ingestion or injection. The majority of data are summarized in the graph at the right; each point gives the ratio of median specific activity to total body burden for one subject; errorbars represent the range of observed values. The dashed and solid curves are ICRP model predictions. The former utilizes the published values of the model parameters; the latter utilizes parameter values deduced to insure a good fit of the model to the data.



Despite their wide scatter, the data points reveal a marked downward trend with time. If ^{226}Ra were lost at the same rate from soft tissue as from the whole body, the data would fall along a straight line of zero slope. Thus, ^{226}Ra is lost more rapidly from soft tissue than from the whole body or from the skeleton and the cumulative dose approaches its final value more quickly in soft tissue than in bone.

The adjustments made to the ICRP model parameters have a significant impact on the predicted retention and on dose calculations based on the model. Five of the parameters were changed from their published values, as shown below. The effective retention

Parameter	Published	Adjusted
β	0.608	0.625
w	1.31	1.43
f_c	3.00	5.64
υ	1.50	1.52
r	0.997	0.995

0019788

Prepared By: R. A. Schlenker Argonne National Laboratory

integrals in units of days, using the adjusted parameter values are given in the following table for ^{226}Ra and for ^{228}Th, the first alpha emitter in the ^{228}Ra decay series. To convert these

Period of Integration (Yr)	^{226}Ra	^{228}Th
1	6.3	1.5
50	9.0	7.7
∞	9.1	7.7

integrals into absorbed dose (rad) for one microcurie of intake to blood, multiply by 0.0045 (^{226}Ra) or 0.0051 (^{228}Th).

The retention integrals exclude contributions from other members of the decay series and thus give minimum dose estimates. These minima, for female radium dial workers who contracted breast cancer, lie in the range 0-45 rad.

The dosimetry of other members of the decay series is quite complex and presents a number of problems which are yet to be solved. Two among these are estimation of the ^{222}Rn content of the soft tissues in the case of ^{226}Ra exposure, and estimation of the ^{224}Ra content in the case of ^{228}Ra exposure.

A third problem is non-uniformity in the distribution of radium throughout the body's soft tissue mass. Insight into the differences between organs can be gained from natural levels of the alkaline earths in body tissues. Using such data, absorbed dose conversion factors (rad/μCi intake to blood) have been derived for 27 organs. A sampling of these is given below for ^{226}Ra. Notice

Organ	Factor
Eye	0.3
Thyroid	0.3
Ovary	0.1
Breast	0.06
Muscle	0.02
Total Soft Tissue	0.04

that the factors for breast and total soft tissue do not differ greatly. This implies that the breast dose from ^{226}Ra alpha particles is not greatly different from the average value for the whole soft tissue mass and, therefore, that the range 0-45 rad given above is realistic for the breast.

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Radium Project Registry

Widespread industrial and medical usage of radium in the United States began about 1913. The highest exposures of workers in the radium dial industry occurred before 1925, when the toxic effects of radium became known and safety measures were adopted. Up to about 1930, other persons acquired significant body burdens through deliberate intake of radium for supposed therapeutic effects. During World War II, a revival of the radium dial industry led to the exposure of another large group of people at lower levels of radium intake. The people thus exposed constitute one of the few large groups of humans with medically significant levels of internally deposited radioactivity.

Follow-up. From the records of earlier radium projects and from search efforts since the Center was formed in 1969, approximately 5000 persons have been identified by name and by type of exposure to radium. This information came from non-selective sources, such as employment records, and from selective sources, such as self-referral or persons named by fellow employees. In some cases, the sources provided little personal data that could be used to locate the persons identified. A summary of the status of persons in the radium registry at the end of 1979 is given in Table 1 by type of exposure. Under "Dial work" are dial painters (mostly women) and non-laboratory employees of the radium dial industry, "Lab" denotes laboratory workers, and "Med" refers to persons who received radium for supposed therapeutic effects. "Measured" persons are those whose radium burden has been determined.

Table 1. Follow-up status of radium cases, by type of exposure

	Dial Work		Lab	Med	Other	Total	
Living							
Measured	~~1377~~	1370	140	30	21	1568	1561
Unmeasured	649	646	32	17	20	718	729
Deceased							
Measured	379	424	121	135	5	640	699
Unmeasured	668	713	111	107	29	915	968
Unlocated	859	754	173	107	163	~~1302~~	1135
Total	3932	[illegible]	577	396	238	~~5143~~	5135

Projected case loads. About half of those exposed to radium in the 1920's are still alive and many are expected to survive for another 10 years or more. In Table 2 are given the numbers of radium cases now alive or of unknown status and the expected survivors in 2, 5, and 10 years. Sex-age-time-specific mortality rates were used. The cases are divided into an

Prepared by A. F. Stehney Argonne National Laboratory

0019190

"early" exposure group (first exposed before about 1930) and a "late" exposure group, mostly women who worked in radium dial plants during World War II.

Table 2. Radium-exposed persons who are alive or of unknown status[1]

	Expected Survivors			
	1980	1982	1985	1990
"Early" exposure				
Located	742	651	517	307
Unlocated	144	125	95	47
	886	776	612	354
"Late" exposure				
Located	1498	1450	1371	1215
Unlocated	711	693	662	602
	2209	2143	2033	1817
Total	3095	2919	2645	2171

[1]Corrected for mortality before 1980

Findings. Radium deposits mostly in bone, and the principal effects are associated with bone and bone surfaces. These are bone sarcomas, carcinomas of the paranasal sinuses and mastoids, and skeletal changes such as bone necrosis and coarsening of trabeculation. Among the measured radium cases, 60 bone sarcomas (2 since 1969) and 29 sinus and mastoid carcinomas (7 since 1969) have been diagnosed. In addition, 24 cases of bone sarcoma and 5 cases of sinus or mastoid tumor in unmeasured radium-exposed persons have been found from search of death certificates and medical records. Among women radium dial painters, a significantly increased incidence of breast cancer has also been found, and there are indications of excess deaths from multiple myeloma, colon cancer, and tumors of the central nervous system.

Future plans. The radium project will continue to register all authenticated cases of radium exposure, so that the potential for evaluation of radium effects will not be lost. The registry provides a pool of cases for study of defined populations and for special purpose studies of individual cases. Continued follow-up of the radium cases will be necessary to complete the study of skeletal effects and to evaluate soft tissue effects which have only recently become apparent. In addition to the skeletal dose, doses from radium in soft tissue and the gastrointestinal tract, and external radiation in the work place will have to be considered.

0019791

October 1980

CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Two Unusual Cases of Accidental Contamination by Actinides

In addition to their use in our major research programs, the whole-body counting facilities of the Center are also available to assist the Occupational Health and Safety Division in the assessment of cases of radioactive contamination which may occur at Argonne. Two recent such cases have served to illustrate some of the problems involved in the measurement of transuranic nuclides (plutonium and americium) in vivo.

The first case was that of a female technician who found her hand to be contaminated with approximately 100,000 dpm of alpha activity after working in a glovebox. The contamination was removed and no additional contamination was found. However, subsequent urine and fecal samples were found to contain a few dpm of alpha activity and she was referred to us for assessment of any inhaled activity. Fortunately, we had performed a routine background count previously, and so her subject background was accurately known. (The determination of a previously-unmeasured individual's subject background is a major source of uncertainty in lung counting for the actinides.)

A lung count with a phoswich detector gave net counting rates of 5.0 ± 1.8 cpm in the 60-keV ^{241}Am energy band, and 6.1 ± 1.0 cpm in the x-ray band (12-24 keV). (Both americium and plutonium emit L x-rays whose principal energies are approximately 17 keV.) These counting rates would correspond to lung burdens of 0.12 ± 0.04 nCi ^{241}Am and 46 ± 16 nCi of plutonium for this individual. Since the maximum permissible lung burden of ^{239}Pu is 16 nCi, there was cause for concern if the observed counting rates were indeed due to activity in the lungs.

However, several facts implied that the activity was external rather than internal. The plutonium to americium activity ratio of the material if it were internal would have been almost 400, which was two orders of magnitude greater than that of the material with which she had been working. No external contamination around the nose or mouth had been detected after the incident, and the amount excreted in the feces was much less than expected if there had been inhalation. Finally, detailed analysis of the photon spectrum observed with the phoswich indicated that the spectrum was not characteristic of internal deposition.[1] The source of the observed net counting rates was eventually traced to about 250 dpm of activity fixed rather firmly on the subject's hair. The important conclusion to be drawn from this episode is that the results of external counting should not be taken by themselves as sufficient evidence for medical intervention. Rather, they must be interpreted in light of all the other information known about a particular case, in order to determine what is most likely the true solution.

The second case was of a rather more serious nature and the external counting results were themselves definitive. In this instance, a male chemist

Prepared by R. E. Toohey

Argonne National Laboratory

0019792

received a puncture wound while attempting to open a sealed glass ampoule of an unknown plutonium solution in a glovebox. When scored, the ampoule exploded and sent a piece of contaminated glass through the glove and into the left thumb.

In order to guide the attending physician in excising contaminated tissue, the depth of the material deposited in the thumb had to be known. This could be determined from measurements made with a xenon-filled proportional counter. This counter is able to resolve the L_α x-ray at 13.6 keV from the L_β x-ray at 17.2 keV, and since the linear attenuation coefficient of soft tissue changes by almost a factor of two between these two energies, the amount of tissue covering the activity can easily be determined by observing the intensity ratio of the two lines. The observations of the thumb wound are summarized in the table. The major part of the activity initially in the thumb has been removed by excision and chelation therapy, but counts of the liver and skull indicate that this individual contains approximately 20 nCi of plutonium (one half the maximum permissible body burden) deposited in the liver and skeleton. Assessment of this case and of the effectiveness of further chelation therapy is continuing.

Table: Actinide Activity in Thumb Wound

Measurement number	Days since incident	$^{239,240}Pu$	^{241}Am	Depth in thumb, mm
		nCi		
1	0	35.1 ± 4.2	8.2 ± 0.6	2.7 ± 0.5
2	1	20.7 ± 5.7	5.0 ± 0.8	3.0 ± 0.6
First excision				
3	1	7.8 ± 3.0	1.4 ± 0.4	2.3 ± 0.5
Second excision				
4	3	<0.1 ± 0.8	0.6 ± 0.1	5.1 ± 1.0
5	9	0.6 ± 0.5	0.5 ± 0.1	5.9 ± 1.2
6	14	3.1 ± 0.2	0.2 ± 0.1	7.6 ± 1.5
7	38	0.6 ± 0.8	0.3 ± 0.1	10.8 ± 2.2

Reference

1. R. E. Toohey and J. Rundo, A rapid method for estimating the effective depth of internally deposited ^{241}Am, in Diagnosis and Treatment of Incorporated Radionuclides, Proceedings of a Seminar, Vienna, 8-12 Dec 1975, (IAEA, Vienna, 1976) pp 277-284.

0019793