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SURVIVAL, CAUSES OF DEATH, AND ESTIMATED TISSUE DOSES IN A GROUP OF HUMAN BEINGS INJECTED WITH PLUTONIUM*

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ABSTRACT

To determine the relationship between urinary excretion and plutonium body content, 18 persons of short life expectancy were injected with plutonium between 1945 and 1947. Seventeen of these 18 individuals have been identified; eight were found to have survived for at least eight years and four are still alive today (1975). The causes of death of 13 of these individuals have been determined from death certificates; none appear to be related to the administered plutonium. Doses to the liver and to the cells on the surface of bone have been calculated for these plutonium cases. The liver doses do not appear to be high enough to be carcinogenic, but comparison of the bone-surface doses with radium doses that have induced bone tumors indicates that six of these cases have received doses high enough to be considered carcinogenic. However, no bone tumors have yet appeared.

INTRODUCTION

In order to obtain information relating plutonium body content to plutonium excretion in human beings, hospitalized individuals of relatively short life expectancy were given intravenous injections of plutonium in 1945-1947, and excreta were collected and measured for as long as they remained hospitalized (Langham et al. 1950). Following the initial discovery by Durbin (1972) that some of these individuals were still living, she and, subsequently, the Center for Human Radiobiology at the Argonne National Laboratory made an effort to identify, trace, and learn the whereabouts of all of these unique cases. The purpose of this report is to detail what is known today about these individuals and their causes of death, and to estimate the liver and bone doses accumulated during their lifetimes.

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DOSE CALCULATIONS

The calculated radiation doses to the liver and to the skeleton are based on the assumption, inferred from ICRP (1972), that the fractional retention of plutonium in the organs of interest is

$$R(t) = e^{-\lambda t}.$$

Here t is the time after administration of plutonium and λ is the effective elimination constant, obtained from the appropriate radioactive decay and biological elimination constants.

The organ dose is then given by

$$D(T) = f \cdot I \cdot Q \int_0^T dt \cdot R(t),$$

where I is the amount of plutonium injected, in μCi,

f is the fraction of the injected isotope in the organ,

Q is the appropriate dose rate per unit activity, in rad/μCi-day,

T is the time after injection, in days, and

D(T) is the average α-ray dose in rad.

The value of f used for both skeleton and liver is 0.45, as recommended by ICRP (1972). The initial injection levels were obtained from the original report on the cases by Langham et al. (1950), from Durbin's reviews of the data (1972) and, for case Cal-I, the original report by Crowley et al. (1946). The existing information on this last case is reviewed in Appendix A.

The time T over which the doses were calculated was from the date of injection to the date of death or, for those cases that are still alive, to July 31, 1975. The calculated liver and bone doses are tabulated in Table I; the complete details of the dose calculations are shown in Appendix B. Table I also lists the injection levels and the dates of injection and death.

SURVIVAL AND CAUSES OF DEATH

Survival time was calculated from date of injection, as given by Langham et al. (1950), to date of death as given on the death certificate. Of the 18 cases, one remains unidentified, four are still alive (as of 7/31/75), and death certificates have been obtained for the remaining thirteen.

The causes of death as given on the death certificates are listed in Table II.

Table 1. Calculated skeletal and liver doses

Case	Plutonium isotope	Injected dose		Dates		ΔT (day)	Calculated α-ray dose (rad)	
		μCi/kg	Total μCi	Injection	Death		Skeletal	Liver
Cal-I*	238	0.060	3.5	05/14/45	01/09/66	7 545	575 } 580	1440 } 1460
	239	0.0008	0.046				7.7	19
Cal-II‡	239	0.011	0.169	04/26/46	01/06/47	255	2.5	8.7
Cal-III†	238	0.0013	0.095	07/18/47	Living	10 240**	11	26
Chi-I	239	0.0052	0.40	04/26/45	10/03/45	160	1.5	4.2
Chi-II	239	0.15	5.9	12/27/45	01/13/46	17	3.5	8.5
Chi-III	239	~0.085	5.9	12/27/45		~ 170‡	24	70
HP-1	239	0.0040	0.28	10/16/45	01/12/60	5 201	33	85
HP-2	239	0.0045	0.31	10/23/45	04/04/48	894	6.5	18
HP-3	239	0.0043	0.30	11/27/45	Living	10 838**	100	210
HP-4	239	0.0054	0.30	11/27/45	04/29/47	518	5.4	13
HP-5	239	~0.0044	0.31	11/30/45	04/29/46	150	1.1	3.1
HP-6	239	~0.0044	0.33	02/01/46	Living	10 772**	76	180
HP-7	239	0.0057	0.39	02/08/46	10/27/46	261	3.5	8.6
HP-8	239	0.0073	0.40	03/09/46	Living	10 736**	140	280
HP-9	239	0.0061	0.39	04/03/46	07/02/47	455	4.2	12
HP-10	239	0.0053	0.38	07/16/46	06/02/57	3 974	34	91
HP-11	239	~0.0056	0.40	02/20/46	02/26/46	6	0.057	0.16
HP-12	239	~0.0044	0.29	04/10/45	04/13/53	2 925	20	52

*See Appendix A

‡Skeletal mass estimated to be 2000 g and liver mass estimated to be 580 g.

**To July 31, 1975

†This individual was injected with 0.095 μCi ^{238}Pu (VI) nitrate intramuscularly in the gastrocnemius muscle. After a mid-thigh amputation four days later, 46.6% of the injected plutonium was recovered from 69.5 g of tissue considered to constitute the injection site (Durbin 1972). The dose calculation here is based on the assumption that the remaining 53.4% of the injected material was retained in the body. However, subsequent measurement of plutonium excreted in the urine has demonstrated that the excretion for this case is two orders of magnitude lower than that of case HP-3 (Rundo 1974). Therefore, these dose levels must be considered absolute upper limits, while the actual doses might be very much lower.

‡Died approximately 170 days after injection (Durbin 1972). Doses calculated to 170 day.

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Table II. Causes of death as given on death certificates

Case	Sex	Birth	Age at injection*	Age at death	Cause of death Contributory causes
Cal-I	M	10/11/86	58	79	Cardio-respiratory failure (12 hr) Pulmonary edema (2 day); arteriosclerotic heart disease with decompensation (1 wk)
Cal-II	M	05/26/41	4	5	Sarcoma of knee (9 mo)**
Cal-III	M	01/26/11	36		(Living)
Chi-I	M	03/30/77	68	68	Recurrence of cancer of chin and metastasis to lungs (1 yr)**
Chi-II	F	01/25/89	56 [55]	56	Metastatic carcinoma of aberrant breast tissue (5 mo)**
Chi-III	M				(Lost to study after ~ 170 day)†
HP-1	M	05/20/78	67	81	Bronchopneumonia (3 day) General arteriosclerosis (unknown interval)
HP-2	M	08/05/97	48 (49)	50	Hypertensive encephalopathy (days) ? Cerebral hemorrhage (days); hypertensive cardiovascular disease (years);** hemophilia (years)**
HP-3	F	04/01/97	48 (49)		(Living)
HP-4	F	02/09/27	18	20	Cushing syndrome (6 yr)** Adenoma-pituitary (6 yr)
HP-5	M	01/04/89	56	57	Bronchopneumonia (2 day) Amyotrophic lateral sclerosis (2 yr, 6 mo)**
HP-6	M	11/14/01	44 (45)		(Living)
HP-7	F	05/07/86	59	60	Pulmonary failure Pneumonia; left base rheumatic heart; thyrotoxic goiter
HP-8	F	09/09/04	41		(Living)
HP-9	M	08/18/81	64 (66)	65	Terminal bronchopneumonia (days) Laryngeal edema and debilitation (days); dermatomyositis (years)**
HP-10	M	10/31/93	52	63	Arteriosclerotic heart disease**
HP-11	M	01/29/77	69 (68)	69	Bronchopneumonia (days) Cirrhosis of liver (years); congestion of viscera (days); arteriosclerosis (years)
HP-12	M	03/17/90	55 (53)	63	Heart failure (3 wk) Auricular fibrillation (3 wk)

*Age at injection calculated from known date of birth. Numbers in ( ) are ages given in LA-1151, if different. Numbers in [ ] are ages given in Durbin 1972, if different.

**Diagnosed illness at time of injection.

†Believed to be suffering from Hodgkin's disease (E. R. Russell, private communication).

DISCUSSION

From the listed causes of death it appears that plutonium cannot be considered to be a contributing cause for any of these cases. These individuals were all gravely ill at the time of the plutonium injections; some died in the hospital shortly after the injections, others survived only to succumb eventually to their diagnosed illnesses. The cases with long survival are, of course, more apt to show the effects of this bone- and liver-seeking isotope. No bone- or liver-related malignancies appear in the listed causes of death or the contributing causes on the death certificates, except for those cases in which it was the diagnosed disease at the time of the injection. Further, to our knowledge today (1975), the cases still living show no plutonium-related effects.

Bone tumors have been induced by radium in human beings, and the risk from plutonium can be estimated from these cases. From the U.S. radium cases who have carried ^{226}Ra or ^{228}Ra or a mixture of the two isotopes, the lowest average skeletal dose associated with a bone tumor is 888 rad (Rowland et al. 1973). The shortest time between first exposure and the diagnosis of a bone tumor was seven years (Rowland et al. 1973). From the more recent study of the effects of ^{224}Ra in man, an average skeletal dose of only 90 rad was observed as the lowest dose associated with a bone tumor, and four years was the shortest time between acquisition and diagnosis (Spiess and Mays 1970).

Following Spiess and Mays (1970), we have converted these "lowest" average skeletal doses into the average doses delivered to the 10-μm-thick volume of cells adjacent to the bone surfaces; here we will call these the average endosteal doses. The relationships between average skeletal dose and this average endosteal dose are listed in Table III (Marshall et al. in press). Since all of our average skeletal doses for plutonium were calculated by use of the new ICRP bone masses--5000 g for males, 3400 g for females (ICRP 1974)--these lowest average skeletal doses associated with bone tumors for ^{226}Ra and ^{224}Ra must first be recalculated on this basis.

The calculation for ^{226}Ra is straightforward; the case with the bone tumor at 888 rad was female with an assumed 5-kg skeleton. Therefore, we recalculate the average skeletal dose to be

$$888 \text{ rad} \times \frac{5 \text{ kg}}{3.4 \text{ kg}} = 1310 \text{ rad}.$$

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The average endosteal dose is thus 1310 rad x 0.45 = 590 rad.

Table III. Relationships between average skeletal dose and bone surface dose for ^{224}Ra, ^{226}Ra, and ^{239}Pu*

	^{224}Ra**	^{226}Ra†	^{239}Pu††
$\frac{\text{Surface dose rate}}{\text{Average skeletal dose rate}}$	8.9	0.45	12.8

*From Marshall et al. in press.

**^{224}Ra and 100% of three α-emitting daughters, 26.46 MeV per decay.

†^{226}Ra and 30% of three α-emitting daughters, 10.53 MeV per decay.

††^{239}Pu, 5.149 MeV per decay.

The calculation for ^{224}Ra is somewhat more complex. The case with the lowest dose was male with an assumed 7-kg skeleton; the recalculated average skeletal dose is thus 90 rad x $\frac{7\text{ kg}}{5\text{ kg}}$ = 126 rad. However, the 90-rad value was based on the assumption that 20% of the injected radium decays in the skeleton, whereas Marshall's conversion factors followed ICRP (1973), in which only 11.5% of the injected dose decayed in the skeleton. We thus determine the average skeletal dose for this case to be 126 rad x $\frac{11.5\%}{20\%}$ = 72 rad. The average endosteal dose is then 72 rad x 8.9 = 640 rad.

We thus expect the lowest average endosteal dose at which plutonium might induce bone tumors in man to be of the order of 600 rad. Table IV lists those cases surviving for at least four years, their average skeletal doses, and their average endosteal doses calculated on the assumption that all of the plutonium resides on the bone surfaces.

Table IV. Skeletal radiation doses in plutonium cases surviving more than four years

Case	Survival (days)	Average skeletal dose (rad)	Surface dose (rad)
Cal-I	7,545	580	7,420
Cal-III*	10,240	11	141
HP-1	5,201	33	422
HP-3	10,838	100	1,280
HP-6	10,772	76	973
HP-8	10,736	140	1,790
HP-10	3,974	34	448
HP-12	2,925	20	256

*See footnote (†) to Table I

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Eight of these 18 cases survived longer than the four-year period noted above as the shortest induction interval for a radium-induced bone tumor; indeed, all of these eight cases actually survived at least eight years and four are still alive 28-29 years after receiving plutonium. Four of these cases received average endosteal doses considerably larger than 600 rad, while two more received doses near enough to this value to be considered to be at considerable risk. From the ^{226}Ra cases one can generalize that about 30% of the cases who have accumulated average endosteal doses greater than 600 rad have a bone-related malignancy. Since a few radium-induced tumors have appeared as late as fifty years after first exposure, whereas the longest exposures in this small plutonium-injected group are now only approaching thirty years, we would predict that between a sixth and a third of the six cases at risk should now have bone tumors. In view of the small numbe of cases at risk, the fact that no tumors have appeared is certainly not conclusive evidence that these radiation doses from plutonium are not tumorigenic.

One might argue that burial of surface-deposited plutonium by appositional bone growth would protect the layer of cells on bone surfaces and thus reduce the risk of bone-deposited plutonium. This is undoubtedly true, but since the same process occurs in the case of ^{226}Ra in bone, the risk estimate derived from experience with this radioisotope takes account of this process. We conclude, from the lack of bone tumors in these cases, the bone-tumor risk from plutonium is no greater than that from radium, and might be less.

Human experience with liver-deposited alpha-emitting isotopes seems to be limited to the radiological contrast medium, Thorotrast. It appears that intravenously injected Thorotrast induces three distinct types of liver tumors: hemangioendotheliomas, bile-duct tumors, and hepatic-cell tumors (Swarm 1968; da Silva Horta 1973). Specific dosimetry on a case-by-case basis is not available, but some general conclusions regarding liver dose can be drawn from the data. Liver dose rates in Thorotrast cases range from 13 to 41 rad/year (Kaul 1973); the average liver dose in ten cases of liver cancer was 509 rad, while the average liver dose in ten cases of hemangioendotheliomas was 609 rad (Faber

1973). Since only one of the cases listed in Table I had a liver dose greater than these values, it is perhaps not surprising that liver tumors have not been seen in these plutonium cases.

Therefore, these cases serve to demonstrate that plutonium does not appear to be more carcinogenic than expected and, as far as bone is concerned, may actually be less carcinogenic than expected.

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APPENDIX A

The Plutonium Injection for Cal-I

There is uncertainty about both the amount and isotopic composition of the plutonium injection of patient Cal-I.

(a) Langham et al. (1950) quoted a calculation by J.G. Hamilton: the injected dose of ^{238}Pu and ^{239}Pu was equivalent to 103 μg of ^{239}Pu (6.3 μCi of total alpha activity). Hamilton had used an injected alpha activity of 120,000 counts/sec as originally reported by Crowley et al. (1946), an unknown alpha counting efficiency (probably 0.5), and a ^{238}Pu half-life of 50 yr, the best estimate available at the time (Seaborg et al. 1942).

(b) Durbin (1972) recalculated the Cal-I dose and obtained a value of 5.2 μCi of alpha activity (Cal-I weighed 58 kg, so the values reported were 0.0896 μCi/kg of ^{238}Pu and 0.002 μCi/kg of ^{239}Pu). Consultation of the original data sheets, which were still in the Lawrence Berkeley Laboratory files, revealed that the injected dose had been 68,000 counts/sec. Evidently, the original report of Crowley et al. (1946) had erroneously shown the dose in units of counts/sec rather than disintegrations/sec. However, Durbin's calculation also contained some false assumptions--the values used for the ^{238}Pu half-life (86.4 yr) and for alpha counting efficiency (0.35) were modern values and not those appropriate to the time the original study was done (1945-1946), and it was erroneously assumed that the isotopic composition of the plutonium solution as stated by Crowley et al. (1946) had been measured directly and was accurately known.

A re-examination of the original data sheets and an inquiry into the methods used at the time of the study for preparation of ^{238}Pu and for alpha measurement reveal the following:

(1) The injected dose is most likely that stated on the original data sheets--68,000 counts/sec.

(2) At the time of the study, alpha activity was measured at the Crocker Laboratory with a parallel-plate ionization chamber which has

a counting efficiency of 0.5 (Jaffey 1954). Samples were mounted on platinum disks from which there is backscatter, so that the total alpha counting efficiency was probably 0.517 as calculated by Jaffey (1954).

(3) The ^{238}Pu had most likely been prepared in the 60-in. cyclotron at the Crocker Laboratory by deuteron bombardment of natural uranium (R.E. Connick, private communication). The energy of the deuteron beam was probably close to 16 MeV, because that beam energy produces the most ^{238}Pu with the least contamination by other plutonium isotopes (Jaffey 1949).

(4) According to Jaffey (1949), who recalculated the yield data of Seaborg et al. (1949) and Kennedy et al. (1949) for the reaction of 16-MeV deuterons on uranium, the ratio of alpha activities of the isotopes ^{239}Pu and ^{238}Pu is 0.013. At that time there was no method of separating the plutonium isotopes, so the isotopic composition of the plutonium in the Cal-I injection solution would have been that of the irradiated target. Furthermore, at that time the isotopic composition of the plutonium could have been characterized only by analysis of the radioactivities--their kinds, energies, intensities, and half-lives--and any values given for the masses of plutonium isotopes in the injection solution must have been obtained from calculations involving the alpha activities and the estimates of the half-lives then in use (50 and 24,000 yr, respectively, for ^{238}Pu and ^{239}Pu).

Using the available facts and some assumptions that we believe are consistent with the history and circumstances of the production of the injected ^{238}Pu-^{239}Pu sample, we have recalculated the Cal-I dose as follows.

The injected alpha activity was 68,000 counts/sec measured with an overall efficiency of 0.517, so the total injected dose was $(68{,}000\ c/sec)/(0.517 \times 3.7 \times 10^4\ d/sec/\mu Ci) = 3.55\ \mu Ci$ of alpha activity. If the ratio of the alpha activities was $^{239}Pu/^{238}Pu = 0.013$, then the injection was 3.5 μCi of ^{238}Pu and 0.046 μCi of ^{239}Pu.

APPENDIX B

Dose Calculation

The organ doses were calculated by assuming an exponential retention function $R(t) = e^{-\lambda t}$ and that the dose is given by $D(T) = f \cdot I \cdot Q \int_0^T dt \cdot R(t)$, as indicated in the text.

The appropriate dose rate per unit activity is calculated from the expression

$$Q = \frac{51.2 \cdot \bar{E}}{m}$$

where Q is the average alpha-ray dose rate per unit activity (in rad/day-μCi),

m is the organ mass (in g), and

$\bar{E}$ is the appropriate average alpha-ray energy (in MeV/disintegration).

The values employed for the relevant quantities are as follows.

(1) Organ masses (ICRP 1974)

	Organ mass (g) For males	For females
Skeleton	5000	3400
Liver	1800	1400

(2) Alpha energies

Isotope	Alpha energy (MeV)	Branching ratio (%)
^{238}Pu	5.499	72
	5.456	28
	$\bar{E}$ = 5.487	
^{239}Pu	5.157	73.3
	5.145	15.1
	5.107	11.5
	$\bar{E}$ = 5.149	

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(3) Calculated Q values

Organ	Sex	^{238}Pu (rad/day-μCi)	^{239}Pu (rad/day-μCi)
Skeleton	Male	0.0562	0.0527
"	Female	0.0827	0.0776
Liver	Male	0.156	0.147
"	Female	0.201	0.189

(4) Half-lives

Isotope	Radioactive half-life (yr)	Biological half-life (yr) (ICRP 1972)		Effective half-life (yr)
^{238}Pu	87.7	Liver	40	27.5
		Skeleton	100	46.7
^{239}Pu	24,390	Liver	40	39.9
		Skeleton	100	99.6

REFERENCES

Crowley, J., Lanz, H., Scott, K., and Hamilton, J.G., 1946. A comparison of the metabolism of plutonium (Pu^{238}) in man and the rat. Metallurgical Laboratory Report CH-3589, 15 pp.

da Silva Horta, J., 1973. Tumours developed in people injected with thorium dioxide (Thorotrast) (Portuguese experience). Pages 233-247 in Proceedings of the Third International Meeting on the Toxicity of Thorotrast, The Finsen Institute, Copenhagen 25-27 April 1973, Risø Report No. 294. Danish Atomic Energy Commission Research Establishment Risø, Copenhagen.

Durbin, P.W., 1972. Plutonium in man: a new look at the old data. Pages 469-530 in Radiobiology of Plutonium, J.W. Press, Salt Lake City, Utah. See also Lawrence Berkeley Laboratory Report UCRL-20850, 141 pp.

Faber, M., 1973. Dose effect relations in hepatic carcinogenesis. Pages 308-316 in Proceedings of the Third International Meeting on the Toxicity of Thorotrast, The Finsen Institute, Copenhagen 25-27 April 1973, Risø Report No. 294. Danish Atomic Energy Commission Research Establishment Risø, Copenhagen.

ICRP, 1972. International Commission on Radiological Protection, Report of a Task Group of Committee 2, The metabolism of compounds of plutonium and other actinides. ICRP Publication 19, Pergamon Press, Oxford, 59 pp.

ICRP, 1973. International Commission on Radiological Protection, Report of a Task Group of Committee 2, Alkaline earth metabolism in adult man. ICRP Publication 20, Pergamon Press, Oxford, 92 pp.

ICRP, 1974. International Commission on Radiological Protection, Report of a Task Group of Committee 2, Reference man. ICRP Publication 23, Pergamon Press, Oxford, 480 pp.

Jaffey, A.H., 1949. Half-life of Pu^{238} by direct decay measurement. Pages 89-99 in The Transuranium Elements, McGraw-Hill Book Co., New York.

Jaffey, A.H., 1954. Radiochemical assay by alpha and fission measurements. Pages 596-732 in The Actinide Elements, McGraw-Hill Book Co., New York.

Kaul, A., 1973. Mean organ dose rates in man following intravascular injection of Thorotrast. Pages 40-51 in Proceedings of the Third International Meeting on the Toxicity of Thorotrast, The Finsen Institute, Copenhagen 25-27 April 1973, Risø Report No. 294, Danish Atomic Energy Commission Research Establishment Risø, Copenhagen.

Kennedy, J.W., Perlman, M.L., Segre, E., and Wahl, A.C., 1949. Formation of the 50-year element 94 from the deuteron bombardment of U^{238}. Pages 79-83 in The Transuranium Elements, McGraw-Hill Book Co., New York.

Langham, W.H., Bassett, S.H., Harris, P.S., and Carter, R.E., 1950. Distribution and excretion of plutonium administered intravenously to man. Los Alamos Scientific Laboratory Report LA-1151, 45 pp.

Lloyd, E., and Hodges, D., 1971. Quantitative characterization of bone: a computer analysis of microradiographs. Clinical Orthopaedics 78:230-250.

Marshall, J.H., Groer, P.G., and Schlenker, R.A., 1976. Dose to endosteal cells and relative distribution factors for radium-224 and plutonium-239 compared to radium-226. Health Physics (in press).

Rowland, R.E., Keane, A.T., and Lucas, H.F., Jr., 1973. A preliminary comparison of the carcinogenicity of ^{226}Ra and ^{228}Ra in man. Pages 406-420 in Radionuclide Carcinogenesis, U.S. Atomic Energy Commission Office of Information Services, Washington, D.C.

Rundo, J., Starzyk, P.M., Sedlet, J., Larsen, R.P., Oldham, R.D., and Robinson, J.J., 1974. Plutonium in the excreta of three subjects 10^4 days after injection. Pages 136-141 in Radiological and Environmental Research Division Annual Report, ANL Report 75-3, Part II, Argonne National Laboratory, Argonne, Illinois.

Seaborg, G.T., Wahl, A.C., and Kennedy, J.W., 1949. Nuclear properties of 94^{238} and 93^{238}. Pages 13-20 in The Transuranium Elements, McGraw-Hill Book Co., New York.

Spiess, H., and Mays, C.W., 1970. Bone cancers induced by ^{224}Ra (Th X) in children and adults. Health Physics 19:713-729.

Swarm, R.L., 1968. The neoplasms of the liver following the deposition of thorium dioxide. Pages 117-120 in The Dosimetry and Toxicity of Thorotrast, IAEA Report 106, International Atomic Energy Agency, Vienna.

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