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REANALYSIS OF DATA RELATING TO THE HANFORD STUDY OF THE CANCER RISKS OF RADIATION WORKERS

By

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Summary

A study of workers in the nuclear industry who had linked records of external radiation doses and certified causes of death (1944-72 deaths of Hanford workers) was followed by a similar analysis of a larger sample of Hanford data (1944-77 deaths). The second study included one test which showed that, in surveys of the delayed effects of low level radiation, comparisons between observed and expected doses of cancer cases (CMD method) are more informative than comparisons between observed and expected cancer deaths of exposed workers (SMR method). A second test (which took the form of a Mantel-Haenszel analysis and included exposure period and internal radiation among the controlling factors) showed that there were genuine differences between the radiation doses of two groups of certified deaths (cancers and non-cancers).

Both studies produced evidence of a cancer hazard from low doses of external radiation even when delivered at low dose rates. According to the second study approximately 5% of the cancer deaths of Hanford workers were radiation-induced and these extra deaths were probably concentrated among cancers of the bone marrow, lung and pancreas.

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A recent study of men and women who were repeatedly exposed to measured doses of low level radiation before dying of cancers and other causes (Hanford data)[1] has raised uncertainties about the cancer hazards of workers in the nuclear industry by producing risk estimates of a different order of magnitude from ICRP 26 recommendations.[2] The analysis of Hanford data was designed to take full advantage of the fact that for all badge-monitored workers there were annual doses of external or penetrating radiation. It therefore followed unusual lines and was open to criticism by advocates of an alternative and more familiar method.

It was clearly important to know which was the more reliable method and to observe the effects of simultaneous control of several factors with radiation or cancer associations. Therefore we first tested the relative efficiency of the two methods, and then applied the more efficient one to a larger sample of Hanford data after applying a Mantel-Haenszel test[3] to the null hypothesis of no difference in the external radiation doses of cancers and non-cancers.

The method actually used in the earlier study compared observed with expected radiation doses and had as cases and controls cancer and non-cancer deaths (Comparative Mean Dose or CMD Method). The alternative was to compare observed with expected cancer deaths and to have as cases and controls exposed and non-exposed workers (Standardized Mortality Ratio or SMR Method). The earlier study was based on 3,520 men and 412 women who died between 1944 and 1972, and the larger sample of Hanford data included 4,694 men and 575 women who died between 1944 and 1977 (table 1).

Test of the Relative Efficiency of the SMR and CMD Methods

(1) Let a population of size N be exposed to a radiation dose distribution of f(x) with a mean dose (R) and a variance (V) such that:

$$R = \int_0^\infty xf(x)dx \text{ and } V = \int_0^\infty (x-R)^2 f(x)dx.$$

(2) Let the risk of a cancer death increase linearly with the radiation dose from level A at zero dose (normal cancer risk) according to doubling dose (D), such that the risk for dose x is A(1+x/D).

(3) Let n be the number of cancer deaths actually observed in a study population with a mean radiation dose r.

Given these conditions the necessary size of the population for detecting a given doubling dose (at a given size and power of the test) will depend upon which approach is taken. Thus the SMR method will require a known value of A which can usually be obtained from official sources (e.g., in Britain, the male cancer death rate is currently in the region of 2,600 per million). Under the hypothesis of no radiation effect, the number of cancer deaths actually observed (n) would have a Poisson distribution with AN as the mean value. Therefore, provided N was large enough to apply a normal approximation to the Poisson distribution, the condition for significance would be

$$n > AN + t\sqrt{AN}$$

where t is the critical normal deviate corresponding to a given significance level (or size of the test).

One now requires the power of the test, or the probability of the significance condition being satisfied under the hypothesis of some radiation effect (with D as the doubling dose). Under this hypothesis the number of cancer deaths actually observed (n) would have a Poisson distribution with AN(1+R/D) as the mean value. Therefore, the required power would be

$$\Phi\left[\frac{AN + t\sqrt{AN} - AN(1+R/D)}{\sqrt{AN(1+R/D)}}\right]$$

where Φ is the integrated normal distribution.

If the power of the test is given the convenient value of 1/2 (so that one is equally likely or unlikely to detect a significant difference between actual and expected cancer deaths), the argument to Φ will become zero — or $t\sqrt{AN} = ANR/D$ — and the necessary size of the base population will be $\frac{t^2D^2}{AR^2}$. Therefore, assuming: (i) a doubling dose of 15 rads; (ii) a mean radiation dose of 1.6 rads; (iii) a normal cancer risk of 2,600 per million; and (iv) a 5% level of significance, the SMR method would require a population of 135,000, or one which was approximately 5 times larger than the original Hanford population.

The corresponding calculations for the CMD method are as follows: On the hypothesis of no radiation effect the mean cancer dose (r) would have an approximately normal distribution with mean R and variance V/n. Therefore, the condition for significance would be $r > R + t\sqrt{V/n}$. On the alternative hypothesis (of some radiation effect with D as the doubling dose) the mean radiation dose (r) would have an approximately normal distribution with $\frac{R+(R^2+V)/D}{1+R/D}$ as the mean value (see Mancuso, et al., 197 and the condition for a power of 1/2 would be $t\sqrt{V/n} = \frac{R+(R^2+V)/D}{1+R/D} - R$. E allowing the approximate mean value of n to be AN the necessary size of the base population would be $\frac{t^2(D+R)^2}{AV}$. Therefore, to detect a doubling dose of 15 rads with a mean radiation dose of 1.6 rads (and the additional information that for Hanford males $\sqrt{V} = 3.6$ rads) the CMD method would only require a population of 32,700, or one similar in size to the present Hanford population.

Finally, the general formula for the efficiency of the CMD metho (compared with the SMR method) is $\frac{D^2V}{(D+R)^2R^2}$. Therefore should the radia tion doses have a wide scatter about the mean, as was certainly the case in Hanford data, comparisons between actual and expected radiation doses of cancer cases should be more reliable than comparisons between actual and expected cancer deaths of exposed workers.

Following this vindication of the CMD method steps were taken to ensure that factors other than external radiation were not influencing th results.

Avoidance of Spurious Associations

In the original study and again in the larger sample of Hanford data the mean radiation doses were higher for cancers than non-cancers (table 2). Therefore the first question requiring a reliable answer was whether these were genuine findings or the result of accidental differences between two groups of certified deaths.

In the earlier study the radiation dose differences remained after controlling separately for five possible sources of bias, including age at death. In the repeat analysis they remained after simultaneous control of the following factors: sex, age at death, date of death, internal radiation and exposure period (tables 3 & 4). The second test took the form of a Mantel-Haenszel analysis which also showed that:

(i) relative risks for different levels of external radiation were only slightly altered in the controlled as compared with the crude analysis (changing from a relative risk of 1.35 to 1.26 in the highest dose category);

(ii) the progressive component for seven dose levels was suggestive of a dose-dependent effect without threshold (i.e., a stochastic effect, see ICRP 26);

(iii) controlling for internal radiation -- which was strongly correlated with external radiation and therefore suspected of causing most if not all of the dose difference between cancers and non-cancers[4 & 5] -- actually strengthened the external radiation effect;

(iv) Hanford females were more cancer sensitive than Hanford males; and

(v) cancers accounted for a higher proportion of deaths between 50 and 60 years than of earlier or later deaths.

Detection of Cancers with Definite Radiation Effects

Since the null hypothesis of no difference in the radiation doses of cancers and non-cancers was rejected by the Mantel-Haenszel test there were strong reasons for suspecting that some of the cancer deaths were radiation-induced. But the question remained: were the extra deaths evenly distributed between the different malignant diseases or concentrated in radiosensitive organs or tissues?

Even in the larger sample of Hanford data there were insufficient numbers to treat the 59 malignant diseases listed under different ICD numbers as separate entities. In the earlier study we allowed the choice of suitable groups to be influenced by the radiation doses. In the repeat analysis the choice was determined solely by ICRP 14[6] or the publication which included a totally independent classification of radiosensitive tissues under the following headings: I. High Sensitivity Established; II. High Sensitivity Apparent; III. Low Sensitivity; and IV. Not Classified (table 5).

In the larger sample of Hanford data the first and second categories included 456 males and 30 females and the third and fourth categories included 287 males and 59 females. For males the mean radiation doses (reading from I to IV) were 405,244, 152 and 93, and for females they were 0, 115, 60 and 83.

Mean Radiation Doses by Pre-Death Years

For three quarters of all the certified deaths of badge-monitored men in table 1 there were records of annual radiation doses for at least 15 years before death (tables 6 & 7). For each of these years the mean cumulative radiation dose (which was strongly correlated with the number of separate exposures) was greater by a significant amount for cancers than non-cancers (table 8 & figure 1), but only two of the four cancer groups (I & II) were responsible for these differences (table 8 & figure 2). For females there were similar findings but owing to the small numbers differences between cancers and non-cancers only achieved statistical significance towards the end of the time scale (table 9 & figure 3).

Estimated Doubling Doses for Cancers Showing Definite Radiation Effects

Comparisons between the two methods had shown that, given a population of 30,000, the CMD method would be more efficient than the SMR method. Therefore the same formula for estimating the doubling dose was used in the repeat analysis as in the earlier study (see Appendix ii of the 1977 report).

On the first occasion only 2 of 9 groups of malignant diseases showed definite evidence of a radiation effect (cancers of bone marrow and pancreas) but lung cancer also showed doubtful evidence of this effect. For these three groups the estimated doubling doses were 0.8, 7.4 and 6.1 rads. On the second occasion there was definite evidence of a radiation effect for groups I and II and for the following subgroups: (i) myeloma and myeloid leukemia with a doubling dose of 3.6 rads; (ii) lung cancer with a doubling dose of 13.7 rads (figure 4); and

(iii) cancers of the pancreas, stomach and large intestine with a doubling dose of 15.6 rads. For one component of group II (lymphoma and reticulum cell sarcoma) there was no evidence of any radiation effect among the male cases. However the female cases included the woman with the highest dose (1,573 centirads) and for all female cancers the estimated doubling dose was 8.6 rads.

For all male cancers the doubling dose was higher in the repeat analysis (33.7 rads) than in the earlier study (12.2 rads). As, however, the lowered estimate of risk still allowed 35 of the 743 male cancers to be radiation-induced (and the expected number was 4.8)[2] there remained a wide gap between risk estimates based on workers in the nuclear industry and ones based on A-bomb survivors and patients with ankylosing spondylitis.

Effect of Age on the Cancer Induction Effects of Radiation

On the basis of the earlier findings we concluded that sensitivity to the cancer induction effects of radiation decreased with age before 30 years and increased with age thereafter. On the basis of the repeat analysis (figure 5) we concluded that more data was needed before we could be certain of age trends before 30 or after 60 years, but between these ages there was definite evidence of an increase in sensitivity (figure 5).

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Checking the Validity of Hanford Based Risk Estimates

In the earlier study we used differences between actual and expected cancer deaths (or the SMR method) to test the validity of the risk estimates derived from the CMD analysis. In the repeat analysis we compared relative risks from the Mantel-Haenszel analysis, first, with the corresponding risks in a crude analysis (table 4) and then with the doubling dose for all male cancers (figures 6 & 7).

The first test showed that the risk estimates were only slightly altered in the controlled as compared with the crude analysis (changing from a relative risk of 1.35 to 1.26 in the highest dose group), and to the second showed that seven points on the curve for relative risks at different dose levels in the controlled analysis were clustered reasonably close to the doubling dose projection line (assuming a linear model).

Discussion

In 1974 Milham discovered an excess of cancer deaths among Hanford workers who died between 1950 and 1973[7] and three years later we confirmed this finding in a larger sample of Hanford data and also produced evidence of a radiation effect for three cancers (myeloma, pancreas and lung).[1] Each report was followed by a peer review (commissioned by ERDA) purporting to show that there was no certain evidence of any radiation effects in Hanford data. However, on the first occasion a significant difference between exposed and non-exposed workers (and a strict increase of effect with radiation dose) was found,[8] and on the second occasion two cancers with radiation effects (myeloma and pancreas) were found after adjusting for the effects of sex, age at death and date of death.[4] Also the present study has confirmed the earlier findings and shown that there is virtually no chance of a significant dose difference between cancers and non-cancers being the result of accidental differences between two groups of certified deaths.

Consequently we not only agree with Milham that "an occupational hazard exists for Hanford workers" but would also think that future ICRP recommendations should be based, not on A-bomb survivors and radiotherapy patients, but on workers in the nuclear industry.

Critics of Hanford data were well-acquainted by ICRP recommendations and evidently saw no reason to doubt the validity of the risk estimates contained in the most recent publication.[2] Therefore they expected bone marrow to be more sensitive to the cancer induction effects of radiation than other tissues and expected some effect from low level radiation. However they also expected the bone marrow effect to take the

form of myeloid leukemia, and expected the dose response curve to be highly sigmoid — thus making it unlikely that Hanford works (which has kept radiation doses far below ICRP maximal permissible doses), would have any radiation-induced cancer deaths among its employees.

These expectations reflect the experiences of two populations (A-bomb survivors and radiotherapy patients) who differ from any population of workers in the nuclear industry in at least two respects: they had much higher rates of non-cancer mortality, and they were only briefly exposed to relatively large doses of radiation.

The first difference is clearly important because data from the Oxford Survey of Childhood Cancers have shown that the precancer state is associated with lowered immunological competence to such an extent that children are in grave danger of dying from secondary infections and accidents before the true state of affairs can be recognized.[9] As a result of these "latent period deaths" serious discrepancies between cancer initiation rates and cancer mortality rates may be introduced,[10] thus making it peculiarly unsafe to base any radiation risk estimates on populations with exceptionally high non-cancer death rates.

The second difference could be the reason why bone marrow effects have taken the form of myeloid leukemia in three populations with brief exposures to relatively large doses of radiation (i.e., A-bomb survivors, early entrants to Hiroshima and Nagasaki after the explosions, and radiotherapy patients) and the form of myeloma in two populations with prolonged exposures to small doses (i.e., radiologists[11] and Hanford workers). It is true that we know very little about the effects of dose

fractionation per se. On the other hand clinical experience suggests that the greater the insult the greater the probability of diffuse disease and the smaller the insult the greater the probability of localized disease.

Although "cumulative radiation doses" played an important role in the CMD analysis of Hanford data this does not imply any cumulative effects of the exposures. For Hanford workers even one exposure to a sizeable dose of radiation was a rare event. Therefore the total radiation dose of each worker was strongly correlated with the number of separate exposures, or the number of times that there was any probability of a stochastic effect.

According to ICRP 26 the mortality risk factor for radiation-induced cancers is about $10^{-2}Sv^{-1}$ (as an average for both sexes and all ages) and the corresponding figure for leukemia is $2 \times 10^{-3}Sv^{-1}$. However, a recent follow-up of "early entrants" (or persons who entered Hiroshima and Nagasaki less than 4 days after the explosions and therefore combined brief exposure periods with relatively low rates of non-cancer mortality), has produced a much higher figure for leukemia, namely $18 \times 10^{-3}Sv^{-1}$.[12] Consequently the Hanford study is no longer the only one to question the validity of the risk estimates contained in ICRP 26.

Since two populations with low rates of non-cancer mortality have produced higher risk estimates than two populations with high rates it is no longer safe to assume that the dose response curve is highly sigmoid.[2] On the contrary since any non-stochastic effects of radiation necessarily have some effect on non-cancer mortality it is possible that

linear extrapolation for high doses will slightly underestimate the risk at low doses.

Finally an important reason for suggesting that future estimates of risk be based on Hanford data is because this population is currently the only one to provide a suitable model for studying the effects of low doses of radiation delivered at low dose rates to individuals whose non-cancer death rate is not increased.

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TABLE 1

Badge-Monitored (External Radiation)	Urine-Monitored (Internal Radiation)	Certified Deaths (1944-77)			Uncertified Deaths	Survivors
		Cancers	Non-Cancers	All Causes		
Monitored Males	Not Monitored	264	1,188	1,452	67	4,386
	Monitored (negative)	194	782	976	28	2,739
	Monitored (positive)	285	1,029	1,314	34	10,884
	Total	743	2,999	3,742	129	17,929
Monitored Females	Not Monitored	33	95	128	20	2,114
	Monitored (negative)	21	50	71	8	1,155
	Monitored (positive)	35	57	92	7	2,487
	Total	89	202	291	35	5,756
All Monitored Workers	Not Monitored	297	1,283	1,580	87	6,500
	Monitored (negative)	215	832	1,047	36	3,894
	Monitored (positive)	320	1,086	1,406	41	13,371
	Total	832	3,201	4,033*	164	23,765
Not Monitored	Males	181	771	952	46	2,362
	Females	88	196	284	51	3,191
	Total	269	967	1,236	97	5,553

*This is the population of badge-monitored workers included in later analyses.

TABLE 2

ex	Certified Causes of Death	ICD 8th Revision	Cases	Penetrating Radiation[1] Total	Mean
	Cancers	140-209	743	150,952	203
	Cardiovascular	390-458	1,988	314,636	158
	Respiratory	460-519	198	29,578	149
s	Digestive	520-577	140	31,694	226
	Violence	800-999	424	55,985	132
	Other Causes	Residue	249	38,804	156
	All Causes	000-999	3,742	621,649	166
	Cancers	140-209	89	7,901	89
	Cardiovascular	390-458	106	6,518	61
	Respiratory	460-519	15	808	54
les	Digestive	520-577	16	912	57
	Violence	800-999	36	975	27
	Other Causes	Residue	29	872	30
	All Causes	000-999	291	17,986	62
All Non-Cancer Deaths		Males	2,999	470,697	157
		Females	202	10,085	50

ll doses in centirads.

TABLE 3

Factors	Levels	Cases (Cancers)	Controls (Non-Cancers)
Sex	Male	743	2,999
	Female	89	202
Final Age	Under 40 years	38	206
	40-49 years	96	396
	50-59 years	223	707
	60+ years	475	1,892
Death Year	1944-54	69	284
	1955-59	79	327
	1960-64	123	575
	1965-69	181	715
	1970-77	380	1,300
Internal Radiation	Not Monitored	297	1,283
	Monitored (negative)	215	832
	Monitored (positive)	320	1,086
Exposure Period	Under 2 years	280	1,223
	Over 2 years	552	1,978
External Radiation	Under 8 Centirads	256	1,068
	8-31 Centirads	131	592
	32-63 Centirads	119	428
	64-127 Centirads	123	448
	128-255 Centirads	91	320
	256-511 Centirads	48	147
	over 511 Centirads	64	198

TABLE 4[1]

st Factors	Levels	Cancer Deaths Observed	Cancer Deaths Expected	t-values O-E	t-values Progressive Component	Relative Risk[2]
x	Male	478	496.6	-3.0*		1.00 (1.00)
	Female	81	62.4	+3.0*	-	1.57 (1.78)
nal Age	Under 40 years	34	46.4	-2.3*		0.61 (0.73)
	40-49 years	92	97.3	-0.7		0.93 (0.96)
	50-59 years	220	188.8	+3.0*		1.30 (1.25)
	60+	468	481.4	-1.2	+0.8	1.00 (1.00)
ath Years	1944-54	66	61.7	+0.7		0.99 (0.83)
	1955-59	78	77.9	+0.0		0.91 (0.83)
	1960-64	120	138.1	-2.0*		0.77 (0.73)
	1965-69	178	183.7	-0.6		0.88 (0.86)
	1970-77	360	340.6	+1.7	+0.9	1.00 (1.00)
ternal diation	Not Monitored	249	251.6	-0.3		1.00 (1.00)
	Monitored -ve	201	197.8	+0.4		1.06 (1.12)
	Monitored +ve	273	273.6	-0.1	+0.1	1.00 (1.27)
posure riod	Under 2 years	245	250.5	-0.8		1.00 (1.00)
	Over 2 years	229	223.5	+0.8	-	1.12 (1.22)
ternal diation	Under 8 centirads	235	240.6	-0.6		1.00 (1.00)
	8-31 centirads	129	146.5	-1.9		0.86 (0.92)
	32-63 centirads	119	108.9	+1.2		1.15 (1.15)
	64-127 centirads	121	117.9	+0.4		1.14 (1.14)
	128-255 centirads	91	89.9	+0.2		1.10 (1.19)
	256-511 centirads	47	43.9	+0.6		1.08 (1.36)
	over 511 centirads	64	58.8	+0.9	+2.0*	1.26 (1.35)

ignificant at the 5% level or higher.

)The observed number of cancer deaths in the results of a Mantel-Haenszel analysis are necessarily smaller than those in the basic data because of the necessity of excluding from the fully-controlled analysis non-informative cases (or cases without controls matched for every factor except the one of immediate interest). The methodology necessitating this exclusion is described in the appendix to Kneale and Stewart (1976).[13]

)Uncontrolled risk estimates in brackets, see table 3.

TABLE 5

ICRP Classification of Cancers	ICD Nos. (8th Rev.)	Males Cases	Males Mean R Dose in Centirads	Females Cases	Females Mean R Dose in Centirads
High Sensitivity					
I. Established					
(a) Bone Marrow	203	10	861	1	0
	205	15	125	-	-
(b) Thyroid	193	1 (26)	44 (405)	- (1)	- (0)
II. Apparent					
(a) Lymph Nodes	200-1	33	136	2	117
Reticular Tissue	202	7 (40)	29 (117)	1 (3)	1,573 (602)
(b) Pharynx	146-9	10	481	-	-
Lung	162-3	215 (225)	258 (268)	10 (10)	52 (52)
(c) Pancreas	157	52	391	5	13
Stomach	151	44	240	2	109
Large Intestine	153	69 (165)	132 (242)	9 (16)	80 (63)
III. Low Sensitivity					
Mouth and Salivary	140-5	15	133	-	-
Esophagus	150	19	43	-	-
Small Intestine	152	2	32	-	-
Liver and Gall Blad.	155-6	18	252	2	230
Nose and Larynx	160-1	14	143	-	-
Bone, C.T. and Skin	170-3	18	149	6	30
Testis and Penis	186-7	3	114	-	-
Kidney	189	23	178	2	44
Eye and CNS	190-2	28	170	5	35
Other Endocrine	194	- (140)	- (152)	- (15)	- (60)
IV. Unclassified					
Rectum	154	20	101	2	30
Other Digestive	158-9	2	38	1	3
Breast	174	1	0	19	120
Uterus and Ovaries	180-4	-	-	11	37
Prostate	185	52	119	-	-
Bladder	188	13	87	-	-
Lymphatic Leukemia	204	3	19	3	70
Other Haemopoetic	206-9	8	23	3	1
Ill Defined	195-9	48 (147)	93 (96)	5 (44)	140 (83)
All High Sensitivity Groups		456	253	30	111
Residual Cancers		287	123	59	78

TABLE 6

e-Death Years	Non-Cancers	Cancers (ICRP Classification) I	II	III	IV	All Cancers
29	377	3	56	11	32	102
28	513	4	72	16	39	131
27	642	4	99	23	44	170
26	767	5	119	28	49	201
25	929	8	148	39	56	251
24	1,059	9	173	45	70	297
23	1,200	11	196	51	79	337
22	1,330	13	212	58	85	368
21	1,470	14	227	66	92	399
20	1,600	15	247	73	97	432
19	1,735	16	260	77	100	453
18	1,854	20	273	83	106	482
17	1,983	20	288	87	110	505
16	2,096	20	305	93	114	532
15	2,198	20	327	97	118	558
14	2,287	21	337	100	123	581
13	2,363	22	352	103	124	600
12	2,442	22	365	108	128	623
11	2,509	22	372	114	130	638
10	2,578	22	380	118	132	652
9	2,640	22	393	122	134	671
8	2,702	22	402	125	136	685
7	2,762	23	408	126	138	695
6	2,806	23	411	129	140	703
5	2,854	26	418	131	140	715
4	2,891	26	424	134	141	725
3	2,927	26	426	138	141	731
2	2,953	26	429	138	144	737
1	2,987	26	430	138	146	740
0	2,999	26	430	140	147	743

TABLE 7

Pre-Death Years	Expected Mean Doses	Actual Mean Doses of Cancer Cases[1] I	II	III	IV	All Cancers
29	21	52	27	31	25	28
28	22	44	30	37	26	28
27	23	49	29	23	26	28
26	26	41	32	26	27	30
25	27	26	33	26	32	31
24	30	25	30	29	28	32
23	34	34	39	31	30	35
22	37	31	44	31	34	40
21	40	39	50	30	38	43
20	44	47	55	33	40	47
19	48	59	63	38	42	54
18	51	57	68	46	46	59
17	56	83	75	52	51	67
16	61	112	85	57	55	75
15	66	140	100	63	59	83
14	71	161	105	69	61	91
13	77	179	116	76	64	100
12	84	204	125	89	66	109
11	91	235	138	93	68	119
10	98	271	150	97	71	128
9	106	313	160	102	73	137
8	114	346	173	111	75	147
7	121	365	185	121	78	158
6	129	386	1987	128	80	167
5	137	374	203	137	83	175
4	143	389	214	142	86	183
3	148	403	229	143	90	190
2	152	413	234	149	92	197
1	155	414	242	153	95	202
0	157	414	244	153	96	204

[1] For cancer classification see table 5 and for expected doses see non-cancers in table 6. All radiation doses in centirads.

TABLE 8

Pre-Death Years	t-Values for the Difference Between Observed and Expected Doses*				
	Ca I	Ca II	Ca III	Ca IV	All Cancers
29	+1.4	+1.1	+0.5	+0.3	+1.5
28	+1.0	+1.4	0.0	+0.4	+1.5
27	+1.2	+1.3	-0.2	+0.3	+1.3
26	+0.7	+1.5	-0.2	+0.1	+1.3
25	-0.1	+1.4	-0.5	+0.6	+1.2
24	-0.3	+1.1	-0.3	-0.4	+0.5
23	0.0	+1.1	-0.3	-0.7	+0.4
22	-0.3	+1.5	-0.7	-0.5	+0.5
21	-0.1	+1.7	-0.9	-0.4	+0.7
20	+0.1	+1.8	-0.8	-0.5	+0.8
19	+0.4	+2.1	-0.7	-0.6	+1.1
18	+0.2	+2.1	-0.4	-0.6	+1.3
17	+0.8	+2.3	-0.3	-0.5	+1.6
16	+1.4	+2.5	-0.3	-0.6	+1.9
15	+1.8	+2.7	-0.2	-0.7	+2.1
14	+2.0	+2.9	-0.3	-0.8	+2.2
13	+2.1	+3.1	-0.2	-0.9	+2.3
12	+2.3	+3.0	+0.1	-1.1	+2.4
11	+2.5	+3.2	0.0	-1.3	+2.4
10	+2.8	+3.3	-0.1	-1.4	+2.5
9	+3.2	+3.3	-0.0	-1.6	+2.4
8	+3.6	+3.6	0.0	-1.9	+2.7
7	+3.7	+3.7	+0.0	-1.9	+2.8
6	+3.6	+3.7	0.0	-2.0	+2.7
5	+3.4	+3.7	+0.0	-2.1	+2.7
4	+3.5	+3.9	+0.0	-2.3	+2.8
3	+3.5	+4.1	-0.1	-2.2	+2.9
2	+3.6	+4.4	-0.1	-2.4	+3.1
1	+3.6	+4.7	-0.1	-2.4	+3.2
0	+3.5	+4.6	-0.1	-2.4	+3.1

*Due to the skewness of the dose distribution normal approximations to one-sided significance levels for t-values only apply if the number (n) in Table 6 exceeds the given value. Thus:

n > 20 and t > 1.7 means p < 0.05

n > 50 and t > 2.3 means p < 0.01

n > 200 and t > 3.0 means p < 0.001

TABLE 9

Pre-Death Years	Exposed Females		Mean R Dose for all Cancers		t-v
	Non-Cancers	Cancers	Expected	Observed	0
29	34	11	10	29	+1
28	41	19	9	18	+1
27	52	26	8	14	+1
26	63	29	9	19	+1
25	74	32	9	21	+1
24	84	37	11	21	+1
23	94	39	13	23	+1
22	103	43	16	23	+1
21	118	46	17	25	+1
20	124	49	20	24	+0
19	133	50	21	27	+0
18	139	57	23	26	+0
17	152	62	23	25	+0.
16	156	64	24	27	+0.
15	163	66	25	29	+0.
14	166	68	37	32	+0.
13	170	69	30	37	+0.
12	172	72	33	44	+0.
11	176	75	36	52	+1.
10	179	76	39	60	+1.
9	182	78	41	64	+1.
8	183	79	43	69	+1.
7	186	80	45	73	+1.
6	190	82	46	75	+1.
5	193	86	47	77	+1.
4	196	87	47	81	+1.
3	201	87	47	84	+1.
2	201	88	49	85	+1.
1	201	89	49	88	+1.
0	201	89	50	88	+1.

*See table 8.

TABLE 10

	Cancers	Cases	Doubling Dose in ~~Centi~~rads[1]			Pre-Death Years[2]	
			Estimate	95% Confidence Limits		Exceptional Ones	Maximum t-value
	Myeloma and Myeloid Leukemia	25	3.6	1.7	10.3	15-0	3.7*
	Lymphoma and Reticulum Cell Sarcoma	40	-	-	-	-	0.7
	Lung Cancer	215	13.7	7.3	28.7	20-0	3.7*
	Pancreas, Stomach and Large Intestine	165	15.6	7.3	55.0	8-0	2.7*
	All High Sensitivity Groups (I & II)	456[3]	13.9	8.4	21.2	21-0	5.3*
	Other Cancers (III & IV)	287	-	-	-	-	0.6
	All Cancers	743	33.7	15.3	79.7	16-0	3.2*
s	All Cancers	89	8.7	2.6	-	4-0	1.9*

uming a linear model (see Mancuso, et al., 1977).

eptional years when the total radiation dose was significantly higher than the corresponding e for non-cancer deaths.

luding cancers of thyroid and pharynx (see table 5).

table 4.

Figure 1

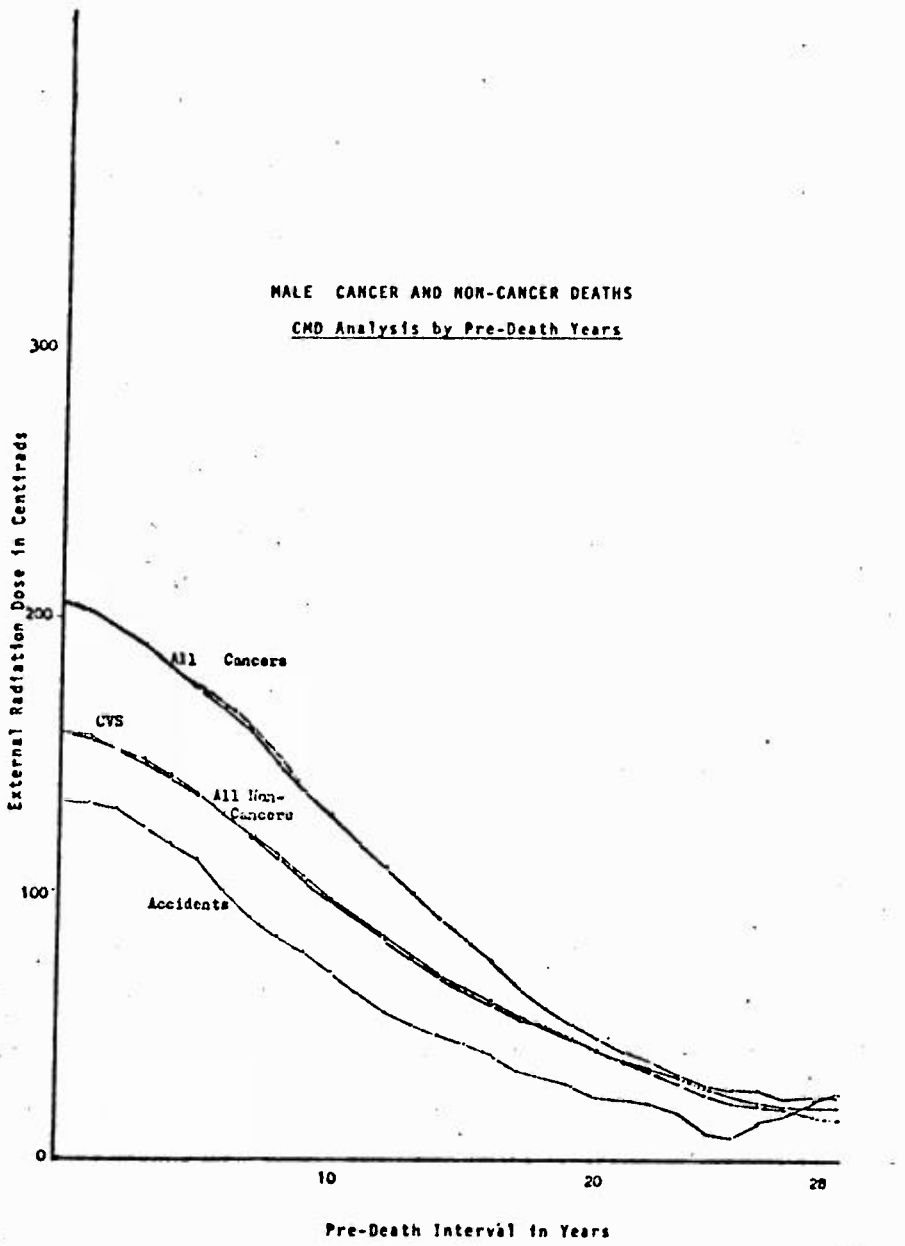

Figure 2

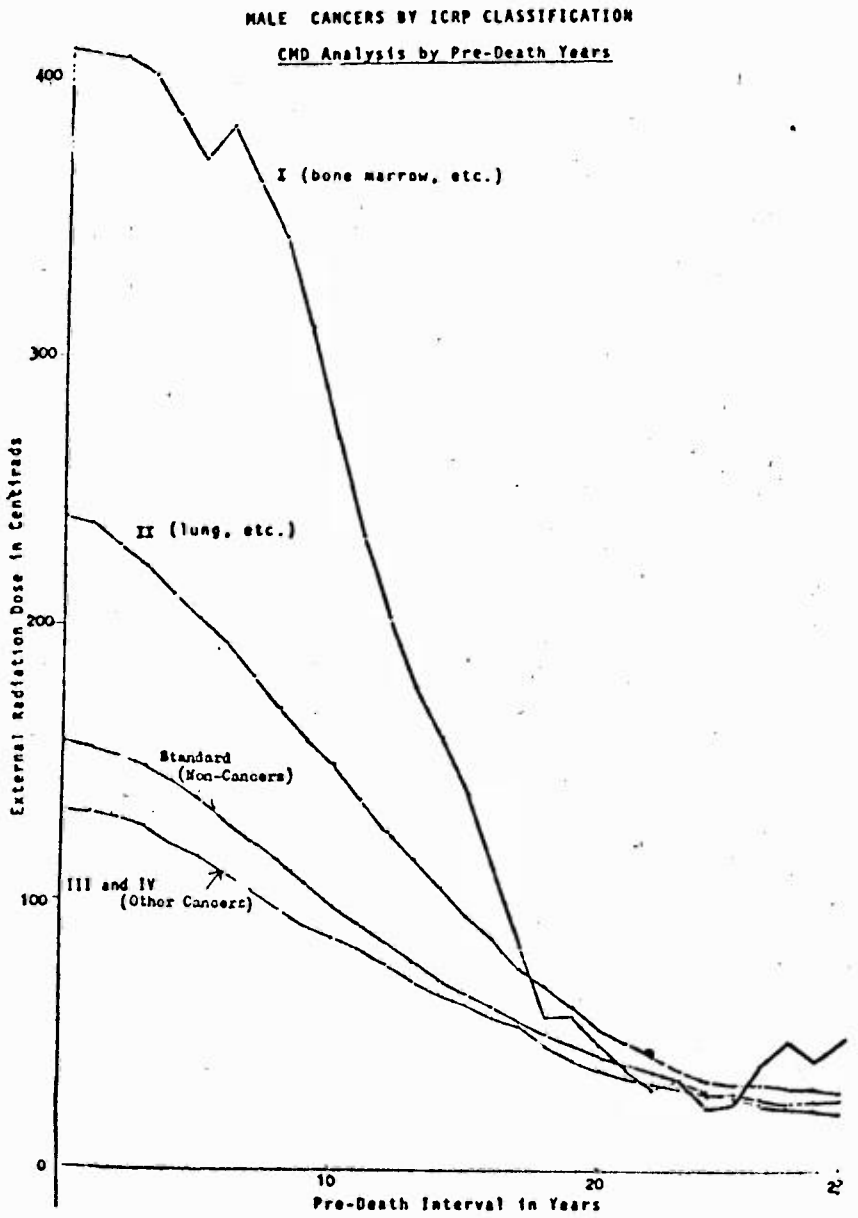

Figure 3

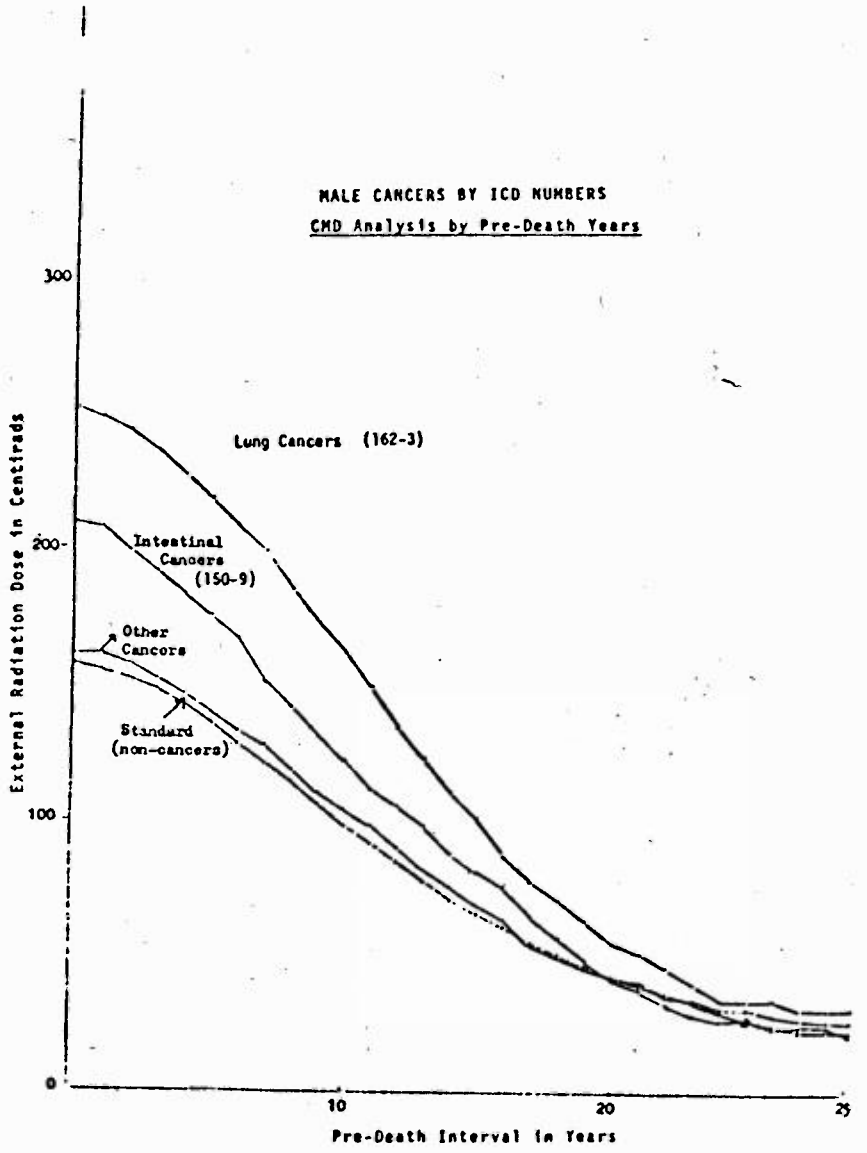

Figure 4

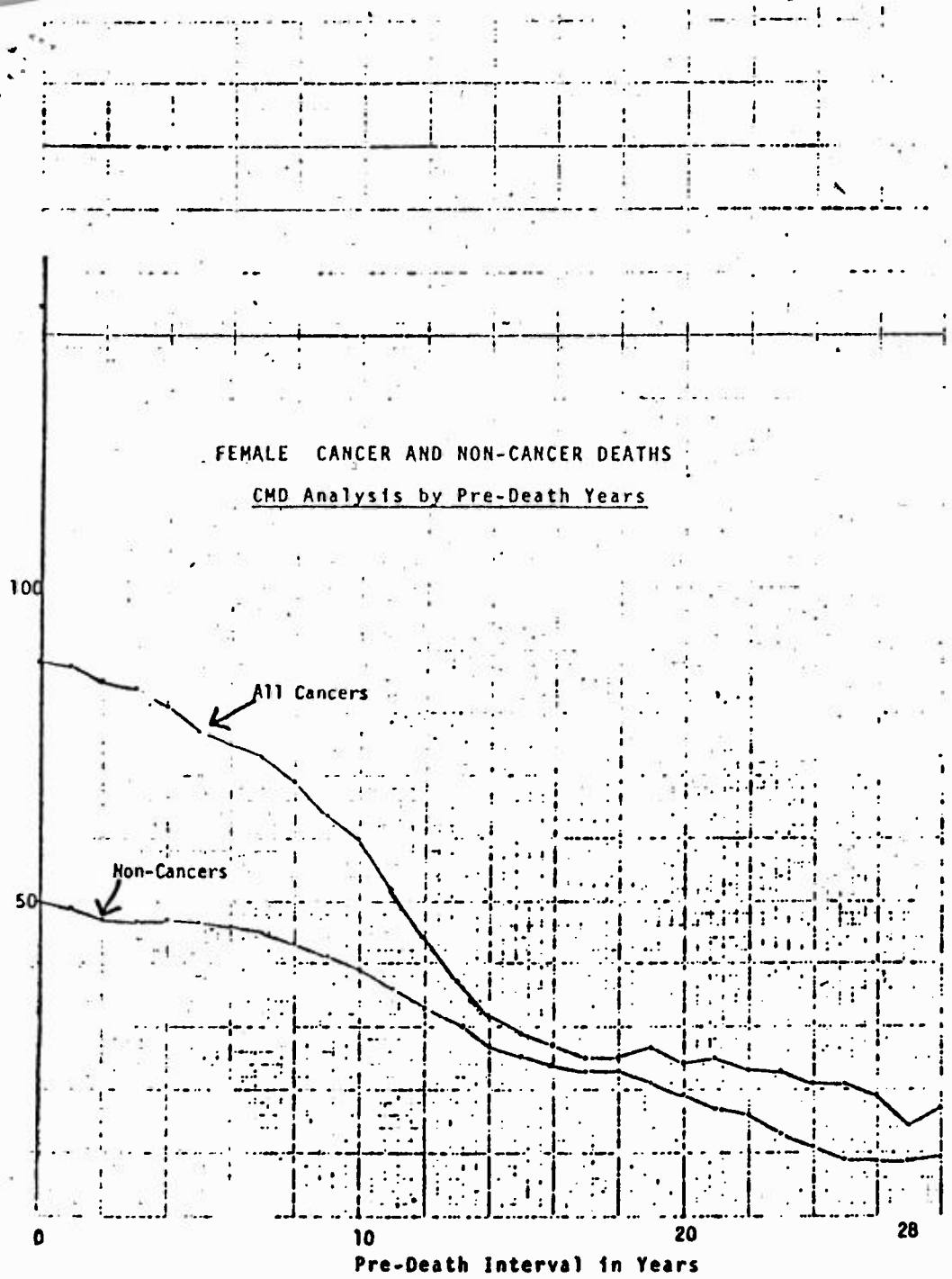

Figure 5