The Hanford Data– A Reply to Recent Criticisms

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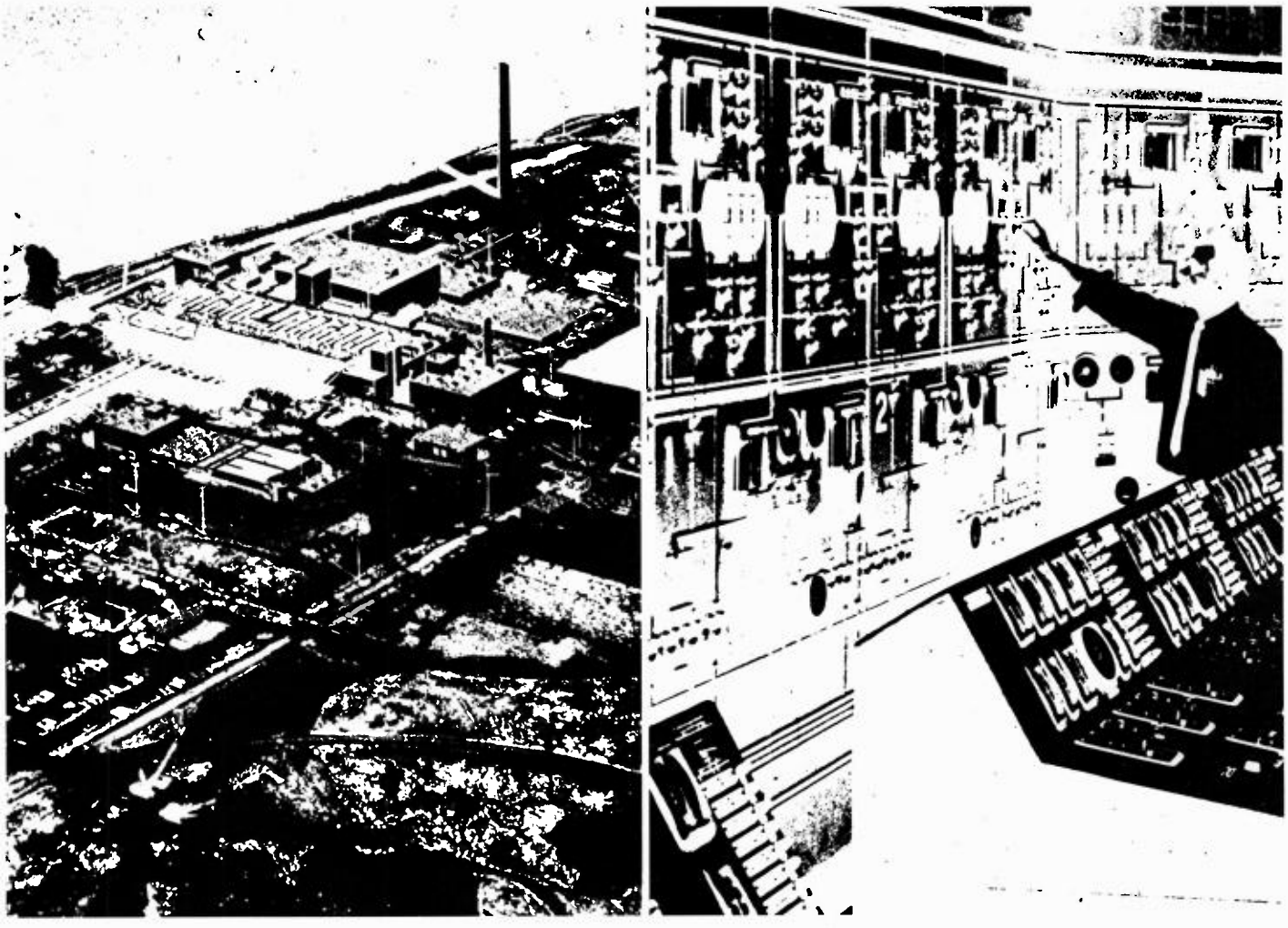
According to the National Radiation Protection Board of Britain (NRPB) scientists from many disciplines view the conclusions reached by Mancuso and his associates in their studies of radiation workers as invalid. It is clear from the official report that two of the three cancers, which were found by Mancuso et al *to be a special risk for workers in the nuclear industry, also feature as radiation-related diseases in their analyses of essentially the same records (Hanford data). But even so, the general consensus of opinion is that there are no grounds for believing that current safety recommendations are based on false premises. In this article the authors respond to criticisms given in the NRPB report and give their reasons for not agreeing with the official opinion.*

The Hanford Plant in the state of Washington, USA. Left is an aerial view of the plant complex and right shows the control room. Photos: Critical Mass, Washington DC.



The Hanford study, which surveyed workers in the nuclear industry, has aroused much controversy because it is the source of risk estimates for cancer effects at rather low radiation levels. Furthermore, these estimates are much greater than those based on A-bomb survivors and radiotherapy patients which are widely used in the setting of safety standards for radiation workers.

The Hanford Works, located in the state of Washington, USA, manufactures plutonium, and the workers have been kept under continuous surveillance since 1944. It was originally intended to compare the mortality experiences of workers in both safe and dangerous occupations with Official Statistics of Mortality (the so-called SMR or Standardized Mortality Ratio method). However, this method is slow to recognize small differences between observed and expected deaths and requires more information about the method of selecting workers for different jobs than was contained in the survey data. However, the data included annual doses of external (or penetrating) radiation and death certificates of workers who lived for many years after their initial and final exposures. Therefore, it was important to compare the radiation doses of workers who had fatal cancers with those who died from other causes (the so-called CMD or Comparative Mean Dose method).

A preliminary analysis of men who died either from cancers (670 cases) or from other certified causes (2850 controls) before 1973, showed that the mean cumulative dose was significantly higher for the cases (1.38 rads) than the controls (.99 rads), see Reference 1. This study, (known as the MSK analysis and named after the authors of the report) showed that there were sufficient data: A) to identify some of the more radiosensitive cancers; B) to quantify the radiosensitivity of these neoplasms; C) to obtain estimates of the latent periods of these cancers; D) to observe the effect of age on sensitivity to cancer induction by radiation.

Further work was needed to be quite certain that the positive findings from three types of cancers (myeloma, pancreas and lung) were not artifacts due to indirect associations between the exposures and the cancers. Therefore, as soon as the necessary records were available, an analysis was applied to 4051 workers who died before 1977 (the so-called KSM analysis). This allowed simultaneous control of five factors: sex, date of birth, date of death, exposure period, and monitoring for internal radiation.

In the first (MSK) analysis, 813 men who were never issued with radiation badges were accidentally included among 1425 workers with zero doses, but the second (KSM) analysis was restricted to workers who had at least one badge reading. This difference is largely responsible for the fact that the MSK risk estimates were appreciably higher than the KSM estimates. However, even the revised estimates were 10 to 20 times higher than estimates approved by the International Committee for Radiation Protection (ICRP).

There have been several rebuttals of these claims which are summarized in a recent report from the National Radiation Protection Board of Britain (NRPB) (2). According to this "catalogue of criticisms" there are five main charges: First, the MSK and KSM analyses of the Hanford data do not provide a valid statistical interpretation of the data, mainly because the approach is wrong (Comparative Mean Dose or CMD analysis) but also because the study population is too small and too narrowly based. Second, claims have been made which disappear when the data are properly standardized and are incompatible with the observed frequency of leukaemia deaths and the observed doses for these cases. Third, there is no justification for the conclusion that sensitivity to cancer induction by radiation is exceptionally low between 20 and 40 years of age. Fourth, the bioassay data serve no useful purpose because they confuse true and false positive findings. Fifth, the only cancers showing any evidence of radiation effects in other (independent) analyses of Hanford data are myeloma and cancer of the pancreas, therefore the observed effects are probably due tc hidden associations between other carcinogens and the radiation exposures.

Confirmed deaths of Hanford workers who were on the 1944–74 payrolls and also wore film badges still stand at 4051 (1944–77 deaths, see Table 1). The following account is based on an earlier analysis of these deaths (3) but is directed towards answering the criticisms listed above.

Table 1. Hanford populations of badge monitored workers (1944–74 cohorts).

Sex	Bioassays[1]	Study Population[2]			Other Workers		Total
		Cancers	Non-Cancers	Total	Alive	Dead[3]	
Males	Yes	480	1821	2301	13 623	51	15 975
	No	267	1191	1458	4386	59	5903
	Total	747	3012	3759	18 009	110	21 878
Females	Yes	56	107	163	3642	15	3820
	No	33	96	129	2114	19	2262
	Total	89	203	292	5756	34	6082
Total	Yes	536	1928	2464	17 265	66	19 795
	No	300	1287	1587	6500	78	8165
	Total	836	3215	4051	23 765	144	27 960

[1] Bioassays = Routine monitoring for internal depositions of radioactive substances.
[2] Workers with linked records of radiation doses and certified causes of death.
[3] Death benefit claims lodged but no death certificate data.

RADIATION DOSES OF CANCERS AND NON-CANCERS

In the group of 4051 confirmed deaths there were 837 workers whose total radiation doses were less than 10 millirads (so-called zero doses because they differed by an insignificant amount from background radiation as measured by a standard badge) and a further 2006 workers who received less than 1 rad (Figure 1). Only two men had doses of more than 49 rads and only two women had doses of more than 10 rads. The exceptional cases were as follows: pulmonary embolus with no verified cancer (49.9 rads), cancer of the pancreas (52.9 rads), cancer of breast (14.4 rads) and lymphosarcoma (15.4 rads).

Table 2. Cancer and non-cancer deaths by sex and radiation dose.

Sex	Certified cause of death	8th Revision	Cases	Radiation Dose (in Rads)	
				Total	Mean
Males	Cancer	140–209	747	1551	2.08
	Non-cancer	Residue	3012	4719	1.57
	Total	140–999	3759	6271	1.67
Females	Cancer	140–209	89	79	0.89
	Non-cancer	Residue	203	101	0.50
	Total	140–999	292	180	0.62
Total	Cancer	140–209	836	1630	1.95
	Non-cancer	Residue	3215	4820	1.50
	Total	140–999	4015	6450	1.59

Table 3. Proportions of cancers and non-cancers at different points on a log scale of radiation dose.

Factor	Levels	Certified deaths			% Cancers
		Cancer	Non-cancer	Total	
External radiation	<0.08 rads	256	1071	1327	19.3
	0.08– rads	131	595	726	18.0
	0.32– rads	119	430	549	21.7
	0.64– rads	123	452	575	21.4
	1.28– rads	94	320	414	22.7
	2.56– rads	48	148	196	24.5
	5.11+ rads	65	199	264	24.6
	TOTALS	836	3215	4051	20.6

Table 4. Cancers and non-cancers, factors other than external radiation.

Factors	Levels	836 Cancers	3215 Non-cancers	% Cancers
Bioassays	Nil	300	1287	18.9
	Negative	214	836	20.4
	Other[1]	322	1092	22.8
Exposure period	Under 2 years	280	1225	18.6
	Over 2 years	556	1990	21.8
Sex	Male	747	3012	19.9
	Female	89	203	30.5
Age at death	Under 40 years	38	206	15.6
	40–49 years	96	397	19.5
	50–59 years	224	712	23.9
	60+ years	478	1900	20.1
Year of death	1944–54	69	284	19.5
	1955–59	79	327	19.5
	1960–64	123	575	17.6
	1965–69	181	716	20.2
	1970–77	384	1313	22.6

[1] *ie:* False positive = finding due to accidental contamination of the specimen. or: True positive = finding due to internal deposition.

Table 5. Mantel-Haenszel Analysis of the independent effects of external radiation.

External radiation dose (in rads) (log scale)	Observed	Expected[1]	*t*-values (O-E)	Relative risk	
				Mantel-Haenszel[2]	Crude[3]
Under 0.08	235	240.6	−0.6	*1.00*	*1.00*
0.08–0.31	129	146.5	−1.9	0.86	0.92
0.32–0.63	119	108.8	+1.2	1.15	1.15
0.64–1.27	121	117.8	+0.4	1.14	1.14
1.28–2.55	91	89.8	+0.2	1.10	1.19
2.56–5.11	47	43.8	+0.6	1.08	1.36
Over 5.11	64	58.8	+0.9	1.26	1.35
Progressive component[2]			*1.994**		

[1] These figures are based on the radiation doses of exactly matched cases and controls. There were 5 controlling factors and a total of 240 control factor levels (see Table 4, also Reference 9). For 30 of the cancer cases there were no exactly matched controls. Therefore the observed numbers in this table are smaller than the ones in Table 3.
[2] See Reference 7.
* The 5 % level of significance starts at 1.96.
[3] See Table 4.

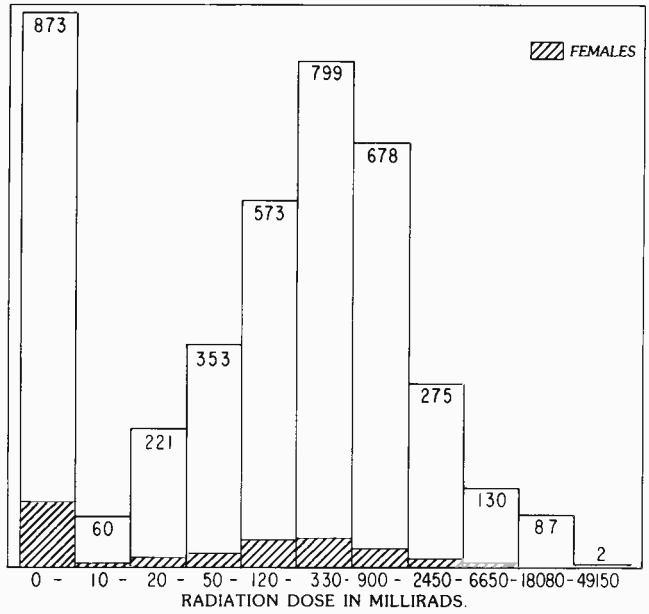


Figure 1. Log distribution of radiation doses.

The group of confirmed deaths included 837 cancers and 3215 non-cancers, and the corresponding mean doses were 1.95 rads (cancers) and 1.50 rads (non-cancers). Both males and females contributed towards this significant difference (Table 2), and the proportion of cancers was higher for the highest dose level (24.6 percent) than for the lowest level (19.3 percent) (Table 3).

According to the NRPB report, this impression of a rising risk with rising dose is an artifact caused by non-standardization for factors which have cancer and radiation associations. However, *simultaneous* control for all the factors listed in Table 4 failed to have this effect and actually left the fully matched cancers with a dose gradient almost as steep as the one for all cancers (Table 5).

The NRPB report also implies that we have made improper use of the bioassay data. This idea evidently comes from scientists who are unaccustomed to seeing records put to purposes for which they were not originally intended. However, this is common practice among epidemiologists. For example, the original purpose of the bioassays was to obtain quick recognition of any worker who had accidentally inhaled or ingested any radioactive substance. In Table 4 the bioassay *records* are being used to distinguish between safe and dangerous occupations. From this point of view either true or false positive tests are indicative of dangers which have no relevance to workers who are never tested and are only of minor importance to workers whose tests are consistently negative.

CHOICE OF NON-CANCER DEATHS AS CONTROLS FOR CANCER CASES

There was better representation of women in the population of live workers (23 909 workers with 5790 or 24 percent women) than in the population of dead workers (4051 workers with 292 or 7 percent women). This difference is related to the fact that Hanford deaths are

radiation.

Test factors	Test factor levels	Cancer cases			Relative risk	
		Observed	Expected	*t*-values	Mantel-Haenszel	Crude
Bioassays	Nil	249	251.6	−0.3	*1.00*	*1.00*
	Negative	201	197.8	0.4	1.06	1.12
	Other	273	273.6	−0.1	1.00	1.27
Exposure period	Under 2 years	245	250.5	−0.8	*1.00*	*1.00*
	Over 2 years	229	223.5	+0.8	1.12	1.22
Sex	Male	478	496.6	−3.0*	*1.00*	*1.00*
	Female	81	62.4	+3.0*	1.57	1.78
Age at death	Under 40 years	34	46.4	−2.3*	0.61	0.73
	40–49	92	97.3	−0.7	0.93	0.96
	50–59	220	188.8	+3.0*	1.30	1.25
	60+	468	481.4	−1.2	*1.00*	*1.00*
Year of death	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	*1.00*	*1.00*

Controlling Factors: External radiation (with 7 levels) (see Table 3) and all other factors in Table 4 apart from the stated test factors.

* Significant at the 5 % level.

identified through death benefit claims, and is a reminder that women have fewer dependants and smaller investments in National Insurance than men. It is important because it implies the existence of an *unknown* number of deaths which may never be traced. Therefore, a statistical analysis which relies upon internal comparisons (CMD or Comparative Mean Dose method) is better suited to Hanford data than an analysis which requires comparisons between observed and expected deaths (SMR or Standardized Mortality Ratio method). Nevertheless, the least appropriate method is the one repeatedly recommended by the NRPB and other critics of the MSK and KSM analyses (2).

The proportion of workers who were monitored for internal as well as external radiation was much higher for live than dead workers (73 percent and 60 percent). This finding is probably related to the fact that between 1944 and 1974 there was a steady increase both in the levels of external radiation (as measured on film badges) and in the number of exceptionally dangerous occupations (as measured by the results of repeated bioassays, 4). It is also a reminder that it is a general policy in the nuclear industry to exclude from all dangerous occupations anyone who has ever shown any signs of radiation sensitivity. Therefore, since there was no classification of Hanford occupations which accurately reflected the external radiation hazard (and the possibility of selective recruitment of exceptionally fit persons into exceptionally dangerous occupations) it was decided that the best controls for workers with fatal cancers were other workers whose death certificates stated that they had died from other causes (non-cancer deaths).

The three point classification of bioassay records was useful inasmuch as it showed that when the dose of external radiation was held constant (and there was also control of other factors in Table 4), the cancer deaths ceased to be unevenly distributed between the three bioassay levels (see Tables 1 and 6). Table 6 also shows some other effects of controlling for factors other than external radiation. For example, we can now see that 1) cancer deaths were evenly distributed between short- and long-term workers; 2) females were more cancer-prone than males; and 3) deaths between 50 and 60 years were more strongly biased in favor of cancers than were deaths in younger or older age groups.

RADIOSENSITIVITY AND RADIATION DOSES

Both the MSK analysis of deaths between 1944 and 1972 and the KSM analysis of 1944–1977 deaths, came to the conclusion that approximately 5 percent of the cancer deaths were radiation induced (1,3) and that the extra deaths were all cancers of radiosensitive tissues (see Tables 7 and 8). According to the NRPB assessment of the two situations there was a much smaller proportion of radiogenic cancers (0.5 percent) and there were no extra deaths apart from myelomas and pancreatic tumors. In Table 9 these cancers, which accounted for 86 of the 552 cancers of radiosensitive tissues, (see sensitivity grades I and II) are analyzed separately.

The two cancers which have shown unmistakable signs of being dose-related in all analyses of Hanford data, had exceptionally high doses. Thus, there were 11 cases of myeloma with a mean dose of 7.83 rads, and 57 pancreatic tumors with a mean dose of 3.58 rads. Removal of these cases barely affected the female cancers, and it still left grade I cancers of males with a higher dose (2.22 rads) than grade II cancers (2.15 rads). In short, even omission of all myelomas and pancreatic tumors caused no change in the ranking of mean doses for three sensitivity ratings, and still left the dose for 484 male and female cancers in grades I and II (2.04 rads) higher than the doses for 284 cancers in lower grades (1.24 rads) and 3215 non-cancers (1.50 rads).

Table 7. Radiosensitivity rating of adult tissues and organs (see References 8 and 9).

Relative sensitivity of adult tissues to cancer induction by radiation		ICD Nos. (8th Revision)
High sensitivity: –		
I Established	Bone marrow & thyroid	203; 205–9; 193
II Apparent	Lymph nodes and reticular tissue	200–2; 204
	Pharynx and bronchus	146–9; 162–3
	Pancreas, stomach, large intestine and breast	151; 153; 157–9 174
Low sensitivity: III	Oesophagus and small intestine	150; 152
	Nose, middle ear, sinuses & larynx	160–1
	Lip, tongue, mouth & salivary gland	140–5
	Liver, gallbladder & bile duct	155–6
	Testis, penis & kidney	186–7; 189
	Skin, connective tissue & bone	170–3
	Eye, brain & nervous tissue	190–2
	Other endocrine (excluding thyroid)	194
Not classified: IV	Ovary and uterus	180–4; 174
	Prostate and bladder	185; 188
	Other and unspecified cancers	195–9

Table 8. Distribution of Hanford cancers by radiosensitivity of affected tissues.

Radiosensitivity ratings of cancer deaths[1]		Males		Females	
		Nos.	Total dose in rads	Nos.	Total dose in rads
	(ICD Nos.)				
I	203	10	86.10	1	–
	205–9	24	55.11	3	0.04
	193	1	0.44	–	–
II	200–2; 204	43	47.48	6	20.16
	146–9	13	50.41	–	–
	162–3	213	557.83	10	5.21
	151	45	106.65	2	2.18
	153–4	90	112.53	11	7.82
	157–9	54	204.08	6	0.68
	174	1	–	19	22.81
III & IV	140–5	12	17.63	–	–
	150; 152	24	11.13	–	–
	155–6	18	45.36	2	4.60
	160–1	14	20.08	–	–
	170–3	18	26.82	6	1.80
	180–9	91	117.55	13	4.95
	190–2	28	47.60	5	1.75
	194–9	48	44.64	5	7.02
	TOTAL	747	1551.44	89	79.02

[1] See Table 7.

Table 9. Differences between myelomas and cancer of pancreas and other cancers of radiosensitive tissues.

A) Myeloma (203) and cancer of the pancreas (157)						
Sensitivity ratings[1]	Males		Females		Total	
	Nos.	Mean R*	Nos.	Mean R*	Nos.	Mean R*
I	10	8.61	1	–	11	7.83
II	52	3.91	5	0.13	57	3.58
II & IV	–	–	–	–	–	–
Σ	62	4.67	6	0.11	68	4.27
B) Remaining cancers						
I	25	2.22	52	1.08	484	2.04
II	407	2.15	–	–	–	–
II & IV	253	1.31	31	0.65	284	1.24
Σ	685	1.84	83	0.94	768	1.75
Non-cancers	3012	1.57	203	0.50	3215	1.50
C) All cancers						
I	35	4.05	4	0.01	39	3.65
II	459	2.35	54	1.09	513	2.22
II & IV	253	1.31	31	0.65	284	1.24
Σ	747	2.08	89	0.89	836	1.95

[1] See Table 7.
* R = Radiation dose in rads.

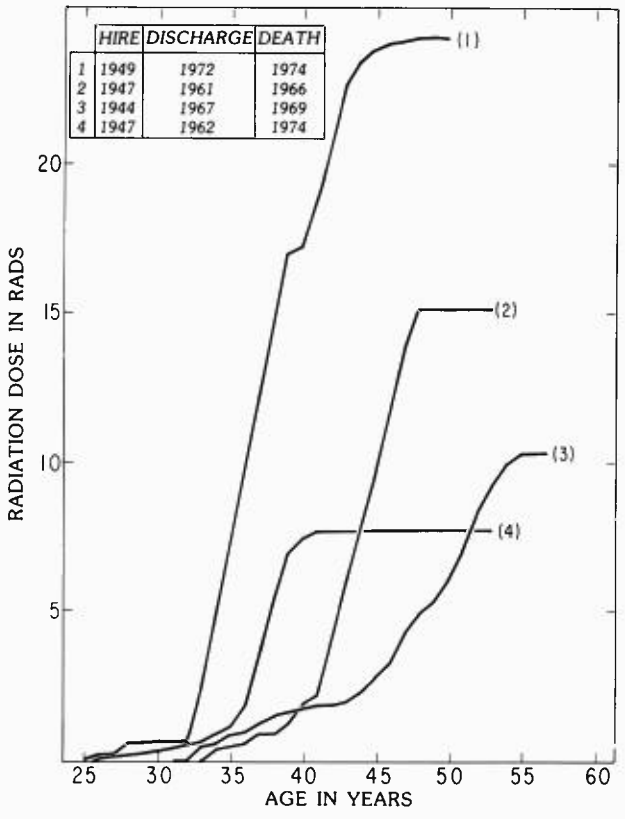


Figure 2. Age trend of radiation doses for four long-term workers.

DOSE TRENDS

Thus far we have only considered the total amount of radiation received by each worker. But records of annual doses, exposure periods and follow-up periods can also be used to observe dose trends on two time scales: one measuring intervals between birth and exposure (age scale) and the other intervals between exposure and death (pre-death scale).

Intervals between entering and leaving the industry (exposure periods) were usually, but not necessarily, much shorter than intervals between entering the industry and dying (follow-up periods). Therefore, although it was not possible for a worker's dose at, say, 60 years to be lower than his previous year's dose (Figure 2), the mean dose for all men aged 60 years could be lower than the mean dose for the previous year if a high proportion of workers with high doses died in the interval (Figure 3). Likewise, although individual doses on the pre-death scale could never be lower for short intervals when compared to long pre-death intervals, mean doses for several workers could be lower if there was a concentration of high doses among workers with short follow-up periods (see Figure 4).

The four individuals whose cumulative doses are shown in Figure 2 had in common the fact that they belonged to early cohorts (1944–1949) and worked for several years in high risk occupations. Their dose records show that for such workers there was usually a short period of low doses followed by a much longer period of high doses, and even for men who died before 60 years of age there was usually a period of several years between leaving the industry and dying.

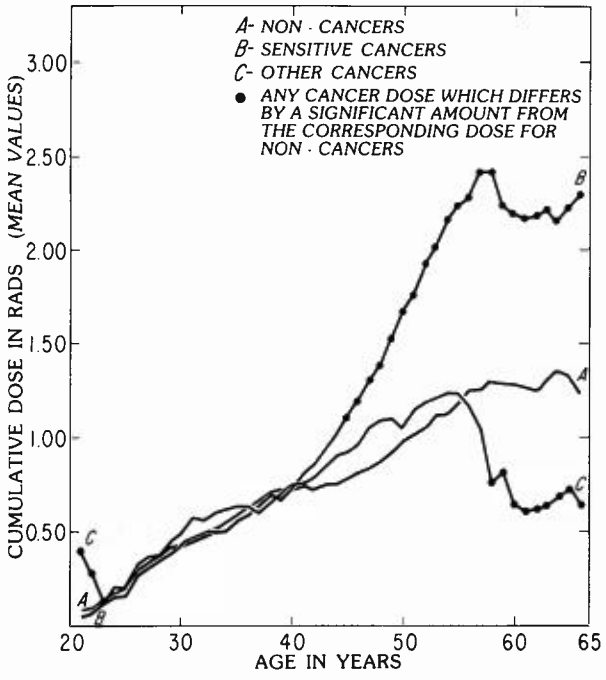


Figure 3. Age trend of cumulative radiation for three groups of male workers.

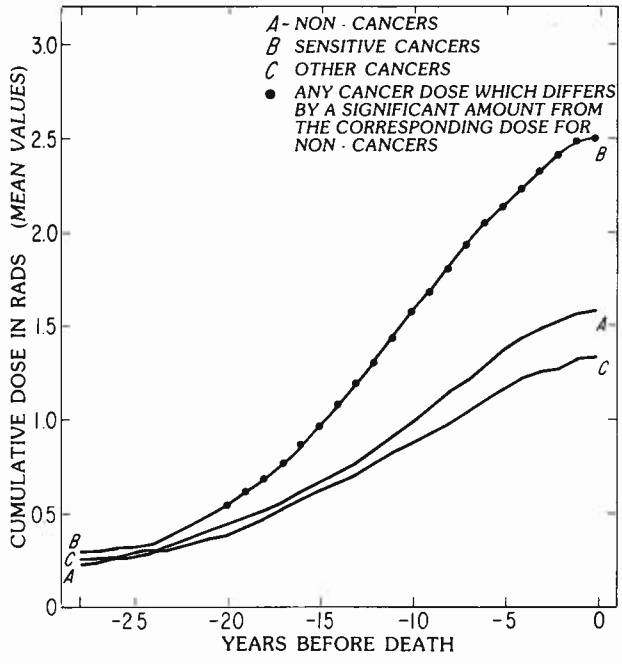


Figure 4. Cumulative radiation doses by stated pre-death rates.

Figure 3 shows the age trend of mean doses for all men in the following categories: A) 3012 non-cancers; B) 494 cancers of sensitive tissues; and C) 253 cancers of other tissues. Until 40 years of age the mean doses for the three groups were very alike but from 40 to 56 years the trend was much steeper for B than C cancers, and also steeper for C cancers than non-cancers. After 56 years of age there was a sharp reversal of the dose trends for the two groups of cancers, which affected C more than B cases, and was accompanied by a much lower ratio of C cancers to non-cancers among men who had high doses and Washington State Social Security numbers (.028) than among other men with equally high doses (.060). Therefore, it is possible that there was under-reporting of cancers by doctors who were accustomed to treating Hanford workers and anxious not to cause unnecessary distress to relatives of patients who might know of the connection between cancers and radiation. Whatever the reason for the shortage of high doses among men with C cancers who lived for more than 55 years, it explains why the mean cumulative dose was lower for group C cancers (1.24 rads) than non-cancers (1.50 rads).

Figure 4 is based on the same individuals as Figure 3, but it shows how the length of the pre-death period affected the dose levels. There were 2112 men who died before 60 years of age, and 1872 men who were followed for less than 20 years (3). In Figure 4 there were no reversals of dose trends. Therefore, although high doses and early cancer deaths often went hand in hand, there was little or no tendency for higher doses to be associated with exceptionally short follow-up periods. Figure 4 also shows that, when measured backwards from death, mean doses were consistently higher for men with group B cancers than for other men, but the difference was only statistically significant (3) the last 19 years before death. This finding is consistent with most of the radiogenic cancers having latent periods of more than 20 years and has not been challenged either by the NRPB or by other critics of the MSK and KSM analyses.

RISK ESTIMATES FOR CANCERS OF RADIOSENSITIVE TISSUES

The figures in Table 10 are for two sets of male deaths: sensitive cancers (or group B cases) and non-cancers (or group A controls). They show the mean cumulative doses for 15 age levels and thus allow one to see that differences between the two groups only became significant after 44 years of age, and were much greater by 57 years than they were 10 years earlier.

From 21 to 39 years, dose levels were consistently higher for controls (*ie* non-cancers) than cases (*ie* sensitive cancers), but none of the differences were statistically significant. Thereafter, there was an ever increasing difference in the opposite direction until, by 57 years of age, the mean cumulative dose was twice as high for cases (2.42 rads) as controls (1.26 rads).

These findings are consistent with much higher levels of resistance to cancer effects of low level radiation among young than old men. Therefore, although the MSK and KSM analyses of Hanford data would allow some cancers

Table 10. Age related risk estimates for radiosensitive cancers.

Age	(A) Non-cancers (controls)			(B) Radiosensitive cancers (cases)		
	Nos.	Mean cumulative dose in rads	Standard deviation	Nos.	Mean cumulative dose in rads	Doubling[1] dose
21	46	0.084	.309	7	0.054	–
24	166	0.179	.515	20	0.147	–
27	289	0.345	.857	48	0.308	–
30	446	0.440	1.052	79	0.431	–
33	636	0.510	1.342	111	0.500	–
36	856	0.626	1.811	162	0.586	–
39	1030	0.732	1.989	195	0.695	–
40	1097	0.724	1.875	209	0.740	–
41	1188	0.748	1.995	225	0.806	81.90
42	1250	0.719	2.043	242	0.857	36.75
45	1399	0.791	2.247	270	1.108*	18.69
48	1565	0.868	2.513	295	1.390*	13.84
51	1688	1.018	2.966	310	1.769*	12.76
54	1759	1.130	3.234	303	2.174*	10.42
57	1741	1.262	3.057	285	2.417*	7.64

* Any cancer dose which differs by a significant amount from the corresponding dose for non-cancers.
[1] Doubling dose: the amount of radiation which is needed to exactly double the normal risk of developing a radiosensitive cancer assuming linearity of dose response (see Reference 10). For method of estimation see Reference 1.

Table 11. Mantel Haenszel Analysis of two groups of respiratory deaths[1].

Dose levels in rads	Lung cancer deaths[2]			Other respiratory deaths[2]		
	Observed	Expected	t	Observed	Expected	t
under 0.01	31	44.3	−2.33*	28	30.8	−0.59
0.01–	24	24.6	−0.14	15	16.5	−0.41
0.08–	35	39.4	−0.81	21	26.2	−1.16
0.32–	27	29.2	−0.46	17	19.8	−0.69
0.64–	37	32.5	+0.89	28	21.0	+1.71
1.28–	28	25.0	+0.67	19	15.0	+1.14
2.56–	15	12.4	+0.80	9	6.8	+0.90
5.12–	15	8.0	+2.67*	4	4.2	−0.10
10.24–	6	4.8	+0.60	2	2.4	−0.29
20.48–	6	4.2	+0.91	2	2.1	−0.06
40.98–44.21	1	0.7	+0.38	–	0.3	−0.54
Progressive Component[3]			**3.42****			**1.31**

[1] Control Group = all non-cancer deaths.
Controlling factors (and levels): sex with (2) levels
See Table 6. age at death with (4) levels
year of death with (5) levels
[2] ICD Nos. (8th revision)
Lung cancers: 162–3
Other respiratory: 490–519
[3] See Table 5.
* Significant at the 5 percent level.
** Significant at the 1 percent level.

with long latent periods to be caused by background radiation we should not expect such cancers to have an appreciable effect on mortality before 65 years of age.

Finally, since the findings for lung cancer were more firmly established in the KSM analysis than in the earlier analysis we are adding a table to show that, although there were no records of smoking habits in Hanford data, it is unlikely that the observed association between radiation doses and lung cancers was the result of an independent association between levels of radiation and smoking (Table 11). This table shows the results of dividing respiratory deaths with smoking associations into two groups (malignant and non-malignant) and comparing both groups with all non-cancer deaths. The comparisons take the form of a Mantel-Haenszel analysis (with three sets of controlling factors) and show A) for lung cancers there is a rising risk with rising dose; and B) for other respiratory deaths there is no such trend. Therefore, it is probably safe to include not only bone marrow and pancreas but also lungs among tissues which are exceptionally sensitive to cancer induction by radiation.

DISCUSSION

For many workers in the nuclear industry there is a constant risk of exposure to small doses of external or penetrating radiation, and for some workers there is also a risk of internal depositions of radioactive substances. Therefore, all workers wore film badges and some had routine urine analyses or body counts (bioassays). For Hanford workers there is a possibility of linking film badge readings and bioassays with dates and causes of death. However, this linkage requires knowledge of death benefit claims and leaves one uncertain whether, in the absence of such a claim, an ex-worker is alive or dead. Therefore, from the point of view of discovering whether there is a cancer hazard from low level radiation, there is clearly more to be learned from a CMD analysis of Hanford data (Comparative Mean Dose method) than an SMR analysis (Standardized Mortality Ratio method) (3).

Thus far all CMD analyses of Hanford data have been restricted to workers with known dates and causes of death. This is due to altered working procedures between 1944 and 1970, which probably affected young recruits more than old recruits. This has destroyed what little value the original (Census) classification of Hanford occupations ever possessed. A more meaningful classification is being prepared, but until this is completed the best controls for cancer cases are deaths without any sign of cancer having been the cause of death.

Much has been made of the fact that neither the first CMD analysis of Hanford data (MSK) nor the second one (KSM) includes live workers. But the real reason why these analyses have been so severely criticized is because their conclusions run counter to the idea that even at low dose levels the cancer most likely to be caused by external radiation is myeloid leukaemia.

Only 11 of the 3520 male deaths included in the MSK analysis were ascribed to myeloid leukaemia and though the mean cumulative dose for these cases (1.22 rads) was higher than the corresponding dose for 2850 non-cancers (0.99 rads) it was only a fraction lower than the dose for solid tumors (1.30 rads). Even so, it was finally concluded that: the proportion of radiogenic cancers was in the region of 5 percent; the most sensitive tissues were bone marrow, pancreas and lung; and this sensitivity increased progressively with adult age but was almost non-existent for young men. Much the same conclusions emerged from a similar analysis of deaths between 1944

and 1977 (KSM). But on this occasion there was more certainty that the extra deaths were radiation induced, and more certainty that there were extra cancers affecting lungs as well as bone marrow and pancreas.

In the study population of A-bomb survivors assembled on behalf of the Atomic Bomb Casualty Commission (ABCC data) there were more persons at risk, and a much wider range of doses than in the study population of Hanford workers (5). Therefore, we have repeatedly been told that risk estimates based on ABCC data are necessarily more reliable than ones based on Hanford data. The former currently stem from an analysis of deaths from 1950–1974 (5, 6) which is supposed to show that all non-cancer effects of the A-bomb radiation were exhausted before October 1950; that a doubling of background radiation would add less than 1 percent to the risk of a cancer death, and that at least one-third of the extra cancers would be myeloid leukaemias.

The analysis actually shows 1) a rising trend of mortality with rising dose for three causes of death (leukaemia, other blood diseases and other cancers); 2) a flat rate for other non-cancer deaths; 3) lower rates of non-cancer mortality for Hiroshima than Nagasaki; and 4) lower rates for Hiroshima than one would expect on the basis of national statistics. Therefore, although the official interpretation of ABCC data has never been questioned it could be very wide of the mark.

The death rate from 1945 to 1950 for first day survivors was exceptionally high, also the extra deaths were concentrated in the highest dose groups and they included literally thousands of deaths ascribed to bone marrow depression. Therefore, short of assuming that all persons of the same age and sex are equally likely to die during the aftermath of a nuclear explosion, and that all persons with radiation damage to bone marrow and other internal organs either died before 1950 or *completely* recovered, the official interpretation of ABCC data must be wrong for the following reasons:

- The assumption that only a rising trend of mortality with rising dose is indicative of extra radiation deaths would have been correct if there had been no gap between the exposure date and the follow-up period. But there was a 5-year gap accompanied by a high, dose-related death rate which must have had a selective effect on weaker individuals. Therefore, from 1950 onwards only a falling trend of mortality with rising dose would be indicative of no further radiation effects.
- There must have been every degree of damage to bone marrow and other internal organs. Therefore, both the rising trend of mortality for non-malignant blood diseases and the flat rate of mortality for other non-cancer deaths should be regarded as evidence of bone marrow depression causing either aplastic anaemia or permanent loss of the body's immunological system.
- Bone marrow is the tissue of origin for localized and diffuse cancers (ie myelomas and myeloid leukaemia) and one effect of bone marrow depression is conversion of slow growing tumors into acute, metastatic diseases. Therefore, in populations exposed to large doses of external radiation (or internal emitters) we should expect high incidences of myeloid leukaemia, and in populations exposed to low doses a high incidence of myelomas.

Therefore, before jumping to the conclusion that all risk estimates which are materially different from ABCC estimates are necessarily wrong, we should be asking ourselves whether any population (human or animal) which has received tissue destructive doses of radiation is suitable for studying low dose effects.

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