HANFORD DATA - A REPLY TO RECENT CRITICISMS

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Summary

According to a recent report from the National Radiation Protection Board of Britain a majority of nuclear scientists are content that risk estimates for cancer effects of man-made radiation should remain based upon a relative risk analysis of the mortality experiences of A bomb survivors (ABCC data) although an alternative source (based upon workers in the nuclear industry) is indicative of a much higher risk at low dose levels. Since none of the reasons which have been given for this preference bear close inspection there could be a hidden flaw in the ABCC data.

Introduction

We recently had occasion to show that risk estimates based on radiation doses of workers in the nuclear industry (Hanford data) are radically different from earlier estimates based on A bomb survivors (ABCC data)[1,2]. There have been several rebuttals of these claims which are summarized in a recent report from the National Radiation Protection Board of Britain[3]. According to this "catalogue of criticisms"

(i) The MSK and MKS analyses of Hanford data do not provide a valid statistical interpretation of the data. This is mainly because the approach is wrong (CMD analysis) but also because the study population is too small and too narrowly based.

(ii) 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.

(iii) There is no justification for the conclusion that sensitivity to cancer induction by radiation is exceptionally low between 20 and 40 years of age.

(iv) The bioassay data serve no useful purpose because they confuse true and false positive findings.

(v) The only cancers showing any evidence of radiation effect in two independent analyses of Hanford data were myeloma and cancer of the pancreas, therefore the observed effects are more likely to be due to other industrial hazards than to radiation per se.

Confirmed deaths of Hanford workers who were on the 1944-74 payrolls and also wore film badges now stand at 4051 (1944-77 deaths, see table 1). The following account is based on an earlier analysis of these deaths[2] but is directed towards answering the criticisms listed above.

Choice of Non-Cancer Deaths as Controls for Cancer Cases

There was better representation of women in the original population of live and dead workers (27,960 workers with 6082 or 22% of women) than in the population of confirmed deaths (4051 workers with 292 or 7% of women). This difference is related to the fact that deaths were 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 one which requires a full complement of deaths (SMR or Standardized Mortality Ratio method). Yet, the least appropriate method was the one repeatedly recommended by critics of the MSK and MKS analyses[3].

Between 1944 and 1974 there was an increase both in the level of annual radiation doses as measured on film badges[4], and in the number of workers who were monitored for internal irradiation (bioassay records). Therefore, workers who were monitored for both sources of

radiation (table 1) were commoner among live than dead workers (73% and 60%). This difference and the fact that there was no classification of Hanford occupations which accurately reflected radiation hazards, were the reasons why both the MSK analysis of 1944-72 deaths of workers with and without film badges and the MKS analysis of 1944-77 deaths of workers with film badges were restricted to workers whose records included a full complement of 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 (fig. 1). Only two men had doses of more than 49 rads and only two women had doses of more than 10 rads. One of the exceptional cases was a man whose death was ascribed to a pulmonary embolism (non-cancer), and the others were cancers of tissues which are known to be exceptionally sensitive to cancer induction by radiation (i.e. lymphnodes, pancreas and breast, see table 7).

In the group of confirmed deaths there were 837 cancers and 3215 non-cancers. The corresponding mean doses were 1.95 rads (cancers) and 1.50 (non-cancers). Both males and females contributed towards this significant difference (table 2) and the proportion of cancers was higher at the upper end of the dose scale (24.6%) than at the lower end (19.3%) (table 3).

According to NRPB 79 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 signally failed to have this effect and actually left the fully matched cases with a dose gradient almost as steep as the one for all cancers (table 5).

The suggestion that we made faulty use of the bioassay data must have come from scientists who are unaccustomed to seeing records put to purposes for which they were not originally intended, but this is common practice in epidemiology. For example, the original purpose of the bioassays was quick recognition of workers who had accidentally inhaled or ingested any fission products. In table 4 they are being used to distinguish between relatively safe and relatively dangerous occupations. From this point of view both true and false positive tests are indicative of dangers which do not apply to workers who are never tested or have consistently negative findings.

The three point classification of bioassay records was useful in as much as it showed that when the external radiation doses were held constant (and there was also control of other factors in table 4) the cancer deaths ceased to be unevenly distributed between the three levels (see tables 1 and 6). Table 6 also shows other effects of controlling for extraneous factors. Thus, it shows that (i) cancer deaths were evenly distributed between short and long term workers; (ii) females were more cancer-prone than males; and (iii) death between 50 and 60 years were more strongly biased in favour of cancers than deaths at earlier or later ages.

Radiosensitivity and Radiation Doses

Both the MSK analysis of 1944-72 deaths and the MKS analysis of 1944-77 deaths came to the conclusion that approximately 5% of the cancer deaths were radiation induced[1,2] and that the extra deaths were all cancers of radiosensitive tissues (tables 7 and 8). According to the NRPB assessment of the situation there was a much smaller proportion of radiogenic cancers (0.5%) and the extra deaths were either myelomas or cancers of the pancreas. In table 9 these cases and 484 other cancers of radiosensitive tissues (see sensitivity grades I and II) are analysed separately.

The two cancers which all analysts of Hanford data agree are dose-related, had exceptionally high doses (myelomas 9.83 and pancreas 3.58 rads), but their removal barely affected female cancers, and only reduced the highest of three mean doses for male cancers from 4.05 to 2.22 (grade I), and the second highest from 2.35 to 2.15 (grade II). Therefore, omission of all myelomas and pancreatic cancers caused no change in the ranking of mean doses for three sensitivity ratings and left the mean dose for 484 cancers in grade I and II (2.04 rads) higher than the mean for 284 cancers in other grades (1.24) or the mean for 3012 non-cancers (1.50).

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 (fig. 2), the mean dose for all workers aged 60 years could be lower than the mean dose for the previous year if individuals with exceptionally high doses died between their 59th and 60th birthdays (fig. 3). Likewise, pre-death doses for individuals could never be lower for short than long intervals, but mean doses could be lower if some workers with short follow-up periods had high doses (fig. 4).

The individuals in figure 2 belonged to early cohorts (1944-49); worked for several years in dangerous occupations, and died in their 50s. Their dose trends in one direction (age) showed that each worker had a short period of low doses followed by a long period of high doses, and that intervals between discharge and death ranged from 2 to 12 years.

Figure 3 shows the same (mean) dose trend for males in the following groups: non-cancers (A); cancers of sensitive tissues (B); and other cancers (C). 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 sensitive cancers than other cancers,and also steeper for other cancers than non-cancers. Between 53 and 57 years of age there was a sharp reversal of trend for other cancers which is the reason why the mean dose for this group is smaller than the mean for non-cancers (table 9).

Figures 3 and 4 are based on the same number of workers (3759, see table 1) but one shows how early deaths affect mean doses (fig. 3) and the other how short follow-up periods affect mean doses (fig. 4). In fact, there were 2112 male deaths before 60 years of age, and 1872 men who were followed for less than 20 years[2]. In figure 4 there were no reversals of dose trends. Therefore, although high doses were often associated with early deaths (fig. 3) they were rarely if ever associated with short follow-up periods. Figure 4 also shows that, when measured backwards from death, mean doses were consistently higher for sensitive cancers than other cancers or non-cancers; and for 19 years the cancer : non-cancer difference was statistically significant[2]. This finding is consistent with some of the radiogenic cancers having latent periods of more than 20 years and has not been challenged by the NRPB or other critics of the MKS analysis.

Risk Estimates for Sensitive Cancers

The figures in table 10 are for two sets of male deaths: sensitive cancers and non-cancers. They include mean doses for 15 age levels and thus allow one to see that significant differences between the two groups were confined to older age levels.

From 21 to 39 years doses were consistently higher for non-cancers than cancers though no one of the differences was statistically significant. Thereafter, there was an ever increasing difference in the opposite direction until, by 57 years of age the mean (total) dose (in rads) was twice as high for the cancers (2.417) as the non-cancers (1.262).

These findings are consistent with low sensitivity to cancer effects of radiation during early adult life and high sensitivity in old age. Therefore, although the MKS analysis of Hanford data would allow some cancers to be caused by background radiation it would not allow these cancers to have much effect on mortality before 60 years of age.

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

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 small risk of internal depositions of chemicals, some of which may be radioactive. Therefore, all workers wear film badges and some have 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 learnt from a CMD analysis of Hanford data (Comparative Mean Dose method) than an SMR analysis (Standardized Mortality Ratio method)[2].

Thus far all CMD analyses of Hanford data have been restricted to workers with known dates and causes of death. This is because changed ways of working between 1944 and 1970 (which evidently had different effects on older and younger workers) had rendered quite useless the original classification of workshops and occupations. New classifications are being prepared but until these are completed it is necessary to have, as controls for cancer cases, all non-cancer deaths.

Much has been made of the fact that neither the first CMD analysis of Hanford data (MSK) nor the second one (MKS) includes live workers. But the real reason why these analyses have been so severely criticised by nuclear scientists is because they reach conclusions which are impossible to reconcile with certain theories based upon the mortality experiences of A bomb survivors (ABCC data).

For example, it is widely believed that the cancer most likely to be caused by radiation is myeloid leukaemia and that all radiation risks (even ones due to non-lethal mutations) can be reduced by dose fractionation or slowing of the dose rate. Only 11 of the 3520 male deaths included in the first CMD analysis of Hanford data were ascribed to myeloid leukaemia and for these cases the mean cumulative dose (122 centirads) was higher than the corresponding dose for 2850 non-cancers (99 centirads) but lower than the dose for 659 other cancers (138 centirads). Nevertheless, it was finally concluded that (i) the proportion of radiogenic cancers was in the region of 5%; (ii) the most sensitive tissues were bone marrow, pancreas and lung; and (iii) 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 1944-77 deaths (MKS). 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 there were more persons at risk, more deaths and higher doses than in the study population of Hanford workers[5]. Therefore, it is arguable that risk estimates based on CMD analysis of Hanford data are less reliable than ABCC estimates. The latter are currently based on a relative risk analysis of 1950-74 deaths[5] in which the risk factors are 8 levels of T-65 estimates of external radiation[6]. Therefore, they are based on two untested assumptions. The first assumption is that after 5 years there was only one source of further trouble from the explosions, namely, measured doses of external radiation, and the second one is that earlier effects of the explosions had left each sub-group of the survivor population with the same level of resistance to disease as they would have had if there had been no explosions.

Therefore, ABCC risk estimates have made no allowance for the following facts and possibilities:

(1) Following a large explosion there is usually a period of several months or years when death rates are high and the extra deaths are concentrated among persons who were nearest to the explosions and persons with low levels of resistance to disease. Therefore, whatever the cause of a large explosion, one can be certain that the overall level of resistance to disease will be higher in the survivor than the original population, and that the difference will be proportional to the distance from the explosion.

(2) Following a nuclear explosion there will be the usual troubles plus extra troubles from internal depositions of radioactive substances (alpha emitters) and external radiation (gamma radiation and neutrons). The radiation lesions will take two forms: tissue damage or immediate effects, and non-lethal mutations or delayed effects. Tissue damage should be more obvious and more immediate when caused by external than internal radiation, and it is possible that following the 1945 explosions internal radiation effects were small and therefore confined to bone marrow.

(3) Even if the effects of internal radiation were more widespread, bone marrow would probably suffer most because it is a very diffuse as well as very sensitive tissue. Therefore, we should expect the cancers caused by the 1945 explosions to include a high proportion of myeloid leukaemias, and/other long term effects to include permanent loss of immunological competence with or without more obvious signs of bone marrow damage (e.g. myelofibrosis and aplastic anaemia).

Therefore, before jumping to the conclusion that any set of risk estimates which is materially different from ABCC estimates must be wrong, radiation protection boards should be seeking answers to a number of outstanding problems. For example, it is now doubtful whether any population (human or animal) which has received tissue destructive doses of radiation is suitable for studying delayed effects of low doses. Therefore, it is

necessary to ask what should be done to make the maximal use of all extant and future dose records for radiation workers. It is also far from certain that the maximal use has been made of ABCC data, and therefore necessary to ask what more could be learnt by comparing A bomb survivors with their Japanese contemporaries and not making the doubtful assumption that there was only one source of radiation effects.

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TABLE 1

Hanford Populations of Badge Monitored Workers (1944-74 cohorts)

Sex	Bioassays[1]	Study Population[2]			Other Workers		
		Cancers	Non-Cancers	Total	Alive	Dead[3]	Total
Males	Yes	480	1821	2301	13623	51	15975
	No	267	1191	1458	4386	59	5903
	Total	747	3012	3759	18009	110	21878
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	17265	66	19795
	No	300	1287	1587	6500	78	8165
	Total	836	3215	4051	23765	144	27960

(1) Bioassays = Routine monitoring for internal depositions of radio-sensitive substances.

(2) Workers with linked records of radiation doses and certified causes of death.

(3) Death benefit claims lodged but no death certificate data.

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	1,551	2.08
	Non-Cancer	Residue	3,012	4,719	1.57
	Total	140-999	3,759	6,271	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	1,630	1.95
	Non-Cancer	Residue	3,215	4,820	1.50
	Total	140-999	4,015	6,450	1.59

TABLE 3

Proportions of Cancers and Non-Cancers at
Different Points on a Log Scale of Radiation Dose

Factor	Levels	Certified Deaths			
		Cancer	Non-Cancer	Total	% Cancers
External Radiation	<0.08 rads	256	1,071	1,327	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	3,215	4,051	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)
i.e.: 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)	Obs.	Exp.(1)	t-values (O-E)	Relative Risk Mantel-Haenszel(2)	Relative Risk 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 Mantel and Haenszel, 1959).(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 Mantel, 1963.(7)

* The 5% level of significance starts at 1.96.

(3) See table 4.

TABLE 6

Mantel-Haenszel Analysis of the Independent Effects of Factors other than External Radiation

Test Factors	Test Factor Levels	Cancer Cases Obs.	Exp.	t-values	Relative Risk 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.

TABLE 7

Radiosensitivity Rating of Adult Tissues and Organs[1]

Relative Sensitivity of Adult Tissues to to Cancer Induction by Radiation(1)		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

(1) See ICRP 14[8] and Mole, 1978.[9]

TABLE 8

Distribution of Hanford Cancers by Radiosensitivity of Affected Tissues

Radiosensitivity Ratings of Cancer Deaths(1)		Males Nos.	Males Total Dose in Rads	Females Nos.	Females 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

(1) 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
III & IV	-	-	-	-	-	-
Σ	62	4.67	6	0.11	68	4.27

(2) Remaining Cancers

I	25	2.22	52	1.08	484	2.04
II	407	2.15				
III & 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

(3) All Cancers

I	35	4.05	4	0.01	39	3.65
II	459	2.35	54	1.09	513	2.22
III & IV	253	1.31	31	0.65	284	1.24
Σ	747	2.08	89	0.89	836	1.95

(1) See table 7.

TABLE 10

Age Related Risk Estimates for Radiosensitive Cancers

Age	Non-Cancers (Controls) Nos.	Mean dose in Rads	Standard Deviation	Radiosensitive Cancers (Cases) Nos.	Mean dose in Rads	Doubling[1] dose
21	46	0.084	30.906	7	0.054	-
24	166	0.179	51.572	20	0.147	-
27	289	0.345	85.682	48	0.308	-
30	446	0.440	105.234	79	0.431	-
33	636	0.510	134.179	111	0.500	-
36	856	0.626	181.054	162	0.586	-
39	1030	0.732	198.925	195	0.695	-
40	1097	0.724	187.479	209	0.740	-
41	1188	0.748	199.504	225	0.806	81.90
42	1250	0.719	204.287	242	0.857	36.75
45	1399	0.791	224.701	270	1.108*	18.69
48	1565	0.868	251.287	295	1.390*	13.84
51	1688	1.018	296.613	310	1.769*	12.76
54	1759	1.130	323.412	303	2.174*	10.42
57	1741	1.262	305.712	285	2.417*	7.64

* Any cancer dose which differs by a significant amount from the corresponding dose for non-cancers.

(i) Doubling Dose: the amount of radiation which is needed to exactly double the normal risk of developing a radiosensitive cancer (see BIER, 1972) (10)
For method of Estimation see Mancuso, Stewart and Kneale, 1977[1].

Table 11

Mantel Haenszel Analysis of Two Groups of Respiratory Deaths[1]

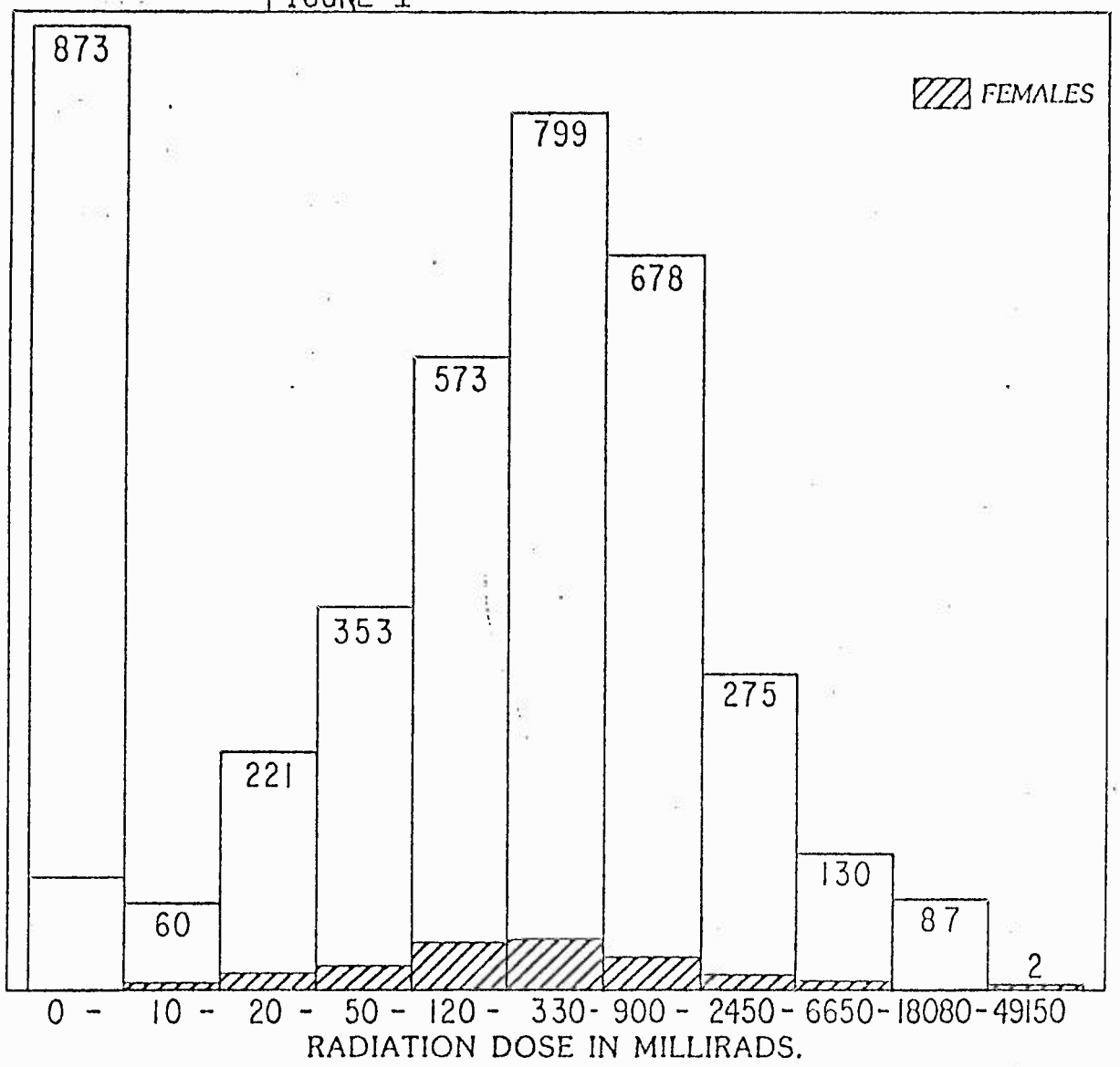
Dose in rads Levels	Lung Cancer Deaths[2]			Other Respiratory Deaths[2]		
	Obs.	Exp.	t	Obs.	Exp.	t
under 0.01	31	44.3	-2.33*	28	30.8	-0.59
0.01 -	24	24.6	-0.14	15	16.5	-0.41
0.08 -	35	39.4	-0.81	21	26.2	-1.16
0.32 -	27	29.2	-0.46	17	19.8	-0.69
0.64 -	37	32.5	+0.89	28	21.0	+1.71
1.28 -	28	25.0	+0.67	19	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 +	1	0.7	+0.38	-	0.3	-0.54
Trend Statistics			3.42**			1.31

(1) Control Group = all non-cancer deaths

Controlling factors and levels: sex (2)
age at death (4)
year of death (5)

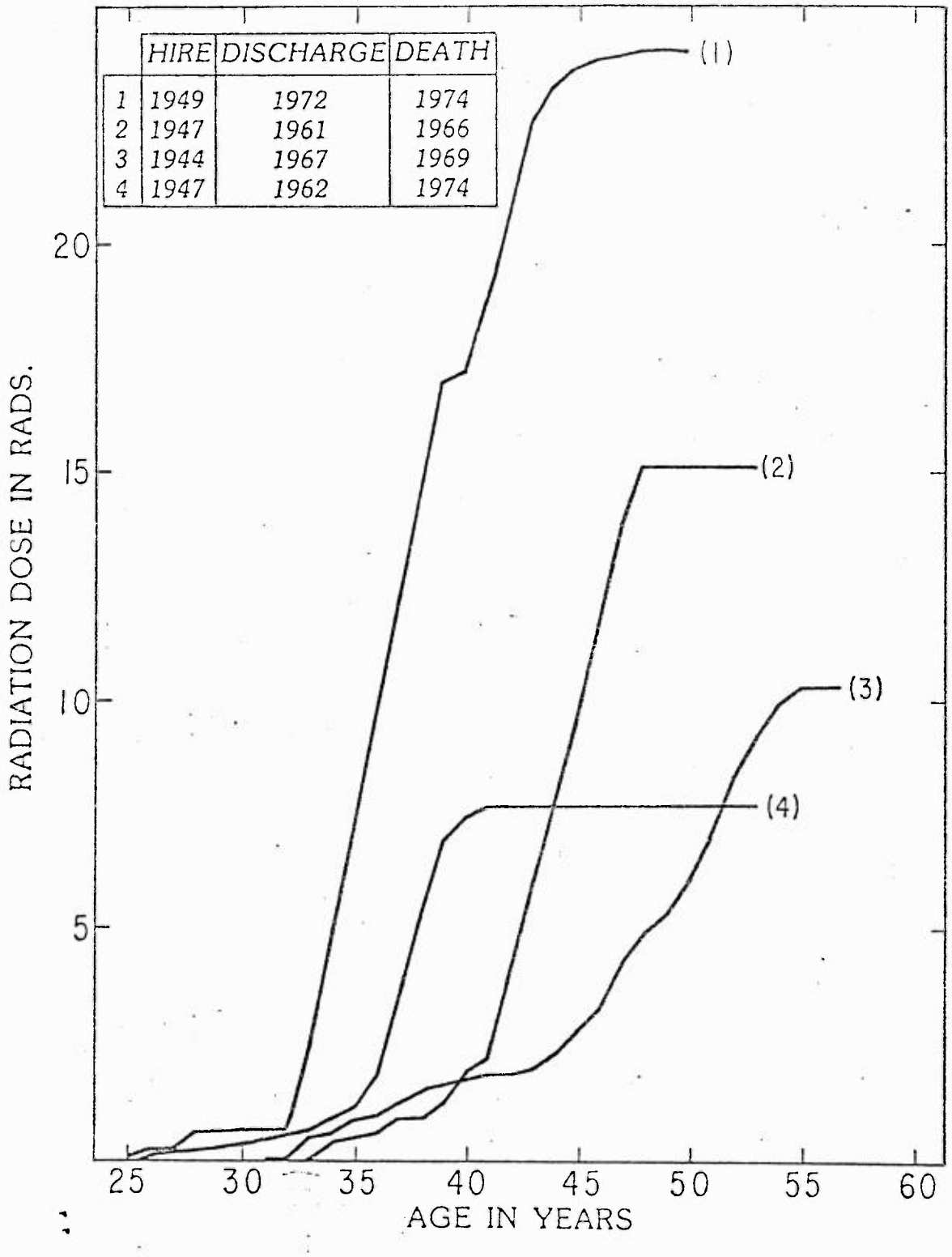
(2) ICD Nos. (8th revision) Lung cancers: 162 - 3
Other respiratory : 490 - 492

FIGURE 1



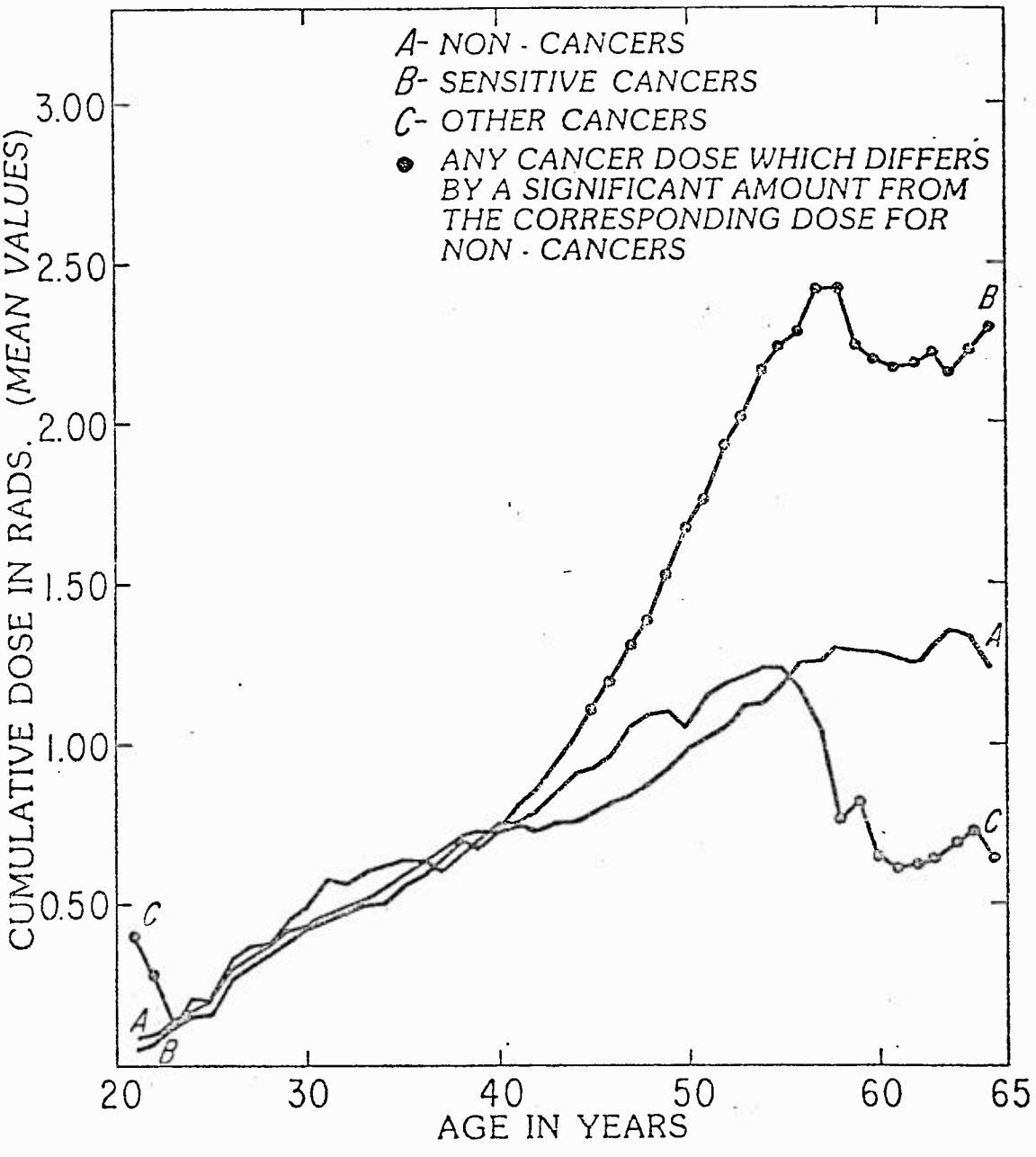
Log Distribution of Radiation Doses

FIGURE 2



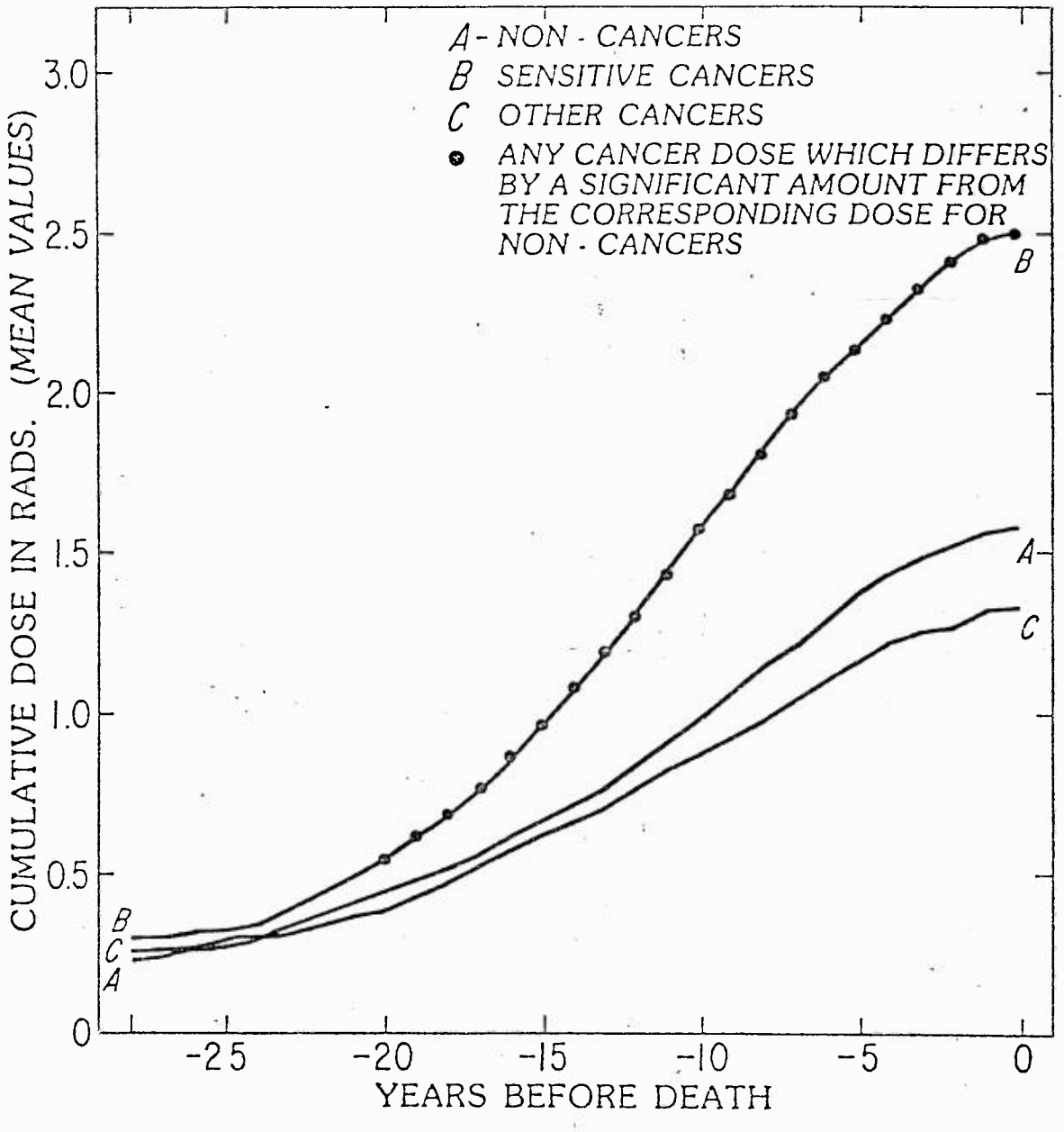
Age Trend of Radiation Doses for 4 Long Term Workers

FIGURE 3



Age Trend of Cumulative Radiation for Three Groups of Male Workers

FIGURE 4



Cumulative Radiation Doses by Stated Pre-Death Rates