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Occupational Exposures to Ionizing Radiations

Relations between exposure age and cancer risk

by

Alice Stewart

George Kneale

Department of Public Health
and Epidemiology
University of Birmingham
Edgbaston
Birmingham B15 2TT
UK

Tel: 0121 414 3367
Fax: 0121 414 3630

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Abstract

The records of nuclear workers at Hanford include annual doses of external radiation for the period 1944 and 1978 and cancer-related deaths before 1990. The results of including these data in a risk set analysis by the Kneale methodology are described together with the results of high lighting the experiences of 56 workers who died in 1969. The findings of this analysis confirm an earlier impression of a cancer risk at low dose levels which is largely the result of exposures after 55 years of age causing extra cancer deaths after 75 years of age. Also confirmed is an earlier impression of no histological difference between the extra radiogenic cancers and the idiopathic cancers. The implications of these findings for A-bomb data are discussed.

Introduction

Though recent work by Stewart and Kneale (1,2) has made it unlikely that A-bomb survivors are "representative human beings" (3). Gilbert and her associates remain firmly of the opinion that risk estimates based on A-bomb data, which are indicative of no risk at low dose levels, are directly applicable to nuclear workers. Thus, they recently had occasion to say that "current data from Hanford indicate that low level radiation exposure risks are consistent with no risk, with predictions by the ICRP or BEIR V, and with risks that are several times these predictions", and, for workers at Hanford, Oak Ridge and Rocky Flats, "the overall risk estimate for leukemia was negative, while the estimate for all cancers except leukemia was almost exactly zero" (4,5).

There has been no attempt by Gilbert *et al* to use these data to study relations between exposure age and cancer risk. However, in one of the 1993 publications there is a table which shows that for Hanford and Oak Ridge workers whose life span exceeded 75 years there was a positive correlation between cancer risk and radiation dose (5). In Rocky Flats data there was no evidence of this correlation, and it had no counterpart in A-bomb data. Therefore, Gilbert *et al* decided that: "although it is possible that the observed increase with age results from a cause-and-effect association, it seems more likely that this effect has resulted from bias in the data such as bias in ascertainment of deaths, bias in the assignment of cause of death, bias related to smoking and other potentially confounding factors for which data were not available, or bias related to dosimetry".

This lame conclusion was clearly the result of A-bomb data repeatedly leaving an impression of a greater cancer risk for persons who were under

50 years of age when exposed than for later exposures (6). But, it was made without reference to fact that Kneale and his associates have repeatedly found evidence of a cancer risk for Hanford workers (7,8), and have recently shown that this was largely the result of exposures after 50 years of age causing of extra cancer deaths after 72 years of age (8).

The Kneale *et al* findings for Hanford workers conflict not only with alternative analyses of exactly the same data but also with the 1991 recommendations of ICRP and BEIR V (9,10). Therefore, there is clearly a need for further testing of the Kneale methodology (11), and of a recent suggestion that both relations between exposure age and cancer risk, and the types of cancer caused by radiation, are different in high and low dose situations (12).

Materials and Methods

The data included in the following analysis have already been described (8). They relate to 27,395 men and 8,473 women whose exposure ages ranged from 18 to 65 years and whose exposure dates ranged from 1944 to 1978. For these occupational exposures the total dose, over and above the background dose, was 831 Sv. For individual workers the average dose rate was less than 1 mSv per annum and only 18 men ever recorded more than 50 mSv in a single year. By the end of 1989 the ascertained number of cancer-related deaths was 2,045, and included in this series were 191 non-fatal cancers (Table 1).

In the 1993 analysis of Hanford data there were numerous controlling factors, also an attempt to estimate the usual interval between cancer induction and cancer death (so called 'lag period') (8). By having only the two controlling factors that are essential for any cohort analysis (death date and death age), and having a fixed lag period of 15 years (Table 2), the complex analysis of 1993 was replaced by a much simpler one. The basic methodology was unchanged (11), but it was much easier to follow since instead of thousands of risk sets there were only the 334 sets produced by classifying the cancer cases by the death years (46 levels) and the death ages (11 levels). The corresponding cancer cases and matched controls are shown separately in Tables 3 and 4.

For these risk sets the overall numbers of cases and matched controls (CMC totals) ranged from 58 for one of the sets containing a single cancer-related death after 75 years of age (year 1958) to 3,770 for the only set containing 9 cancer-related deaths between 50 and 55 years (year 1977). The much larger number of controls than cases was the result of the Kneale methodology 1) defining a cancer case as 'a cancer-related death in a given year'; 2) having as matched controls of the cases in each risk set all fellow members of the same birth cohort who were still at risk of a (later) cancer death; 3) treating 'ascertained deaths' either as a reason for immediate transfer to the case series (cancer-related deaths) or as a reason for removal, the following year from the study population (other deaths); 4) restricting the 'operative dose' of each risk set to the dose received by all members of the set by the end of the previous calendar year (see the CMC doses in Table 5), and 5) having, as the expected dose for each risk set, the mean CMC dose (see the average CMC doses in Table 5 and the expected doses in Table 6).

The figures in these Tables only relate to 10 risk sets in 1969 (or 56 of the 2,045 cancer-related deaths), but the figures for any one year should make it easy to follow each step of the analysis. For example, in 1969 there were (as usual) no cancer-related deaths before 30 years of age (Table 3). For deaths at later ages, the CMC numbers ranged from 357 (over 75 years) to 3,539 (45-49 years) and the equivalent case numbers were 3 and 4 (Tables 3 and 4). For all (lag and pre-lag) exposures, the corresponding overall doses were 1,691 and 99,654 mSv for CMC, (Table 5); 10 and 123 mSv for cases (Table 6), and 14.2 and 112.6 mSv for the expected doses assuming no difference between cases and matched controls (Tables 5 and 6). Also included in these tables are separate figures for lag and pre-lag doses, and separate figures for pre-lag doses after division into four exposure ages (under 35; 35-44; 45-54, and 55+ years). With these subdivisions, which necessarily left the exposure age subgroups with a different range of death ages, there were 7 sets of observed and expected doses including 5 for the pre-lag period exposures.

For the 10 risk sets containing the 1969 cancer-related deaths, the observed and expected doses for the pre-lag period were 53 and 46.8 mSv for exposures before 35 years of age (ratio 1.13); 80 and 95.0 mSv for exposures between 35 and 45 years (0.84), 150 and 114.4 mSv for exposures between 45 and 54 years (1.31), 37 and 28.2 for exposures after 55 years (1.31), and 320 and 284.4 mSv for all pre-lag exposures (1.13). For the lag period only the observed and expected doses were 924 and 989.3 mSv (0.93), and for all (lag and pre-lag) exposures they were 1,244 and 1273.7 mSv (0.98).

For the complete series of 2,054 cancer cases the ratio of observed to expected doses was 1.01 for all lag and pre-lag exposures; 0.78 for the lag period and 1.22 for pre-lag period (Table 7). After division of the pre-lag doses into four exposure age groups, the ratios were 1.05 (under 35 years), 1.15 (35-44 years), 1.11 (45-54 years) and 1.78 (over 55 years). Also shown in Table 7 are the findings for two shorter follow-up periods, i.e. 1944 to 1975 with 796 ascertained cases, and 1944 to 1984 with 1,703. For each of these follow-up periods the O:E ratio was higher for pre-lag than lag exposures, and for pre-lag exposures after 55 years of age, they were higher for the longest follow-up period (1.78) than for the shorter periods (1.34 and 1.46).

By applying a Mantel-Haenszel chi-square test to the observed and expected doses for all pre-lag exposures after 55 years of age (36,930 and 30,303 mSv), the difference was found to be highly significant ($p < 0.0001$). Therefore, despite the need for several subdivisions of the pre-lag exposures, there was strong statistical support for the conclusion that there is a cancer risk at low dose levels which, during adult life, progressively increases with exposure age.

Types of radiogenic cancers

Though there have never been any signs of a special relationship between leukemia and radiation in Hanford data, the impression of *no* histological differences between the radiogenic and idiopathic cancers is a recent one (8). Therefore, in Table 8, both the main series of 2,054 cases, and the cases whose pre-lag doses for exposures after 55 years of age exceeded the expected dose (163 'high risk' cases), are given an ICD classification, with separate identification of fatal and non-fatal cancers.

In the main series there were less than 10% of deaths after 75 years of age (Table 3), and in the high risk series there were, by definition, no younger cases. This age difference is the most likely reason for the proportion of non-fatal cancers being much higher for the high risk cases (17.3%) than the main series cases (7.0%), and the proportion of prostate tumors also being higher in the high risk series (14.7%) than in the main series (8.3%). This is so, because in the main series both the deaths ascribed to prostate tumors and the non-cancer deaths that had these tumors as a contributory cause, had an older age distribution than the other cancer-related deaths. Therefore, even with no histological differences between the radiogenic and idiopathic cancers, we would expect the former to be biased in favour of prostate cancers. In short, although the extra cancer deaths of A-bomb survivors were biased in favour of leukemia (and on the young side) neither of these findings was reproduced in Hanford data. On the contrary according to these data the cancers caused by occupational exposures to radiation mainly affected workers who were over 75 years of age when they died and were histologically indistinguishable from other cancers in the same age range.

Discussion

Between the bombing of Hiroshima and Nagasaki, in August 1945, and collection of A-bomb data, which began in October 1950, there was ample time for deaths from acute effects of the radiation to have age related effects. These early deaths required high doses and are probably the reason why, in the life span study cohort of A-bomb survivors, the proportion of high doses was much lower for persons who were under 10 or over 50 years of age in 1945 than for the intervening age groups; and in the much smaller cohort of *in utero* children, there was gross under-representation of near conception exposures in the highest of four dose groups (2). According to Stewart and Kneale, the first bias makes it unsafe to base risk estimates for nuclear workers on A-bomb data, and the second bias makes it much harder to detect cancer effects of fetal irradiation in A-bomb data than in the Oxford Survey of Childhood Cancers (OSCC data) (13,14).

Both in Hanford data, where a possible cause of extra cancer deaths was repeated exposures to small doses of external radiation, and in OSCC data, where a possible cause of extra cancer deaths was obstetric radiography, we now have evidence of a cancer risk at low dose levels. We also have evidence that although, in both situations, the extra cancers had a distinctive age distribution, they had the same cell types as the idiopathic cancers. Thus, in Hanford data where the trouble seems to be coming exposures after 50 years of age, and in OSCC data, where it is largely the result of third trimester exposures, the radiogenic cancers had the older age distribution and were histologically indistinguishable from the idiopathic cancers.

These findings are a reminder that fatal cancers are probably the result of a long drawn out process involving both loss of immunological competence (which allows infections to be competing causes of death) and mutations (whose commonest cause might well be natural radioactivity or background radiation). Therefore, both the present findings for occupational exposures, and the earlier findings for prenatal x-rays could be the result of a) background radiation being a common cause of idiopathic cancers; b) small doses of man-made radiation having the same effect as natural radioactivity, and c) the cancer risk from mutant clones having strong associations with age and environmental factors *via* competing causes of death (15).

According to these suggestions, which amount to no more than a working hypothesis, both relations between exposure age and cancer risk, and the types of cancers caused by man-made radioactivity, might depend upon whether the extra radiation was or was not sufficient to have an exclusively high dose effect, namely, immune system damage. For occupational exposures and diagnostic radiography there would be little or no risk of such damage. However, known effects of the Hiroshima and Nagasaki bombs included deaths from acute bone marrow depression (16) and possible effects include abortions (2) and a need for extra leucocytes that was met by a rapid outpouring of mutant cells and followed by extra deaths from myeloid leukemia (17).

These observations make it relevant to ask whether A-bomb data should remain as the principal source of risk estimates for occupational exposures and other low dose situations. According to this method, which is the basis of ICRP and BEIR V recommendations, the non-cancer death rate of the LSS cohort should be independent of the radiation dose, and the proportion of high dose survivors should be independent of the exposure age. Even according to the Radiation Effects Research Foundation, the first condition has not been met (18). Therefore, the present findings for Hanford workers could be a further sign that both the types of cancer caused by radiation, and the relations between exposure age and cancer risk, are different in high and low dose situations.

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Table 1

Specifications of the 2054 cancer-related deaths.

Specifications		Cancer Cases			%
		Fatal[a]	Non-Fatal[b]	Total	Non-Fatal
Birth Dates	1875-89	50	14	64	21.9
	1890-99	220	43	263	16.3
	1900-09	517	60	577	10.4
	1910-19	656	54	710	7.6
	1920-29	312	15	327	4.6
	1930-39	85	5	90)	4.4
	1940+	23	-	23)	
Death Dates	1944-59	152	3	155)	4.9
	1960-64	122	11	133)	
	1965-69	205	17	202	8.4
	1970-74	272	34	306	11.1
	1975-79	360	42	402	10.4
	1980-84	449	56	505	11.1
	1985-89[c]	323	28	351	8.0
Cancer Sites (ICD Nos)	Digestive (150-159)	484	45	529	8.5
	Respiratory (160-163)	581	49	630	7.8
	Genito-urinary (180-189)	242	54	296	18.2
	RES neoplasms[d] (200-209)	198	20	218	9.2
	Other & unspecified	358	23	381	6.0
	Total	1863	191	2054	9.3

a Cases where cancer was the stated cause of death

b Cases where cancer was a contributory cause of a non-cancer death

c Incomplete identification of death certificate data, mainly affecting deaths outside the State of Washington

d RES neoplasms include leukemia, myeloma and lymphoma

Table 2

Recorded doses of the 2045 cancers cases by pre-death interval and exposure age.

Pre-Death Interval years	Exposure Age years	Radiation Dose in mSv Total	Mean
0-14 (lag period)	All ages	223,081	108.6
15+ (pre-lag period)	under 35	49,960	24.3
	35-44	108,650	52.9
	45-54	128,115	62.4
	55+	83,268	40.5
Total	pre-lag	369,993	180.1
	lag and pre-lag	593,074	288.7

Table 3

Cancer cases by death age and death year (334 risk sets)

Calendar years	Cancer Cases by Death Ages in years											Total
	25-	30-	35-	40-	45-	50-	55-	60-	65-	70-	75+	
1944	–	–	–	–	–	1	–	–	–	–	–	1
1945	–	1	–	–	–	–	–	1	–	–	–	2
1946	–	–	–	–	–	–	–	1	–	–	–	1
1947	–	1	–	–	–	1	–	1	–	–	–	3
1948	–	1	–	1	1	–	1	1	–	–	–	5
1949	–	1	–	1	1	–	2	2	–	–	–	7
1950	–	–	–	1	–	–	1	3	–	–	–	5
1951	1	1	–	–	1	4	1	1	2	–	–	11
1952	–	1	–	–	1	2	3	1	1	–	–	9
1953	–	1	1	2	3	2	3	3	1	–	–	16
1954	–	–	2	–	1	2	3	1	–	3	–	12
1955	–	2	–	1	1	2	1	4	1	–	–	12
1956	–	–	1	1	2	1	2	1	4	1	–	13
1957	–	–	1	–	2	3	5	2	1	2	–	16
1958	–	1	1	4	3	2	1	4	4	1	1	22
1959	–	–	–	–	5	3	3	4	2	2	1	20
1960	–	–	–	2	1	3	3	4	5	2	2	22
1961	–	–	4	4	1	1	–	4	5	–	3	22
1962	–	1	2	–	4	4	6	1	5	4	2	29
1963	–	–	–	1	1	3	7	10	6	2	1	31
1964	1	–	–	2	5	1	3	7	5	4	1	29
1965	–	–	–	2	2	4	4	6	7	8	2	35
1966	–	–	–	–	1	4	6	3	2	4	6	26
1967	–	1	3	1	–	7	11	6	7	4	2	42
1968	–	–	2	4	2	1	7	11	11	3	2	43
*1969	–	1	2	1	4	3	8	10	12	12	3	56
1970	–	1	–	–	4	10	12	8	10	4	8	57
1971	–	–	–	2	1	8	14	13	9	11	9	67
1972	1	–	–	2	3	4	8	13	10	7	10	58
1973	–	1	1	3	4	7	6	12	12	8	8	62
1974	–	–	–	2	2	10	9	9	7	7	16	62
1975	–	–	2	1	3	4	12	15	13	8	9	67
1976	–	–	–	2	2	7	6	24	9	16	14	80
1977	–	3	–	–	1	9	7	17	9	16	16	78
1978	1	–	1	3	3	6	10	19	16	13	17	89
1979	–	–	2	3	6	4	7	16	17	15	18	88
1980	–	–	–	1	2	7	15	14	21	21	21	102
1981	–	–	1	2	2	3	7	12	29	16	14	86
1982	–	–	1	–	3	2	19	9	22	16	29	101
1983	–	–	–	2	4	3	10	11	34	15	24	103
1984	–	–	–	3	2	4	11	17	25	21	30	113
1985	–	–	–	–	3	2	12	13	21	19	32	102
1986	1	–	–	2	5	6	7	11	11	22	37	102
1987	–	–	–	–	–	3	3	11	9	14	20	60
1988	1	–	–	–	1	2	4	3	8	10	17	46
1989	–	–	1	–	–	2	–	4	10	8	16	41
	(6)	(18)	(28)	(56)	(93)	(157)	(260)	(343)	(383)	(319)	(391)	2054

* see Tables 5 and 6

Table 4

Matched controls in the 334 risk sets of Table 3

Calendar years	Matched Controls for Each Risk Set										
	25-	30-	35-	40-	45-	50-	55-	60-	65-	70-	75+
1944	-	-	-	-	-	188	-	-	-	-	-
1945	-	1324	-	-	-	-	-	104	-	-	-
1946	-	-	-	-	-	-	-	136	-	-	-
1947	-	1731	-	-	-	593	-	170	-	-	-
1948	-	2030	-	1301	990	-	419	206	-	-	-
1949	-	2107	-	1453	1116	-	476	240	-	-	-
1950	-	-	-	1593	-	-	526	287	-	-	-
1951	2504	2498	-	-	1254	1008	600	330	139	-	-
1952	-	2654	-	-	1463	1050	691	376	165	-	-
1953	-	2781	2656	2283	1596	1171	730	432	196	-	-
1954	-	-	2727	-	1752	1288	801	484	-	78	-
1955	-	3151	-	2644	1947	1244	1049	533	274	-	-
1956	-	-	2977	2822	2127	1432	1071	593	311	128	-
1957	-	-	3061	-	2348	1609	1118	671	341	150	-
1958	-	3019	3164	2945	2500	1747	1238	711	384	176	57
1959	-	-	-	-	2627	1852	1342	787	427	200	79
1960	-	-	-	2981	2734	1986	1265	1022	473	233	93
1961	-	-	3355	3084	2883	2112	-	1020	531	-	122
1962	-	2696	3267	-	2915	2317	1571	1044	600	283	140
1963	-	-	-	3289	2997	2476	1680	1156	610	312	176
1964	1777	-	-	3436	3018	2609	1786	1243	678	348	196
1965	-	-	-	3524	3045	2730	1926	1181	883	379	233
1966	-	-	-	-	3156	2876	2062	1321	877	429	267
1967	-	2169	2966	3443	-	2923	2259	1469	918	463	296
1968	-	-	2793	3385	3386	3015	2417	1585	1017	489	322
*1969	-	2232	2579	3340	3539	3048	2548	1693	1089	551	357
1970	-	2208	-	-	3623	3084	2667	1825	1041	722	408
1971	-	-	-	3206	3625	3211	2818	1937	1156	711	467
1972	1452	-	-	3099	3575	3295	2852	2122	1293	745	510
1973	-	2004	2459	2942	3493	3428	2936	2271	1405	823	529
1974	-	-	-	2823	3479	3613	2986	2416	1505	862	594
1975	-	-	2658	2742	3458	3707	3054	2512	1636	844	760
1976	-	-	-	2772	3475	3732	3216	2652	1749	967	781
1977	-	2697	-	-	3457	3761	3360	2715	1916	1091	825
1978	2740	-	2887	3075	3390	3745	3532	2817	2052	1216	909
1979	-	-	2825	3080	3189	3709	3701	2864	2179	1308	1009
1980	-	-	-	3098	3056	3646	3782	2957	2271	1410	1143
1981	-	-	2846	3000	2999	3571	3763	3109	2423	1502	1254
1982	-	-	2947	-	3013	3472	3713	3228	2483	1637	1403
1983	-	-	-	2868	3042	3341	3638	3371	2578	1761	1554
1984	-	-	-	2801	3049	3144	3606	3526	2634	1877	1686
1985	-	-	-	-	3068	3024	3546	3584	2725	1966	1855
1986	795	-	-	2823	2974	2959	3478	3565	2878	2096	2016
1987	-	-	-	-	-	2966	3379	3534	2983	2168	2221
1988	295	-	-	-	2846	3002	3258	3475	3149	2281	2530
1989	-	-	2656	-	-	3017	-	3473	3306	2400	2840

* see Tables 5 and 6

Table 5

Overall (CMC) doses for the 10 risk sets including 56 cancer related deaths in 1969

Pre-Death Interval Years	Exposure Age Years	Death Age Years		30-	35-	40-	45-	50-	55-	60-	65-	70-	75+
				Age Range of Cancer-Related Deaths in Years — Cumulative Dose (mSv)									
Under 15 Years (lag period)	All Ages	30+	a	40317	53441	84095	82984	72553	70932	42357	18958	2566	207
			b	*18.1*	*41.4*	*25.2*	*93.8*	*71.4*	*222.7*	*250.2*	*208.9*	*55.9*	*1.7*
Over 15 years (pre-lag period)	under 35	30-59	a	61	2461	11746	16670	10134	4027	-	-	-	-
			b	*0.0*	*1.9*	*3.5*	*18.8*	*10.0*	*12.6*	-	-	-	-
	35-44	50-69	a	-	-	-	-	6402	11541	6114	1488	-	-
			b	-	-	-	-	*6.3*	*36.2*	*36.1*	*16.4*	-	-
	45-54	60+	a	-	-	-	-	-	-	3438	4528	1860	436
			b	-	-	-	-	-	-	*20.3*	*49.9*	*40.5*	*3.7*
	55+	70+	a	-	-	-	-	-	-	-	-	894	1047
			b	-	-	-	-	-	-	-	-	*19.4*	*8.8*
	All ages	30+	a	61	2461	11746	16670	16536	15568	9553	6016	2754	1484
			b	*0.0*	*1.9*	*3.5*	*18.8*	*16.3*	*48.8*	*56.4*	*66.3*	*59.9*	*12.5*
Lag and Pre-Lag Exposures	All ages	30+	a	40378	55902	95841	99654	89089	86500	51910	24974	5320	1691
			b	*18.1*	*43.3*	*28.7*	*112.6*	*87.7*	*271.5*	*306.6*	*275.2*	*115.8*	*14.2*
Nos. of persons (CMC Totals)				2232	2579	3340	3539	3048	2548	1693	1089	551	357

a Total CMC doses for 10 risk sets containing one or more of the 56 cancer-related deaths in 1969

b Average CMC dose (these are the expected risk set doses for the 56 cancer-related deaths in 1969 (see Table 6)

Table 6

Observed (0) and expected (E) doses for the 10 risk sets in Table 5

Pre-Death Interval Years	Exposure Age Years	Death Age Years		30-	35-	40-	45-	50-	55-	60-	65-	70-	75+	Total Dose
				Cancer Death Age Range in Years — Observed and Expected Doses (mSv)										
Under 15 Years (lag period)	All Ages	30+	0	21	61	9	83	20	157	370	176	27	0	924
			E	*18.1*	*41.4*	*25.2*	*93.8*	*71.4*	*222.7*	*250.2*	*208.9*	*55.9*	*1.7*	*989.3*
Over 15 years (pre-lag period)	under 35	30-59	0	-	-	3	40	3	7	-	-	-	-	53
			E	*0.0*	*1.9*	*3.5*	*18.8*	*10.0*	*12.6*	-	-	-	-	*46.8*
	35-44	50-69	0	-	-	-	-	0	42	25	13	-	-	80
			E	-	-	-	-	*6.3*	*36.2*	*36.1*	*16.4*	-	-	*95.0*
	45-54	60+	0	-	-	-	-	-	-	28	35	87	0	150
			E	-	-	-	-	-	-	*20.3*	*49.9*	*40.5*	*3.7*	*114.4*
	55+	70+	0	-	-	-	-	-	-	-	-	27	10	37
			E	-	-	-	-	-	-	-	-	*19.4*	*8.8*	*28.2*
	All ages	30+	0	0	0	3	40	3	49	53	48	114	10	320
			E	*0.0*	*1.9*	*3.5*	*18.8*	*16.3*	*48.8*	*56.4*	*66.3*	*59.9*	*12.5*	*284.4*
All exposures		30+	0	21	61	12	123	23	206	423	224	141	10	1244
			E	*18.1*	*43.3*	*28.7*	*112.6*	*87.7*	*271.5*	*306.6*	*275.2*	*115.8*	*14.2*	*1273.7*
No. of Cancer Cases				1	2	1	4	3	8	10	12	12	3	56

0 Observed doses of the cancer cases in each risk set

E Expected doses (see average CMC doses in Table 5)

Table 7

Observed and expected doses for cancer-related deaths in three overlapping periods

Follow-up Period (No. of Cancer Cases)	Exposure Period	Exposure Age Years	Dose in mSv Obs (O)	Dose in mSv Exp (E)	Ratio O:E
	Lag and Pre-Lag	all ages	13975	14921	0.94
	Lag period only	all ages	10234	11602	0.88
1944-1974 (796)	Pre-lag only	all ages	374	3319	1.13
		under 35	642	696	0.92
		35-44	1477	1221	1.21
		45-54	1121	1028	1.09
		55+	501	374	1.34
	Lag and Pre-Lag	all ages	40441	47223	0.86
	Lag period only	all ages	19631	26546	0.74
1944-1984 (1703)	Pre-lag only	all ages	20810	20677	1.01
		under 35	2929	3305	0.89
		35-44	6694	6581	1.02
		45-54	6917	7875	0.88
		55+	4270	2916	1.46
	Lag and Pre-Lag	all ages	59292	58816	1.01
	Lag period only	all ages	22362	28513	0.78
1944-1989 (2054)	Pre-lag only	all ages	36930	30303	1.22
		under 35	4996	4709	1.06
		35-44	10803	9409	1.15
		45-54	12810	11519	1.11
		55+	8321	4666	1.78

Table 8

Comparisons Between two Series of Fatal and Non-fatal Cancers

Types of Cancer (ICD Nos.)	Cancers[a] All Cases Nos.	%	High Risk Cases[b] Nos.	%	% of High Risk Cancers Fatal	Non-Fatal
Digestive (150-159)	529 (45)	25.8	32 (7)	19.6	5.2	15.6
Respiratory (160-163)	630 (49)	30.7	50 (8)	30.7	7.2	16.3
Prostate (185)	170 (38)	8.3	24 (9)	14.7	11.4	23.7
Other GU (180-189)	126 (16)	6.1	12 (2)	7.4	9.1	12.5
RES neoplasms (200-209)	218 (20)	10.6	16 (2)	9.8	7.1	10.0
Other & unspecified	381 (23)	18.5	29 (5)	17.8	6.7	21.7
Death Years						
1944-84	1703 (163)	82.9	64 (25)	39.3	5.9	15.3
1985-89	351 (28)	17.1	99 (8)	30.1	11.7	28.6
Total	2054 (191)	100.0	163 (33)	100.0	7.0	17.3

a ( ) = Non-fatal cancers (see footnote to Table 1)

b Cancer cases whose pre-lag doses for exposures after 55 years of age exceeded the expected dose.