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Radiation Risks of Nuclear Workers and Other Low Dose Situations

by

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ABSTRACT

Re-analysis of Hanford data by a method which identified all the exactly matched controls of all the cancer cases, has produced evidence of a cancer risk at supposedly safe dose levels. The extra cancers are evenly distributed between different types of neoplasms but as a result of mounting sensitivity to carcinogenic effects of radiation with mounting age, as well as long intervals between cancer induction and death, the radiogenic cancers are concentrated among the older cases. The much lower risk for A-bomb survivors than for nuclear workers is probably the result of gross under representation of older persons in the higher dose subgroups of the LSS cohort - or selection effects of the early deaths.

Key words: Cancer: Radiation: Epidemiology.

In 1990 BEIR V addressed the problem of 'health effects of exposure to low levels of ionizing radiation' and came to the conclusion that when dose rates fall to the levels which are typical of occupational exposures and background radiation, spontaneous repair of mutational damage reduces the cancer risk[1]. Therefore, although they recommended that risk estimates for such exposures be derived from a life span study cohort of A-bomb survivors (LSS data), they also recommended a reduction allowance of 2 or thereabouts, to allow for a 'dose rate effectiveness factor' or DREF.

According to BEIR V only a small proportion of cancer deaths are the result of a life time exposure to background radiation, and the possibility of no risk from this source has not been ruled out. Therefore, it occasioned no surprise when an analysis of 1945-1981 deaths of 36,235 badge monitored workers at Hanford found no evidence of any cancer effects in spite of there being a total dose of 831 Sv[2] - since this was equivalent to less than seven years exposure to background radiation. However, a later analysis of 1944-1986 deaths of these workers has not confirmed these negative findings[3]. On the contrary, it has served as a reminder that it is far from certain that linear extrapolation of high doses overestimates the cancer risks of small, repeated doses.

The first sign of trouble from the supposedly safe exposures of workers in US nuclear facilities dates back to 1977, when Mancuso, Stewart and Kneale (MSK) first examined Hanford data[4]. On that occasion comparisons between workers who had died from cancer and workers who had died from other causes showed that the former had a higher average dose than the latter. Also established was the fact that this difference was largely the result of radiation received after 40 years of age (and more

than 10 years before deaths) by men who subsequently developed three types of cancer, namely, myeloma and cancers of pancreas and lung.

The sponsor of the 1977 analysis of Hanford data was the US Department of Energy or DOE. This department refused to accept the MSK findings as evidence of a cancer risk and felt justified in this opinion when Saunders showed that live workers had higher doses than cancer cases[5], and Marks *et al* showed that the workforce as a whole had exceptionally low rates of mortality for all diseases including cancer[6]. These rebuttals of the MSK analysis marked the beginning of a long drawn out dispute about the correct interpretation of Hanford data, or a controversy which still has Kneale and Gilbert as the principal exponents of opposite opinions, and the nuclear establishment firmly on the side of Gilbert[7].

After publication of the 1977 analysis, Kneale had only limited access to Hanford data and was much hampered by being unable to correct obvious errors in the recording of job descriptions. Nevertheless, in 1981 and again in 1984, he showed the results of including these workers in an analysis which a) relied upon internal comparisons and included several levels of internal radiation monitoring (IRM) among the controlling factors; b) assumed that factors influencing our perceptions of radiation effects include exposure ages and pre-death interval and c) distinguished between neoplasms on tissue which rated high in an ICRP classification of 'sensitivity to cancer induction by radiation' (A cancers), and other and unspecified neoplasms (B cancers)[8 & 9].

For the A cancers, or the group which included neoplasms of digestive, respiratory and haemopoietic tissues, there was definite evidence of a radiation effect. However, for B cancers (as well as non-cancer deaths) there was a negative dose trend (or evidence of insufficient control of dose related factors other than radiation). Therefore, for A and B cancers combined there was no certainty of any radiation effect. Kneale was naturally anxious to observe the effects of controlling for socio-economic status (SES)instead of IRM levels but was in no position to do this. Therefore, although he continued to use Hanford data to perfect a method of statistical analysis which required identification of "risk sets within cohorts" (see below), there was a period of several years when he could do nothing further to influence opinions about cancer risks of low level radiation.

Gilbert was in a much stronger position to influence these opinions since she had the full backing of and was in a position to replace the faulty job descriptions with a standard SES classification of Hanford occupations[2]. She was aware that internal comparisons were more informative than external comparisons, but, unlike Kneale - whose tests of cancer effects were based on the experiences of individual workers, Gilbert computed her 'test statistics' from three or four dose level groups, and continued to compare Hanford workers both with national statistics and with the life span study (LSS) cohort of A-bomb survivors.

By 1990, Gilbert had extended her analysis of risk factors to workers in three nuclear facilities (Hanford, Oak Ridge and Rocky Flats)[10]. She remained firmly of the opinion that in these facilities there had not been any erosion of a strong 'healthy worker effect' (HWE) by any harmful

effects of the radiation, and equally certain that there was no discrepancy between these negative findings and risk estimates based on LSS data. However, as a result of DOE being forced to concede that it was not in the public interest for holders of their epidemiological contracts to be the only source of risk estimates for nuclear workers, Gilbert has not had the last word in the Hanford controversy. In 1986 an agreement between DOE and the Three Mile Island Public Health Fund was signed which ensured gradual release of all records of epidemiological importance and, by January 1992, there had been sufficient release of Hanford data for Kneale to include this cohort in a new analysis[3].

A novel feature of the 1992 analysis of Hanford data (1944-86 deaths) was a system of numbering which produced "risk set formation" of the data by bringing together workers who had in common the ten 'essential controlling factors' in Table 1. Only the sets which included cancer cases were of any use, but from these could be obtained observed and expected doses for each year of age (and each predeath period) based on the records of the cancer cases and all closely matched controls.

For example, the risk set in Table 2 (which has three cancer deaths) was restricted to workers who had the following factors in common: they were white males who had worked at Hanford for more than three years; were hired in 1950 or 1951; had no offsite exposures, and had ceased work more than three years before the end of the follow-up period (1986). They also belonged in the lowest of five socio-economic levels and were born in the period 1895-99. The first man to die (from cancer) had an interval of less than 3 years between leaving Hanford and dying. Therefore, for this case, there were only two exactly matched controls (see the first demarcation

line in Table 2). For the second death (which was also a cancer case) there were 10 exactly matched controls, and for the third cancer death (which came sixth in the list of all deaths) there were 5 controls. Finally, for each cancer case there were observed and expected doses for 13 years (1951-63) and 13 ages (51 to 64 years), and for one of the cases there was an interval of 19 years between leaving Hanford and dying.

With Hanford data in risk set formation it was possible to calculate the probability of each set having the observed numbers of cancer deaths and thus discover that there was a significant excess of these cases. This critical finding was followed by numerous tests of cancer and radiation related factors whose purpose was to discover a) the amount of radiation needed to double the normal cancer risk assuming a linear dose response (doubling dose), b) the shape of the dose response curve (exponent factor) and c) the effects of varying exposure age, exposure year and interval (or predeath period).

The results of these tests were consistent with there being a dose related cancer risk, and with the extra radiogenic cancers having the same cell types as normal or non-radiogenic cancers. In addition, there was evidence of a) greater sensitivity to carcinogenic effects of the radiation after than before 50 years of age (exposure age effect); b) better recording of doses after than before ~~1930~~ 1960 (exposure year effect); c) an interval of several years between cancer induction and cancer death (interval or cancer latency effect), and d) non-linearity of dose response (with a power law exponent of less than 1.0). As a result of these factors the radiogenic cancers were concentrated among the older cases, and the model of relative risk finally chosen by Kneale had in addition to two main

parameters (i.e. radiation dose and power law exponent), three subsidiary parameters (i.e. exposure age, exposure year and interval).

With this model the cancer risks of individual workers can be estimated by weighting annual doses with estimated effects exposure age, exposure year and interval, as in Table 3. The two sets of actual and 'cancer effective doses' in this table are taken from an earlier risk model which only had two subsidiary parameters (exposure age and interval). But they serve to show that, the main difference between Kneale and BEIR V risk models is that the former attach much greater importance to exposure age than the latter. The reason for this important differences requires an understanding of why the LSS cohort of A-bomb survivors has always been regarded as a suitable population for estimating low dose effects, and why this might be a mistake.

The LSS cohort was assembled five years after the bombing of Hiroshima and Nagasaki, and is a source of risk estimates which, for a long time, assumed that no selection effects of the massively high death rates of 1945-46 had lasted for more than five years[11]. Today they are based on the assumption that selection against non-cancer deaths has lasted longer than 5 years, but that this has not appreciably affected the cancer risk[12]. However, it is only necessary to divide the LSS cohort into 8 dose levels on the T65 scale (or 7 levels on the DS86 scale) to see that the proportion of high dose survivors (over 1 Gy) is much smaller for persons who were under 10 or over 50 years of age in 1945 than for the intervening age groups (Fig. 1).

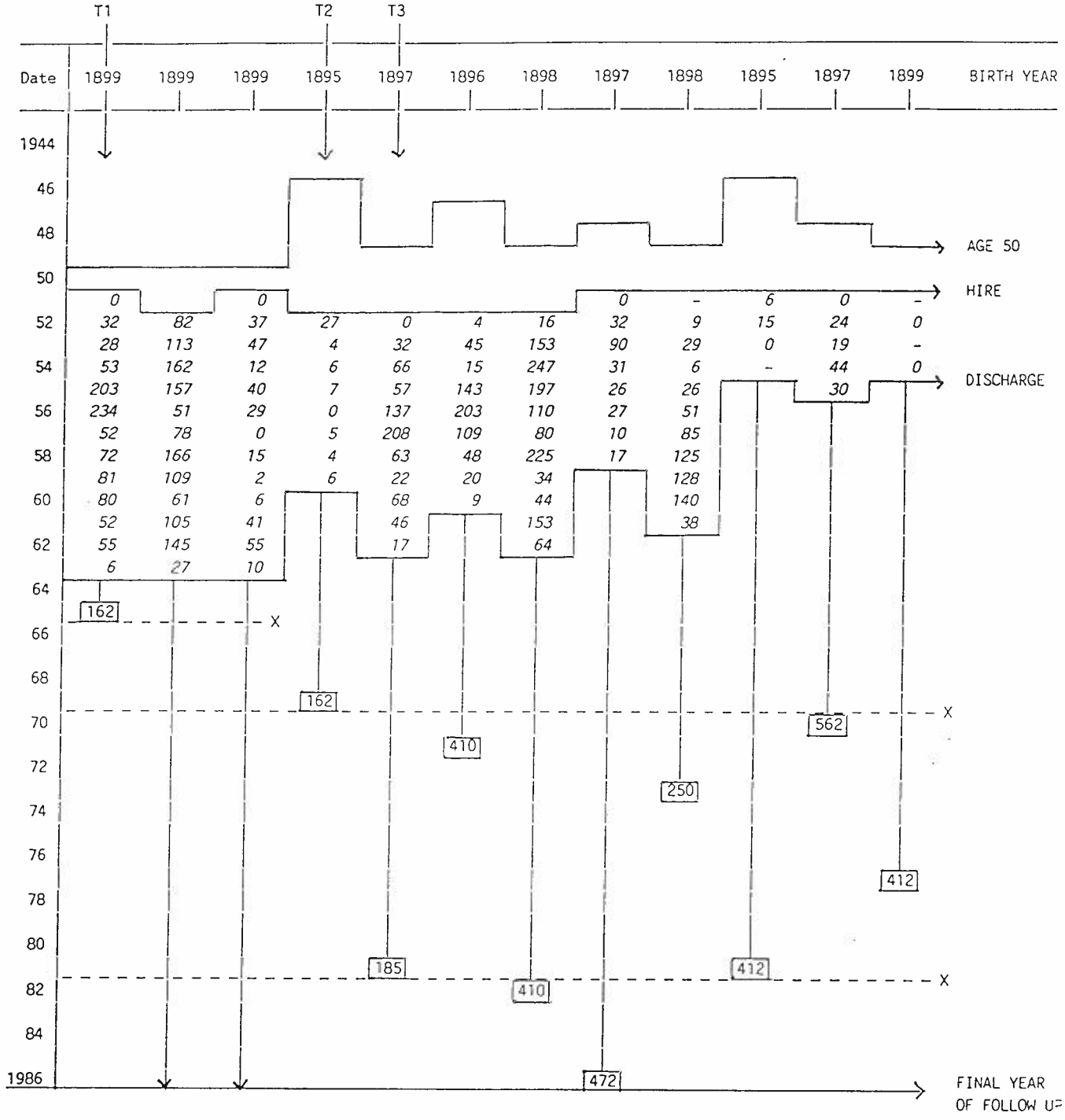
The LSS cohort originally included equal numbers of persons from four zones (measured from each hypocentre) and each zone was matched for size, age and sex. Therefore, the age group differences in Fig. 1 could only be the result of children and old persons experiencing more deaths from subacute effects of the bombing than young or middle aged adults, and thus, leaving the study cohort short of persons who (by virtue of their age in 1945 and their exposure positions) were most at risk of dying either from radiogenic or non-radiogenic cancers in the first 20 or 30 years of follow-up.

Such age group differences would be expected and their effect on the LSS cohort would justify the Stewart and Kneale assumption of partial cancellation of the selection effects by residual effects of extensive marrow aplasia[13]. They would also make it unnecessary to expect uniformity between A-bomb survivors and nuclear workers, and reasonable to assume that the Kneale findings for such workers are just as plausible as the Oxford Survey of Childhood Cancer findings for prenatal x-rays[14]. Finally, the OSCC findings include evidence that background radiation is an numerically important cause of naturally occurring cancers. Therefore, instead of perpetuating the idea that man-made additions to this source of radioactivity are of no practical importance, we should be doing everything possible to make the public understand that the opposite is true, and that any addition to background radiation automatically adds to population loads of cancer and genetic damage.

Table 1. Essential Controlling Factors for Kneale Model

Factors	Levels	
Sex	2	Male or female
Race	2	White or other
Birth Year	20	5 year intervals from 1870 to 1964
Hire Year	13	2 year intervals from 1944-1978
Employment period	2	Under or over 3 years
Post exposure interval	2	Under or over 3 years
Facility	2	With or without offsite exposures
Discharge Status	2	With or without definite termination date
Possible years of Death	43	1944 to 1986
Social Class	5	Census classification of main occupation: 1-199 Professional 200-229 Managerial 300-399 Clerical 400-599 Craftsmen 600+ Other blue collar

Table 2. Specifications of a Risk Set with Three Cancer Cases



T = TEST CASES or cancer deaths; _ _ _ _ X Demarcation lines for three risk sets.

Table 3. Actual and Cancer Effective Doses of two Hanford Workers

1) Birth 22.7.25 Hire 18.8.53 Death 21.3.61 Cause of Death : Large Intestine Cancer

Years	Age	Pre-death years	Actual Dose 0.1 mSv	Modulating Factors Age	Modulating Factors Latency	Cancer Effective Dose	Relative Risk
1953	28	8	-	-	-	-	-
1954	29	7	190	0.25	0.56	27	-
1955	30	6	70	0.29	0.51	10	-
1956	31	5	1050	0.33	0.44	152	-
1957	32	4	1220	0.38	0.37	172	-
1958	33	3	1830	0.43	0.29	228	-
1959	34	2	1970	0.49	0.20	193	-
1960	35	1	1250	0.54	0.10	68	-
1961	36	0	70	0.62	0.00	0	-
			Σ 7650			Σ 850	<1.26

2) Birth ~~3.4.97~~ 4.6.69 Hire 23.10.47 Death ~~4.6.69~~ 3.4.97 Cause of Death : Lung Cancer

1947	50	22	-	-	-	-	-
1948	51	21	250	3.9	0.98	965	-
1949	52	20	50	4.4	0.97	213	-
1950	53	19	1240	5.0	0.96	5952	-
1951	54	18	1480	5.7	0.94	7930	-
1952	55	17	1750	6.5	0.93	10579	-
1953	56	16	930	7.7	0.90	6445	-
1954	57	15	380	8.6	0.88	2876	-
1955	58	14	240	10.0	0.86	2064	-
1956	59	13	60	11.1	0.83	553	-
1957	60	12	40	12.5	0.81	405	-
1958	61	11	200	14.0	0.77	216	-
1959	62	10	20	16.0	0.73	234	-
1960	63	9	-	-	-	-	-
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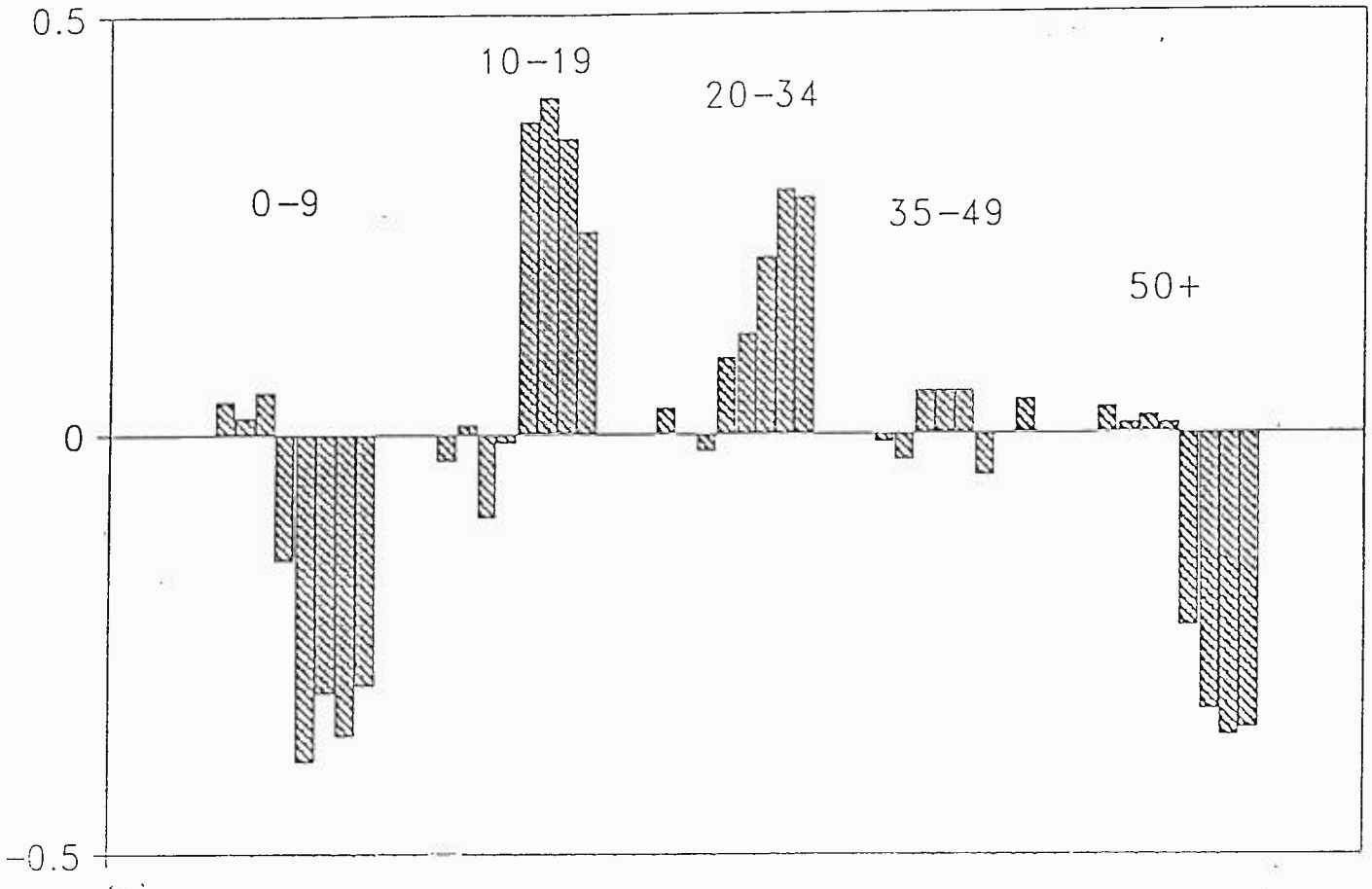
1969	72	0	Σ 6640	-	-	Σ 38432	2.59

Fig 1

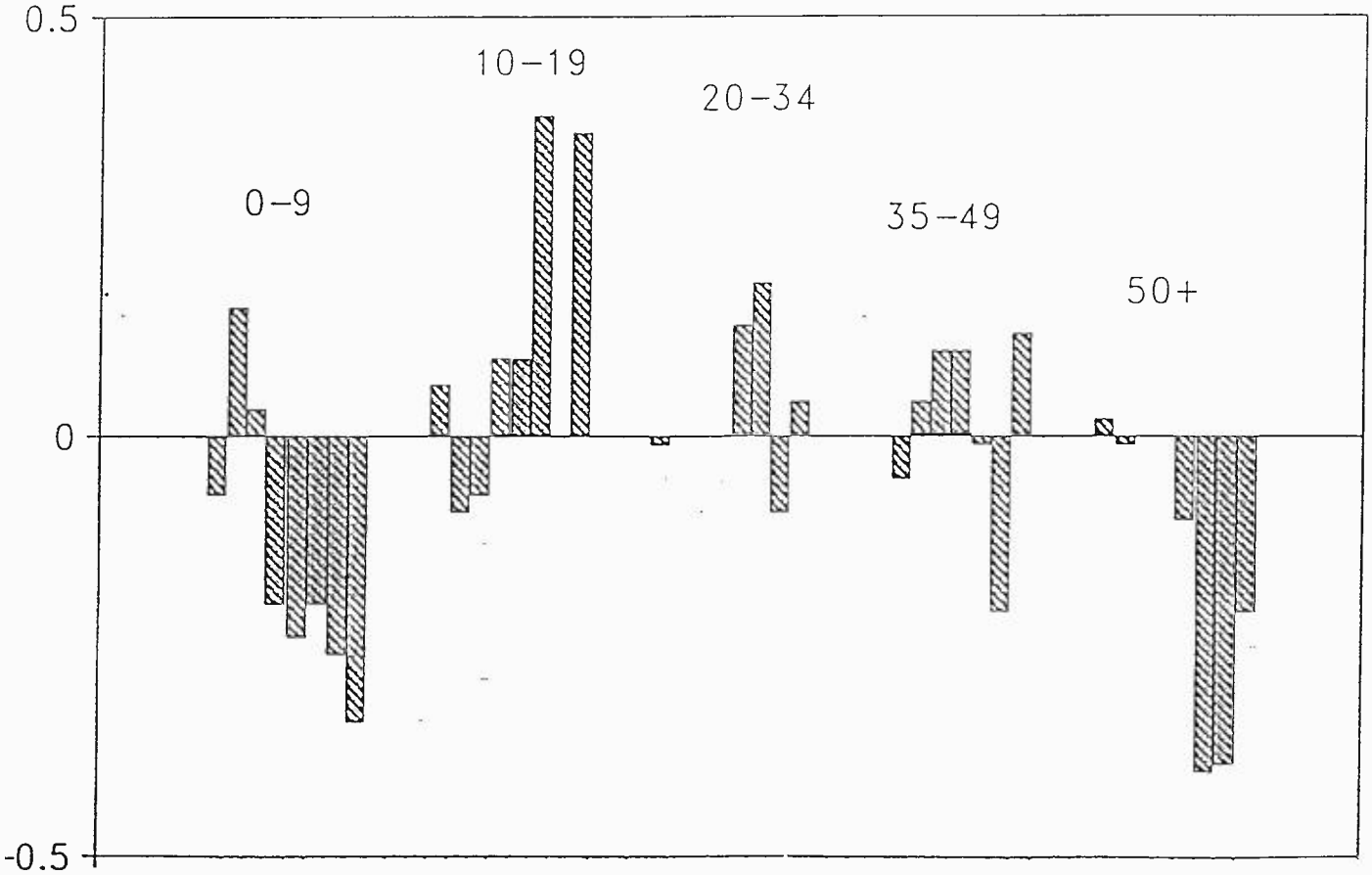
LSS Cohort

(a) T65 Dose Distribution for 5 Exposure Age Groups



(b)

(2) DS86 Dose Distribution for 5 Exposure Age Groups



Vertical Axis : Ratio of Observed to Expected Nos.
Horizontal Axis T65 : 0-, 1-, 10-, 50-, 100-, 200-, 300-, 400+

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