

Delayed effects of A-bomb radiation: a review of recent mortality rates and risk estimates for five-year survivors

ALICE M STEWART

From the Cancer Epidemiology Research Unit, Department of Social Medicine, University of Birmingham, Edgbaston, Birmingham B15 2TT

SUMMARY A review of published data relating to A-bomb survivors has led to the conclusion that since they were based on the mortality experiences of five-year survivors estimates of radiation effects should have been controlled for two opposing forces—namely, selective survival of exceptionally fit individuals during the period of heavy acute mortality and residual disabilities. Both effects were dose-related and beyond question, and the disabilities probably included the effects of incomplete repair of bone marrow damage. Therefore, in addition to differences between high and low dose being largely obliterated, there was probably distortion of cancer effects. The two opposing forces are clearly the reason why the change from the high mortality rates of 1945–6 to the low rates of the 1950s was not accompanied by a change from a positive to a negative association with dose, and imperviousness to the residual disabilities is probably the reason why sudden deaths of previously healthy individuals (exemplified by suicides) were an exception to this rule. Finally, impairment of bone marrow function probably accounts for the early epidemic of myeloid leukaemia; the apparent absence of other cancers at this time, and the relatively high, dose-related death rates for blood diseases other than leukaemia.

The question of how best to obtain estimates for cancer and other mutational effects of ionising radiation is engaging public attention.^{1,2} After a long period of assuming that estimates for A-bomb survivors—which were internally consistent and in close agreement with other observations—were of direct relevance to radiation workers and the general public,³ a survey of workers in the nuclear industry has produced estimates that are an order of magnitude higher than the earlier ones and have different implications for solid tumours and leukaemias.^{4–6} There have been numerous attempts to discredit the worker-based estimates,^{7–10} but this time it is the survivor-based estimates that are under fire.^{11,12}

The source of these estimates is a life span study (LSS) population of about 80 000 people, identified through census records five years after the bombing of Hiroshima and Nagasaki. To be more precise: the two cities were bombed in August 1945; the follow-up began in October 1950, and, for purposes of estimating radiation effects, the study population was eventually restricted to 79 736 people with

estimated (T65) doses. In this population those who were within range of potentially lethal doses of radiation were better represented (about 100%) than those from more distant locations (about 30%). Therefore the mean radiation dose (24 rads) is higher than the estimate for all five-year survivors (about 285 000 with an estimated mean dose of 17 rads). Even so there were 34 634 with near zero doses and a further 20 502 with estimated doses of less than 10 rads (table 1).

From time to time these dose-level subgroups have been compared with national statistics (standardised mortality ratio or SMR analysis),^{13–15} but all estimates of radiation effects are based on internal comparisons similar to the ones in table 1 and now available for six calendar year periods (relative risk analysis with control for sex, exposure age, and city).¹¹ On these terms the only causes of death that have ever shown any evidence of radiation effects are (a) leukaemia, with special emphasis on early years (1950–8) and the more acute forms of myeloid leukaemia; (b) other blood diseases, whose involvement could be an artifact due to difficulty in

Table 1 Relative risk analysis of 1950–74 deaths for the purpose of detecting radiation effects (from Beebe et al⁶)

Dose levels (rads)	Relative risk ¹							All deaths (1950–74)	Individuals at risk (1950)	Death rate
	All causes	All neoplasms	Leukaemia	All injuries	Suicide	Other diseases	Blood diseases			
0	1.00	1.00						8 607	34 643	23.3
1–9	1.00	1.01	1.00	1.00	1.00	1.00	1.00	4 933	20 502	24.1
10–49	0.99	1.06	1.41	0.87	0.64	0.98	1.35	3 635	14 520	25.0
50–99	0.98	1.07	1.77	1.00	0.98	0.96	1.43	1 025	4 032	25.5
100–199	1.08	1.25	5.22	0.94	0.81	0.96	2.61	740	3 112	23.8
200–299	1.06	1.72	10.80	0.55	0.12	0.94	4.08	317	1 404	22.6
300–399	1.01	1.48	18.90	0.62	0.00	0.91	4.59	138	640	21.6
≥400	1.32	2.12	23.13	1.33	0.40	1.10	6.96	248	883	28.1
Deaths No	19 646	4031	144	1210	395	14 405	117	19 646	79 736	24.6
Deaths %	100.0	20.5	0.7	6.2	2.0	73.3	0.6	—	—	—
Test ² statistic	0.000*	0.000*	0.000*	0.720	0.999†	0.610	0.000*	—	—	—

¹ With control for sex, exposure age, and city.

² Test of increasing linear trend: *significant increase in risk with rising dose.
†significant decrease in risk with rising dose.

distinguishing between aplastic anaemia and leukaemia, and (c) solid tumours, now the cause of more radiogenic deaths than leukaemia but with much weaker involvement than myeloid leukaemia and little in the way of dose-related mortality rates before 1960.

The mortality rate for diseases other than cancer has never shown any signs of being either raised or dose-related.^{11–14} Therefore in a series of mortality reports, originally sponsored by the Atomic Bomb Casualty Commission (ABCC) and now the responsibility of the Radiation Effects Research Foundation (RERF) (and culminating in the relative risk analysis of 1950–74 deaths,¹¹) there have been many reiterations of the following claims. The first conclusion is that there has been no involvement of five-year survivors in non-cancer effects of the radiation and therefore a normal risk of dying from natural causes (including cancers) at all dose levels since October 1950. The second conclusion is that the much earlier involvement of leukaemia than solid tumours is purely the result of latent period differences, and the much greater involvement of myeloid leukaemia than other neoplasms is because bone marrow is more sensitive to cancer induction by radiation than other tissues. Hence a general conclusion—namely, that we can expect early warning of any cancer hazard for radiation workers, and that this will take the form of extra deaths from myeloid leukaemia.

This interpretation of the mortality experiences of A-bomb survivors has held sway for many years and is still the basis of all safety recommendations approved by the International Commission on Radiation Protection (ICRP).¹⁶ It is, however, dependent on the findings for blood disease being an artifact and, more importantly, on a survivor population, which began by being strongly biased in

favour of exceptionally healthy individuals, losing this advantage in under five years.

Early effects of the bombing

Death from hitherto unknown syndromes, all caused by extensive destruction by the radiation, of bone marrow and other internal organs, and heralded by the now classic “radiation sickness” symptoms, was the fate of many who had either survived more immediate effects of the bombing—for example, blast injuries and radiation burns—or had no evidence of radiation effects apart from the sickness.¹⁷ Therefore, over and above the usual reasons why all survivor populations are necessarily biased in favour of exceptionally healthy people (survival of the fittest or healthy survivor effect), there was a brand new reason exerting direct effects on general haemopoiesis and the immune system.

Both the usual and the exceptional components of the healthy survivor effect were strongly dose-related. Therefore the question we should be asking ourselves is why the change from the fantastically high death rate of the first six months to the near normal rate of the 1950s was not accompanied by a change from a positive to a negative association with dose (table 1). The high dose levels of the life span study population (assembled in October 1950) were the ones most strongly biased in favour of people highly resistant to diseases and injuries in August 1945. Therefore, in the normal course of events we would have expected them to have much the lowest death rates. This reasonable expectation was, in fact, fulfilled for one cause of death—namely, suicide. Therefore there is a subsidiary as well as a main problem—namely, why deaths from self-inflicted injuries differed from other accidental deaths.

Though early deaths of first-day survivors were numbered in thousands, many eventually recovered from their injuries or illnesses with or without obvious residual disabilities.¹⁷ So it is inconceivable that no member of the life span study population had any residual disabilities and possible that the less obvious ones included incomplete repair of bone marrow damage and consequent effects on infection sensitivity and blood diseases.

Net effect of two opposing forces

According to this hypothesis some cancellation of the healthy survivor effect by residual disabilities is an inevitable consequence of all natural and man-made disasters. In the case of Hiroshima and Nagasaki the addition of marrow damage to more obvious disabilities both prevented full expression of mutational effects of the radiation (either cancers or inherited defects) and left the non-cancer death rate reflecting almost equal pressures by two opposing forces—namely, selection in favour of exceptionally fit individuals and incomplete healing of blast injuries and tissue-destructive effects of the radiation.

On these terms all dose-level comparisons would require some control of the healthy survivor effect and, without this, one would be left with a (false) impression of no mutational effects at low dose levels. Furthermore, given near equality of the two opposing forces, it would be easy to overlook certain possibilities. For example, the temporary absence of a dose-related death rate for solid tumours could be due to extra, radiogenic cases (whose latency periods had been shortened by the bone marrow damage),

falling into gaps created by shortages of normal cancers or cases initiated before August 1945, which therefore became merged with all the other causes of extra death during the period of heavy acute mortality. Likewise, the early epidemic of myeloid leukaemia could be a direct consequence of bone marrow damage and therefore something that would not be expected to follow exposure to worker doses or background radiation.

Residual bone marrow damage

Evidence for or against there being residual bone marrow damage would require careful testing of specific hypotheses. Therefore there is little of value to be found in reports that have never envisaged this possibility. Beebe *et al*¹⁵ however, after completing their relative risk analysis of 1950–74 deaths, did proceed to test a new hypothesis that had grown out of experimental work. The hypothesis was that exposure to ionising radiation accelerates the natural aging process.¹⁵ and the test by Beebe *et al* was prefaced with the following statement: “In this report we examine the mortality experience of the A-bomb survivors more closely than has been done before, using recent tabulated information on deaths through September 1974, and explore the question of bias from the heavy acute mortality.”

In practice evidence of the postulated effect of the radiation was sought in a series of dose-level comparisons (with national statistics setting the standards of normality) after exclusion, firstly, of all deaths from neoplasms and injuries and, later, of deaths from blood diseases other than leukaemia.

Table 2 SMR analysis of 1950–74 deaths for the purpose of testing the hypothesis of radiation accelerated aging (from Beebe *et al*)¹⁴

Dose level (rads)	Standardised mortality ratios						Total excluding blood	At risk No	%
	Tuberculosis	Cerebro-vascular	Other cardio-vascular	Digestive	Blood	Residue			
Hiroshima									
0	94	76	89	98	134	88	84	29 943	49.5
1–9	111	75	89	88	153	87	83	13 796	22.8
10–49	122	72	84	97	226	78	80	10 761	17.8
50–99	99	79	105	94	240	79	85	2 718	4.5
100+	97	80	92	113	460	91	88	3 252	9.8
Total	103	75	89	94	178	86	83	60 470	100.0
Nagasaki									
0	150	93	105	104	148	123	107	4 700	24.4
1–9	171	98	102	122	101	112	108	6 706	34.8
10–49	188	98	115	102	35	103	107	3 759	19.5
50–99	106	84	130	102	102	85	95	1 314	6.8
100+	146	90	110	108	550	109	104	2 787	21.3
Total	160	95	110	105	162	110	106	19 266	100.0
No of deaths									
Hiroshima	753	3355	2261	1238	94	3524	11 131	11 225	77.9
Nagasaki	336	925	604	328	23	964	3 157	3 180	22.1
Total No	1089	4280	2865	1566	117	4488	14 288	14 405	100.0
%	7.5	29.7	19.9	10.9	0.8	31.2	99.2		100.0

The later exclusions were made after division of diseases other than neoplasms into six diagnostic groups had produced five sets of what were deemed to be negative findings in a mortality analysis that recognised two groups of survivors (Hiroshima and Nagasaki) and five dose levels (table 2). For the group as a whole there was unmistakable evidence of a rising trend of mortality in Hiroshima but not Nagasaki, also evidence of exceptionally low SMRs in the oldest age group (over 50 years in 1945).

Finally, in their discussion of these findings and other reports, Beebe *et al* admit that with mortality "falling rapidly with increasing distance from the hypocentre, selection is an undubitable fact not an issue." They made no attempt to control for this factor, however, and ignored a second axiom—namely, that after extensive injuries some permanent crippling is a fact not an issue. In short, as a way of testing the only new hypothesis for many years, Beebe and his associates have used their original method without control for any new factors and without even a passing thought for their own exceptional findings for suicide (table 1).

In a survivor population sudden deaths would have less chance of being influenced by residual disabilities than other modes of dying and, if the disabilities included bone marrow damage, there could be a substantial difference between sudden deaths due to violence and later deaths from less overwhelming injuries. It is unfortunate that the decision not to include injuries in the test of the new hypothesis has left us without any SMRs for deaths from self-inflicted injuries. Nevertheless, there is much of interest to be found in table 2.

Thus for the five large groups of non-cancer deaths the rates were consistently lower for survivors at Hiroshima, who included under 10% of those whose doses were high enough to cause tissue damage (over 100 rads), than survivors at Nagasaki with over 20% of such people. Furthermore, in both cities the group of non-cancers with the highest proportion of sudden deaths (cerebrovascular accidents) had exceptionally low SMRs; the group most directly concerned with general haemopoiesis (blood diseases) had much the highest ratios, and the only identifiable infection (tuberculosis) had ratios that were well above average, also much higher for Nagasaki than Hiroshima survivors.

City differences

The fact that survivors at Hiroshima have always had lower death rates than those at Nagasaki is supposed to result from unidentified socioeconomic factors.¹³ The differences in table 2, however, are suggestive of stronger opposition to the healthy worker effect by

residual disabilities in Nagasaki than Hiroshima. In favour of this hypothesis are, firstly, the higher dose levels and, secondly, although there were more sudden deaths after the first than the second bomb, the August to December death rate for first-day survivors was much higher in Nagasaki than Hiroshima (table 3). Therefore, from the point of view of delayed effects of the radiation, it is probably of more importance that the first bomb exploded over a delta and the second bomb over a narrow valley surrounded by hills, than that the two bombs were not exactly alike.¹⁸

Blood diseases

The group of blood diseases, with aplastic anaemia as the main component, is the only diagnostic group in table 2 with a raised and dose-related death rate. This has always been so,¹¹ yet we are asked to believe that all exceptional findings for blood diseases other than leukaemia are the result of misdiagnoses.¹⁵ In support of this hypothesis is the fact that a review of 59 cases by the leukaemia registry in Hiroshima led to 13 cases being reclassified as leukaemia (9) or other cancers (4). The rejected cases had relatively high doses. Nevertheless, their exclusion still left more observed than expected deaths and the same dose-level ranking of SMRs (table 4). Table 4 also shows that applying similar "corrections" to the remaining 58 cases still left evidence of a dose-related effect.

These findings make it impossible to agree with Beebe *et al*¹⁵ that "a superficial association between mortality from diseases of blood and blood forming organs and radiation rests entirely on the carcinogenic effects of radiation, mainly the leukaemogenic effect." This statement, like so many conclusions of ABCC reports, is indicative of an obsession with possible mutational effects of ionising radiation to the exclusion of all else. Yet it was widely known that the threshold dose for bone marrow damage was relatively low and that aplastic anaemia is the most easily recognised effect of such damage.

Table 3 *Early effects of the bombing: differences between the two cities (from Ohkita⁷)*

Early effects of bombing	Hiroshima		Nagasaki	
	No	% *	No	%
First day				
Fatal injuries	45 000	17.6	22 000	12.7
Other injuries	91 000	35.7	42 000	24.1
Not injured	119 000	46.7	110 000	63.2
Total	255 000	100.0	174 000	100.0
Next four months				
Fatal injuries	19 000	20.9	17 000	40.5
Other injuries	72 000	79.1	25 000	59.5
Total	91 000	100.0	42 000	100.0
1950 Census population of survivors			285 000	

Table 4 *SMR analysis of blood disease deaths: effect of removing suspect cases (from 8th Mortality Report^a and Beebe *et al*^b)*

Diseases of blood and blood-forming organs	Dose level (rads)	Original notifications			Revised diagnosis		
		Observed	Expected	SMR	Observed	Expected	SMR
Leukaemia registry cases	0-9	30	22.9	131	29	22.9	127
	10-99	15	7.3	205	12	7.3	164
	≥100	14	2.3	609	5	2.3	217
	Total	59	32.5	182	46	32.5	142
Other cases	0-9	33	22.6	146	32	22.6	142
	10-99	15	7.1	211	12	7.1	169
	≥100	10	2.3	435	4	2.3	174
	Total	58	32.0	181	48	32.0	150

Estimates of radiation effects

If the mortality experiences of A-bomb survivors are being influenced by a healthy worker effect and residual disabilities neither comparisons with national statistics or with a zero dose level will provide the correct standard for estimating radiation effects. What is needed is a cause of death which (a) is not affected by one or other of the opposing forces, and (b) can be compared with national statistics. The first requirement rules out everything except sudden deaths of previously healthy people, and the second rules out sudden deaths but leaves deaths from cerebrovascular accidents as a possible alternative. Unlike suicides, these deaths have never shown any signs of a negative correlation with dose, but for many years the rate for survivors at Hiroshima was about 30% below the national rate. Therefore it is reasonable to assume that as a result of the heavy acute mortality the risk of dying from natural causes was at least 30% below par for all five-year survivors ("correction factor" for the healthy survivor effect).

Since cancers and injuries were not included in the SMR analysis of 1950-74 deaths it is only possible to produce estimates of extra, radiogenic deaths for the period five to 27 years after the bombing (see 7th mortality report with observed and expected deaths for 1950-72¹³). During these years there were 18 526 deaths from all causes and 3744 from cancer. By national standards the corresponding expectations were 20 182 and 3283. Therefore, after correction for the healthy survivor effect, the ratio of observed to expected deaths was $3744 \div 2298$ or 1.62 for cancers and $14\ 748 \div 11\ 829$ or 1.25 for non-cancers. On these terms 1446 or 39% of the 1950-72 deaths from cancer were radiation-induced. For other deaths the corresponding estimates are 2953 or 20%, and for all deaths they are 4399 or 24%.

Included in the relative risk analysis of 1950-74 deaths are estimates of extra radiation deaths for all five-year survivors (roughly 285 000, see table 3). As usual they are based on dose level comparisons within the life span study population, and they assume linearity of dose response. On these terms the

only extra deaths were either leukaemias (192 cases) or solid tumours (223) and accompanying these 415 radiogenic deaths there were about 70 000 from natural causes. Therefore, even on the unlikely assumption that (a) there was no involvement of deaths from cerebrovascular accidents in residual disabilities, (b) there were no radiation-induced deaths after 1972, and (c) all extra deaths were contained within the life span study population, the revised estimates for extra radiogenic deaths are over 10 times higher than the ones currently approved by ICRP.¹⁶

Discussion

According to the "silent forces" hypothesis, the fact that the life span study population of A-bomb survivors has always recorded relatively low rates of general mortality is a direct consequence of the exceptionally high rates during the immediate aftermath of the explosions. During this period of devastation the risk of dying either from natural causes or special effects of the A-bomb must have been exceptionally high for anyone who, for whatever reason, was already in a poor state of health or nutrition. Among those who were eligible for inclusion in the study population of five-year survivors there must have been too few naturally weak individuals and too many naturally strong—that is, exceptionally healthy—ones and a general bias in favour of well-to-do families.

This effect of the bombing is admitted by Beebe *et al*,¹⁵ but neither they nor the authors of earlier reports have been prepared to admit that, given this inevitable bias, their findings can no longer be accepted at face value.

The present review has found even the firmest of earlier conclusions unacceptable, not only for conjectural reasons but also because the findings for blood diseases other than leukaemia are not adequately explained in terms of misdiagnosis and therefore require delayed effects of the radiation to include tissue damage as well as mutations. Furthermore, since the principal target of all

radiation effects (immediate or delayed) is bone marrow, there was, over and above the usual reasons for expecting a (dose-related) healthy survivor effect, a special factor—with direct effects on the immune system and general haemopoiesis—which was contributing both to the healthy survivor effect and to residual disabilities.

This brings us to the nub of the problem: can the straightforward analyses of dose-related death rates—which are the basis of current beliefs about the health risks of radiation workers—be regarded as robust under the conditions that undoubtedly existed. On reflection it is easy to see that they are not. For example, the idea that an increased prevalence of myeloid leukaemia will necessarily be the first sign of any ill effects from radiation-induced mutations makes no allowance for the possibility that, in the case of A-bomb survivors, bone marrow damage both aided rapid development of post-bomb cancer inductions and added to an already high risk of latency deaths for earlier inductions. Therefore, the fact that the 1950–8 mortality rates for most forms of cancer were not dose-related is important, since this is exactly the finding one would expect if most of the extra, radiogenic cases were filling gaps caused by pre-1950 (latency) deaths of non-radiogenic cancers. In favour of this interpretation of the data is evidence from a survey of childhood cancers which suggests that defects of the immune system are concomitants of the cancer process and have often been the cause of latency deaths ascribed to respiratory infections.¹⁹

In the Hanford study the risk of dying from natural causes was found to be much lower for the workers than for the nation as a whole, also lower for workers in high- than low-risk occupations.^{6,9} Unlike the healthy survivor effect, however, whose dose-related consequences were caused by the radiation, the healthy worker effect (also dose-related) predated all exposures and was clearly the result of deliberate recruitment of exceptionally healthy people into the more dangerous jobs.⁶ A healthy worker effect is common to many industries.²⁰ Therefore detection of mutational effects of radiation should be much easier in a worker than a survivor population. This has proved to be so, and the nature of the most distinctive effect of the worker doses (multiple myeloma) suggests that we are right to assume that bone marrow is exceptionally sensitive to cancer induction by radiation but wrong to assume that without bone marrow damage the resulting cancers will be myeloid leukaemias.

Thus far only a small fraction of the data relating to workers in the nuclear industry has been examined for evidence of radiation effects, and none has yet been examined for evidence of second or third generation effects. There is no shortage, however, of

radiation worker dose records, and where they are most plentiful (in the United States) there are also unusually good opportunities for relating the data both to death certificates of ex-workers (through Social Security claims) and to medical records of mentally or physically defective offspring of workers (through the same system). Therefore research workers should be dissuaded from taking the short cut advocated by Land² (since this requires extrapolation of high-dose observations to low-dose levels) and encouraged to make full use of data already collected during the course of a special study—that is, a study of the lifetime health and mortality experiences of employees of AEC Contractors)²¹ which was, incidentally, the source of the Hanford data.

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