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LATE EFFECTS ON A POPULATION EXPOSED TO ACTIVITY RELEASED INTO THE ENVIRONMENT

P Kay and J A Reissland

ABSTRACT

The long-term effects resulting from an accidental release of radioactive material are considered. The magnitude of the release is defined in terms of the percentage of total excess cancer mortalities relative to the number that would have been expected in the same population had the accident not occurred. The increased risk is expressed as the change in life expectancy as a function of age at the time of exposure. Loss of life-years per person are calculated for an increase of 0.1%, 1%, 10% and 100% in cancer deaths and are quoted for ages 1, 10, 20, 30, 40 and 50 years at the time of exposure. The variation of the effect with age may be summarised using the ratio of the reduced life expectancy to the reduced life expectancy for a person aged zero at the time of the accident. For the initial external dose, this ratio exhibits a linear decrease up to about 45 years at the rate of about 2% per year so, for example, the increased risk for a person aged 40 years at exposure is 20% of the increased risk of a child just born.

Similar estimates are made for a dose which is prolonged over 50 years, to represent doses from internally incorporated radionuclides.

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1. INTRODUCTION

In the event of a release of radioactive material into the environment, the population in the path of the plume will be subjected to an increased risk of death from certain causes. This report describes a procedure for assessing the long-term effects of the accident by estimating the number of excess cancer deaths which are induced in the exposed population by the dose of radiation they receive. The dose experienced consists of two components; an external dose from the radioactivity of the passing cloud and the material deposited, and a protracted internal dose from material inhaled and ingested. Here we shall consider the effects of the external dose accumulated during the year following the accident and, separately, the effects of the full dose commitment represented by the dose versus time relationship shown in Figures 1 and 2. Figure 1 is representative of the annual dose to lung and Figure 2 to bone marrow (the latter being appropriate to estimate induced leukaemias). Both correspond to the exposure of a person 1 km from the point of release. Also considered is a uniform dose over 50 years as the other extreme to the external dose all in the first year. By comparison of these two, the effects of internal and external dose can be separated (for the long-lived nuclides).

In calculations for radiological protection purposes, it is necessary to estimate the total (or average) effect and to identify the section of the community which incurs the greatest risk. This report is concerned with relative risk for different age groups and, in the following, age normally means age at the time of the release. The population at risk is assumed to have an age distribution (men, women and children) similar to the population of England and Wales and the home population statistics contained in the Registrar General's Review for 1973 have been used (Figure 3). Differences in the standard mortality rates for males and females are not taken into account, all properties being averaged over the two sexes.

In making the estimates reported here, the level of risk is chosen to produce a predetermined number of excess deaths defined as a percentage of the number of cancer deaths that would have been expected without the accident. Thus, the product of the dose (in rads) and the risk coefficient (the number of radiation-induced cancer deaths per rad) is adjusted by an iterative procedure until the required number is predicted. Then the corresponding age distribution of the effect of the accident can be extracted and is expressed here in terms of effective life shortening per person of a given age due to induced cancer deaths lowering their expected average age at death.

The concept of reduced life expectancy has been chosen to describe the effect in preference to numbers of cancer deaths. The latter is dependent on the population which is exposed and, hence, this must be specified. Reduced life expectancy may be quoted for a person of specified age and is quantitatively more meaningful than increased probability of dying of cancer. It is also a convenient measure of the impact of the accident on a population.

The results presented in Section 4 demonstrate the relative risk of the young and old and indicate the possibility of simplifying approximations in estimating population effects. These deductions are dependent on the assumptions made in the modelling described in the following two sections.

2. NATURAL MALIGNANCIES

Figure 4 shows the distribution of deaths from all causes which have been used for the natural deaths of the population. Figures 5 and 6 (dashed lines) show the corresponding distribution for lung cancer deaths and for leukaemia, respectively. These all derive from death rates given by the Registrar General (1973). These rates are assumed to have remained applicable every year following exposure. Data from the same source have been used to evaluate the expected number of cancer mortalities in the exposed population. This number is used as the reference for the number of radiation-induced deaths. It should be emphasised that this is the number expected without the accident; when the accident is taken into account there will be less non-radiation-induced cancers because some cancer deaths which would have occurred will be pre-empted by a radiation-induced death. In the following discussion the term 'percentage excess' of cancers means the number of radiation-induced cancers (as a result of the accident) expressed as a percentage of the number of cancer deaths that would have been expected had the accident not occurred. Thus, the percentage excess cancers is a measure of the magnitude of the accident.

The death rates corresponding to Figure 4 may be used to construct a survival curve showing the probability that a person of a given age will still be alive as a function of time in the future. These are shown for various ages in Section 4.

3. RADIATION-INDUCED MALIGNANCIES

The period of risk following exposure to ionising radiation is not well established. It is reasonably well agreed that there will be a latent period of zero risk after exposure followed by a finite risk extending over some considerable time. The literature does not contain adequate data to predict the form of this time variation. Some guidance in making a choice is available from the times of appearance of cancers and leukaemias among the survivors of the Japanese atomic bombs. The data suggest that about 90% of the deaths due to radiation-induced leukaemia had occurred by 1972. These did not begin to appear for at least 5 years after the bomb but they reached a peak quickly and thereafter decreased steadily. The negative slope has not been included since it is partially accounted for by the underestimating of radiation-induced leukaemias due to deaths from other causes. In order to generate over-estimates rather than underestimates, a rectangular time variation has been taken, as described below. Significant numbers of induced deaths due to other cancers appeared later than the leukaemias and the data suggest that by 1972 perhaps half of them had occurred. For these the increase had been slower but it was clear that the effects extended over much greater times following exposure. For reasons similar to those for the leukaemia deaths, the time variation was taken to be rectangular but starting later and lasting longer. The simple models shown in Figure 7 have been used; for all cancers a latent period of 15 years followed by uniform risk for 30 years (dashed line); for leukaemia a 5-year latent period and finite risk extending up to 25 years after exposure.

As discussed in the previous section, separate values for the risk coefficient and the dose are not relevant; however, their product is uniquely determined by the excess deaths and hence, for a given risk coefficient, the dose necessary to produce the effect can be quoted. To give an indication of the magnitude of the accident which will cause the effect, the average doses necessary are quoted assuming a risk coefficient of 10^{-4} radiation-induced cancer deaths per rad averaged over a standard population (this is based on observations on Japanese atomic bomb survivors (Goss, 1974)). For example, to produce 100% excess cancer (see Section 2, ie, to double the risk of dying through cancer) requires an external dose during the year following the accident of 3500 rads per person. If the dose is accumulated uniformly over a 50-year period the dose required to double the risk is 7500 rads (received by a person who survives for 50 years after the accident).

The probability (normalised to unity) that a radiation-induced lung cancer death will occur at a particular age is shown in Figure 5 (solid line) along with the corresponding curve for the natural incidence (dashed line). This histogram has been calculated for the 100% excess cancer accident using the dose-time relationship as in Figure 1 and the risk-time relationship as in Figure 7 (dashed line). A clear difference in the age distribution is demonstrated: for example, a person aged under 25 years at exposure has an order of magnitude increase in risk of cancer death at 40 years compared with the risk had the accident not occurred. However, this must be taken in perspective with the absolute risk of dying due to cancer at 40 years, viz. about 5 deaths per 10,000 persons per year. The same comparison is made in Figure 6 for leukaemias. It is seen here that the radiation-induced leukaemias appear earlier on average than the lung cancers shown in Figure 5, although the peak for lung cancers occurs at a lower age (45-50 years compared with 55-60 years for leukaemias). This happens because the lung cancers are considerably more numerous and influence the population significantly. Figure 6 also shows the clear difference in the age distribution of induced and spontaneous leukaemia deaths. Probability of death from leukaemia at age 40 (for a person under 35 years at the time of the accident) is about five times greater than if the release of activity had not occurred; the spontaneous rate at age 40 is just under 3 leukaemia deaths per 100,000 persons per year. It should be remarked that although a standard population of men, women and children has been worked with, mortalities due to lung cancer (without the radiation exposure) are very unevenly distributed between the sexes, male deaths due to lung cancer being a factor of 5 greater than in females. The radiation-induced lung cancers are expected to be evenly distributed between the sexes. Thus, since population averages were used to define the accident, women will experience an increased risk relative to the no-accident situation, which is greater than that for men. This is not so for leukaemias, which are more evenly distributed.

4. AGE VARIATION OF INCREASED RISK

The effect of the accident is assessed as modified life expectancy. The life expectancy of a person aged i years is defined as

$$L(i) = (n_i + n_{i+1} + n_{i+2} + \dots)/n_i \qquad \dots\dots\dots\dots (1)$$

where n_i is the number of people aged i,

n_{i+1} is the number out of the original n_i still alive 1 year later (ie, who survive until i+1),

n_{i+2} is the number who survive for 2 years, etc.

Life expectancy predicts a mean age of death (L(i) + i for a group of given age i) which increases with the age of the group. The life expectancy, L(i), may be calculated with ($L_R(i)$) and without ($L_N(i)$) the accident and the effect of the accident expressed as the difference, $L_N(i) - L_R(i)$ ($\equiv \Delta L(i)$).

The decrease in life expectancy, $\Delta L(i)$, has been calculated for all i up to 80 years and for accidents corresponding to 0.1%, 1%, 10% and 100% excess, ie, radiation-induced deaths (relative to the number of cancer deaths expected without the accident, as described in Section 2).

Considering first the case of external dose accumulated during the year following the accident, Table 1 shows $\Delta L(i)$ for sample ages and Figure 8 shows the ratio, $\Delta L(i)/\Delta L(o)$, for four magnitudes of accident falling closely on the same curve. From these results (solid line) it can be seen that the effect decreases linearly with age at a rate of about 2.1% per year up to about the age of 45. Beyond that the risk flattens to zero, being identically zero at 80 years, since this model assumes a 15-year latency and everybody dying before 95 years. It is seen also from Table 1 that the effect measured as reduced life expectancy is closely proportional to the excess death rate at all ages shown. Thus, using a figure of 13 years' reduced life expectancy for a newborn child for an accident which produces 100% excess cancer deaths, together with a 2.1% decrease per year 'age at exposure', enables estimates corresponding to any accident and any age to be made. That is, for an accident which results in x% excess cancer deaths, the reduced life

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expectancy of a person aged i is

$$\Delta L(i) = 13(1 - 0.021i)\frac{x}{100} \qquad \dots\dots\dots\dots\dots\dots\dots\dots (2)$$

Figure 8 also shows the corresponding curve for a protracted dose (dashed line). This curve is always below the acute dose curve and is well fitted by the quadratic

$$\Delta L(i) = 12.3(1 - 0.0356i + 0.000323i^2)\frac{x}{100} \qquad \dots\dots\dots (3)$$

Figure 9 shows the survival curves for sample ages for the 100% case. Also shown (dotted line) for age 1 is the 10% case which is the only one distinguishable on the scale from the no-accident curve. The solid curves show the probability of surviving against years after the time of the accident and the broken lines show the same probability taking account of the accident.

The last column of Table 1 shows the reduced life expectancy corresponding to a dose accumulated uniformly over 50 years producing 100% excess cancers. The dose necessary is over twice that for the previous case of the accumulation all in the first year but a smaller loss of life-years is seen despite the same total number of radiation-induced deaths from cancer. Figure 10 compares the survival curves of the protracted dose (solid line) with the acute dose (dashed line) for ages 1 and 30. For age 1 at exposure the probability of survival for 54 years is greater for a protracted dose, but survival longer than this is less for a protracted dose. This is due to the full effects of the dose not being effective for the first 45 years. For a person aged 30 at exposure there is no cross-over since the average person aged 30 does not expect to live long enough to experience the full dose.

5. COMMENTS

The main objective of this work is to investigate the age distribution of the effects of exposing a standard population to radiation. There is no involvement in assumptions about the type of accident nor in its probability; the sole concern is with the distribution of an assumed total effect over the age at time of exposure. Intuitively, the greatest risk is expected to be incurred by the younger among the exposed since they have a greater probability of living long enough to experience the full long-term effects of their exposure. This is demonstrated by Table 1 and Figures 8, 9 and 10. The magnitude of the ratio of the risk between young and old is quite striking. Figure 8 shows, for example, that an exposed 40-year old incurs only 20% of the risk of a newborn baby.

We have chosen to express the effects of the accident in terms of years of life lost per person of given age. This is a quantity whose significance is easily grasped, as well as being population independent. This does not mean that all children aged 1 at the time of exposure will die the specified number of years earlier than they would have done had the accident not occurred. It means that during each year following exposure, some children who were 1 year old when the accident occurred will die of radiation-induced cancers (on this model none during the first 15 years following exposure or after 45 years). This results in a reduction of the total number of person life-years. This total is averaged over all children exposed at the age of 1 year to represent a reduced life expectancy.

The survival curves shown in Figures 9 and 10 illustrate the same differential risk. In addition, they show the relationship with the survival probabilities that would have existed had the accident not occurred. The curves show the probability of still being alive x years after the accident for sample ages. The area between the two curves for a given age is a measure of the impact of the accident on a person of that age at exposure; the greater the area the greater the impact. The curves are drawn for the largest accident considered, 100% excess cancers. Even 10% excess produces survival curves which are barely distinguishable from the no-accident situation. The 10% case for age 1 is shown in Figure 9 as a comparison and shows that, even for the most significant age, the radiation-induced curve is close to the natural curve.

There is a simple relationship between the reduction in life expectancy for different ages and for different magnitude accidents (measured by the percentage total excess cancers induced by the radiation). This is summarised by equation (2) in the previous section for dose received during the first year following the accident. For the dose spread over 50 years the ratios are less at all ages and fall on a smooth curve that is closely approximated by the quadratic equation (3).

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Table 1

The reduced life expectancy (equation (1)) for a series of accidental releases of radioactive material for persons aged i at the time of the release

Reduced life expectancy (years), ΔL(i)					
Excess deaths (see Section 2)	0.1%	1%	10%	100%	100% (uniform dose 50 years)
Age (i)					
1	0.013	0.13	1.3	12.9	12.3
10	0.010	0.10	1.0	10.3	8.6
20	0.007	0.07	0.74	7.49	5.2
30	0.005	0.05	0.47	4.87	2.7
40	0.003	0.03	0.25	2.66	1.1
50	0.001	0.01	0.10	1.11	0.35
Population mean (over all ages)	0.005	0.048	0.48	4.9	3.7
Equivalent dose/person based on risk coefficient of 10^{-4}/rad	3 rads	31 rads	307 rads	3515 rads	7442 rads

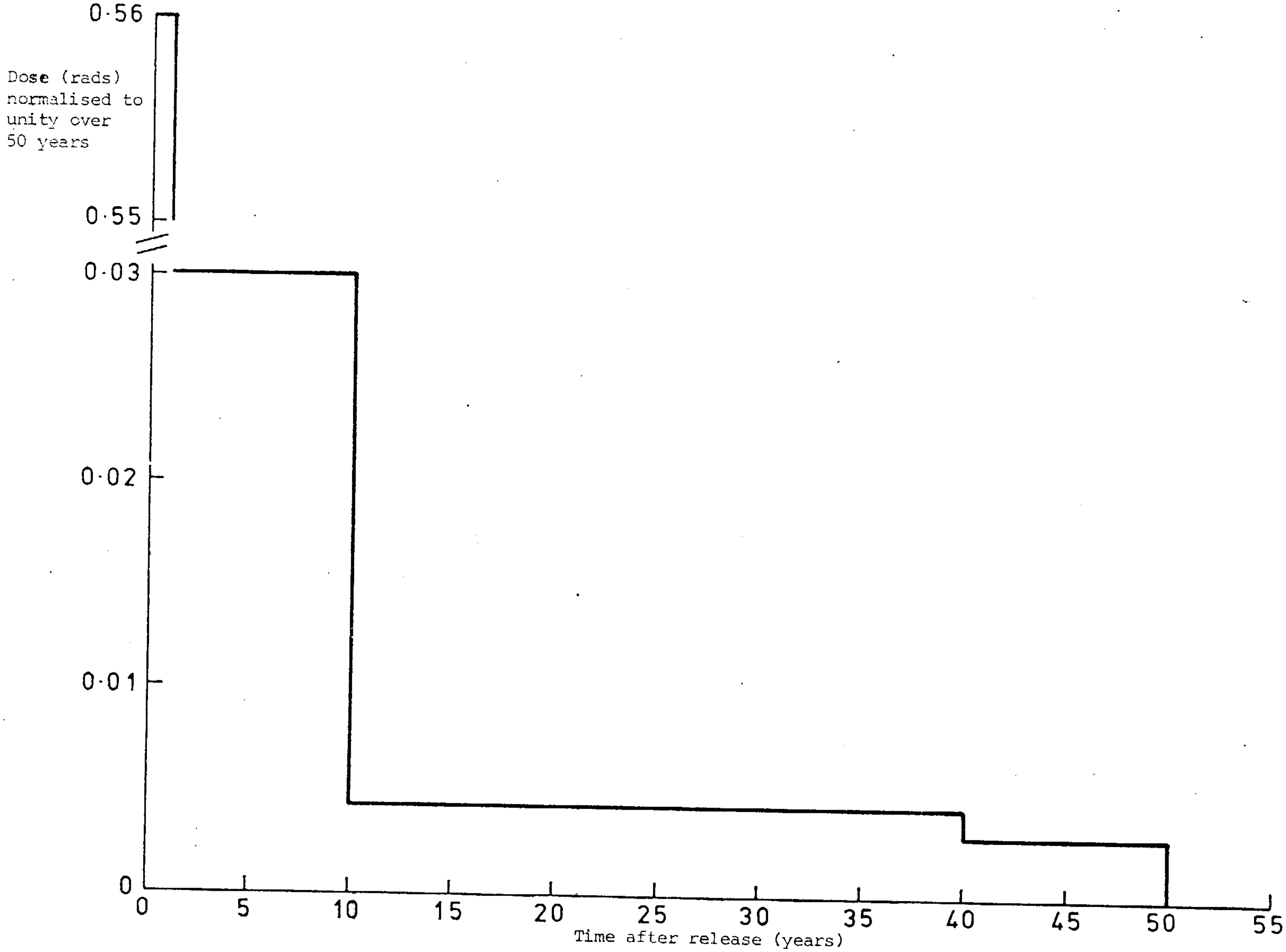

Figure 1. Annual dose to lung as a function of years after the release of activity (note the change of scale necessary to show both the short-term and protracted doses)

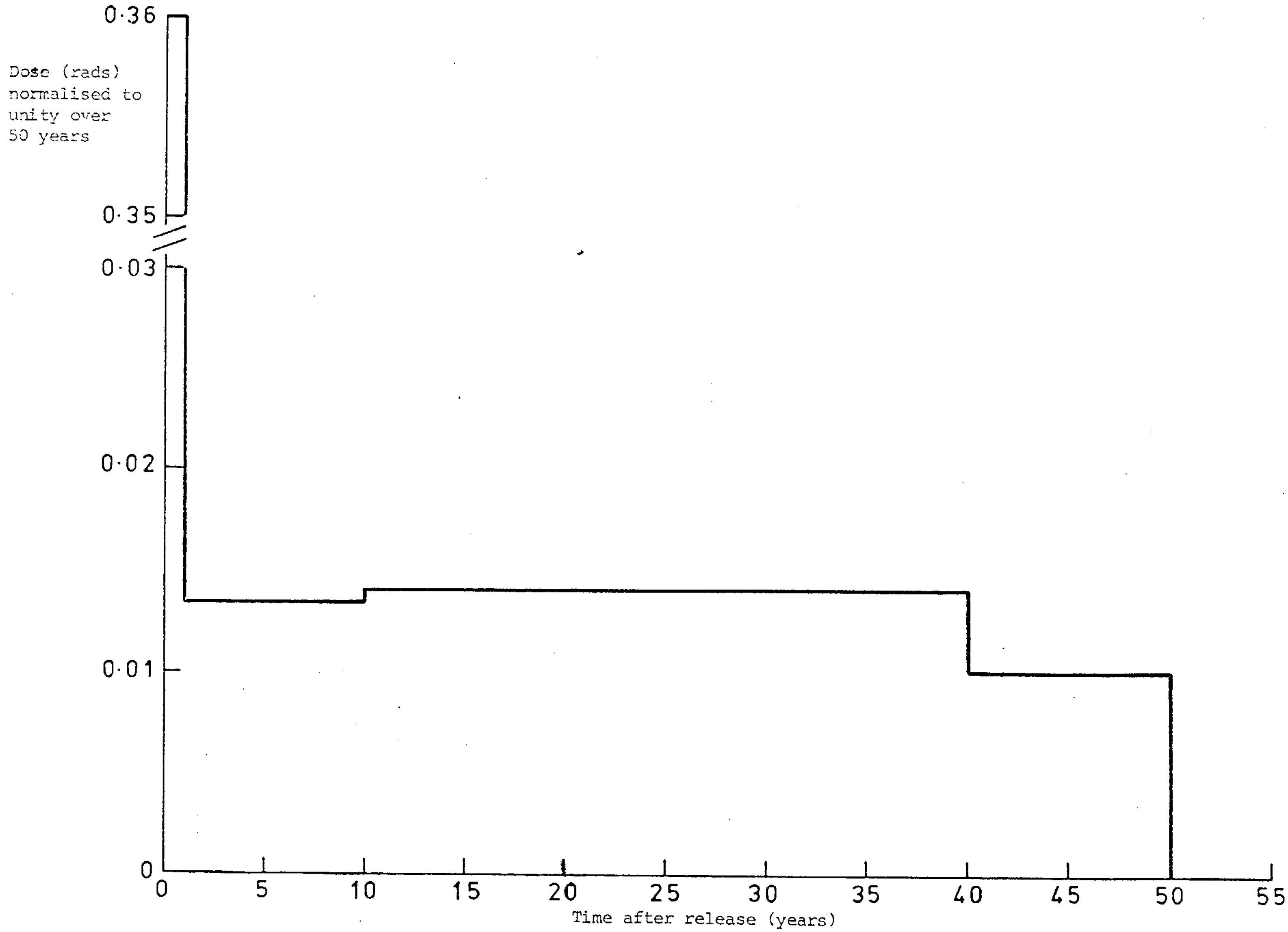

Figure 2. Annual dose to bone marrow as a function of years after the release of activity (note the change of scale necessary to show both short-term and protracted doses)

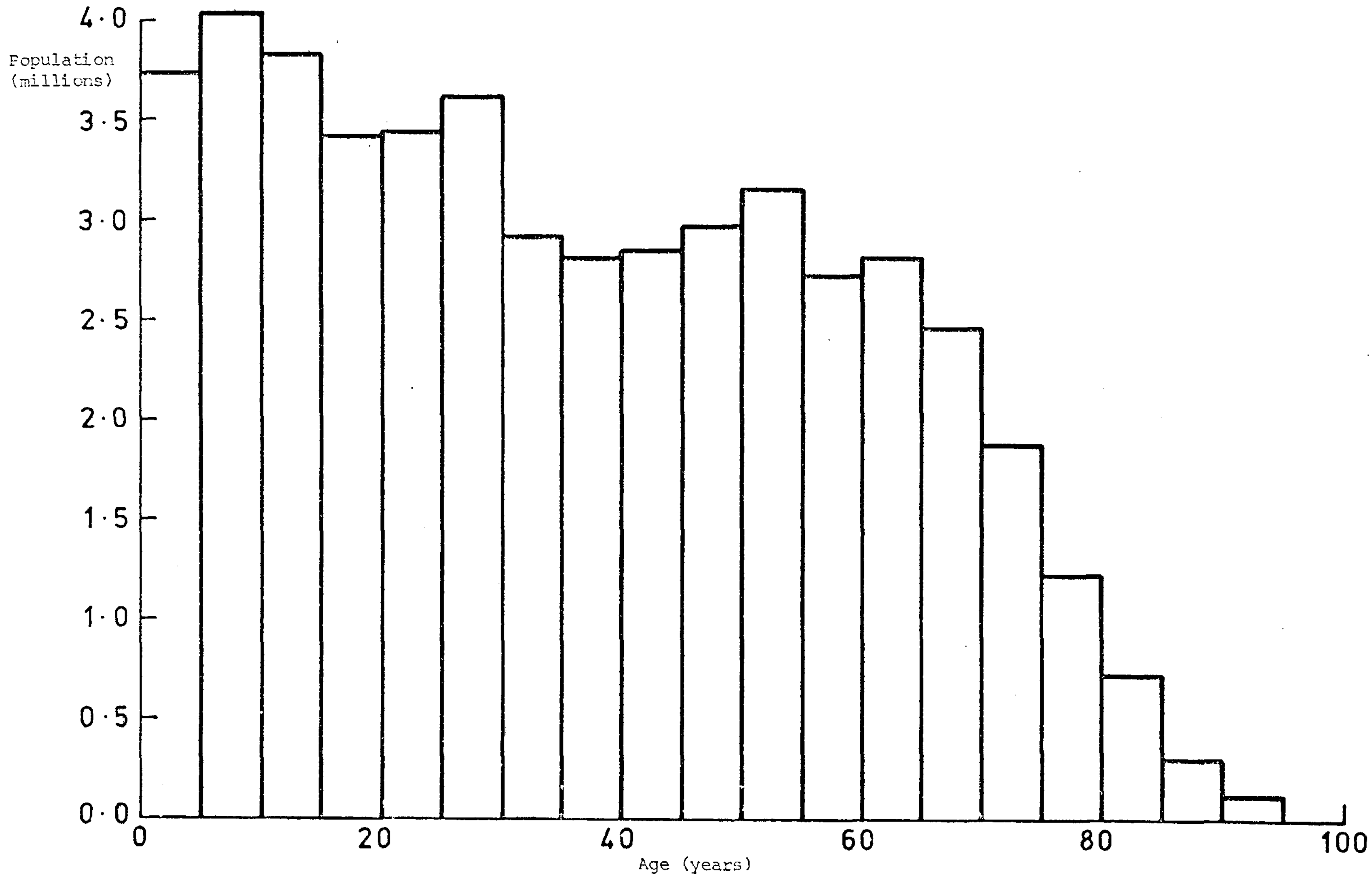

Figure 3. Estimated home population (1973)

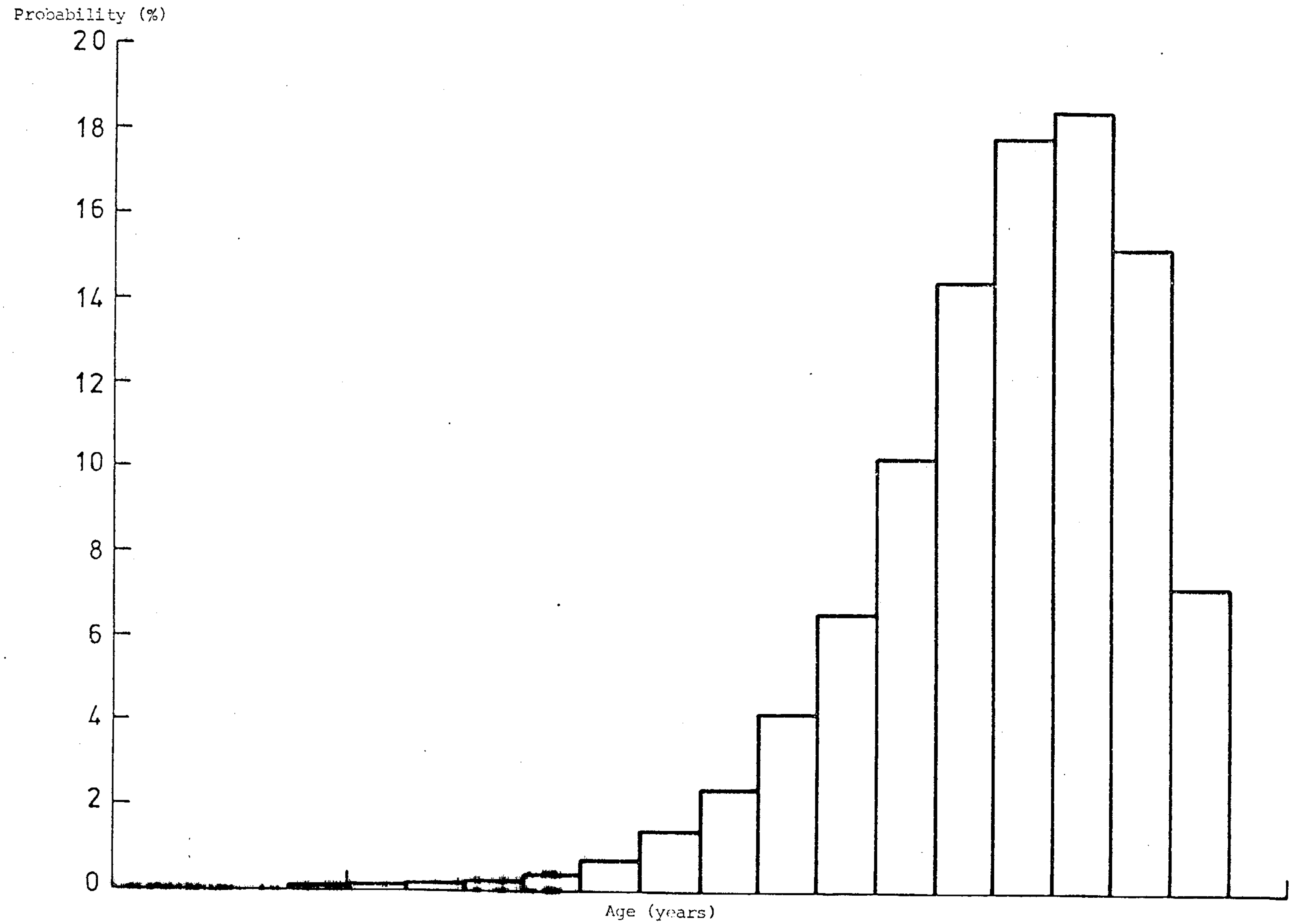

Figure 4. Probability of a death (all causes) occurring in a given age group

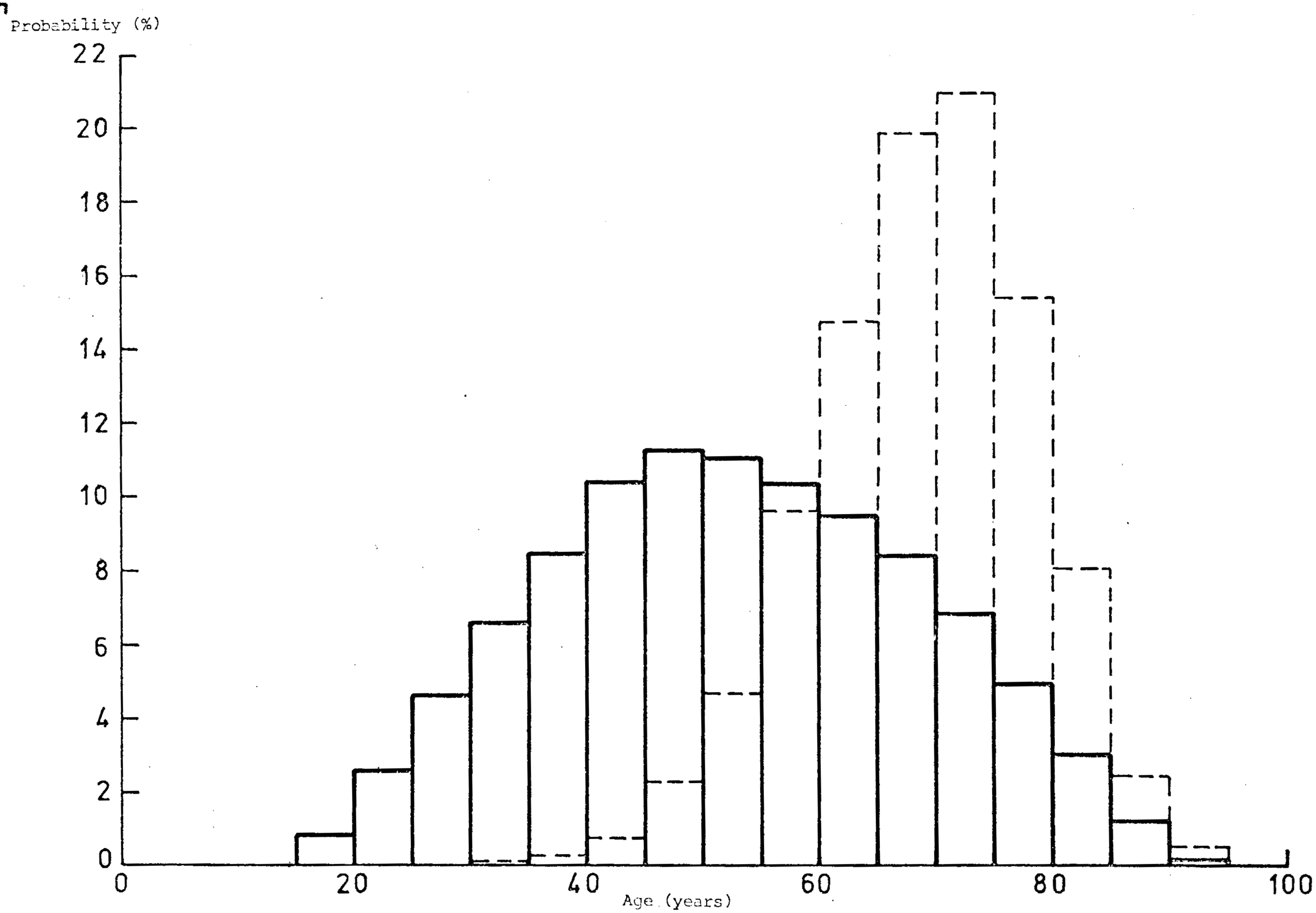

Figure 5. Comparative age distributions: deaths due to lung cancer, with and without the release of activity

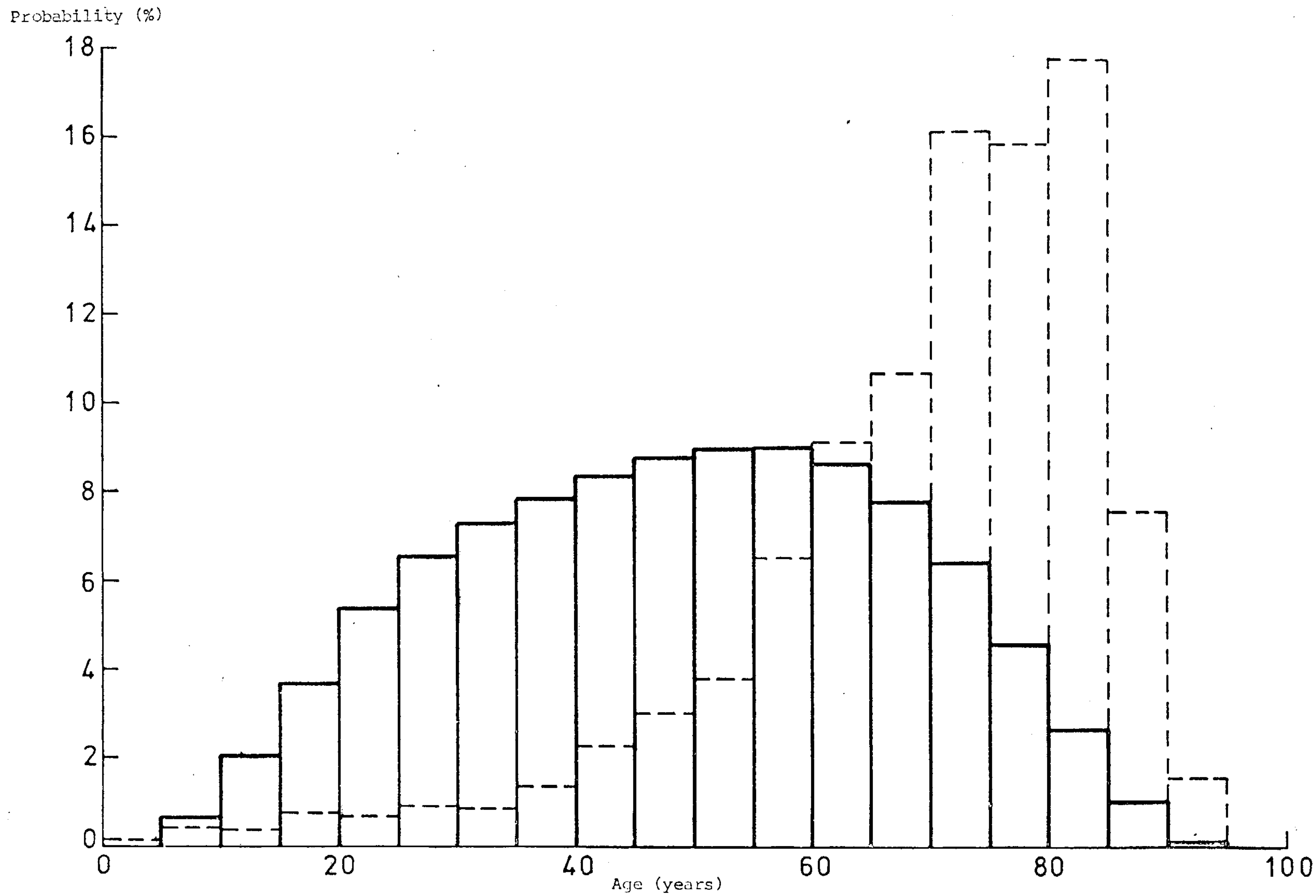

Figure 5. Comparative age distributions: deaths due to leukaemia, with and without the release of activity

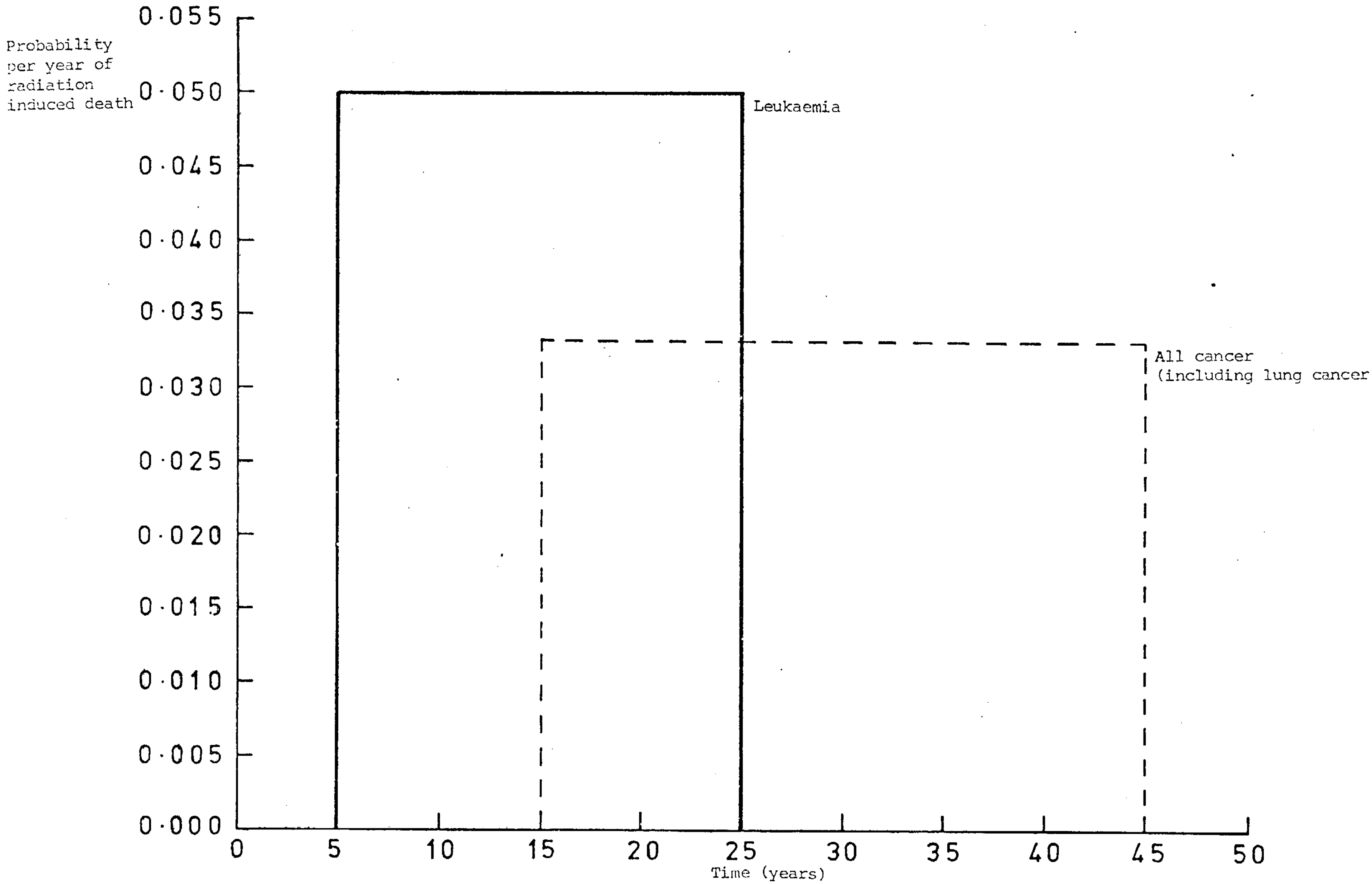

Figure 7. Risk-time relationship

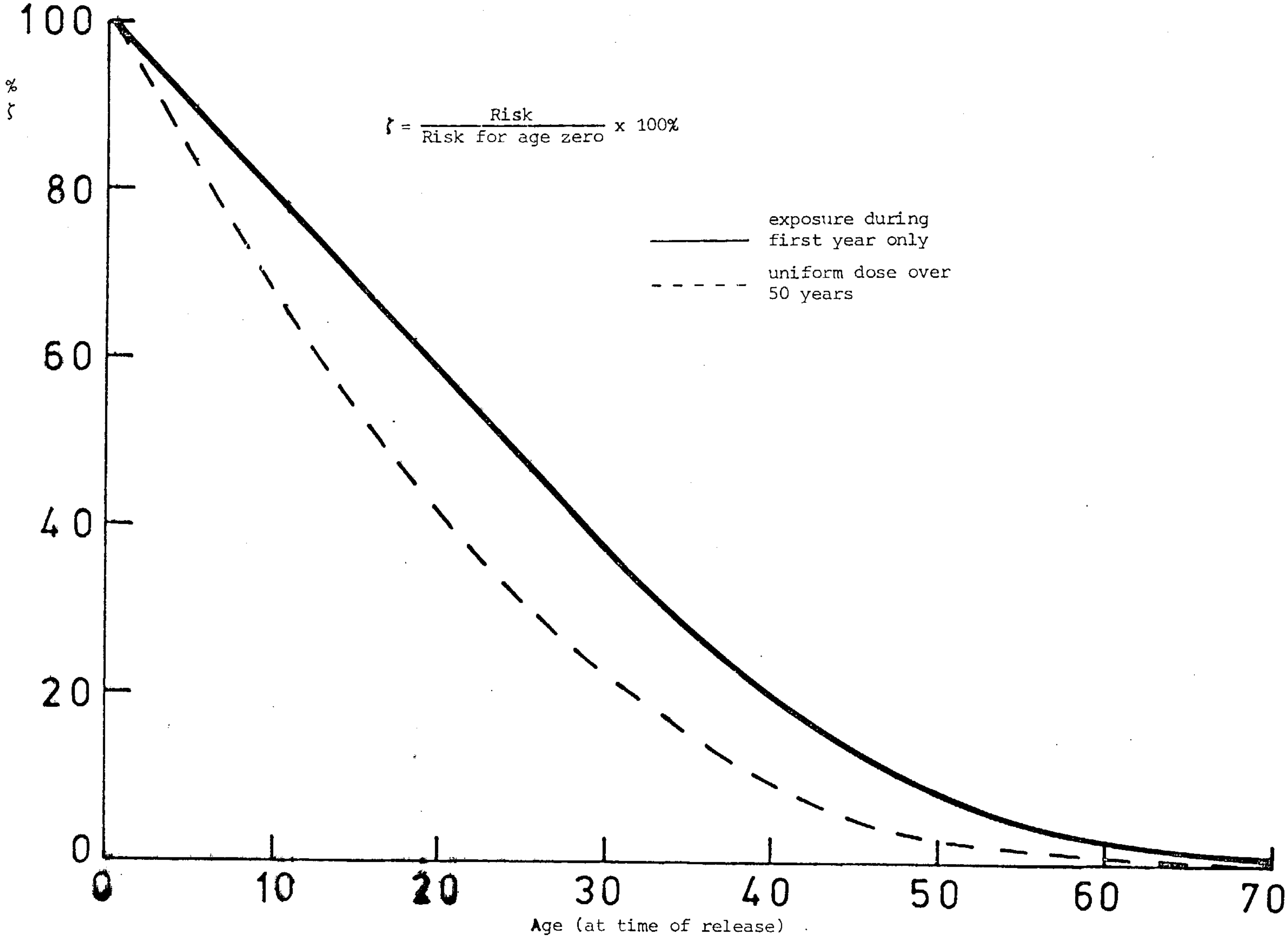

Figure 8. Comparative risk with age at time of the release

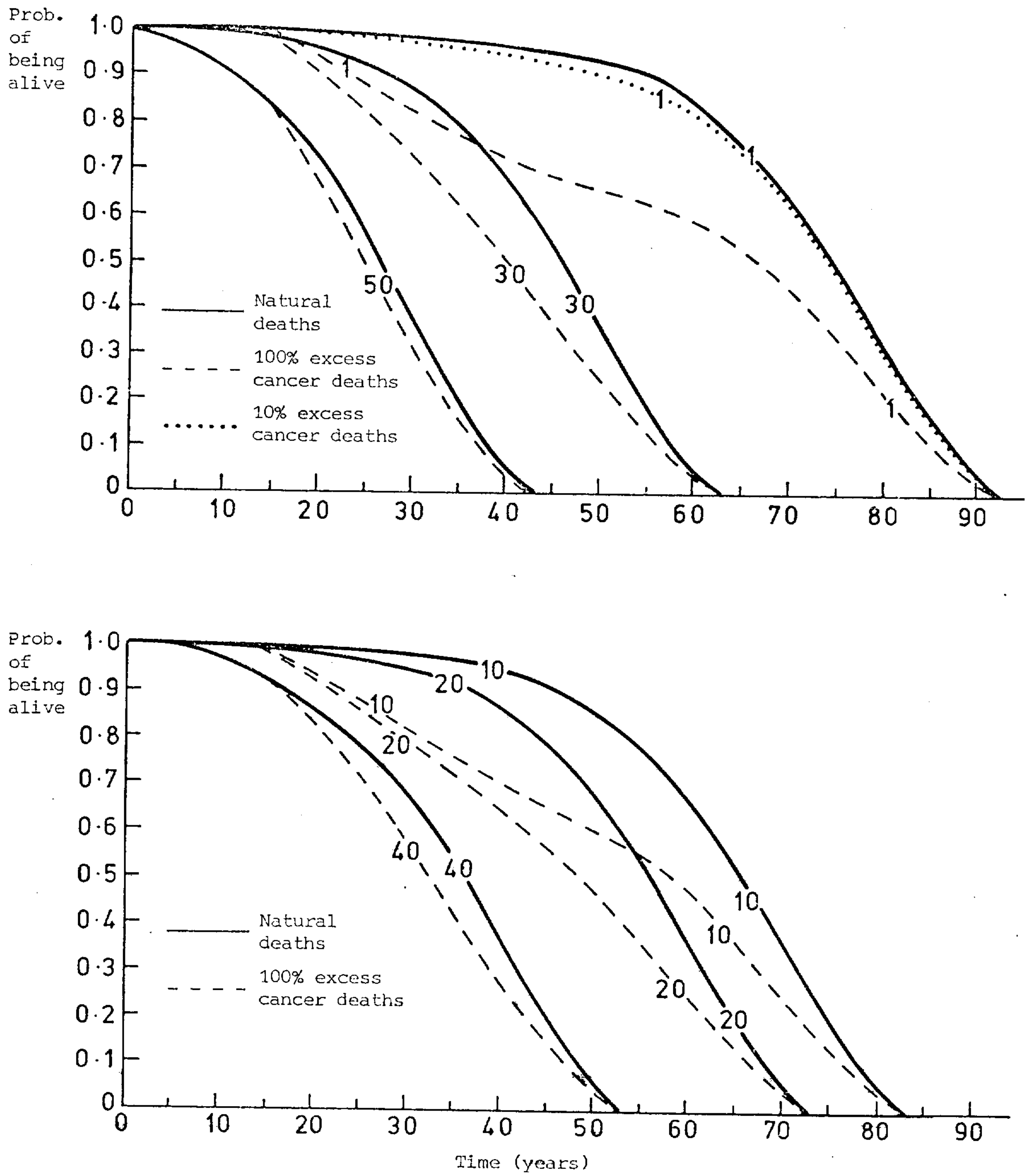

Figure 9. Survival probabilities comparing release and no-release situations. The numbers on the curves show age at time of release

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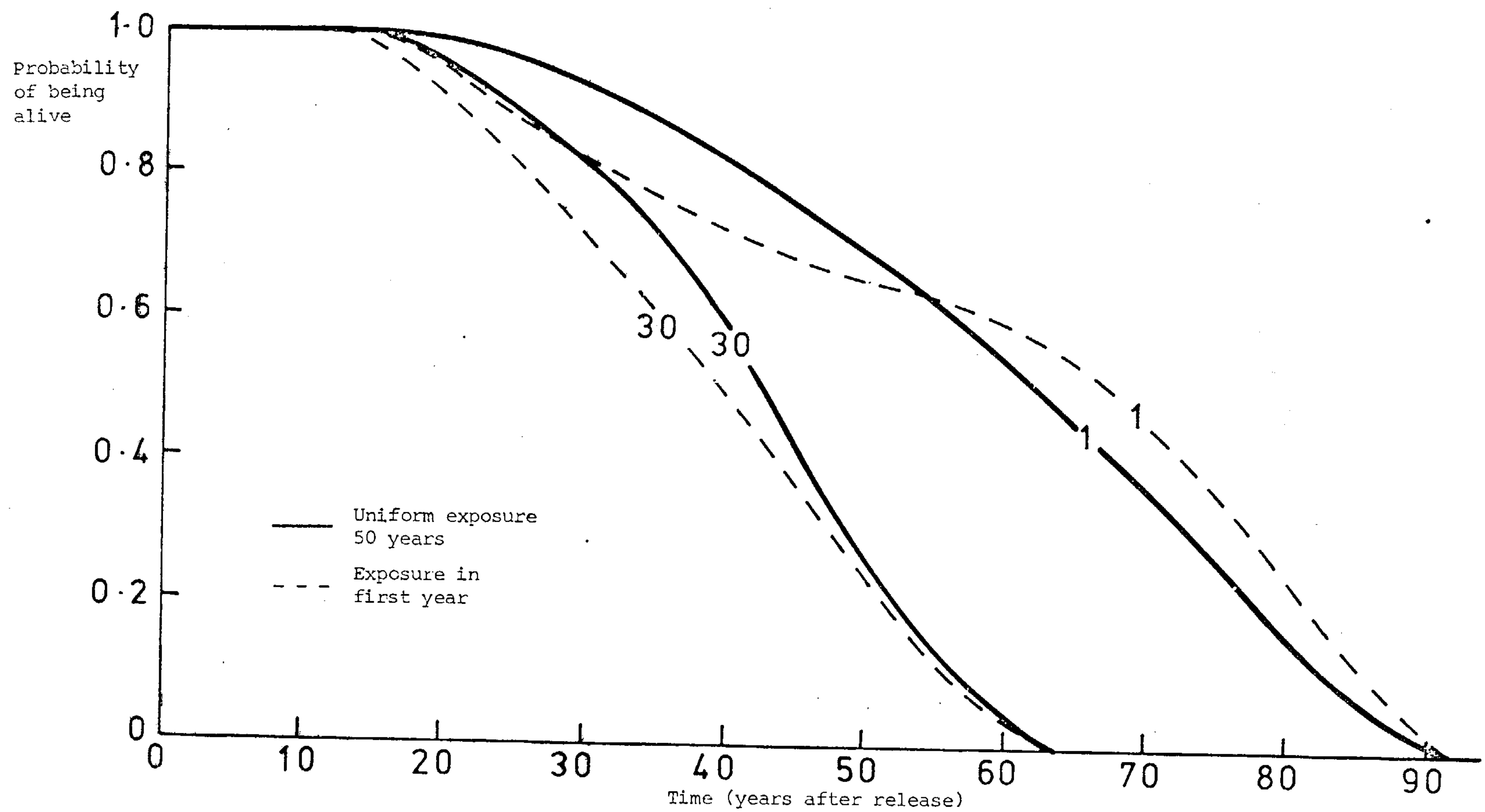

Figure 10. The survival probabilities for individuals of various ages as a function of time, comparing short- and long-term exposures

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