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# **Age at death from a radiation-induced cancer based on the Marshall model for mortality period**



### *P.J. Thomas*

*Safety Systems Research Centre, Queen's School of Engineering, University of Bristol, Queen's Building, University Walk, Bristol, BS8 1TR, United Kingdom*

#### a r t i c l e i n f o

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#### a b s t r a c t

Results presented elsewhere in this issue of *Process Safety and Environmental Protection* point to the radiation-induced loss of life expectancy following severe nuclear accidents being lower than generally feared. But this leaves open the question of the loss of life expectancy amongst radiation cancer victims, even if fortunately there are likely to be few of them. Addressing this question, the research presented here finds that the average radiation cancer victim will live into his or her 60s or 70s, depending on how long the radiation exposure lasts, based on data from the UK life tables. Between 8 and 22 years of life expectancy will be lost, well below the 42 years taken away on average by an immediately fatal accident, such as a car crash or rail crash. Not only are the results useful in their own right, but they inevitably call into question once again the concept of the Value of a Prevented Fatality still used for cost-benefit analyses in the UK on a "one size fits all" basis, which disregards the amount of life expectancy lost. This problem with applying the VPF in the context of radiological protection is additional to the gross flaws previously uncovered in the value assigned to the VPF in the UK. It is clear that the VPF should not be used as a criterion for cost-benefit analysis in radiological protection.

An important feature of the results presented is that they apply to any exposure to radiation between a point dose and a constant annual dose that does not cause radiation sickness. The figures presented, for both point and constant annual exposures, are equally valid whether the dose is a few mSv or a few hundred mSv. Nor is the outcome affected by the magnitude of the coefficient used to convert radiation dose into risk.

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#### **1. Introduction**

In his pioneering study, Lord Marshall [\(Marshall](#page--1-0) et [al.,](#page--1-0) [1983\)](#page--1-0) calculated the loss of life expectancy after a severe nuclear accident and radioactivity release by assuming that any death caused by a radiation-induced cancer from a non-acute dose of radiation would occur at a random point between times  $\omega_1$  and  $\omega_2$  after exposure. He used a uniform distribution starting at  $\omega_1$  = 10 and finishing  $\omega_2$  = 40 years after exposure to represent the probability from death from any type of cancer caused by the radiation exposure. The possibility that the probability density for death from a radiation cancer will stay raised beyond 40 years is catered for conservatively in this formulation, in the sense that the Marshall model will predict a greater loss of life expectancy due to radiation exposure in such a circumstance. Any hazard that is delayed past 40 years is effectively brought forward.

Confirmatory evidence for the Marshall range,  $\omega_1$  = 10 and  $\omega_2$  = 40 years, comes from Chernobyl, where, while an excess of childhood thyroid cancer cases was detected as soon as 4 years after the 1986 accident [\(Thomas,](#page--1-0) [1997\),](#page--1-0) the mean latency period before diagnosis was found to be 17 years with a standard deviation of 10 years [\(Thomas](#page--1-0) [and](#page--1-0) [Zwissler,](#page--1-0) [2003\).](#page--1-0) Moreover, the figures are for first diagnosis rather than death, and, indeed, medical treatment will have produced full remission most cases (at least 70% of cases, [Thomas](#page--1-0) [and](#page--1-0) [Zwissler,](#page--1-0) [2003,](#page--1-0) and possibly as many as 98%, [UNSCEAR,](#page--1-0) [2000\).](#page--1-0) Table A.4.2 of ICRP Publication 103 ([ICRP,](#page--1-0) [2007c\)](#page--1-0) contains five estimates in the range 90% to 93.4% for the fraction of people

E-mail addresses: [pjt3.michaelmas@gmail.com](mailto:pjt3.michaelmas@gmail.com), [philip.thomas@bristol.ac.uk](mailto:philip.thomas@bristol.ac.uk)

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expected to survive a radiation-induced thyroid cancer. Clearly any deaths (affecting only a small fraction of the total cases diagnosed) would lag on incidence and diagnosis, but would likely fall within the Marshall time limits in most cases. But ICRP Publication 103 lists 14 different types of radiation-induced cancer in Table A.4.1, each of which will have different characteristics, including latency period. That those latency periods can be many decades long is shown by evidence from the Life Span Study of the Japanese survivors of the atomic bomb explosions ([Preston](#page--1-0) et [al.,](#page--1-0) [2003,](#page--1-0) [2007\).](#page--1-0) Marshall's choice of a uniform probability density between 10 and 40 years after exposure for death from radiation-induced cancer makes a reasonable allowance for the different latency periods associated with the large number of radiation-induced cancers that are possible. This is corroborated by a study made by [Richardson](#page--1-0) [and](#page--1-0) [Ashmore](#page--1-0) [\(2005\)](#page--1-0) of the records of 40,000 Canadian radiation workers (86% men, 14% women), who, since the 1950s, had received low level radiation doses. Cumulative whole body doses averaged 14mSv, with the 90th percentile being 35mSv. They found that for cancers other than lung and leukaemia there was a delay of between 5 and 15 years between exposure and the first radiation-induced mortalities and this was followed by a roughly uniform risk of death to 40 years and beyond. For lung cancer the delay was between 10 and 20 years, after which the risk of death was roughly uniform out to 30–35 years, when the risk fell to zero. They found for leukaemia that there was no lag on exposure (although they considered this unrealistically short) and the risk was constant out to 30–33 years, after which it dropped to zero.

The perturbations to the hazard rate that follow from Marshall's model are well suited to and have been adopted for use in J-value calculations ([Thomas](#page--1-0) et [al.,](#page--1-0) [2006,](#page--1-0) [2010\).](#page--1-0) Modifications to the coefficients have been introduced to account for the latest recommendations of the International Commission on Radiation Protection [\(ICRP,](#page--1-0) [1990,](#page--1-0) [2006,](#page--1-0) [2007a,b,c;](#page--1-0) [Thomas](#page--1-0) [and](#page--1-0) [Jones,](#page--1-0) [2009a\)](#page--1-0) and the method has been extended to take account of prolonged exposures as well as the one-off dose considered by Marshall ([Thomas](#page--1-0) et [al.,](#page--1-0) [2006,](#page--1-0) [2007;](#page--1-0) [Jones](#page--1-0) et [al.,](#page--1-0) [2007a,b;](#page--1-0) [Jones](#page--1-0) [and](#page--1-0) [Thomas,](#page--1-0) [2009;](#page--1-0) [Thomas](#page--1-0) [and](#page--1-0) [Jones,](#page--1-0) [2009b\).](#page--1-0) As explained in Section 4.2 of [Waddington](#page--1-0) et [al.](#page--1-0) [\(2017\),](#page--1-0) the calculated loss of life expectancy accords well with results from an independent computer code produced by the Centre d'étude sur l'Evaluation de la Protection dans le domaine Nucléaire (CEPN), as reported in [Lochard](#page--1-0) [and](#page--1-0) [Schneider](#page--1-0) [\(1992\).](#page--1-0) However, given that the values of  $\omega_1$  and  $\omega_2$  were chosen as representative (but conservative), the present work includes sensitivity studies, in the first of which  $\omega_1$  was reduced to 5 years, while  $\omega_2$  was increased to 45 years in the second.

The loss of life expectancy experienced across the population affected by radiation exposure is typically very small. For example, the members of the public most under threat from Chernobyl, namely the 116,000 people evacuated in the first phase, can be calculated to have lost 9.3 days of life expectancy as a result of the 1986 accident. This figure may be compared with the 3¼ year shortfall in population-average life expectancy experienced by the inhabitants of Manchester in the North of England when compared with that of the inhabitants of Harrow in London. It may be further compared with the estimated 4½ months by which Londoners' lives would on average be extended if the capital's air pollution could be reduced to rural levels (9 months at birth, [Lord](#page--1-0) [Darzi,](#page--1-0) [2014\).](#page--1-0) Given that most people will prefer life to death, the population-average change in life expectancy provides a very good indicator of the size of harm, whether that harm results from industrial pollution or radiation pollution, for example. The average change in life expectancy over an appropriately defined population is the figure needed for a J-value analysis of measures to mitigate radiation exposure, where the J-value method of assessment has been validated against national ([Thomas,](#page--1-0) [2017\)](#page--1-0) and pan-national data [\(Thomas](#page--1-0) [and](#page--1-0) [Waddington,](#page--1-0) [2017a\).](#page--1-0) However further useful information may be gained by calculating the loss of life expectancy for those who actually contract a radiation induced cancer that proves fatal.

The change in life expectancy will depend on the profile of the radiation dose with time. For simplicity this paper will consider that the dose is uniform over time, but will vary the time of exposure,  $T_R$ , to give an indication of the sensitivity of the results to exposure time. Thus  $T_R$  may be set to 10 years, 20, 50, 100 or 10,000 years.

Meanwhile, choosing T<sub>R</sub> as 0.01 years (4 days) will give a good characterisation of the effects of a dose coming from a one-off release. This is verified using a separate approach applied to a point exposure. It will further be argued that these dose profiles, continuing exposure at a constant rate and a point exposure, bracket a range of other dose profiles, such as generated by fallout after a big nuclear accident, and give indicative results that are generally representative of those cases also.

The paper proposes a number of mathematical models for analysing the loss of life expectancy among those members of the public who will die as a result of exposure to nuclear radiation. Such unfortunate people, in practice a small fraction of those exposed even after a major accident, are defined as "radiation cancer victims".

The layout of the paper is as follows. Section [2](#page--1-0) derives the conditional probability density function for contracting cancer at age, *x*, given that the radiation cancer victim's exposure started at age, *a*, named here his "starting age". This is then combined with a general form of the probability density for the mortality period, which is the difference,  $m = y - x$ , between the age at death, y, and the age, x, of cancer induction. The combination leads to the radiation cancer victim's probability density for death at age, *y*, given his starting age, *a*.

Section [3](#page--1-0) specifies that the mortality period should obey the Marshall model, where the probability density for the mortality period is a rectangular function, uniform between time limits:  $\omega_1$ , which represents the minimum delay between cancer induction and death, and  $\omega_2$ , which represents the maximum time that death can be delayed on induction. The conditional probability density for death at age, *y*, given first exposure at age, *a*, is then derived for starting ages that are sufficiently low for all possible mortality periods to play out before the person dies of extreme old age. Extreme old age,  $\alpha_0$ , is set to 101 years in the calculations of this paper. Section [4](#page--1-0) extends the treatment so that the conditional probability density for death at age, *y*, given that radiation exposure starts at age, *a*, can apply to all starting ages, without limit.

Section [5](#page--1-0) uses the conditional probability density derived in Section [4](#page--1-0) to find the expected age at death, E (Y|A = a), for a radiation cancer victim of starting age, *a*, who is alive at the start of the exposure. The variance, *var*  $(Y|A = a)$ , is also found.

Section [6](#page--1-0) introduces the steady state population model, where there is a turnover of people, but the overall number stays steady. Such a model can be used to represent to reasonable accuracy both national and urban populations. The section applies the steady-state model in deriving the probability density for starting age amongst radiation cancer victims who are alive at the start of the radiation exposure.

The probability density for starting age derived in Section [6](#page--1-0) may be applied along with the expected age at death,  $E(Y|A = a)$ , for a radiation cancer victim of starting age, *a*, with the conjunction allowing the unconditional expected age at death for radiation cancer victims, E (Y), to be found. This process is detailed in Section [7.](#page--1-0) The unconditional variance of age at death for radiation cancer victims, var (Y), is also found in Section [7.](#page--1-0) These values apply to those alive at the time the radiation exposure starts.

But a prolonged radiation exposure will have an effect not only on those alive at the start of the exposure, but also on those born into it. It is now necessary to extend the treatment of Section [6](#page--1-0) and find the probability density for starting age amongst radiation cancer victims for both those alive at the start of the exposure and those born into it. This is carried out in Section [8.](#page--1-0)

Section [9](#page--1-0) uses this probability for starting age in an analogous fashion to Section [7,](#page--1-0) and the unconditional average age at death for radiation cancer victims, E (Y), is found for the total population of radiation cancer victims, both those alive at the start of the exposure and those born into it. The corresponding unconditional variance of age at death for radiation cancer victims, var (Y), is also found in Section [9](#page--1-0) for the total population of radiation cancer victims subjected to a continuing radiation dose.

Section [10](#page--1-0) treats the simpler problem of finding the average age at death for radiation cancer victims exposed to a point dose of radiation. E (Y) and var (Y) are derived. Since the point dose is the limiting case of a continuing dose as the dose period is reduced towards zero, the numerical results can be used as a check on the values produced under the continuing dose case.

## ِ متن کامل مقا<mark>ل</mark>ه

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