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Childhood cancers near German nuclear power stations: hypothesis to explain the cancer increases

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In early 2008, the very large Kinderkrebs in der Umgebung von Kernkraftwerken [Childhood Cancer near Nuclear Power Plants] (KiKK) study in Germany reported increases in leukaemias and solid cancers among children living near all German nuclear power plants (NPPs). This study, previously described in *Medicine, Conflict and Survival*, has triggered debates in many countries as to the cause or causes of these increased cancers. An accompanying article reports on the recent developments on the KiKK study including the responses by German radiation agencies, and the results of recent epidemiological studies near United Kingdom and French nuclear installations. This article outlines a possible explanation for the increased cancers. In essence, doses from environmental NPP emissions to embryos/foetuses in pregnant women near NPPs may be larger than suspected, and haematopoietic tissues may be considerably more radiosensitive in embryos/foetuses than in newborn babies. The article concludes with recommendations for further research.

Keywords: cancer; carbon-14; congenital malformations; discharges; embryo; emissions; foetus; leukaemia; nuclear power stations; pregnancy outcomes; radiation; radioactivity; radionuclides; relative risk; tritium

Introduction

In early 2008, the KiKK study[1,2] in Germany reported a 1.6-fold increase in solid cancers and a 2.2-fold increase in leukaemias among children living within 5 km of all 16 German nuclear power plants (NPPs). The study was very large: it examined 593 under-fives with leukaemia and 1766 controls. Its remarkable findings, previously described in *Medicine, Conflict and Survival*[3] have triggered debates among many scientists as to their cause or causes – see the accompanying article on the KiKK debate. (Previous studies on childhood leukaemias near NPPs are listed in the accompanying article published in this edition of the journal.) The increased risks do not seem to

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be explained by the radiation from NPPs as official estimated doses from NPPs appear to be too low.

Possible explanations for increased cancer incidences near nuclear power plants

It has been known at least since the late 1950s[4] that radiation exposures can result in increased leukaemias and that environmental exposures to radiation are a risk factor for leukaemia[5–7]. In 1984, the first reported incidence[8] of increased leukaemias near nuclear facilities occurred near the Windscale, now Sellafield, nuclear reprocessing plant in Cumbria, UK. This was followed by several ecological and case–control studies[9–11] which suggested an association between NPPs and raised incidences of childhood leukaemia among those living nearby.

At the same time, the 'clustering' of diseases and other health effects is a natural phenomenon, and increased cancer rates have been found near sites unrelated to nuclear power, for example near UK New Towns[12] and sites proposed for power stations but not actually developed[13]. For this reason, epidemiologists tend to avoid the term 'cluster' as disease incidences are often spatially and temporally heterogeneous from natural causes.

Although such non-random distributions could have possibly occurred at one or two NPP sites, it is very unlikely that they occurred near all 16 German NPP sites. Nevertheless, care has to be taken in ascribing a causal relationship between increased cancers and proximity to nuclear power stations.

Even before the KiKK study, two large studies indicated an association between NPPs and child leukaemias. In 1999, Laurier and Bard[14] examined the literature on childhood leukaemias near NPPs world wide. They listed no fewer than 50 studies (29 ecological, 7 case–control and 14 national multi-site studies). Many of these revealed small increases in childhood leukaemia near NPPs although most of the data in the ecological studies were not statistically significant. In 2007, Baker and Hoel[15] in a meta-analysis assessed data from 17 research papers worldwide covering 136 nuclear sites in the UK, Canada, France, United States, Germany, Japan and Spain. In children up to nine years old, leukaemia death rates were from 5–24% higher, and leukaemia incidence rates were 14–21% higher. These findings were statistically significant. Post-KIKK in 2008, Körblein carried out a meta-analysis[16] of leukaemias near most NPPs in Germany, France and the UK. He also found an increased risk of child leukaemia deaths near NPPs with a relative risk of 1.33 which was statistically significant (single tailed $p = 0.0246$).

Various hypotheses have been put forward to explain these cancer increases near nuclear installations including coincidence; a postulated virus from population-mixing (the Kinlen hypothesis); the response to the lack of childhood immunity to infectious diseases (the Greaves hypothesis[17]); parental pre-conception irradiation (the Gardner hypothesis[18]); genetic

predisposition to cancer; synergistic effects between radiation and unnamed chemicals or combinations of these factors. Some remain little more than suggestions: others have not been supported by the KiKK study. Although some hypotheses are vigorously promoted by individuals, none commands widespread support.

The first step towards elucidating a possible explanation is the KiKK study's finding that increased risks were directly linked with proximity to NPPs. Therefore, we need to examine those aspects of the normal operation of NPPs which might result in increased exposures and risks. These include:

- direct radiation, such as gamma rays and neutrons, from reactor cores;
- 'skyshine' from reactor neutrons being reflected back to earth by N, C and O atoms;
- electro-magnetic radiation from power lines near NPPs;
- water vapour emissions from cooling towers at about half the 16 German NPPs;
- parental pre-conception irradiation of nuclear workers;
- radioactive contamination of nuclear workers' homes, for example, by workers clothing; and
- radioactive releases to the environment.

The increased risks could also be due to a combination of the above factors, and there may well be interactions between environmental exposures we are yet to understand. For example, synergistic effects may exist between radiation and chemicals that may act to increase cancer risks[19,20]. Unfortunately, these explanations were not explored by recent German, UK and French studies on KiKK (see accompanying article in this issue), or indeed by the KiKK study itself. A widely discussed explanation – radioactive releases from NPPs – was prefigured by two aspects of the KiKK study: the use of NPP chimneys as the measuring point for distances to cases and controls, and the extension of the study area to sectors downwind of NPPs.

However, this explanation was discounted by the KiKK study because current official estimates of the radiation doses from NPP releases were too low, typically by three orders of magnitude, to result in the cancer risks observed by the KiKK study. But how reliable are official dose and risk estimates?

Uncertainties in dose estimates

Published radiation doses to members of the public near NPPs are invariably very low (usually in the range 10^{-3} to 10^{-4} mSv per year): these are estimates not measurements. How these estimates are derived is not widely understood by scientists, and not at all by members of the public.

In fact, the methodology is quite complicated, as they are derived using at least four computer models in sequence:

- models for the generation of fission/activation products in reactor cores – the emission data published by utilities are derived from these models;
- environmental transport models for radionuclides, including weather models;
- human metabolism models which estimate nuclide uptake, retention and excretion;
- dose models which estimate radiation doses from internally retained nuclides.

Each of these models would derive a range of results log-normally distributed, that is, they would be skewed to the right as in Figure 1. This means that, although the real value could be larger or smaller than the median value, in practice some very high values could result. However, in most cases only the median value is used.

The problem is that each model's central result is inherently uncertain (the real result lying within the shown distribution). The uncertainties from each model have to be treated together to gain an idea of the overall uncertainty in the final dose estimate[21]. Further uncertainties are introduced by unconservative radiation weighting factors, dose rate reduction factors, and tissue weighting factors in official models[22]. The cumulative uncertainty in dose estimates could be very large as recognized by the report of the UK Government's CERRIE Committee[23].

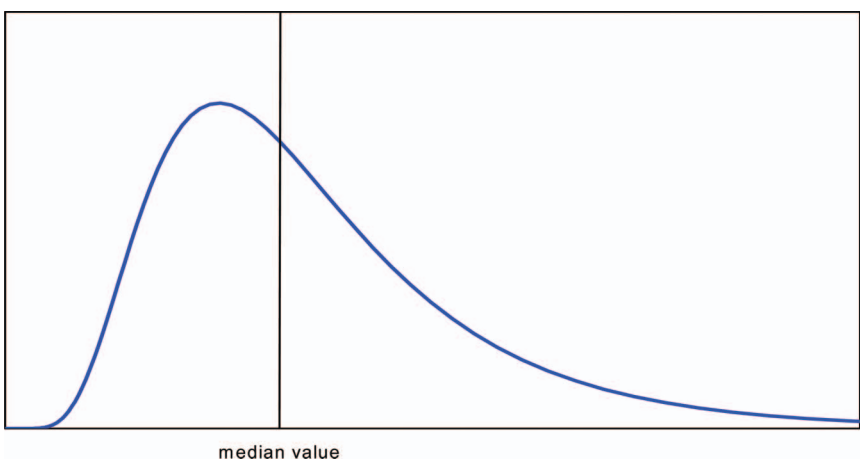

Figure 1. Log-normal distribution (generic example, axes not labelled).

This does not mean that official dose estimates are necessarily incorrect, but they may contain unquantified uncertainties which could be large. This means that where evidence (for example, from KiKK) suggests that dose estimates may be incorrect, we should reassess the matter. In particular, when we examine the possible reasons for the wide gulf between estimated risks and the risks observed by KiKK, we should not dismiss radiation exposures as a possible cause on the grounds of low official dose estimates without first carefully examining them.

Uncertainties in risk estimates

Uncertainties exist with estimated risks as well as estimated doses. This is because risk models have to be used to estimate the likely level of cancers, but uncertainties could exist in these models as well. For example, current official risks are derived mainly from the Japanese survivors of the atomic bombs in 1945. However, some consider that these risk estimates may be unreliable for a variety of reasons. One is that the Japanese bomb survivor study was started five years after the bomb blasts, which means that deaths in the first five years were not counted[24]. Another is that the risks estimated from a sudden external blast of high energy neutrons and gamma rays are not applicable to environmental releases which result in chronic, slow, internal exposures often to low-range beta radiation. Uncertainties in official risk models also derive from the application of risks from a Japanese to a European population, from the application to adults only, from the application of age and gender-averaged risks, and from the practice of arbitrarily halving risks to take account of cell studies suggesting lower risks from low doses and low dose rates (i.e. applying a Dose and Dose Rate Effectiveness Factor). However, it is difficult to quantify these uncertainties and to give a figure which may indicate how much the current fatal cancer risk estimate for adults (5 per cent per Sv effective dose) may be an underestimate or overestimate.

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Radioactive releases from nuclear power plants

Operational radionuclide releases from NPPs, including emissions to air and discharges to rivers or seas, are suspected to be a causal factor in

cancer increases near NPPs, although this is rarely discussed in the literature[25]. It is a common misconception that liquid discharges to the sea (or to rivers from German NPPs) are more important than emissions to air. In fact, the latter cause most of the doses (both individual and collective) from NPP releases, and this is certainly the case with German reactors. An important factor is the emission rate: large emissions over a few hours result in higher individual doses than the same amount spread over many days.

The largest emissions from German NPPs are H-3 (tritium) in the form of radioactive water vapour and water, and C-14 mainly in the form of radioactive carbon dioxide. These substances clearly interact with the biosphere, including humans. In addition, large quantities of radioactive noble gases, including Kr-85, Ar-41 and Xe-133, are emitted from NPPs; however these are not, at present, considered to interact with the biosphere to a significant extent.

NPP emissions result in elevated nuclide concentrations in vegetation and foodstuffs nearby. This is seen in Figure 2 which indicates tritium concentrations in vegetation and food moisture near Canadian NPPs. This is shown as comparable data for German NPPs are not available to the public. Figure 2 shows that the distance dependency of tritium concentrations is similar to the distance dependency for cancer risk. Although tritium emissions from Canadian heavy water reactors are quantitatively larger than from German PWR and BWR reactors, the same qualitative pattern of raised nuclide concentrations in vegetation and food is expected to occur near German reactors.

Hypothesis: *in utero* exposures from environmental releases

The KiKK findings have prompted much debate among scientists throughout Europe as to the cause(s) of the increased leukaemia cases near German NPPs. Indeed, it is a primary task of science to attempt to explain observed phenomena which are apparently at odds with received wisdom or, in this case, with the current understandings of radiation risks. It is for this reason that the following hypothesis is suggested to explain the risks shown by the KiKK study.

It is that 'spikes' in NPP releases may result in the labelling of the embryos and foetuses of pregnant women living nearby at high concentrations. Such high nuclide concentrations could occur in long-lived cells and could result in large exposures to radiosensitive tissues and subsequent cancers. This suggestion was first made by the late Professor Edward Radford, the former Chairman of BEIR III Committee in the US. He mooted it 30 years ago during testimony to the Ontario Select Committee on Hydro Matters[26] which then was examining the possible health effects of tritium discharges from nuclear facilities near Toronto in Canada.

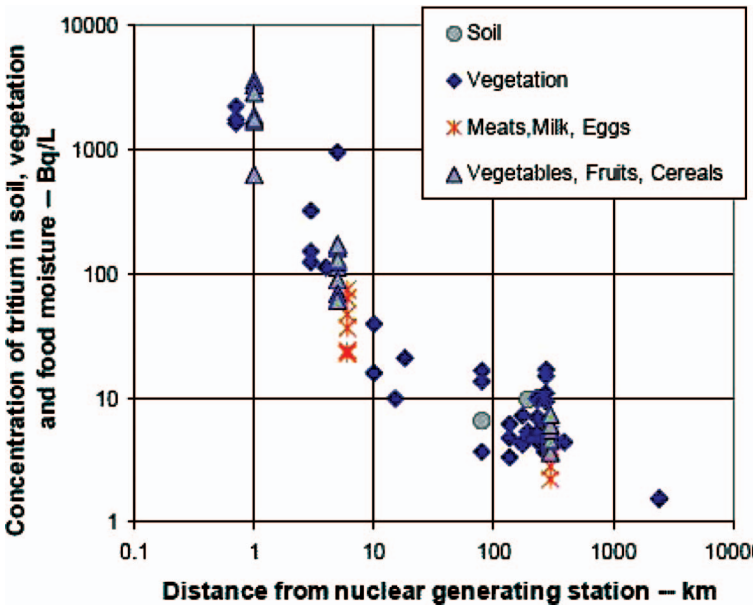

Figure 2. ^{3}H concentrations in vegetation and food moisture near NPPs.
Source: Reproduced with permission from the Canadian Nuclear Safety Commission from *Tritium in the Canadian Environment: Levels and Health Effects*. Report RSP-0153-1. Prepared for the Canadian Nuclear Safety Commission under CNSC contract no. 87055-01-0184 by Ranasara Consultants and Dr Richard Osborne. Data from Health Canada (2001) Environmental Radioactivity in Canada. Radiological Monitoring Report. Ottawa, Government of Canada.

Spikes in the emissions of radioactive carbon and hydrogen (as carbon dioxide and water vapour) occur at NPPs when their reactors are opened (approximately once a year) to replace nuclear fuel. Figure 3 indicates ^{14}C concentrations in air near a German PWR nuclear power station in recent years. Tritium and noble gases will be released at the same time as ^{14}C. It can be seen that gaseous releases are episodic with spikes occurring about once per year.

In order to assess our hypothesis, we discuss below a number of considerations which would need to be shown in order to demonstrate biological plausibility. These include:

- the nature of the emissions from NPPs (mostly carbon (^{14}C) and hydrogen (^{3}H));
- the bio-accumulation of ^{3}H and ^{14}C in embryos and foetuses;
- the increased radiosensitivities of embryos and foetuses; and
- the apparent increased radiosensitivity of pre-natal haematopoietic cells.

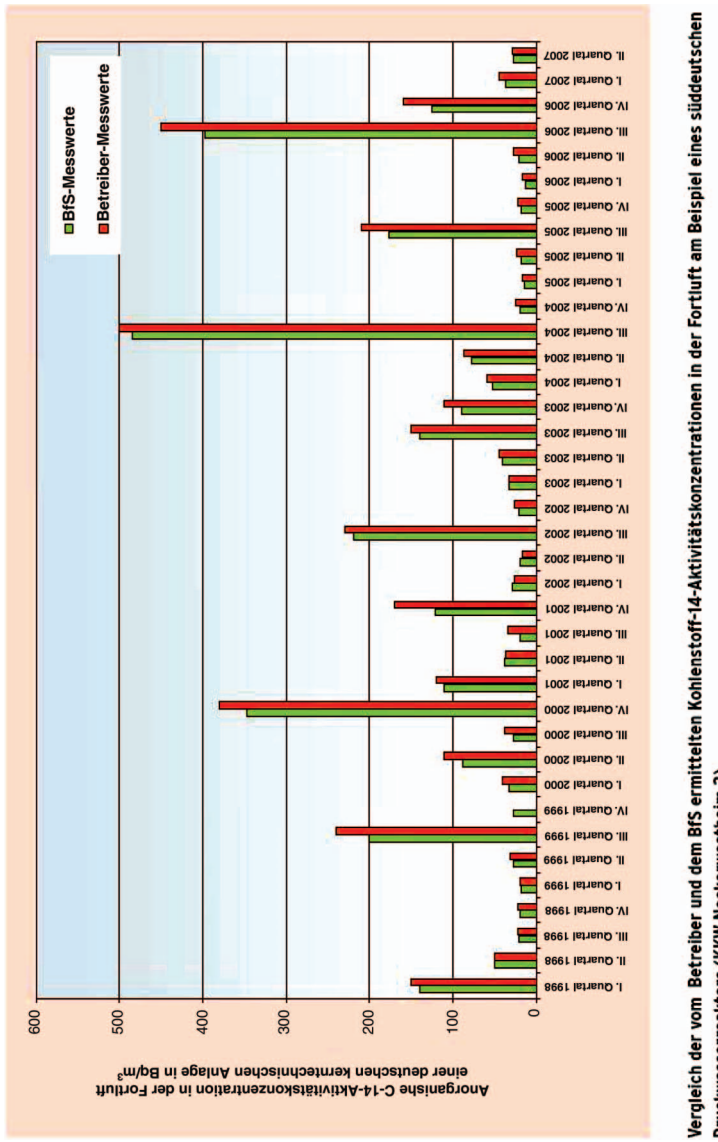

Figure 3. Quarterly ^{14}C releases from the Neckarwestheim2 nuclear power station.
Source: Jahresbericht (Annual Yearbook) 2007, Bundesamt fur Strahlenschutz, Berlin.

Major emissions: carbon (^{14}C) and hydrogen (^{3}H)

As stated above, the largest nuclide emissions from NPPs are radioactive carbon (^{14}C) and hydrogen (^{3}H). These nuclides move quickly through the environment as water, water vapour and carbon dioxide; they rapidly exchange with stable H and C; and they recycle in all biota. Figure 2 indicates tritium concentrations in food and vegetation near NPPs. Similar concentrations are expected for carbon-14.

Organically bound tritium (OBT) and organically bound carbon (OBC) are formed by embryos and foetuses taking up H and C atoms during new cell production and during metabolic reactions. The results are that embryos and foetuses near NPPs may be labelled at the levels of ambient (i.e. environmental) ^{3}H and ^{14}C concentrations. That is, mothers near NPPs may give birth to babies with raised concentrations of these nuclides.

^{3}H and ^{14}C bio-accumulation in embryos and foetuses

Stather et al.[27] have estimated that, following tritium intakes by the mother during pregnancy, tritium concentrations in her foetus are 60 per cent higher than in herself. As a result, the Health Protection Agency now estimates[28] that doses in embryonic and foetal tissues are raised by factors of 1.5 to two compared with adult tissues following exposures to air releases of tritiated water vapour (HTO). Both studies showed similar increases for ^{14}C.

Increased radiosensitivities of embryos and foetuses

The best data on the radiation risks of *in utero* exposures, that is on the radiosensitivity of embryos and foetuses, are from the UK Oxford Survey of Childhood Cancer (OSCC) carried out by the pioneering radiation epidemiologist, Alice Stewart, in the 1950s–1980s[29]. Recently, Wakeford[30] comprehensively reviewed the OSCC and more than 30 similar studies worldwide. The latter studies confirmed the presence and size of the risks of *in utero* radiation initially found by Stewart. Wakeford and Little[31] estimated from OSCC and other data that the ERR of leukaemia in children aged under 15 from abdominal exposures to X-rays was 51 per Gy (95% CI: 28, 76).

If we apply this risk estimate to the KiKK situation, three corrections are needed. First, the leukaemia risk in under-five year-olds (as in KiKK) is higher than in under-15 year-olds because the peak years for leukaemia diagnoses are in children aged two to three years. This would result in the average relative risk being greater by a factor of perhaps $\sim$1.5. Also, most ($>$90%) OSCC exposures were in the last trimester, and it has been estimated[32] that risks from exposures in the first trimester are perhaps five times greater than those from exposures in the last trimester.

These risks arose from external X-rays, whereas the KiKK risks are hypothesized to arise from internal exposures to radionuclides. There are few estimates of the risks arising from internal *in utero* exposures. However, Fucic et al.[33] have recently suggested that *in utero* risks from internal nuclides were four to five times greater than from *in utero* X-rays[1]. Summing these factors, we postulate that the relative risk of child leukaemia in nought to five-year olds from internal nuclides in the first trimester near NPPs would be:

$$\mathrm{RR} = 52\,\mathrm{per\,Gy(OSCC)} \times 1.5(0-5\,\mathrm{year\text{-}olds}) \times 5(1\,\mathrm{st\,trimester}) \times 5(\mathrm{internal}\ \textit{vs.}\ \mathrm{X\text{-}rays}) = \sim 2\,\mathrm{per\,mGy}$$

This suggests that human embryos and foetuses are much more radiosensitive to radiation than currently acknowledged. It also seems to suggest that background radiation of about 1 mGy per year (excluding radon doses) could be a major cause of naturally occurring childhood leukaemia and this has already been proposed[34].

If we were to apply the relative risk for childhood leukaemia of 2.2 observed by KiKK, it would suggest *in utero* doses to embryos in pregnant women near German NPPs of about 1 mGy. Such doses, although very low, are still about 1000 times higher than the official estimated doses of a few μGy to adults from NPP emissions. (Official estimates of exposures *to embryos* near German NPPs do not appear to have been carried out.)

Increased radiosensitivity of pre-natal haematopoietic cells

Finally, we need to consider the different radiosensitivities of various embryonic tissues. As we are primarily concerned with leukaemia which affects white blood cells, our attention is focussed on the haematopoietic[2] system, that is, bone marrow and lymphatic tissues. These tissues contain many stem cells, i.e. they are self-renewing. When they divide, some daughter cells remain stem cells, so the number of stem cells stays about the same. Radiation-caused mutations to stem cells would clearly be damaging to haematopoietic system and could result in increased leukaemia risks. Compared with other organs, bone marrow contains a very high proportion of proliferating stem cells and it is likely to be among the most radiosensitive of embryonic/foetal tissues.

This has been hinted at previously. In 1990, after the Gardner team had published their paternal pre-conception irradiation (PPI) hypothesis, the BMJ published letters questioning aspects of the hypothesis. Dr J. A. Morris stated[35] that, assuming mutations were the main factor[36] causing the observed 10-fold increase in leukaemia incidence observed by Gardner's team, it would require a 100 to 1000-fold increase in the radiation-induced

mutation rate if acting on the germ cell; a 10-fold increase if acting on lymphocytes during early extra uterine life; but only a 1.8-fold increase if acting on lymphocytes throughout intrauterine life. He added the latter seemed the most plausible mechanism even though the exposure pathways were unclear.

Later in 1992, Lord et al.[37] made a similar suggestion when they stated that embryonic haematopoietic cells could be up to 1000 times more radiosensitive than post-natal haematopoietic cells. They added that different mechanisms of inducing this damage operated at different embryonic/foetal stages. More recently, this suggestion has been supported by Ohtaki et al.[38] in their study of chromosome translocation frequencies in the white blood cells of Japanese A-bomb survivors irradiated *in utero*. They found that precursor lymphocytes of the foetal haematopoietic system may be highly radiosensitive, perhaps 100 times more radiosensitive than post-natal lymphocytes. From this study, Wakeford[39] surmised that radiosensitive primitive cells (whose mutation may result in childhood cancers) remain active throughout pregnancy, including during the third trimester. However, this radiosensitivity ceases after birth, although it is not known at present why this is the case.

It is concluded that the increased radiosensitivity of haematopoietic cells before birth might prove to be a major factor in explaining the discrepancy between official dose estimates and the observed level of risks in the KiKK study.

Why were *in utero* exposures not considered in the 1980s and 1990s?

In the 1980s and 1990s, the possible cause or causes of the raised leukaemia incidence near Sellafield in the UK were much discussed. The explanation favoured by the Gardner team[40] which reported the increase was PPI of the sperm cells of Windscale employees. Later, this was discredited for a number of reasons, partly because such fathers were distributed throughout Cumbria but the leukaemia excess was restricted to the village of Seascale, a few kilometres from the Windscale complex.

It is difficult to recreate the situation prevailing nearly 20 years ago, but three possible reasons for discounting *in utero* exposures at the time are as follows. First, if *in utero* exposures were the cause, then we should expect to see other effects such as increased congenital malformations, stillbirths, and neonatal deaths, and various studies[41] did not find these effects. Later studies seemed to show the same dearth of untoward pregnancy outcomes[42]. However, it is now realized that the ascertainment of these outcomes is fraught with difficulties. These include underreporting due to the reluctance (or refusal) to discuss these effects and the difficulty of distinguishing between spontaneous abortions, stillbirths and heavy periods (see discussion by Sara Downs[43] in 1993).

Consequently, these epidemiological studies could have reported false negative results.

Second, although Stewart's findings of increased risks from low X-ray exposures *in utero* are nowadays widely accepted as valid[39], this was not the case in the 1980s and 1990s. In other words, *in utero* exposures were then not widely considered to be unduly risky. But perhaps most important, there was little awareness then of the considerably increased radiosensitivity of erythropoietic tissues in embryos and foetuses. In particular, apparently little attention was given to Morris's letter on the increased radiosensitivity of lymphocytic cells *in utero*.

What about the increases in solid cancers?

The above hypothesis may explain the leukaemia increases, but what about the increase in solid cancers also observed by KiKK? Although the numbers of solid cancers were smaller and not statistically significant, there are good theoretical grounds for expecting solid cancers as well. For example, the OSCC study[14] also found increased solid cancers from *in utero* exposures. The higher risks for leukaemia could be explained by the exceptional radiosensitivity of erythropoietic tissues *in utero* compared with other tissues *in utero*. And this could be explained by the higher concentrations of stem cells in such tissues, as it is thought the majority of stem cells in adults are found in erythropoietic tissues, that is, bone marrow and lymph glands.

Conclusion

To recap, a possible biological mechanism to explain the KiKK observations is that NPP emission spikes result in the radioactive labelling of embryos and foetal tissues in pregnant women living nearby. Such *in utero* concentrations over two to three years both before and after birth could result in the accumulation of relatively high doses in the radiosensitive organs of embryos and foetuses, particularly haematopoietic tissues. Unfortunately, cumulative radiation doses and risks to specific organs and tissues in embryos/foetuses from nuclide uptakes during pregnancy are presently not considered by the ICRP.

In sum, the observed high rates of infant leukaemias in KiKK may be a teratogenic effect from incorporated radionuclides. (Teratogenic effects being those which arise from exposures of toxic substances to embryos and foetuses in the womb.) These effects are often recognized at birth, but infant leukaemia is not easily ascertained at birth. Such babies are born pre-leukaemia and full-blown leukaemias are only diagnosed after birth, perhaps after their bone marrows have accumulated sufficient radioactive decays from the nuclides therein.

Recommendation

Whatever the explanation for the increases, the KiKK study and its implications raise difficult questions, including whether vulnerable people, in particular, pregnant women and women of child-bearing age, should be advised to move away from nuclear facilities. Another question is whether local residents should be advised not to eat produce from their gardens or wild foods, as the food pathway is the largest contributor to local doses.

As a first step, it is recommended that the Bundesamt für Strahlenschutz, the German Government's radiation protection agency, should:

- investigate the dosimetric effects of episodic nuclide emissions from NPPs;
- estimate doses to the bone marrow of developing embryos and the subsequent risks of leukaemia in young children;
- assess the confidence intervals around their dose and risk estimates;
- publish its results.

Notes on contributor

Ian Fairlie is an independent consultant on radioactivity in the environment. He has worked for various UK government departments and regulatory agencies, and advises environmental NGOs, the European Parliament and local authorities. Between 2001 and 2004 he acted as Secretariat to the UK government's CERRIE Committee on internal emitters. Dr Fairlie wishes to thank Dr Alfred Körblein for his valued help in translating German documents and checking various drafts.

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