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A hypothesis to explain childhood cancers near nuclear power plants

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ABSTRACT

Over 60 epidemiological studies world-wide have examined cancer incidences in children near nuclear power plants (NPPs): most of them indicate leukemia increases. These include the 2008 KiKK study commissioned by the German Government which found relative risks (RR) of 1.6 in total cancers and 2.2 in leukemias among infants living within 5 km of all German NPPs. The KiKK study has retriggered the debate as to the cause(s) of these increased cancers. A suggested hypothesis is that the increased cancers arise from radiation exposures to pregnant women near NPPs. However any theory has to account for the >10,000 fold discrepancy between official dose estimates from NPP emissions and observed increased risks. An explanation may be that doses from spikes in NPP radionuclide emissions are significantly larger than those estimated by official models which are diluted through the use of annual averages. In addition, risks to embryos/fetuses are greater than those to adults and haematopoietic tissues appear more radiosensitive in embryos/fetuses than in newborn babies. The product of possible increased doses and possible increased risks per dose may provide an explanation.

1. Introduction

In the early 1950s, Folley et al. (1952) observed an increased risk of leukemia among Japanese bomb survivors. In the late 1950s, Stewart et al. (1958) also observed that radiation exposures can result in increased incidences of leukemia. A number of studies since then (BEIR, 1990; Preston et al., 1994; IARC, 1999) have shown that ionising radiation including medical, occupational and environmental exposures, are a risk factor for leukemia. In addition, older ecological and case–control studies (Forman et al., 1987; Gardner, 1991; Pobel and Viel, 1997) revealed an association between nuclear power plants and childhood leukemia among those living nearby.

In the late 1980s and early 1990s, increased incidences of childhood leukemias were reported near several UK nuclear facilities. Various explanations were offered for these increases, however the UK Government's Committee on the Medical Aspects of Radiation in the Environment (COMARE) concluded in a series of reports (1986; 1988; 1989; 1996) that the cause remained unknown but was unlikely to involve radiation exposures. This was mainly because official estimates for radiation doses from these facilities were too low by orders of magnitude to explain the increased leukemias. Indeed, any theory will have to account for the >10,000 fold discrepancy between official dose estimates from NPP emissions and observed increased risks.

A pattern of epidemiological evidence world-wide now clearly indicates increased leukemia risks near nuclear power plants (NPPs). Laurier and Bard (1999) and Laurier et al. (2008) examined the literature on childhood leukemias near NPPs world-wide. These two studies identified a total of over 60 studies. An independent review of these studies (Fairlie and Körblein, 2010) indicated that the large majority of these studies revealed small increases in childhood leukemia although in many cases these were not statistically significant. Laurier and Bard and Laurier et al., mostly employees of the French Government's Institut de Radioprotection et Sûreté Nucléaire (IRSN), confirmed that clusters of childhood leukemia cases existed near most NPPs but refrained from drawing wider conclusions. Fairlie and Körblein (2010) in their review concluded that the copious evidence indicating increased leukemia rates near nuclear facilities, specifically in young children, was quite convincing.

This conclusion was supported by two meta-analyses of national multi-site studies. Baker and Hoel (2007) assessed data from 17 research studies covering 136 nuclear sites in the UK, Canada, France, the US, Germany, Japan, and Spain. In children up to nine years old, leukemia death rates were from 5 to 24% higher and leukemia incidence rates were 14–21% higher. However their analysis was criticised by Spix and Blettner (2009).

The second meta-analysis by Körblein (2009) covering NPPs in Germany, France, and the UK also found a statistically significant

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increased risk of child leukemias and relative risk of leukemia deaths near NPPs (RR = 1.33; one-tailed p value = 0.0246). Further studies (Guizard et al., 2001; Hoffman et al., 2007) indicated raised leukemia incidences in France and Germany. However COMARE (2005; 2006) declined to support these conclusions.

Later, Bithell et al. (2008) and Laurier et al. (2008) found increases in child leukemias near UK and French NPPs respectively. In both cases, the numbers were low and not statistically significant – i.e. there was a greater than 5% possibility that the observations could have occurred by chance. However instead of reporting these increases, the studies incorrectly concluded that there was "no evidence" (Bithell) and "no suggestion" (Laurier) of leukemia increases near UK and French nuclear reactors, merely because their data lacked statistical significance. These conclusions were incorrect: the authors should have reported the observed leukemia increases but added there was a >5% probability they could have occurred by chance.

In more detail, p values (that is, the probabilities that observed effects may be due to chance) are affected by both the magnitude of effect and the size of study (Whitley and Ball, 2002). This means statistical tests must be used with caution as the use of an arbitrary cut-off for statistical significance (usually p = 5%) can lead to incorrectly accepting the null hypothesis (ie nil effect) merely because it is not statistically significant (Sterne and Smith, 2001): a possible type II error. This often occurs in small studies due to their small sample sizes rather than lack of effect (Everett et al., 1998). Axelson (2004) has pointed out that many epidemiology studies with negative results – statistically speaking, are of questionable validity as they may obscure existing risks.

2. KiKK study

The KiKK study (**K**inderkrebs **i**n der Umgebung von **K**ern**K**raftwerken = Childhood Cancer in the Vicinity of Nuclear Power Plants) found a 120% increase in leukemia and a 60% increase in all cancers among infants and children under 5 years old living within 5 km of all German NPPs (Kaatsch et al., 2008b; Spix et al., 2008). The increase of risk with proximity to the NPP site, tested with a reciprocal distance trend, was significant for all cancers (p = 0.0034, one-sided), as well as for leukemias (p = 0.0044).

KiKK is a large well-conducted study; its findings are scientifically rigorous; its evidence is particularly strong; and the German Government's Bundesamt für Strahlenschutz, which commissioned the study, has confirmed its findings. A BfS-appointed expert group stated (BfS, 2008) "The present study confirms that in Germany there is a correlation between the distance of the home from the nearest NPP [nuclear power plant] at the time of diagnosis and the risk of developing cancer (particularly leukemia) before the 5th birthday. This study is not able to state which biological risk factors could explain this relationship. Exposure to ionising radiation was neither measured nor modelled. Although previous results could be reproduced by the current study, the present status of radiobiological and epidemiological knowledge does not allow the conclusion that the ionising radiation emitted by German NPPs during normal operation is the cause. This study cannot conclusively clarify whether confounders, selection or randomness play a role in the distance trend observed."

One potential problem is that the KiKK study considered residence at the time of leukemia diagnosis and not residence at the time of early pregnancy: if my hypothesis (see below) were correct this would add uncertainty to the findings. The best way to deal with this would be a new study which established residence at the time of early pregnancy.

Table 1
Studies of observed (O) and expected (E) leukemia cases within 5 km of NPPs.

Dataset	O	E	SIR = O/E	90%CI	p-value
Germany	34	24.1	1.41	1.04–1.88	0.0328
Great Britain	20	15.4	1.30	0.86–1.89	0.1464
Switzerland	11	7.9[a]	1.40	0.78–2.31	0.1711
France[b]	14	10.2	1.37	0.83–2.15	0.1506
Pooled data	79	57.5	1.37	1.13–1.66	0.0042

[a] derived from data in Spycher et al. (2011).
[b] acute leukemia cases.

3. Post-KiKK studies

KiKK reignited the childhood leukemia debate (Nussbaum, 2009) and resulted in studies being carried out in the UK (COMARE, 2011), France (Sermage-Faure et al., 2012) and Switzerland (Spycher et al., 2011). Together with a geographical study from Germany (Kaatsch et al., 2008a) using data from the KiKK study region, four datasets now exist of similar design and with the same endpoints, distance definitions and age categories. These four studies have similar findings. In particular, the leukemia increases in the 5 km zone observed in the four studies are very close as shown in Table 1.

Körblein and Fairlie (2012) pooled the data of acute leukemia in children under 5 within 5 km of NPPs from these four studies: the standardized incidence ratios (SIRs) within 5 km are shown in Table 1. Their analysis yielded an overall SIR of 1.37 in the 5 km zone (90% CI: 1.13–1.66, p = 0.0042, one-sided).

To study the shape of the distance dependency of leukemia risk, Körblein and Fairlie (2012) also carried out a joint Poisson regression of the four datasets using linear and linear-quadratic dependencies on reciprocal distance. The linear-quadratic model yielded a better fit to the data – see Fig. 1.

The best fit, judged by the Akaike information criterion, was obtained with a model estimating the excess rate in the 5 km zone relative to the rate in the >5 km zone. The authors found a SIR of 0.95 (0.90–1.00) at distances $r \geq 5$ km. From the ratio of the two SIRs, a relative risk of 1.37/0.95 = 1.44 (p = 0.0018) was obtained. With a one-sided test, the result was highly significant (p = 0.0009). This pooled analysis provides statistically strong evidence of leukemia increases near NPPs which contradicts statements by the above authors to the contrary.

In view of the preponderance of the above evidence, there is little dispute about the association of childhood leukemia incidence with proximity to nuclear facilities. The remaining arguments are about its causes and energy policy implications.

4. What are the causes of increased cancers near NPPs?

The KiKK authors stated "the reported findings were… not to be expected under radiation biological and epidemiological considerations" and that the increase in leukemias "remains unexplained". They added that "no risk factors of the necessary strength for this [KIKK] effect are known for childhood cancer and specifically childhood leukemia". (Kaatsch et al., 2008b).

Since the first leukemia cluster near nuclear facilities was discovered in 1984 near the Sellafield nuclear facility in the UK, there has been much discussion as to possible causes for these cancer increases. However we are little closer to ascertaining them than we were in the 1980s.

Various suggestions have been put forward, including a postulated virus from population-mixing (Kinlen, 2004); an unusual response to infectious diseases in children (Greaves, 2006); genetic predisposition to cancer; or a combination of factors. None of these addresses KiKK's central finding that the increased cancers were

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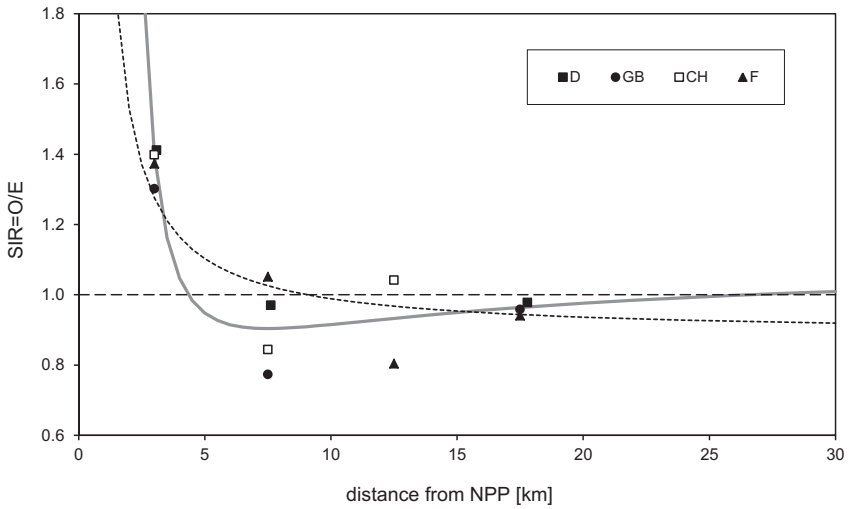

Fig. 1. Leukemia incidence near nuclear plants in Germany (D), Great Britain (GB), Switzerland (CH), and France (F), and results of joint Poisson-regressions with a linear (dotted line) and a linear-quadratic (solid line) dependency on reciprocal distance.

strongly associated with proximity to NPPs. Also, Gardner et al. (1990) had suggested that the significant association of childhood leukemia with distance from Sellafield could be explained by occupational doses received by fathers, but the increased leukemia incidence was restricted to one village, Seascale, ie where the mothers all lived, whereas the fathers lived across a wide area.

The increases could possibly be due to a combination of factors as there may be interactions between environmental exposures we are yet to understand. For example, synergistic effects between radiation and chemicals may act to increase cancer risks.

(Koppe et al., 2006; Wheldon et al., 1989). This reason was not explored by the KiKK study, but risks from the small chemical releases at NPPs are estimated to be very low. Most observers discount effects from chemicals from NPPs although few if any studies have examined the matter, it would appear. On the other hand, radiation exposures can result in increased leukemias and radioactive NPP emissions are large.

Körblein (2008) has put forward another explanation: that the dose-risk relationship may be curvilinear (ie supralinear) instead of linear. This would explain why the effect is limited to the immediate 5 km zone vicinity of NPPs and why radiation spikes, which occur during ~annual episodes of nuclear refuelling, may have a disproportionate effect on radiation risk. The latter point is discussed below.

5. Hypothesis: *in utero* exposures from environmental releases

It is hypothesised that the increased cancers result from radiation exposures to the embryos/fetuses of pregnant women near NPPs from their radioactive releases. This hypothesis was initially mooted (Fairlie, 2010) earlier: this article expands the theory, contains new information on emission spikes and pooled data results, and attempts to explain the $\sim 10^4$–10^5 fold gap between estimated doses and observed risks.

The theory stems from KiKK's observation that the increased solid cancers were mostly "embryonal", ie babies were born either with solid cancers or with precancerous tissues which developed into full-blown tumours after birth. As shown in Fig. 3, this happens with leukemia as well (Fig. 4).

The theory also originates from KiKK's finding that the increased incidences of infant and child leukemias were closely associated with proximity to the NPP chimneys.

The hypothesis is that approximately annual spikes in NPP releases may lead to nuclide intakes by pregnant women living nearby resulting in the labelling of their embryos/fetuses. These nuclide concentrations could be long-lived and result in high doses to radiosensitive tissues in embryos/fetuses and in subsequent cancers. This suggestion was first made by the late Professor Edward Radford, former Chairman of BEIR III Committee, more than 30 years ago during testimony to the Ontario Select Committee on Hydro Matters (Provincial Government of Ontario, 1978) which examined possible health effects of tritium discharges from nuclear facilities near Toronto, Canada.

The hypothesis has five main elements. First, the cancer increases may be due to radiation exposures from NPP air emissions. Second, spikes in NPP emissions may result in large increases in dose rates to populations within 5 km of NPPs. Third, the observed cancers may arise *in utero*. Fourth, both doses and risks to embryos/fetuses may be greater than currently understood. And fifth, pre-

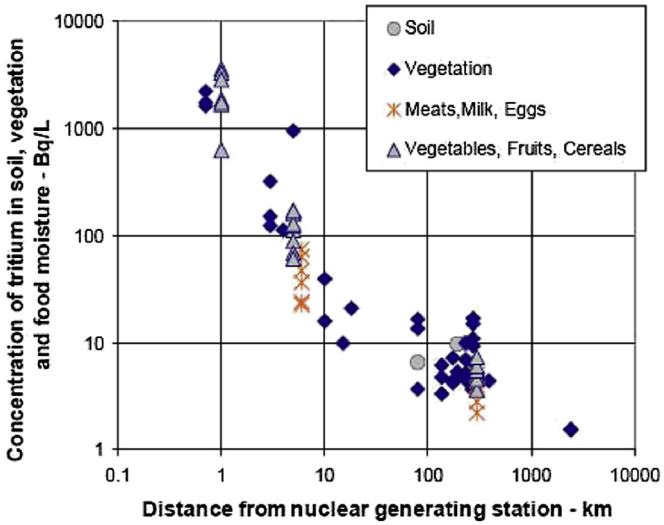

Fig. 2. Tritium concentrations in vegetation food moisture near Canadian NPPs. Source: Reproduced with permission of the CNSC from Tritium in the Canadian Environment: Levels and Health Effects. Report RSP-0153-1. Prepared for the Canadian Nuclear Safety Commission under CNSC contract no. 87055-01-0184 by Ranasara Consultants and Richard Osborne. Data from Health Canada (2001).

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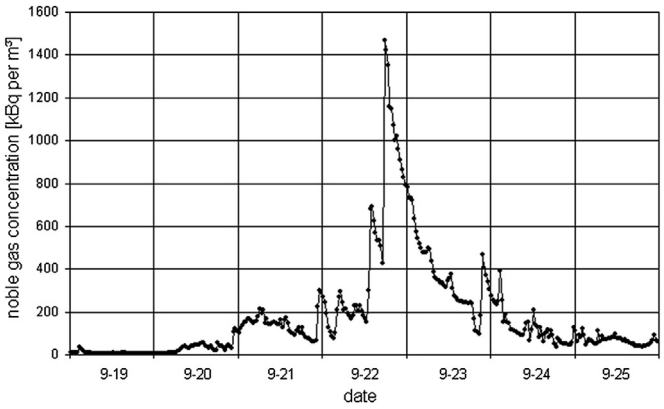

Fig. 3. Noble gas concentrations at Gundremmingen C, September 19–25, 2011

natal haematopoietic cells may be unusually radiosensitive. Together these five factors offer a possible explanation for the 10^4–10^5 fold discrepancy between estimated radiation doses from NPP releases and observed KiKK effects. These factors are discussed below.

5.1. Radioactive releases from NPPs

The main sources of radiation from NPPs are their relatively large nuclide releases. See Table 2. The KiKK study had NPPs releases in mind when it was established as distances were measured from station chimney stacks and the monitored areas were predominantly downwind from the stations.

Other forms of radiation, such as direct gamma rays and neutrons from reactor cores; skyshine from core neutrons reflected back to earth; electro-magnetic radiation from power lines and radioactive contamination of workers' homes, e.g. by workers' clothing have also been considered but none bears close scrutiny when compared with the relatively large nuclide releases from NPPs.

Radioactive releases from NPPs occur through emissions to air and discharges to rivers in Germany and to sea in other countries. Air emissions cause most of the collective dose to humans. Radiation risks from NPP nuclide releases are rarely discussed in the literature. Evrard et al. (2006) estimated leukemia incidences near French nuclear installations using geographic zones based on dose estimates from gaseous emissions. This study did not find an increased leukemia risk in the highest exposure group but it relied on very low dose estimates in the μSv/y range whose derivation was unexplained and which may contain significant uncertainties.

The air emissions of C-14 and H-3 are relatively large and result in elevated nuclide concentrations in vegetation and foodstuffs near NPPs. The half-life of tritium (H-3) is 12.3 years and carbon-14 5730 years. In the absence of German data on tritium

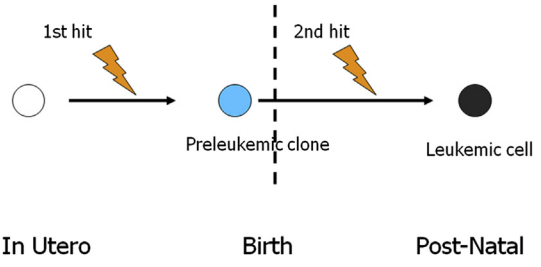

Fig. 4. Scheme for leukemogenesis after Rössig and Jürgens (2008).

Table 2
Indicates GBq amounts of main nuclides emitted to air from a typical German NPP, Gundremmingen B + C, in 2003.

C-14 to air	H-3 to air	Kr-85 to air	I-131 to air
1.1 E+03	1.1 E+03	1.2 E+02	2.50 E−03

Source: European Commission, 2005).

concentrations in vegetation and food moisture near NPPs, Canadian data is shown (Fig. 1). Although tritium emissions from Canadian heavy water nuclear reactors are considerably larger than from PWR and BWR reactors, the same pattern of raised nuclide concentrations in vegetation and food is expected to occur near all reactor types. Tritium concentrations near other reactor types are available in the UK Government's annual RIFE publication (RIFE, 2011).

Fig. 1 shows that the risk–proximity relationship is proportional to $1/r^2$, as the slope (for distances under ~10 km) is approximately minus 2. The observed tritium concentration–distance relationship is similar to the risk–distance relationship within 5 km indicated by the KiKK study's regression analysis.

5.2. Spikes in air emissions of nuclides

Spikes in NPP nuclide emissions occur when reactors are opened and depressurised about once a year to replace spent nuclear fuel with fresh fuel. Fig. 2 indicates half- hourly noble gas concentrations in the chimney stack of Gundremmingen NPP unit C in Germany during September 19–25, 2011, in the 38th calendar week. Tritium, C-14 and other nuclides are expected to be released at the same time as noble gases.

According to the International Physicians for the Prevention of Nuclear War in Germany (IPPNW, 2011) who obtained this data, the normal noble gas concentration during the year is about 3 kBq/m^3. During inspection/refuelling in September 22 and 23, 2011, this increased to 1470 kBq/m^3, i.e. ~500-times the normal concentration. Noble gas emissions during this calendar week were about half of estimated annual releases (Körblein, 2011).

People near and downwind from nuclear power stations may be exposed to higher exposures during these emission spikes than from releases during the rest of the year: estimates range from 20 to 100 times higher. The reason is partly related to the duration of the release, as short-term releases produce narrow plumes. Longer durations mean that the width of the plume increases (widths vary non-linearly as a fractional power of duration) with the result that individual doses decrease per Bq emitted and vice versa. The reason is also partly due to the fact that spikes result in high concentrations in environmental materials and in humans, which have longer retentions and thus higher doses, especially OBT and OBC.

The UK National Dose Assessment Working Group (NDAWG, 2011) published guidance on short-term releases to the atmosphere. This stated that, using the cautious assumption that all the release in a year is over a single short period then "…doses from the assessment of a single realistic short-term release are a factor of about 20 greater than doses from the continuous release assessment." An older study (Hinrichsen, 2001) indicated that doses could be as much as 100-fold greater. The actual dose increase will depend on many factors, including proximity to the reactor, plume width, wind speed, wind direction and the diets and habits of local people. It is implicit here that inhalation doses dominate over ingestion doses. Close to NPPs (ie within a few km) most environmental transport models predict greater inhalation doses than ingestion doses, whereas much further away, the opposite is the case.

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At present, doses to critical groups near NPPs are still calculated using the continuous release assessment method.

5.3. Observed cancers may arise in utero

As stated above, the hypothesis stems from KiKK's observation that the increased solid cancers were embryonal, ie babies were born with cancers. Rössig and Jürgens (2008) have suggested that infant leukemias may be initiated *in utero* and only develop into full leukemia after birth. This is shown schematically in Fig. 3.

5.4. Radiosensitivity of embryos and fetuses from in utero exposures

The best data on radiation risks from *in utero* exposures, i.e. on the radiosensitivities of embryos and fetuses, are from the UK Oxford Survey of Childhood Cancer (OSCC) by Stewart et al. (1956) in the 1950s to 1970s. More recently, Wakeford (2008) comprehensively reviewed the data from OSCC and more than 30 similar studies world-wide. The latter studies confirmed the presence and size of the risks of *in utero* radiation initially found by Stewart in <15 year-olds. Wakeford and Little (2003) had previously estimated from OSCC and other data that the relative risk (RR) of leukemia in children aged under 15 from abdominal exposures to X-rays was 52 per Gy (95% CI: 28, 76).

If we apply this risk estimate to the suggested hypothesis, we need to make three corrections. First, the leukemia risk in <5 year-olds (as determined by KiKK) is greater than in under 15 year-olds because the peak years for leukemia diagnoses in children are in 2–3 year-olds. This would result in the average relative risk being greater by a factor of ~1.5.

Also, most (>90%) OSCC exposures were in the last trimester, and it has been estimated (Stewart et al., 1958) that risks from exposures in the first trimester are perhaps 5 times greater than those from exposures in the last trimester.

In addition, these risks arose from external X-rays, whereas the risks are hypothesised to arise from internal exposures. Few estimates exist, but Fucic et al. (2008) have suggested that *in utero* risks from internal nuclides are 4–5 times greater than from X-rays, although the risk here was spontaneous abortion rather than leukemia. Multiplying these factors, the relative risk of leukemia in 0–5 year olds from internal nuclides in the first trimester could be

$$\begin{aligned} \mathrm{RR} &= 52\ \text{per Gy(OSCC)} \times 1.5(0-5\ \text{yr}-\text{olds}) \\ &\quad \times 5(1^{\text{st}}\text{trimester}) \times 5(\text{internal vs X}-\text{rays}) \\ &= 1,950\ \text{per Gy} = \sim 2\ \text{per mGy} \end{aligned}$$

If correct, this suggests that human embryos and fetuses are considerably more radiosensitive than currently acknowledged. It also suggests that average background gamma radiation of about 1 mGy per year (excluding radon) could be a significant cause of naturally-occurring childhood leukemia. This has already been suggested (Wakeford et al., 2009; Mobbs et al., 2009).

Interestingly, the above relative risk estimate of ~2 per mGy is similar to risk estimates from other studies. Stevenson (2001) observed that the doubling dose for childhood leukemia is about 2 mSv after *in utero* exposures in the first trimester. And Stewart et al. (1956) estimated that the abdominal doses from X-rays to pregnant women in the UK Oxford Survey of Childhood Cancer were of the order of a few mGy.

It is recalled that KiKK found leukemia risks in children near NPPs were ~doubled (RR = 2.19). From the above discussion, this doubling suggests that *in utero* doses in the KiKK study may have actually been several **mSv**, contrary to official German dose estimates (Deutscher Bundestag, 2007) for 1 year-olds of several **μSv** per year, ie 1000 times smaller. I attempt to explain this discrepancy in dose estimates below.

5.5. Increased radiosensitivity of pre-natal haematopoietic cells

Finally, we need to consider the radiosensitivity of the fetal haematopoietic system, i.e. blood-forming cells in bone marrow and lymphatic tissues. These tissues contain stem cells which are self-renewing: when they divide, some daughter cells remain stem cells, so the number of stem cells stays about the same. Radiation-induced mutations to stem cells could result in increased malformation rates of white blood cells.

Bone marrow contains a relatively high number of stem cells and it is likely to be among the most radiosensitive of embryonic/fetal tissues: this has been hinted at on at least three occasions. In 1990, after Gardner et al. (1990) had published their paternal pre-conception irradiation hypothesis, the BMJ published letters questioning aspects of the hypothesis. A letter by Dr J A Morris (1990) stated that, assuming mutations were the cause of the 10-fold increase in leukemia 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. See also Morris (1992). He earlier had stated (1989) the latter seemed the most plausible mechanism even though the exposure pathways were unclear.

A few years later, Lord et al. (1992) indicated the same thing when they suggested 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/fetal stages.

More recently, Ohtaki et al. (2004) suggested the same in their study of chromosome translocation frequencies in white blood cells of Japanese A-bomb survivors irradiated *in utero*. They found that precursor lymphocytes of the fetal haematopoietic system may be highly radiosensitive, perhaps 100 times more so than post-natal lymphocytes. From this study, Wakeford (2008) surmised that radiosensitive primitive cells (whose mutation may result in childhood cancers) remain active throughout pregnancy, including during the third trimester but not after birth, although it is not known at present why this should be the case.

This apparent increased radiosensitivity of haematopoietic cells before birth might be a major factor in explaining the discrepancy between official dose estimates and the observed level of risks in the KiKK study.

6. Can the 10^4–10^5 fold discrepancy in doses/risks be explained?

The explanation that NPP radionuclide emissions may cause cancer increases was dismissed by the German Strahlenschutzkommission (2008). It stated "The additional radiation exposure caused by nuclear power plants is lower, by a factor of considerably more than 1,000, than the radiation exposure that could cause the risks reported by the KiKK Study". The KiKK authors stated "While annual natural radiation exposure in Germany is about 1.4 millisieverts and the annual average exposure from medical examinations is about 1.8 millisieverts per year, radiation exposure near German nuclear power plants is a factor of 1000–100,000 less."

This means that any explanation will have to account for a gap of 4–5 orders of magnitude. Could official dose and risk estimates be incorrect by such a large amount? Initially, this appears unlikely but the above KIKK doses/risks are to children not

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embryos/fetuses: large differences exist in their doses and radiosensitivities.

In order to explain the discrepancy between estimated doses from NPPs and risks observed by KiKK, it is necessary to multiply the estimated risk (fatal cancers per mSv) by the estimated dose (mSv). This means we have to separately examine doses and risks: first we examine dose estimates.

6.1. Incorrect dose estimates

Current dose estimates could be incorrect for the following reasons.

(i) spikes in radionuclide emissions may result in a 20-fold (NDAWG, 2011) to 100-fold (Hinrichsen, 2001) increases in doses to people downwind of NPPs compared to annual averaged doses.
(ii) Stather et al. (2002) have estimated that, following tritium intakes during pregnancy, fetal tritium concentrations are 60% higher than in the mother. Tritium is a major emission from all NPPs, mainly in the form of tritiated water vapour (HTO). It is expected that tritium will be a major contributor to local population exposures. The UK Health Protection Agency (HPA, 2008) has estimated that doses to embryonic and fetal tissues are raised by factors of 1.5–2 compared to adult tissues following exposures to air releases of tritiated water vapour. These studies showed similar increases for ^{14}C.
(iii) Unfortunately official tritium dosimetry is plagued with problems and misunderstandings, as I have shown previously (Fairlie, 2008). The radiation weighting factor (w_R) used by International Commission on Radiological Protection (ICRP) for tritium is still unity despite considerable radiobiological evidence that it should be doubled (AGIR, 2007) or trebled (Fairlie, 2007). In addition, the official ICRP tritium model continues to underestimate doses from organically bound tritium (OBT). In persons chronically exposed to tritium, OBT doses are about four times greater than HTO doses at equilibrium (AGIR, 2007). In the case of chronic but fluctuating exposures to populations near NPPs, it is difficult to estimate OBT doses as equilibria may not occur. A conservative estimate would be to double HTO doses to take account of OBT formation.
(iv) Richardson (2009) has added that, for various metabolic reasons (including that the ICRP model does not account for human growth) radionuclide dose coefficients (Sv per Bq) for infants are approximately 10 times greater than those for adults.

Multiplying together the above dose factors (20 from spikes $\times$ 2 from fetal tritium concentrations $\times$ 2 from RBE $\times$ 2 from OBT $\times$ 10 from human growth $=$ $\sim$1600) which could partly explain the above discrepancy. Obviously this is a very rough estimate and no accuracy is implied, but the above factors indicate that official dose estimates could be incorrect.

Indeed, large uncertainties may exist in estimates of internal doses from intakes of NPP emissions (Fairlie, 2005). This was the main conclusion of the report of the UK Government's CERRIE Committee (2004) on internal radiation risks. Unquantified uncertainties may render dose estimates unreliable especially where evidence exists to the contrary. In other words, when we try to ascertain the reasons for the wide gulf between very small estimated doses and large observed risks, we should not dismiss radiation as a possible reason just because dose estimates are too low. Unfortunately, uncertainties in doses are rarely examined: they were not examined by the above German, UK and French studies on KiKK, or by KiKK itself.

6.2. Incorrect risk estimates

Radiation risks to embryos/fetuses are poorly characterised, especially the risks from uptakes of radionuclides by embryos/fetuses. Richardson (2009) has observed that the hazard from internal radiation exposures increases markedly with younger people. From the Japanese bomb survivor data, he estimated that radiation risks per unit dose are about 10 times greater for infants than for adults. Also, Ohtaki et al. (2004) found that precursor lymphocytes of the fetal haematopoietic system may be highly radiosensitive, perhaps 100 times more so than lymphocytes in infants.

In total, as stated above, we need to multiply the latter increased risk of $\sim$100 together with the $\sim$1000 factor from dose uncertainty. The product could reach the 10^4–10^5 fold difference between estimated doses from NPPs and risks observed by KiKK. Again, no accuracy is implied but a possible explanation has been put forward. It has to be admitted that some other studies, for example from exposures at Chernobyl, Techa River, and the atmospheric test bombs in the 1950s and 1960s, do not reveal such increased levels of childhood leukemias. However this lack of findings could be due to several factors including ascertainment bias, ie were all the leukemia cases found in these studies?

In addition, recent evidence from an environmental review has suggested that current estimates for the radiosensitivities of animals (and perhaps humans) are too low (Garnier et al., 2012). This review showed that radiosensitivities in free-living animals were an order of magnitude higher than those predicted by conventional models which used laboratory animals.

7. Conclusions

A possible biological mechanism to explain the KiKK observations is that NPP emission spikes result in the radioactive labelling of embryo and fetal tissues in pregnant women living nearby. Such nuclide concentrations could result in high exposures to haematopoietic tissues in embryos and fetuses. Cumulative radiation doses and risks to specific organs and tissues in embryos/fetuses from nuclide uptakes during pregnancy are not specifically considered in ICRP publications.

The leukemia increases observed by KiKK and other studies may arise *in utero* as a result of fetal exposures to incorporated radionuclides. It has been suggested that babies are born pre-leukemic and full-blown leukemias are only diagnosed after birth. A radiation spike might produce a pre-leukemic clone, and after birth a second radiation hit might transform a few of these clones into full-blown leukemia cells.

In view of these concerns, it is recommended that the following information should be published for NPPs

- radiation exposures from episodic NPP emissions, i.e. from spikes
- estimates of the resulting radiation doses to bone marrows of developing embryos
- estimates of subsequent risks of leukemia to infants and young children, and
- confidence intervals around these dose and risk estimates

It is also recommended that a wider case–control study of leukemias near European NPPs be established using, as far as possible, the same methodology as the KiKK study, in particular the measurements of precise distances between cancer cases and NPP stacks.

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