Childhood leukemia near nuclear facilities Response to Report 2010:21 for the Swedish Radiation Protection Authority SSM (Wojkik A and Feychting M)

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1. Background

The health effects of radioactive discharges from nuclear sites have been an area of debate since the discovery by a TV company of the child leukemia cluster near the Sellafield reprocessing plant in Cumbria UK in 1983. Since then, childhood leukemia clusters have been found near a significant number of nuclear sites. Though the link to radiation exposure from the various plants has been regularly dismissed on the basis of dosimetric considerations the matter remains a concern of the public who understandably feel that since radiation is the main known cause of the disease, that a causal relationship with radioactive discharges is the most likely explanation. The matter was addressed by the European Committee on Radiation Risk in 2003 and more recently in 2010. The ECRR pointed out that the only impediment to accepting that the discharges caused the excess risk was the belief that the internal chronic low dose exposures of the children or their parents to internal radionuclides could be modeled by reference to external large acute gamma radiation doses experienced by Japanese wartime populations. This, as the committee pointed out, was not science. The ECRR drew attention to the child leukemia clusters that had been discovered (Table 1) and the fact that errors in the radiation risk model of the ICRP which was based on the external doses received by the Japanese lifespan studies of between 400-100 times were necessary. But these levels of error could easily be accomodated by considering the local doses to target DNA from hot particles, from nuclides that had high binding affinity for DNA or for other exposure regimes where the averaging methodology of the ICRP model was inappropriate.

Nuclear Site	Year	Defined ICRP	Notes
		risk	
		multiplier	
^a Sellafield/ Windscale, UK	1983	100-300	Well studied by COMARE: high level of discharge to atmosphere and sea
^a Dounreay, UK	1986	100-1000	Well studied by COMARE: particle discharges to atmosphere and sea.
^a La Hague, France	1993	100-1000	Particle discharges to atmosphere and sea: ecological and case control studies
^c Aldermaston/ Burghfield, UK	1987	200-1000	Well studied by COMARE: particle discharges to atmosphere and rivers
^b Hinkley Point, UK	1988	200-1000	Discharges to offshore mud bank
d Harwell	1997	200-1000	Discharges to atmosphere and river
b Kruemmel,	1997	200-1000	Discharges to atmosphere and river
Germany			

Table 1 Studies establishing excess leukemia and cancer risk in children living near nuclear sites (from ECRR2010).

d _{Julich,}	1996	200-1000	Discharges to atmosphere and river
Germany			
b Barsebaeck,	1998	200-1000	Discharges to atmosphere and sea
Sweden			
^b Chepstow,	2001	200-1000	Discharges to offshore mud banks
UK			
Germany all;	2007	1000	Various types aggregated
KiKK			

^aReprocessing plants discharging to sea; ^bNuclear power station discharging to sea or river; ^cAtomic weapon and nuclear material fabrication plants; ^dAtomic research with discharges to local rivers

In 2007 the German BfS published the much discussed KiKK study which was the result of an investigation of childhood cancer near the 16 German nuclear power plants. The result of that study, which covered a long enough period and a large enough sample to provide persuasive statistical power, was the finding of a significant excess risk of child leukemia inside a distance band radius of 5km from the nuclear plants (Spix et al 2008). The 2010 report by Wojkik and Feychting (Hereafter WF) discuss the issue in a report published by the Swedish SSM.

2. The principal arguments and conclusions of WF

WF embrace and propagate the basic errors of the conventional dosimetric approach to the problem. They conclude that the possible doses to the children or the parents are too low to have caused the leukemias. In particular, they argue that the possible doses from releases from nuclear sites are far below natural background doses. Thus it is possible to say that since there are not child leukemia excesses in areas of higher natural background radiation, it cannot be the radiation exposure from the nuclear sites that is the cause of the leukemias.

In this argument they fall into the trap, which I will elaborate below, of assuming that the concept of "absorbed dose" can be compared across different types of exposure, different nuclides, different fractionations and with a linear relationship between dose and effect.

WF also advance one other argument, and that is that child leukemia excess is found in areas where nuclear plants were about to be built but were not built. Finally, WF conclude, probably correctly, that in Sweden the population size is too small for sufficient statistical power to investigate the issue of child leukemia near nuclear plants.

To summarise, their conclusion is that:

- a. There are no *radiation related* excess child leukemia risks near nuclear plants and any excess found must be due to some other as yet undiscovered cause
- b. There is no point in investigating the issue in Sweden as the populations are too low, given the rate of child leukemia of about 6 per annum per 100,000 children at risk, to obtain any statistically significant result.

Wojkik (who I have met discussed these issues with at the SSM in Stockholm) is a physicist and his expertise is in radiation cell biology. He believes in the concept of absorbed dose being applicable across different types of exposure, external and internal. Feychting is an epidemiologist who has published in the area of electromagnetic radiation and child leukemia.

3. Why WF is wrong

Since the paper by WF was published, new research from France has supported the existence of childhood leukemia excesses near nuclear sites (Sermage-Faurier et al 2012). Thus we can take it as a reality that there is some effect. The question is the cause.

I will deal with the main issue first. The arguments relating to differences in hazard between external and internal radiation exposures have been described at length in ECRR2003 and ECRR2010 and I refer the reader to those publications (downloadable from www.euradcom.org). Briefly, it is the ionization density at the target DNA that causes the genotoxic damage which leads to childhood leukemia and to other late manifestations of genotoxic damage to the DNA, for example adult cancer. It is easy to show that for certain types of internal exposure, especially from man made nuclides emitted by nuclear sites, the ionization at the target is much greater than is modeled on the basis of "absorbed dose" which is an average quantity. These discharges include nuclides which bind to DNA (Strontium-90, Uranium, Tritium) and nuclides whose exposure is in the form of particulates (Uranium, Plutonium) or high atomic number elements which bind to DNA and amplify natural background radiation (Busby and Schnug 2009). Furthermore, it has been shown unequivocally that the use of absorbed dose to quantify the child leukemia yield after internal exposures from Chernobyl are in error by roughly the same amount (400-old) as that needed to explain the nuclear site leukemias (Busby and Scott Cato 2000, Busby 2009). These analyses show that the ICRP approach employed by WF is certainly wrong and that it is the radiation that causes the leukemias since in the case of the infants who were in the womb at the time of the Chernobyl fallout, there is no alternative explanation. The population mixing explanation advanced by WF as a possible cause for the nuclear site leukemias cannot occur in the womb.

The other argument from WF is that excess risk exists at sites where a nuclear power station was planned but not commissioned. This is easily disposed of. Such sites are always near the sea and in areas of high rainfall. Therefore local populations are exposed to radionuclide contamination from fallout and reprocessing plant contamination of coastal sediment. It is of interest that WF argue that there are excess risks at such sites where nuclear plants were planned yet their diagram Fig 1 shows the results of only one such study and the result does not in fact show a significant excess risk or support the argument they use.

4. Where WF is correct and how to take this forward: adult cancer rates.

The argument that the Swedish population is too small for a useful study of child leukemia near nuclear sites may be valid. This assumes that the child leukemia numbers found in a study of the combined populations living near the Swedish nuclear sites finds a modest excess, of about 2-fold the same as that found in Germany and France. But it may be that the results will show much higher rates, in which case, as with the Sellafield cluster, there could be statistical significance.

But there is a better way of dealing with this issue, which has been avoided by all those who have examined it. It is to look at adult cancer rates in those living near nuclear sites. The logic is inescapable. Child leukemia is caused by genetic damage, and this means that the child has been exposed to genotoxic agent (Busby and Fucic 2006, Busby et al 2009). Near the nuclear sites, the assumption is that this is from radioactive discharges. But it is difficult to study this in children as the background rates are very low, about 6 per 100,000 per year.

But if there is a genotoxic agent and if it operates through some enhanced hazard due to internal exposure, then it should also cause increases in adult cancer. In this case, the rates are much higher. For example, the background rate of breast cancer is about 250 per 100,000 and so a study of breast cancer near a nuclear site is much more statistically powerful. I have carried out breast cancer risk studies near four nuclear sites in the UK and found an approximate 2-fold excess in each of these, especially in those living near contaminated coastal sediment (Busby et al 2001, 2002, 2006).

5. Political dimensions

There is now a great deal of evidence that internal radiation exposure carries excess risk for cancer over that which is modeled by the ICRP system (see e.g. The Lesvos Declaration 2009). In 2004 a study of Chernobyl effects in Sweden showed an 11% increase in cancer per 100kBq m⁻² Cs-137 contamination (Tondel et al 2004, 2006). This defines an error in the ICRP model of roughly 600-fold, and can be seen as support for the ECRR2003 risk model which was published one year before it (ECRR2010). The failure of the SSM to take these pieces of evidence seriously, particularly to ignore the infant leukemias after Chernobyl, and to continue to attempt (as with the SSM publication of WF) to dismiss the issue is irresponsible and biased in favour of the nuclear industry. It is to be hoped that the political and legal system in Sweden begins to realise that this issue is one where an urgent change of approach is necessary to protect the Swedish public and that the SSM can no longer be seen as responsible in this area. It is worrying that the Swedish Medical Officer of Health, Lars Eric Holm is ex-head of the ICRP and also of the precursor organization to SSM and this is a clear conflict of interest which should be addressed. Since there is now considerable suspicion in this area, it is suggested that any study carried out involves independent epidemiologists and is transparent.

6. Conclusion

There are real increased risks of child leukemia in those living near nuclear sites. The excess risk is caused by internal exposures to radioactive discharges from the plants. Rather than looking at child leukemia, a study in Sweden should focus on cancer risk in adults living near nuclear plants since these would provide sufficient statistical power and adult cancer, particularly breast cancer is also caused by radiation exposure and would provide a simple test of the hypothesis that emissions from nuclear plants have genotoxic effects on local populations.

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