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Justification of radiation exposures of members of the public and workers: review of existing practices.
New and important information.

Dear Sir/Madam,

Under Article 6.2 of the Council Directive 96/29/Euratom of 13 May 1996 and the Justification of Practices Involving Ionising Radiation Regulations 2004 Part 3 Regulation 10: "Review of existing practices", paragraph 4(a) "acquisition of new and important evidence about [the] efficacy or consequences of a practice" I hereby request a review of the Secretary of State's Decision:

> "Regulatory Justification of the Class or Type of Practice being: "*The generation of electricity from nuclear energy using oxide fuel of low enrichment in fissile content in a light water cooled, light water moderated thermal reactor currently known as the EPR designed by AREVA NP.*"" hereinafter "the Decision".[1]

The evidence I submit bears particularly on the consequences of the practice defined in Paragraph 1.7 of the Decision in terms of *detriment to health, safety and the environment.*

The evidence contradicts the Secretary of State's assertions in para 1.7

> that *the potential detriment is small [and] well understood;*
>
> that *the established regulatory regime … actively and effectively works to keep detriments within acceptable limits;*

[1] https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/47936/666-decision-EPR-nuclear-reactor.pdf

> that *the risk of health detriment from the building and operation of EPRs in the UK is very low.*

The evidence contradicts an implicit assumption made by the Secretary of State in the following passage from para 1.7:

> *As a proportion of the overall radiation to which members of the public are exposed from all sources, including natural sources, the evidence he has reviewed suggests that the contribution from any EPR would be very small*

the implicit assumption being that that the risk of health detriment is necessarily proportional in a linear fashion to the overall radiation to which members of the public are exposed from all sources, including natural sources.

Similarly, the evidence contradicts the assumption that

> *the radiation dose which members of the public would receive from the normal operation of an EPR on an annual basis would be below detectable risk levels in the context of overall radiation exposure.*

New and important evidence on the safety of the radiation risk model upon which EU Directives and domestic regulation depend.

Background

The issue of the genotoxic hazard from internal radionuclides was considered sufficiently important for Environment Minister Michael Meacher and Health Ministers Yvette Cooper to set up the Committee Examining Radiation Risks of Internal Emitters CERRIE in 2001. Mr Meacher was removed from office before CERRIE completed its deliberations and two agreed joint studies which would have assisted the process were cancelled. The final report was not agreed by all the members. Since CERRIE new and important evidence which informs this issue has been published in the peer-review literature.

1. Evidence for the failure of the Hiroshima Studies.

Directives in the European Union and Regulations in the UK depend upon cancer risk factors published by the International Commission on Radiological Protection, an independent NGO. These risk factors are based primarily on the doses and cancer yield of the Japanese Lifespan Study (LSS). This epidemiological study was set up to depend upon comparison of exposed and unexposed individuals and the cancer yield in those exposed compared with unexposed controls. Forensic examination of the methodology and decisions made over the period of the study reveals that significant errors were introduced which resulted in incorrect conclusions being drawn. In particular it appears that the original control group, those who were not in the city at the time of the bombing, was discarded in 1973 when it appeared that their inclusion was suggesting a high level of cancer in the exposed groups. Furthermore, evidence presented in the Royal Courts of Justice in the Pensions Appeals Tribunal (Abdale and Others vs. Secretary of State for Defence; June 13th 2016) showed that all the epidemiological groups were exposed to rainout and subsequent contamination of the city by Uranium nanoparticles. The LSS study did not address internal contamination resulting from inhalation of the nanoparticles. New evidence which has emerged since CERRIE reported in 2004 demonstrates that exposure to Uranium particulates carries

levels of genetic hazard which are not incorporated into the ICRP risk model. The matter is outlined in a letter which has been accepted for publication by a leading peer-review journal and will be printed in December 2016. A draft of the paper is attached as Appendix A.

2. Evidence of genetic damage leading to heritable effects in those exposed to Chernobyl fallout in Europe.

A review of evidence relating to the genetic effects of chronic internal exposure to contamination from the Chernobyl accident was published in a leading peer-review journal in January 2016 [Schmitz-Feuerhake et al., 2016]. It examined the considerable evidence relating to increases in congenital defects and other heritable conditions in Chernobyl-exposed individuals but also discussed other situations where significant excess risk was shown to exist in offspring of exposed parents. The current ICRP radiation risk factor for such effects is obtained from mice because the LSS study (above) was unable to find any heritable effects in children of the exposed groups. However we now see that the chosen comparison groups were unsafe for the purposes of obtaining evidence of harm (see 1 above). The aggregated evidence presented in Schmitz-Feuerhake et al 2016 demonstrates unequivocally an error in the current risk factor for heritable defects of approximately 1000-fold. It shows that heritable defects occur in offspring of those exposed to internal doses of less than 10mSv and furthermore that the dose response is not linear, as assumed by the ICRP and current legislation.

3. The ethical basis of the ICRP and regulations which depend on it.

EU Directives and UK Regulations which control radiation exposures encapsulate a decision to tolerate low levels of risk of cancer and genetic damage. The current 1mSv annual dose limit for members of the public enshrined in EU Council Directive 96/29/EURATOM and its successor 2013/59/EURATOM is based on a permitted level of absolute cancer risk of 1 in 1 million. The current relative cancer risk factor of the ICRP is about 0.5 per Sievert. Thus an exposure of 1 mSv carries with it an excess risk of 0.5/1000 or 1 in 2000. This is considered acceptable to Society. Regarding heritable damage, the current doubling dose published by the ICRP and agreed also by the United Nations Scientific Committee on the Effects of Atomic Radiation UNSCEAR is 1Sv. Thus an annual dose of 1mSv, the limit for effective dose for public exposures under Directive 2013/59EURATOM, carries an excess risk of 1 in 500 of heritable effects in the offspring of parents exposed. This is considered to be acceptable as a side effect of the agreed development of nuclear technology. The new and important evidence referred to above shows that this factor is in error by approximately 1000-fold when applied to internal chronic exposures. This issue is relevant to releases of radioactivity from nuclear plant and other sources, to contamination of the sea and watercourses, and other releases which are currently controlled on the basis of the 1mSv level. The issue may be less relevant to external exposures from X-rays or other external sources.

4. CERRIE

The disagreements which were documented in the CERRIE majority and minority reports and evidence which was brought forward by the various members of the committee can reasonably be revisited in the light of new evidence which has emerged since then; this evidence includes but is not restricted to the above.

5. Evidence presented during consultation leading to the Decision
This submission is made without prejudice to any future challenge to the Decision which may reference advice from Health Protection Agency (HPA) reproduced in Annexes E and F of the Decision.

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p.p. Richard Bramhall

Reference:
Schmitz-Feuerhake, **Busby C**, Pflugbeil P Genetic Radiation Risks-A Neglected Topic in the Low Dose Debate. Environmental Health and Toxicology. 2016. 31Article ID e2016001. http://dx.doi.org/10.5620/eht.e2016001.

Appendix A

Below is a paper which has been accepted for publication by a major journal and will shortly appear. The version here has been anonymised to avoid problems of confidentiality pending publication.
[Note Dec.2018, this is http://www.genetics.org/content/204/4/1627]

The Japanese Lifespan Study as a secure basis for radiation legislation

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It is claimed the Lifespan Study (LSS) of Japanese A-Bomb survivors in Hiroshima and Nagasaki has given definitive information on the relation between exposure and genetic damage, expressed as cancer and heritable effects in offspring of those exposed. The LSS is presented as the gold standard in radiation epidemiology [2]. The LSS results are the basis of legal limits for exposure and are employed to dismiss evidence showing that health effects from Chernobyl [3], Fukushima thyroid cancers [4] and child leukemias near nuclear sites [5] etc., somehow cannot be causal because the "dose is too low". According to the LSS study one must receive a dose of 1Sv (1000mSv, 500 times natural background) to have a 42% excess chance of cancer, and as for the offspring, there have been no increased frequencies of abnormalities or genetic effects detected. Unfortunately there are some worrying problems with the epidemiological methods that were employed, specifically with the key issue of the choice and then abandonment of the control group.

The common understanding of the LSS study is that groups of individuals with known doses are compared over their lifespan with zero dose control groups who were not there. The belief is that:
The ABCC and later RERF assembled a lifespan study LSS cohort of 120,000 individuals (100,000 exposed at various known levels and 20,000 controls Not in the City (NIC) at the time of the bombing).

What is not generally known is that the NIC controls were discarded in 1973 because they appeared to be "too healthy". The 1973 ABCC report wrote:

In order to ascertain the effects of radiation exposure it is necessary to compare the mortality experience of the population exposed to ionizing radiation with a comparison control population. For this purpose a group of people who were not present in the cities was included in the sample. . . .
The mortality experience of the NIC comparison group has been very favourable. . . [and] would have the effect of exaggerating the difference in mortality between the heavily exposed population and the control group. . .
[[6] pp 6-7, ABCC LSS Report 7, 1973]

At that point, in 1973, the original control was discarded in favour of shifting to the lowest dose group as the control, something which should never be done in the middle of an epidemiological study. The substitution with a new lowest-dose control group was followed by the use of mathematical regression methodology. This approach is questionable because of assumptions listed below many of which are now known to be wrong:

- The concept of "absorbed dose" employed by the study is a legitimate measure of biological damage from internal exposures. i.e., that internal exposures can be translated into a "dose" that carries the same biological hazard as the identical external exposure dose.
- The dose response relation is linear or at least monotonic, a necessity for regression.
- There was no fallout, which would have contaminated all the exposed groups equally.
- Acute exposures carry the same proportional hazard as chronic exposures.
- The Japanese survivor population is representative of the general (western) public

These assumptions have been reviewed elsewhere [7,8].
The use of the lowest dose group as control is now standard in all nuclear worker studies [9] which, like the LSS, employ linear regression to establish risk factors. This is because if the national population is employed as a control, the nuclear workers show a "healthy worker effect" (HWE): their relative risks for cancer are lower than the general public. But this does not permit the lowest dose group to be a valid control unless it is also known that there is a linear or monotonic dose response. Also, the true value of the HWE is unknown. The risk factor for cancer obtained from regression is the slope of the best straight line that can be fitted to the excess cancer risk in groups aggregated according to their external dose measured by a film badge. The bigger the dose, the bigger the effect, is the assumption, though the data do not show this.

Another problem is that nuclear workers are from a different social class than the national population, and are fundamentally healthier (as are e.g. physicians, optometrists, soldiers, university lecturers etc.). So their relative risk for cancer should be lower. But how much lower? The epidemiological method used now makes the (unfounded) assumption that the effects of radiation on the lowest dose group can be

set at zero. It is the point (0,0) for the regression line. Two observations are relevant here. First, the lowest dose group (usually with the most individuals in it) is a group of workers who mostly work on the contaminated sites (rather like the Hiroshima survivors did), perhaps inhaling radioactive particles. So they should be compared with similar workers who are from a completely different industry with no radioactive contamination (or with the national population, adjusting for the healthy worker effect).

There is some evidence about the real HWE value from data published by the UK National Radiological Protection Board of the relative risk of cancer in UK nuclear workers stratified by length of time working in the nuclear industry [10]. The level of healthiness (HWE) shifted from about 64% of the National rate at start of employment to nearer 90% after 10 years, i.e., the healthy worker effect rapidly disappeared. This could be seen as an effect of exposure. Use of 64% for the HWE results in significant 30-40% excess risk in the lowest dose group for nuclear workers. To return to the linear dose-response regression point, all the published data stratified by dose group define a dose response that is biphasic: it goes up at the lowest dose, then comes down, then goes gently up again at the high doses. There are plausible biological reasons for this (especially in the case of congenital effects where the end point is seen only after birth, and at some dose level pre-birth viability stops). Drawing a straight line though these data points results in the wrong answer to the question of risk: the risk factors at low dose, medium dose and high dose are different. Thus it is not epidemiologically valid to employ regression methods for nuclear workers, any more than it is for the Hiroshima survivors.

The LSS populations, like the nuclear workers, lived on the contaminated sites of the bombed towns for many years after the bomb. Contamination was a consequence of the black rain [11]. The up-draught from the rising fireball at Hiroshima and Nagasaki sucked in moist maritime air which cooled with altitude and condensed on the 95% un-fissioned Uranium nano-particles created in the plasma. This produced black rain over an area which included all of the dose groups used for the LSS study, for which dose was calculated by distance from the hypocentre. Uranium was measured later in the contaminated areas [12]. The existence of any fallout was denied, and external acute doses were calculated based on distance using data from experiments carried out in the Nevada desert. The last twenty years has seen changes in the understanding of the biological effects of radiation. This includes realisation that for internal exposures to elements that have chemical affinity for DNA, and to nanoparticles, the concept of absorbed dose is worthless [13]. Uranium has a high affinity for DNA and a large number of studies have now shown effects which define large errors in the “dose” based approach [8, 14]. The European Union has recently funded research on this issue [15].

The black rain contamination of Hiroshima and Nagasaki resulted in continuous chronic internal exposure of all the dose groups and controls by inhalation and ingestion of uranium particles. The only accurate way to establish the real effects is to employ a truly unexposed group and abandon regression methods. In 2009 Wanatabe et al. compared age and sex specific cancer rates between 1971 and 1990, using the adjacent Okayama prefecture as a control [16]. This period was chosen because cancer data prior to 1971 is insufficiently accurate. It was found that there were significantly greater levels of cancer in all the exposed groups, including the LSS

lowest dose controls, compared with the Okayama control group but also (to a lesser extent) compared with an all Hiroshima control group. When compared with the Okayama group, the highest cancer effect per unit dose was seen in the 0-5mSv group, the lowest dose LSS group, where there was a 33% excess risk of all cancer in men at external doses estimated at 0-5mSv. The authors write: *the contribution of residual radiation, ignored in LSS, is suggested to be fairly high.* This falsifies all the LSS epidemiology. Similar criticisms were made by Sawada [11, 17] who examined immediate deterministic effects of radiation (epilation, diarrhea), which were reported from areas more than 5km from the hypocentre where black rain fell but where the prompt gamma doses were effectively zero.

Similar control group errors in the LSS genetic studies were addressed long ago by de Bellefeuille, who questioned the sex-ratio results [18]. The LSS researchers focused on sex-ratio, the number of boys born to the number of girls, a well-accepted measure of genetic damage [19]. The direction of the effect depends on whether the mother (egg) or father (sperm) are irradiated. The LSS geneticists reported no apparent genetic damage, but they analysed results from families in which both parents were irradiated, and thus the effects cancelled, and they employed the wrong controls. Use of the NIC controls gives a sex-ratio effect in the expected direction [20]. This issue is discussed in a recent review by Schmitz-Feuerhake et al (2016) of heritable effects reported at very low doses of internal exposure. Results from Chernobyl studies clearly demonstrate that the current genetic risk factor is in error by about 1000-fold, and that the dose-response is not linear. There are significant increases in major congenital malformations in offspring of those exposed to internal doses less than 1mSv [21].

I suggest that this adherence to the LSS as a definitive answer to the public's fears is a result of a scientific culture of acceptance that goes back over a long period of time, and that few researchers have had the time or funding to forensically examine the many (often obscure) reports needed to open up the methodological black boxes. I submit that belief in the validity of the Japanese A-Bomb studies is unsafe, and that the health effects of low level internal exposures to radioactivity should be re-evaluated.

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