Low Level Radiation Campaign
Times Building
South Crescent
Llandrindod Wells
LD1 5DH
lowradcampaign@gmail.com

to: Matt Clarke
Deputy Director
Nuclear Energy Generation and International
BEIS 3 Whitehall Place,
London SW1A 2AW

22nd May 2017 updated from original of 24th April 2017

Dear Matt,

Your letter of 30th March denies our Application for review of the Justification of the proposed European Pressurised Reactor at Hinkley Point. You say we have provided no new and important evidence, and you cite *Regulation 10 of the Justification Regulations*. The relevant Guidance on Regulation 10[1] says, in paragraph 47: ... *An application for a review should provide sufficient evidence to convince the Justifying Authority of the need for a review*. So the hurdle we have to clear is defined by what is *sufficient to convince.* In other words, it's subjective. I have a lot of sympathy for your position; you are not an expert on the effects of radiation and you have applied to Public Health England for advice. You had no alternative but PHE have not served you well because, at some point, any subjective test has to withstand a test of objectivity. The advice PHE have given you is not objective; it is evasive, selective and misleading and it consistently misrepresents our submission.

The misrepresentation starts at the top where (their para 1) PHE reject our outline criticisms of the conventional radiation risk model. Please note they have skipped over the preceding passage in which we said the new evidence contradicts the Justification document[2] - specifically, the Secretary of State's opinion that:

> *the potential detriment is small [and] well understood;*
>
> that *the established regulatory regime … actively and effectively works to keep detriments within acceptable limits;*
>
> and that *the risk of health detriment from the building and operation of EPRs in the UK is very low.*

All these statements are falsified by the evidence we presented — a wide-ranging review[3] published in a reputable peer reviewed journal showing that children whose parents were exposed to Chernobyl fallout have far higher rates of genetic defects than predicted by the International Commission on Radiological Protection (ICRP). To dispose of this evidence PHE would have to show that the high rates don't exist or that they are caused by some other factor. Nowhere in their note do they attempt to do either.

In the rest of this letter I refer to the genetic defects review as *ISF*, after the lead author.

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[1] The Justification of Practices Involving Ionising Radiation Regulations 2004 (SI 2004 No 1769) Guidance on their Application and administration May 2008

[2] See para.1.7 of Justification (dated 2010)

[3] *Genetic radiation risks: a neglected topic in the low dose debate*: Inge Schmitz-Feuerhake, Christopher Busby, Sebastian Pflugbeil Environ Health Toxicol 2016; 31: e2016001. Published online: January 20, 2016

The doses in the reviewed studies were less than 10milliSieverts. According to the ICRP risk model, no increases in birth defects would be detectable at such doses. The discrepancy between what ICRP predicts and what ISF reports is so extreme that it cannot be left hanging; it demands explanation. Thus ISF says

> *We conclude that the current risk model for heritable effects of radiation is unsafe. The dose response relationship is non-linear with the greatest effects at the lowest doses. Using Chernobyl data we derive an excess relative risk for all malformations of 1.0 per 10 mSv cumulative dose. The safety of the Japanese A-bomb epidemiology is argued to be both scientifically and philosophically questionable owing to errors in the choice of control groups, omission of internal exposure effects and assumptions about linear dose response.*

This evidence is the central issue in our application. PHE's response is to ignore it and, without any scientific analysis, reject our outline of why it's a mistake to assume that dose response is linear. They state that:

> *The use of a linear non-threshold dose-risk relationship for assessing cancer and hereditary risk at low doses is the model best supported by the available evidence. This is the international consensus agreed by bodies such as ICRP etc. etc.*

so they simultaneously make both a Type 1 error and a Type 3 error as defined by ISF:

***Type 1**, The dose response problem. For genetic damage, increasing dose will not linearly increase effects since at high doses there will be sterility or fetal loss;*

***Type 3**, The philosophical method problem. If data is interpreted though a particular scientific model, evidence which cannot fit the model will be ignored, dismissed or invisible.*

The scale of the Error in ICRP's Heritable Effects risk factor

PHE complain (paras. 2c, 3, & 4) that they cannot find evidence for our assertion that ISF demonstrates a roughly 1000-fold error in the current risk factor for heritable effects. It's quite simple and I'd expect professional radiation protection specialists to find it obvious. The error in the studies ISF reviewed is the difference between the number of cases reported and the number you'd expect if the ICRP2007 risk coefficient for heritable effects were correct. In other words, it's the number that has to be used to multiply the ICRP coefficient to explain the observed effects.

Before you can quantify such errors you need the doses. National and international authorities including the United Nations estimated doses after Chernobyl, some with whole-body monitoring of the gamma-emitter Caesium-137, others mapping ^{137}Caesium from the air. Levels can be converted to external doses using published methodology.[4] Thus the average doses to the parents are approximately known. For example here's a table from UNESCO:[5]

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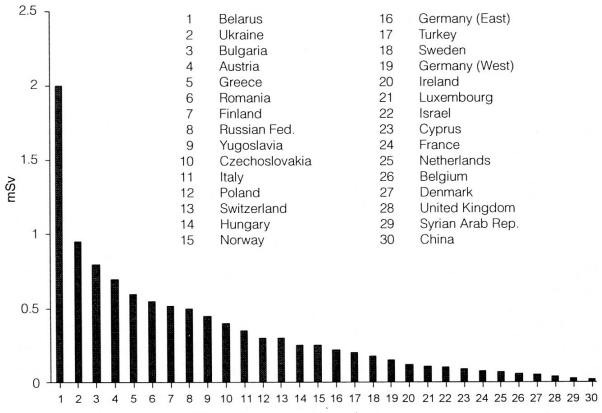
[4] e.g. the US Environmental Protection Agency FGR12 Part II, the program Microshield, the graphs in the UK Handbook of Radiological Protection 1972

[5] Savchenko VK *The Ecology of the Chernobyl Catastrophe: Scientific outlines of an International Programme of Collaborative Research*: UNESCO 1995 p. 83



Average first-year committed effective doses from the Chernobyl accident

The last item of information you need is the ICRP risk factor. It is 0.2% per Sievert, meaning that if a normal human population is exposed to a dose of 1 Sv (1000mSv) the natural rate of heritable defects will increase by 0.2%.

Below is a selection of the results in ISF showing how the Error is calculated.

Belarus: Lazjuk et al. 1997.[6] This paper compared 1992-1994 with 1982-1985 in three regions of different exposures.[7] For this calculation I have looked at the medium exposure region and assumed a conservative mid-range dose level of 5mSv. The excess risk apparent in the data is 49%. That means 5mSv caused a 49% increase. But ICRP states that 1000mSv would cause an increase of 0.2%. If that were true, a dose of 5mSv (200 times smaller) would cause an increase of only 0.001% (0.2 divided by 200). The observed 49 divided by the expected 0.001 is 49,000. Here is the arithmetic.

$$\text{Error} = \left(\frac{\text{ICRP dose standard } \mathbf{1000mSv}}{\text{Fallout dose } \mathbf{5mSv}} \right) \times \text{Effect observed } \mathbf{49\%}$$

$$\text{ICRP standard effect } \mathbf{0.2\%}$$

An example of calculating the scale of the Error in ICRP risk factors for heritable effects (figures used are from Lazjuk et al. 1997, reference 22 from Table 1 *Increase of congenital malformations after exposure by the Chernobyl accident* in *Genetic radiation risks: a neglected topic in the low dose debate* in Inge Schmitz-Feuerhake, Christopher Busby, Sebastian Pflugbeil Environ Health Toxicol 2016; 31: e2016001.)

On a calculator enter 1000 ÷ 5 x 49 ÷ 0.2 = and the answer should be 49000

[6] https://doi.org/10.1002/stem.5530150734

[7] International authorities defined what was meant by low, medium and high levels. The *medium exposure* region covers a wide range between 37 kiloBecquerels per square metre (37kBq/m^2) and 555kBq/m^2 conferring a similarly wide range of annual doses; between 0.44mSv and 6.7mSv.

Belarus: Feschchenko.[8] This study looks at differences between high contamination and low contamination regions and a strict definition of malformations. The differential effect is 30%; the differential between high dose and low can conservatively be put at 10mSv. So in the calculation the figure *ICRP dose standard* remains 1000mSv; the *Fallout dose* is 10mSv; the *Effect observed* is 30%; the *ICRP standard effect* is still 0.2%

Enter 1000 ÷ 10 x 30 ÷ 0.2 = 15000.

Ukraine: Wertelecki.[9,10] This is a prolonged study of estimated cumulative internal doses to the gonads comparing two regions with different contamination levels. The differential internal dose was 26mSv (estimated by whole-body monitoring of gamma from incorporated Caesium 137). The differential effect in Neural Tube Defects (spina bifida etc) is 59%.

Enter 1000 ÷ 26 x 59 ÷ 0.2 = 11346.

I note PHE's use of the word "consistent" in the sentence (their para. 3)

> *... international consensus reviews have not identified consistent health effects in the children of those exposed to Chernobyl fallout.*

The apparent inconsistency of the observations is an artefact arising from the assumption that the effects are linear — in other words proportional to the dose. The observed effects in ISF are not linear. It is fairly obvious that at higher doses foetuses are more likely to be so damaged that they die in the womb, so records of congenital malformations fail to capture the data. The WHO *Chernobyl Forum* noted the same phenomenon:

> *There has been a slow but steady increase in congenital malformations recorded in both high and low contamination areas, but the increase does not show a dose-response pattern ... there were [...] [fewer] congenital abnormalities in the high contamination areas compared with low contamination areas ...* [11]

This *data loss* issue is presented in ISF (p. 9) but PHE's note fails to offer you any advice on it.

Further evidence

The congenital malformations reviewed by IFS are in line with an even more recent review[12] of genome damage, genomic instability and radiosensitivity in people born in Ukraine, Belarus and Russia after Chernobyl.

PHE misdirection on LSS, internal contamination and Uranium

PHE's paragraph 2(b) is smoke and mirrors. We pointed out that the Lifetime Survivors Studies (LSS) of the Hiroshima and Nagasaki bombs provide no information on the health effects of internal exposures. This has long been admitted. It follows that stretching the risk factors obtained from the external studies to predict the effects of internal exposures is questionable. PHE cite the *Brief Description* of the LSS.[13] It doesn't change anything, it is merely an assessment that doses from internal radioactivity would be low, which is not evidence that the effects of internal radioactivity would be small; in other words it assumes that the effects are

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[8] http://www.ncbi.nlm.nih.gov/pubmed/12064450
[9] https://doi.org/10.1542/peds.2009-2219
[10] https://doi.org/10.1111/cga.12051
[11] 2006 report of the World Health Organisation's *Chernobyl Forum Expert Group "Health"*. (pp. 86-87)
[12] Follow-up studies on genome damage in children after Chernobyl nuclear power plant accident: Fucic A, Aghajanyan A, Druzhinin V, Minina V, Neronova E Arch Toxicol *DOI 10.1007/s00204-016-1766-z*
[13] http://www.rerf.jp/shared/briefdescript/briefdescript_e.pdf

linear. PHE's second paragraph — an overview of the components of the fallout — makes the same point and is equally irrelevant.

PHE's next paragraph 2(c) cites two studies by Sakata[14] and Ozasa[15] which could mislead you on the significance of the post-bomb rainfall. PHE is suggesting that we are arguing that the rain itself played a role in damaging health, but in fact the rain is significant only because it establishes that there was a mechanism for delivering fallout to the ground. The Sakata paper confirms this:

> *(The) expert consensus indicates that additional fallout due to gravitational settling was not possible in the absence of rain [...] .*

Apart from this rainout mechanism these studies are completely irrelevant to the case we made. Their essence is that the 93,000 LSS survivors were asked *Were you caught in rain just after the atomic bombing?* The *Yes* and *No* and *Don't know* answers were analysed in terms of the health outcomes and there was no difference. That does not mean the fallout had no effect — it just means getting wet or staying dry were irrelevant. The rain brought down the radioactive particles and then it evaporated so the particles became available to be resuspended and everyone in the region could inhale them, ingest them, and absorb them through skin lesions. This is a chronic cumulative exposure situation that lasted for decades.

PHE has not advised you on the Wanatabe and Sawada studies we cited. Wanatabe's analysis of fallout and cancer[16] found

> *the contribution of residual radiation, ignored in LSS, is [...] fairly high.*

Sawada[17,18] examined hair loss and diarrhoea which are symptoms of high radiation doses and were reported from areas more than 5km from the hypocentre where the black rain fell though the prompt gamma doses were effectively zero. That tells us that the fallout caused acute health effects in people who got no external irradiation. PHE are trying to divert your attention away from that.

Uranium: the Photoelectric Effect and Uranium binding to DNA

In the first three sentences of PHE's 2(c) they are playing down the significance of the interaction between natural gamma and Uranium in body tissue causing high densities of ionisation (Einstein's photoelectric effect). They refer to computer modelling of particles by Tanner et al.[19] Since 2003, when we first raised the photoelectric effect in the context of radiation protection, there have been other computer studies. The following passage from a summary of evidence on radiation risks[20] says

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[14] Sakata R, Grant EJ, Furukawa K, Misumi M, Cullings H, Ozasa K, Shore RE. (2014) Long-term effects of the rain exposure shortly after the atomic bombings in Hiroshima and Nagasaki. Radiat Res. 182:599-606.

[15] Ozasa K, Sakata R, Cullings HM, Grant EJ. (2016) Association of Acute Radiation Syndrome and Rain after the Bombings in Atomic Bomb Survivors. Radiat Res. 185:604-15

[16] Wanatabe T, Miyao M, Honda R and Yamada Y., 2008 Hiroshima survivors exposed to very low doses of A-Bomb primary radiation showed a high risk of cancers. Env. Health. Prev. Med. 13: 264-270

[17] Abdale and Ors. Vs The Secretary of State for Defence. Pensions Appeals Tribunal; Royal Courts of Justice, London June 13th - July 4th 2016

[18] Sawada S., 2007 Cover up of the effects of internal exposure by residual radiation from the atomic bombing of Hiroshima and Nagasaki. Med. Confl. Surviv. 23: 58-74

[19] Tanner RJ, Eakins JS, Jansen JT, Harrison JD. (2012) Doses and risks from uranium are not increased significantly by interactions with natural background photon radiation. Radiat Prot Dosimetry. 151:323-43.

[20] Extract from *Statement on the proposed re-opening and further operation of the San Onofre nuclear plant in California* C. Busby 2012 (San Onofre is now being decommissioned)

Two computer studies by the radiation establishment have conceded that there is an enhancement of dose near such particles [21, 22] but both have shown that the enhancement is finite but modest. The studies are both flawed by the same methodology, which is to dilute the energy into a large volume of tissue. The experimental measurements with gold foil[23] and gold nanoparticles[24] and other computer analyses which examine the dose close to the particles[25] show quite clearly that the effects are those of high enhancement of dose largely predicted by theory.

I note that the Tanner study too finds an enhanced effect. I can't assess whether it suffers the same flaw as the Pattison and Eakins papers (refs 21,22) by diluting the energy into too large a volume of tissue. This topic is already on the agenda for a meeting between members of the BEIS/ Nuclear NGO Forum and CoMARE because PHE's elementary mistakes about the photoelectric effect are incorporated word for word in the Justification of the EPR at Hinkley Point (the subject of our application). I hope for constructive discussion.

The CoMARE agenda also includes Concerted Uranium Research in Europe (CURE) and the question of whether cell culture results show high rates of transformation associated with Uranium particles. This might show whether there are effects in living tissue and cut through the computer modelling that PHE always rely on. The extent to which Uranium binds to DNA in living tissue, which is obviously relevant but is disputed, is another topic for discussion.

Dropping the controls

In PHE's paragraph 2(a) they mislead you over control groups in epidemiological studies. Our Application pointed out that the LSS originally included a control population of NIC (Not In City) people who had been away from the cities when the bombs dropped, so they were not externally irradiated and were almost certainly far less contaminated internally. The controls were scrapped 23 years into the study, which destroyed any chance that LSS might quantify the effects of internal contamination. PHE's note explains that the controls were dropped because:

it was seen that there were significant differences in the underlying disease rates between the exposed survivors and the 'not in city' control group (the control group were markedly more healthy compared to those exposure survivors who received very little exposure from the bombs).

This reveals PHE's thinking — it's the linear assumption again. They assume that survivors who were closest to the hypocentre must have had the greatest damage to their health, and those right on the edge, where dose approached zero, must represent a negligible effect indistinguishable from the unexposed controls. Fallout however was distributed far beyond the distance that the prompt

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[21] Pattison J E, Hugtenburg R P, Green S, (2009) Enhancement of natural background gamma-radiation dose around uranium micro-particles in the human body. J.Royal Society Interface doi: 10.1098/rsif.2009.0300. http://rsif.royalsocietypublishing.org/content/early/2009/09/23/rsif.2009.0300.abstract

[22] Eakins, JS, Jansen J. Th. M. and Tanner R. J. (2011) A Monte Carlo analysis of possible cell dose enhancements effects by Uranium microparticles in photon fields Radiation Protection Dosimetry (2011), Vol. 143, No. 2–4, pp. 177–180 doi:10.1093/rpd/ncq398

[23] Regulla D F, Hieber L B, Seidenbusch M, (1998) Physical and biological interface dose effects in tissue due to X-ray induced release of secondary radiation from metallic gold surfaces. *Radiat. Res.* 150: 92-100.

[24] Hainfeld J F, Slatkin D N, Smilowitz H M, (2004) The use of gold nanoparticles to enhance radiotherapy in mice. *Phys. Med. Biol.* 49: N309-N315.

[25] Howard C V, Elsaesser A, Busby C, (2009) The biological implications of radiation induced photoelectron production, as a function of particle size and composition. *International Conference; Royal Society for Chemistry NanoParticles 2009.*

gamma rays and neutrons could reach and, as Wanatabe and Sawada demonstrate (refs.16,17,18), it caused health effects which show no such trend with distance from the hypocentre. So it is reasonable to conclude that the unexposed NIC controls were scrapped for political reasons when the fact that their health status was better than the low to zero group showed that fallout was a significant hazard.

Similarly we pointed out that the UK nuclear industry worker studies have no unexposed control groups. PHE tell you this is because

> *... nuclear workers ... health is regularly monitored [so they] tend to exhibit significantly lower incidences of disease compared to the general population... .*

There's a paradox; the health of Japanese survivors has been closely monitored too, yet they are less healthy than the general population represented by the NICs. So PHE is telling you that health monitoring is beneficial for UK nuclear workers but has failed to benefit the survivors of Hiroshima and Nagasaki. Can that really be true?

Our Application (page 7) cited control group errors in the LSS genetic studies. De Bellefeuille questioned the sex-ratio results which LSS focused on.[26] The ratio of male to female births is a well-accepted measure of genetic damage and the direction of the effect depends on whether the mother's ovum or the father's sperm are irradiated.[27] The LSS geneticists reported no apparent genetic damage, but they had analysed results from families in which both parents were irradiated, and thus the effects cancelled each other; also they used inappropriate controls. Use of the NIC controls gives a sex-ratio effect in the expected direction.[28] PHE haven't addressed these issues, though they are clearly relevant to the genetic effects (above) that are the main aspect of our application.

The British nuclear test veterans' pensions appeals: High Court June 2016

This section has nothing to do with what PHE says; it has to do with your own letter. You referred to evidence presented in a pensions appeal by veterans who attended nuclear weapons tests in the 1950s. I should declare an interest; LLRC covered costs for the vets' representatives and witnesses. You quoted paragraph 300 of the Court's Decision. I'm pasting the same passage here for everyone to see because it shows how biased the Judge was:

> *Nothing has emerged from the evidence of the [Battersby/Smith] expert witnesses and the materials they cite to throw any doubt on the ICRP model. Indeed our evaluation of this evidence merely confirms the reasons given by others for rejecting it. The positive case that risk assessment using ICRP is flawed is rejected. The rejection is not a matter of preferring one body of scientific opinion to another, but an acceptance of the consensus of scientific opinion against the unscientific assertions of another body of campaigners.*

You should take no comfort from this. The Judge applied a severe test of the role of expert witnesses. He applied it only to the witnesses called by the vets, ruling that as they are associated with the European Committee on Radiation Risk they are not impartial in the sense demanded by court rules in England. On that logic he excluded (i.e. ignored) all their evidence, though all of it

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[26] De Bellefeuille Paul., 1961 Genetic hazards of radiation to man Part I. Acta Radiologica: 56: 65-80]

[27] Scherb H, Voigt K 2011. The human sex odds at birth after the atmospheric atomic bomb tests, after Chernobyl, and in the vicinity of nuclear facilities. Environ Sci Pollut Res. 18:697-707

[28] Padmanabhan VT 2012. Sex Ratio in A-Bomb survivors. Evidence of radiation induced X-linked lethal mutations. In Busby C, Busby J, Rietuma D and de Messieres M Eds. Fukushima: What to Expect. Proceedings of the 3rd International Conference of the European Committee on Radiation Risk May 5/6th Lesvos Greece. Brussels: ECRR; Aberystwyth UK: Green Audit, 2012

is solidly based on papers published in reputable peer reviewed journals. He was also very critical of Carmel Mothersill who had given evidence in previous hearings. On the other hand he backed the ICRP on no evidence at all; I say *on no evidence* because the MoD called witnesses whose fields of expertise were so narrow that none of them could be cross-examined on the problems with the LSS and ICRP — the kind of criticisms I am making here. On these and other grounds the decision has been appealed. The barrister who acted for the Secretary of State for Defence has been reported to the Bar Standards Board for misdirecting the court.[29]

Ethical basis of ICRP recommendations (PHE section 4)

You say[30] PHE have noted a number of errors. I put my hand up to one; it's a typo — the ICRP doubling dose reduces to 1 in 500,000 for hereditary disease, as you say. We mustn't lose sight of the point that, in ISF, where the assessed doses were of the order of 1mSv the observed rates of congenital malformations show this doubling dose number is wrong by about 10,000 times. This means that the real risk from internal exposures to Chernobyl contaminants is about 1 in 50 at 1mSv. That figure is also apparent in the increases of infant mortality in England and Wales and the USA at the time of the global nuclear test fallout. PHE have not addressed this huge discrepancy. PHE reject the cancer risk factor we used - 0.5 per Sievert. They give a different value of 0.05[31] which they do not explain. I guess they are referring to ICRP Publication 103, Table A.4.4.[32] They do not mention that ICRP 103 also contains Table A.4.6 which gives an Excess Relative Risk factor for cancer of 0.5 per Gray (or per Sievert for the sake of discussion). This is the risk figure of 1 cancer in 2000 people per millisievert we cited in the Application. It is not a mistake; the question is which table to use. Table A.4.6 is a relative risk factor. Table A.4.4 appears to be an absolute risk factor, though PHE don't say so. My advisers say relative risk is the appropriate method as it is directly derived from epidemiology. A standard textbook says

> *The natural risk of getting cancer increases with age so the relative risk model predicts a higher number of cancer deaths than the absolute risk model*,[33]

so we can expect a difference in the values but the 10-fold difference is remarkable and demands explanation.

This is not a trivial matter. There is a dose limit for members of the public of 1millisievert per year resulting from all authorised practices.[34] There is also a Health and Safety Executive criterion expressed in terms of deaths per year. A risk level deemed "acceptable" is 1 death per year in a million people, often expressed as *one in ten to the six*.[35] It is based on the method which in 1988 HSE applied to the control of risk from nuclear power stations.[36]

We can compare HSE's "acceptable" death rate criterion with the cancer risk factors. According to the risk factor PHE has chosen, a 1mSv exposure will cause 1 in 20,000 people to

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[29] We have agreed with the Bar Standards Board that this matter is parked pending the outcome of the appeal.
[30] your para (e)
[31] PHE calls it "ICRP recommended factor for the risk to human health (detriment adjusted nominal risk coefficient)" without giving the source or making it clear whether this risk factor is actually for cancer.
[32] "Detriment adjusted nominal risk coefficient for cancer and heritable effects".
[33] Sumner D *Radiation Risks* ISBN 1 870781 04 X
[34] 2013/59/EURATOM *Article 12* Dose limits for public exposure 1. Member States shall ensure that the dose limits for public exposure shall apply to the sum of annual exposures of a member of the public resulting from all authorised practices. 2. Member States shall set the limit on the effective dose for public exposure at 1 mSv in a year.
[35] http://www.hse.gov.uk/risk/theory/r2p2.pdf
[36] The tolerability of risks from nuclear power stations (TOR) HSE Books 1992 ISBN 0 11 886368 1

develop cancer in their lifetime. Half of adult cancer patients in UK die within 10 years of diagnosis.[37] It follows that half of the 1 in 20,000 people will die within 10 years of diagnosis. That equates to 1 death in 40,000 which is 25 times higher than HSE's 1 in a million "acceptable" risk.

Taking a real world situation, doses from Sellafield and Drigg were 0.42mSv in 2015.[38] Using the absolute risk factor favoured by PHE, that dose gives a death rate of 1 in 95,000, which is roughly 10 times worse than the HSE criterion.

Looking at the six nuclear power station sites operating in 2015, the average public dose is 0.021mSv.[39] Using PHE's risk factor that average dose gives a death rate of 1 in 2 million. It's compliant with HSE. If on the other hand the 0.5 per Sv relative risk factor is valid, the six sites are already exceeding the acceptable death rate by 5 times - that is 1 in 200,000. That's just cancer deaths, not counting other diseases or heritable defects.

Finally I bring you back to the genetic effects of exposures to ionising radiation which are the topic of our Application for a Justification review. The BSS Directives require that the information submitted with any Application shall be new and important. The information we sent has emerged from areas affected by Chernobyl. It had not been published when the Justification of the EPR was determined (November 2010, as you say), so it is new. Most people would agree that it is important, and it is relevant because the EPR would routinely emit radioactive species that would affect the human environment. PHE haven't said anything about the genetic effects except by referring to an alleged consensus. Your response likewise fails to address the information. Instead you make peripheral or irrelevant points about other matters. I have responded to this shoal of red herrings in an attempt to cut through the misleading fog it stirs up, but I didn't really need to because our Application stands essentially unanswered.

Yours sincerely

Richard Bramhall

p.s. Today I hear that the Journal of Environmental Protection has just published a new study of infant deaths in Pennsylvania. It compares pre- and post-fracking rates indicating that the counties with high densities of wells have a statistically significant increased rate of deaths. In the same period the rate across the whole state decreased. The process of fracking releases Radium, Uranium and other naturally occurring radioactive materials from shale strata which then contaminate groundwater, so it seems to me that fracking is a "practice" in the sense meant by the BSS Directives and therefore has to be justified. Is there a relevant justification decision?

[37] http://www.cancerresearchuk.org/health-professional/cancer-statistics/survival/all-cancers-combined - heading-Zero

[38] https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/563010/RIFE21.pdf

[39] RIFE 21 for Hartlepool 0.022mSv, Heysham 0.023mSv, Hinkley Point 0.016mSv, Hunterston 0.025mSv, Sizewell 0.021mSv and Torness 0.02mSv.