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5th June 2018

Diane McCrea
Chair, Natural Resources Wales
c/o Kirsty Williams AM

Dear Diane McCrea

Some fundamental scientific issues relevant to Petition P-05-785 *Suspend Marine Licence 12/45/ML to dump radioactive marine sediments from the Hinkley Point nuclear site into Wales coastal waters off Cardiff*

I am grateful to Kirsty Williams, my constituency AM, for grappling with the complex issues underlying the Petition and for agreeing to send you this letter.

Lesley Griffiths said on 23rd May that *there is no scientific basis for any further testing or assessments to be done.* This is completely untrue. There are knowledge gaps and uncertainties underlying the assessment of radiological risk from the dump and in this letter I am setting out to define them and to explain that the gaps arise from false assumptions in modelling some aspects of radiation risk. I believe your Board's remit requires you to appraise the scope and significance of this information. I request a reasoned response. I urge you to suspend the marine licence while you consider this matter because a suspended licence can be reinstated, while releasing and remobilising the radioactivity in the mud is irreversible. Several AMs have invoked the precautionary principle with good reason.

The next section of this letter outlines how the current radiation risk model was developed and why it underestimates the risks of Uranium and Plutonium and fragments of the same elements. The section after that shows that Government advisers are fully aware that fragmentation of alpha emitting elements is problematic. This vindicates the mud dump campaigners' concerns especially in view of the lack of direct information on alpha emitters in the sediment and CEFAS' failure to report to you the indirect information they did find.

Since 1999 I have been involved in discussions with government, industry, regulators, academics, and advisors like COMARE. In a separate document which I shall prepare in the next seven days I'll provide documentary evidence of the increasing irrationality and evasiveness of their responses. Much of the information is recent. My point is that you have to make a decision on protecting the public and the environment at a time when a classic scientific revolution is occurring in the manner described by Polanyi and Kuhn.

Modelling radiation risk

EDF has said the sediment is not a radioactive substance as defined in any legislation. This is true. The issue, as many of the people who oppose the dump are aware, is whether the radioactivity it undoubtedly contains is of a potentially harmful nature. The fault lies in the inadequacy of the risk modelling that underlies the legislation; the pivotal issue is the inappropriate use of averaging in defining dose.

The early years of research on radiation effects concerned X-rays and radium. By the 1920s it was clear that the risks of both were poorly understood and efforts were made to improve protection standards. It is informative to note the difference between X-

rays and radium. X-rays are always external but, as the tragic history of the dial painters showed, radium is very dangerous when ingested, inhaled or absorbed and the 1930s saw strenuous efforts to develop safety standards. They were expressed in terms of microgrammes of radium in body tissue.[1] But at the same time a completely different kind of exposure to radium was developed by a British physicist, Herbert Parker. Known as the Manchester System it involved sealing radium in glass tubes which were carefully placed to irradiate cancerous tissues. Conceptually, this is an external exposure quite different to the radium that perfused the dial painters' bones, and it led to a conceptual turning point in radiation protection when Parker went to Oak Ridge in 1943 to establish a health physics programme for the U.S. atomic energy agencies. He extended the Manchester System's concept, shifting attention away from direct investigation of the effects of specific substances onto a new concept - radiation dose - which he could apply to radiation from all sources to assess workers' total exposure. He defined a unit of dose in ergs per gramme of tissue and called it the Roentgen Equivalent Physical. Its very name reveals the mindset; Wilhelm Roentgen was the discoverer of X-rays (for a long time they were called Roentgen rays). The source of X-rays is always outside the body, so we can see the understanding of dose, and hence risk, was now to be based on an external paradigm.

In 1948 America's Atomic Energy Commission pressed the National Council for Radiation Protection (NCRP) to develop safety standards for the growing nuclear industry. Internal and external radiation were considered in separate sub-committees. The external sub-committee completed its work quite quickly but the other was slowed down by the many complexities of internal contamination — questions about where radioactive elements go once they are inside the body, how long they stay there and what biological damage they do. In 1951 NCRP's Executive grew impatient and closed down the internal committee and the report of the external committee was stretched to cover internal radiation.

In 1950, American influence revived the old International X-ray and Radium Protection Committee (IXRPC) which had been dormant during the war. Only two of its members were still alive and one of those was an American who was Chairman of the American NCRP. In reality it was just an overseas branch of the NCRP but it was renamed the International Commission on Radiological Protection (ICRP) and in 1953 it adopted the NCRP report wholesale. This completed the paradigm switch; all exposures were to be seen in terms of energy deposited in a "phantom" - a schematic model of the human body comprising quite large bags of water representing different organs and tissue types. This was before the shape and function of the DNA molecule had been described.

Estimating the health effects

The Life-Span Studies (LSS) of people who survived the bombing of Hiroshima and Nagasaki are the only substantial studies of human exposures. Survivors' health is still followed up and radiation risk estimates are still based on them. Most people know the LSS studied people who were so close to the explosions that they received instantaneous doses of gamma rays and neutrons (high doses to people near the epicentre and lower doses further away). It is commonly thought that the health of these people was/is compared with so-called NIC control groups who received no radiation because they were Not In the Cities at the time. Few realise that the NIC controls were discarded in 1973 because they appeared to be “too healthy”. After

[1] see http://www.llrc.org/switcheroo.htm for more on the history.

1973 the method was to compare health outcomes in high dose groups nearest the explosion with the lower dose groups further away. So the first criticism of LSS as epidemiology is that it has no unexposed controls; it is like a drug trial that has no placebo group.

The second criticism is that LSS is totally silent on internal radioactivity. The doses were reconstructed in terms of gamma rays and neutrons only. No attempt was made to quantify internal contamination although Black Rain (black with Uranium) was widely though unevenly distributed across the region. LSS has no information on exposures to that or any fallout. Many of the areas where the black rain fell were so far away from the epicentre (>5km) that gamma/neutron doses were zero. Original data include reports of acute radiation sickness (hair loss, purpurea, diahorrea) from those distant areas but those people were not recruited to the LSS. The illness attributable to ingested and inhaled fallout is therefore lost. These criticisms of LSS were published recently in the reputable journal *Genetics*[2] following an opinion piece by Bertrand Jordan. They have not been answered with any relevant argument. Appendix 1 is an example; it's an email exchange between me and Dr. Jordan. I asked him to clarify his comments but his answer is just as baffling and irrelevant as his published reply to Busby.

Nuclear industry workers' studies

Official radiation protection agencies claim that studies of nuclear industry employees give confidence in the standards derived from the LSS, but the worker studies have the same flaws:- they use the lowest dose group as a control and have no unexposed controls. Proper epidemiologists would compare nuclear workers with a different industry — one that required similar qualifications and conferred similar rates of pay but didn't involve being anywhere near radiation sources or sources of radioactive pollution. The worker studies are silent on internal radioactivity:- most of the workers are employed on nuclear sites and are therefore at risk of inhaling radioactive particulates but the various dose groups are defined on the basis of external doses as measured by film badges.

Particles, particulates and Absorbed Dose

The Mud Dump campaigners expressed concern at the possibility that Uranium or Plutonium and other alpha-emitting elements are present in the sediment in particulate form. Before discussing this I want to clarify terms. An alpha particle resulting from the decay of an atom of Uranium or Plutonium is two protons and two neutrons bound together into a subatomic particle identical to a helium-4 nucleus. For the avoidance of confusion, the previous sentence is the only place in this letter where I use the word *particle.* I shall use *particulate(s)* or fragment for pieces of Uranium, Plutonium and Americium or mixtures of such elements. Even the smallest such particulates are massively larger than the subatomic alphas.

Absorbed Dose as employed as a quantity by ICRP is conceived as energy from any source averaged over substantial volumes of tissue, usually equivalent to the whole body or a region like the lung. It takes no account of differences in ionisation density, which is an obvious error where we have to consider particulates in body tissue. It is

[2] Letter from Busby http://www.genetics.org/content/204/4/1627
response by Jordan http://www.genetics.org/content/204/4/1631.
The original article by Jordan is http://www.genetics.org/content/203/4/1505

an averaging error, similar to believing that it makes no difference whether you warm yourself by sitting next to the fire or by eating a burning coal. Uranium and Plutonium are at the extreme of this anomaly. Alphas are relatively massive, they slow down rapidly, travelling just a few cell diameters and depositing all their energy into a minuscule volume of tissue. Appendix 2 is a short paper quantifying the doses delivered to spheres of lymphatic tissue within reach of the alphas emitted by single fragments of various sizes from 0.2 micron up to 5 microns. An example (edited here to match my terminology):

> *For Uranium, the table shows that for particulates as small as 0.2 microns diameter, average annual alpha dose to the lymphatic tissue surrounding them is about the same as (and additional to) the total average natural background dose of 2mSv. It rapidly increases for larger particulates.*

This makes nonsense of "absorbed dose" because it is modelled as an average energy transfer in a volume of tissue usually equivalent to the whole body or a region; for example, tracheo-bronchial lymph nodes are regarded as being part of the lung. EdF's recent statements about the mud dump refer to whole body doses — a presentational ploy intended to minimise the figures. I am not going to discuss EdF's comparisons with bananas, radon or cosmic rays unless you ask me to.

Alpha-emitting particulates are certainly emitted from nuclear power stations. UNSCEAR used to publish data (I don't know whether they still do).[3] I calculated the numbers recorded for Hinkley Point. I assumed they were all 1 micron Uranium Oxide and the answer is 7118millionmillion per year. The three reactors have been running for a total of 119 years (the A station 35 years; the B reactors 42 years each). If they have been emitting at the same rate that's 282thousandmillionmillion particulates small enough to inhale into the deepest parts of ones lungs. CEFAS reported none in the sediment; they used gamma spectrometry which does not detect alphas but it did show the daughters of Uranium and Plutonium. CEFAS failed to report that to you. There are other omissions, as you can see in the attached Green Audit report on the raw digital spectra.[4] (URL in footnote 4 below)
http://www.llrc.org/campaigns/muddump/4Feb2018HinkMudRept.pdf

In December I requested data from NRW and the other three environment agencies in UK on particulates in marine sediments. There is no data, except on debris that's physically large and radioactive enough to be detected by Groundhog beach-cleaning vehicles in the vicinity of Sellafield and Dounreay - that was 300 microns until 2009 when a slightly more sensitive Groundhog came into use.

I am part way through evaluating a Health Protection Agency and an extensive *Supporting Scientific Report*.[5] Some of the information is relevant to the mud dump:

> *It is useful to note that the size of airborne [fragments] determines their penetration within the respiratory tract, with*

[3] http://www.unscear.org/docs/publications/2000/UNSCEAR_2000_Annex-C-CORR.pdf shows the three Hinkley Point reactors released 3.6GBq of radioactive particulates in airborne effluents between 1990 - 1996.

[4] Analysis of Hinkley Point Jetty application mud sample digital spectra supplied by CEFAS in January 2018; Christopher Busby PhD. 4th February 2108
http://www.llrc.org/campaigns/muddump/4Feb2018HinkMudRept.pdf

[5] HPA (now PHE) *Health Risks from Radioactive Objects on Beaches in the Vicinity of the Sellafield Site"* J Brown and G Etherington HPA-CRCE-018 April 2011

> *[fragments] less than 10 micron aerodynamic diameter being able to penetrate more deeply.*

I don't need to comment on that.

> *For [fragment] sizes that are likely to be inhaled, the effective dose resulting from inhalation of a single [one] would be no greater than a few mSv for all age groups, based on currently available information. Corresponding lifetime risks for all age groups are therefore estimated to be very low.*

Of course this idea is based on the ICRP's average Absorbed Dose model. See Appendix 2 for the doses to the tissues that would actually be within the range of the alpha decays.

> *The possibility remains that larger numbers of [smaller fragments ...], perhaps resulting from the sequential break-up of larger [ones], could be inhaled. If small enough (ie, with aerodynamic diameters less than about 30 micron), these could penetrate to and deposit in the lungs. It is recommended that environmental monitoring data should be reviewed to determine whether this potential pathway of exposure needs further evaluation. […] An analysis should be performed to determine whether the sequential break-up of larger particles could give rise to a component of contamination on the beaches or in the local atmospheric environment that is distinguishable from the ubiquitous contamination present in the beach environment. If so, data from routine environmental monitoring programmes should be reviewed to determine if the available data indicate whether this component is present.*
> *Consideration should also be given to the additional monitoring and measurements that might be performed to identify and characterise a possible component of environmental contamination that might result from the sequential break-up of larger particles on the beaches. […]*

The Health Protection Agency (now Public Health England) is pointing out that this type of exposure will increase over time as larger fragments break up. This suggests it's advisable to characterise any particulates that might be at Hinkley Point before they are dug up and dropped into a high energy tidal recirculating system because two mechanisms will make more of the radioactivity available to cause health damage. First, smaller fragments are more environmentally mobile and more respirable. Second, smaller fragments confer proportionally greater doses because radiation only escapes from atoms on or near the surface. Decays from atoms within a fragment are absorbed by the fragment itself, especially if the direction of travel leads through the mass of material. As larger fragments break up, any given amount of particulate matter will become more mobile, be more easily inhaled into the deep lung and the lymphatic system, and will emit more radiation.

I shall write a further letter/report outlining evidence in the peer-reviewed literature showing health effects from weapons test fallout Chernobyl which are not explained by the ICRP risk model. I shall include accounts of how I and colleagues have brought the evidence to the attention of government ministers in the BEIS/NGO Forum, in dialogues such as SAFEGROUNDS, the ONR/NGO Forum, COMARE, the Westminster Government's Justification review process, PHE, the Environment

Agency and other official bodies and co-ordinations. I shall describe, with documentary evidence, the bizarre, unscientific and illogical arguments we have met. The reply I received from Dr. Jordan (see p.3 above) is one example.

Your web-site describes NRW as

> *... committed to seeking new ideas and approaches from both inside and outside NRW, innovation, finding creative solutions, working with others outside the organisation to find practical and pragmatic solutions, learning from mistakes and making improvements where there have been mistakes.*

I hope I have ticked enough of those boxes to persuade you to suspend the licence pending discussions which I shall treat as a high priority.

Yours sincerely

Richard Bramhall

Appendix 1
emails between Richard Bramhall and Bertrand Jordan March 2017

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[Upshot: he just cannot think his way out of the linear box.]

Dear Professor Jordan. I am puzzled by what you wrote in the December 2016 Genetics about Chris Busby's letter in the same issue:

"… Busby argues for a biphasic dose response… If this concerns the effect per milligray, it seems unlikely but conceivable; if it is the absolute effect, it does not make sense: How could a 50-mGy exposure be less damaging than 5 mGy?"

There does not seem to be any distinction between "the effect per milligray" (which you agree may be biphasic), and your Socratic expression "How could a 50-mGy exposure be less damaging than 5 mGy?" (which implies the effect can not be biphasic). Please could you explain? - Busby did, after all, specify a mechanism: "There are plausible biological reasons ... (especially in the case of congenital effects where the end point is seen only after birth, and at some dose level prebirth viability stops)."

Richard Bramhall

Dear Dr Bramhall,

Thank you for your remark. What I meant in my comment is that I can accept the possibility that 5 mGy alone might do more damage that the *5 mGy increment* from 50 to 55 mGy would produce - in that sense the "damage per mGy" value could be higher at very low mGy values than at somewhat higher ones (although to me this is counter-intuitive, low doses giving more leeway for DNA repair). On the other hand, in the build-up leading to a 50 mGy dose, there has to be a stage where the dose is 5 mGy, so I find it impossible to accept that a total dose of 50 mGy could do *less* harm than one of 5 mGy. Maybe I should have spelled that out more clearly.

The argument about loss of viability skewing the results doesn't really hold water here, since we are by definition discussing very low doses for which it is actually difficult to see any effect, loss of viability only occurs at much, much higher doses.

Best wishes,

Bertrand Jordan

Appendix 2

Report (unedited) submitted to the Committee Examining Radiation Risks of Internal Emitters (CERRIE) May 2004 during preparation of the Committee's Final report. I was a member of CERRIE throughout its existence (2001 - 2004). The Workshop referred to was a three-day international event attended by invited guests.

"Nowhere does the spatial anisotropy of dose appear more extreme than when we compare doses from external radiation with doses from particulates. The question of high risk from particles was raised in the 1970s by Arthur Tamplin who drew attention to the problem of doses local to inhaled Plutonium Oxide particles and deduced an enhancement factor of 115,000 for lung cancer. As the main report points out, the available information on hot particles, though interesting, was of little utility in addressing the fundamental problem of the health effects of the lower activity particles most commonly found in the environment. Our problem with the main report is that while it identifies the paucity of data relevant to the Committee's inquiry it underreports the extensive discussion on this important topic both in Committee and at the Workshop.

Radioactive substances from DU weapons, reprocessing plant discharges, weapons tests and accidents in the form of micron sized particles are widely dispersed in the environment. Through inhalation or ingestion they may become internalised and immobilised in tissue, giving rise to local tissue doses which are high, compared with the same amount of energy averaged over the whole body or organ. We have calculated the doses for the two common environmental alpha emitting particulates, Uranium and Plutonium Oxide. (For comparison mean annual whole body doses are about 2mSv.)

Table 1 Doses to sphere of tissue 30 micron radius by one particle of U_3O_8 of various diameters

Particle diam.	Particle vol. cm^3	Mass U_3O_8 (g)	Mass U238 (g)	Activity of particle (Bq)	Hits /day (dose/day mSv)	Hits/year (dose/year)
0.2 μ	4.2×10^{-15}	3.6×10^{-14}	3.06×10^{-14}	3.8×10^{-10}	3.3×10^{-5} (3.96×10^{-3})	0.012 (1.44mSv)
0.5 μ	6.5×10^{-14}	5.6×10^{-13}	4.8×10^{-13}	5.9×10^{-9}	5.1×10^{-4} (0.06)	0.186 (21.9mSv)
1.0 μ	5.2×10^{-13}	4.3×10^{-12}	3.7×10^{-12}	8.8×10^{-8}	7.6×10^{-3} (0.91)	2.77 (332mSv)
2.0 μ	4×10^{-12}	3.5×10^{-11}	2.9×10^{-11}	3.6×10^{-7}	0.031 (3.72)	11.32 (1358mSv)
5.0 μ	6.5×10^{-11}	5.6×10^{-10}	4.75×10^{-10}	5.9×10^{-6}	0.51 (60)	186 (21900mSv)

Assumptions: Uranium Oxide (U238) is in the U3O8 form (density = 8.6); specific activity of U238 = 12.43 MBq/Kg: Alpha decay energy = 4.45MeV; Alpha range = 30 microns. Relative Biological Effectiveness factor for Alphas = 20 (from ICRP) has been used to convert dose in Grays to effective dose in Sieverts.

For Uranium, the table shows that for particles as small as 0.2 microns diameter, average annual alpha dose to the lymphatic tissue surrounding the

particles is about the same as (and additional to) the total average natural background dose of 2mSv. It rapidly increases for larger particles.
Particle sizes from 0.1 to 5 microns are frequent in the environment. The dangerous size range for genetic mutation is between 0.5 and 5 microns for Uranium Oxide since they will cause “Second Event” processes (see below). For Plutonium, the dangerous sizes are smaller and more biologically mobile. Due to the high cell killing capacity of the Pu particles, ironically, the Uranium may be relatively more hazardous than one might expect.

Table 2 Doses to sphere of tissue 30 micron radius by one particle of PuO_2 of various diameters

Particle diam.	Particle vol. Cm^3	Mass PuO_2 (g)	Mass Pu239 (g)	Activity (Bq)	Hits /day (dose/day)	Hits/year (dose/year)
0.05μ	$6.5x\ 10^{-17}$	7.5×10^{-16}	6.5×10^{-16}	1.5×10^{-6}	0.129 (0.02Sv)	47 (7.3Sv)
0.1μ	5.2×10^{-16}	6.0×10^{-15}	5.2×10^{-15}	1.2×10^{-5} Bq	1.03 (0.15Sv)	375 (54 Sv)
0.2μ	4.2×10^{-15}	4.8×10^{-14}	4.2×10^{-14}	9.6×10^{-5} Bq	8.3 (1.2Sv)	3029 (438 Sv)
1.0μ	5.2×10^{-13}	5.9×10^{-12}	5.1×10^{-12}	1.16×10^{-2} Bq	1002 (146Sv)	365800 (53290 Sv)
2.0 μ	4.2×10^{-12}	4.8×10^{-11}	4.2×10^{-11}	0.096 Bq	8294 (1220Sv)	3027310 (445300 Sv)

Assumptions: Plutonium Oxide (Pu239) is in the PuO2 form (density = 11.6); Alpha decay energy = 5.2 MeV; Alpha range = 30 microns. Relative Biological Effectiveness factor for Alphas = 20 (from ICRP) has been used to convert dose in Grays to effective dose in Sieverts.

Plutonium Oxide particles will in a short time produce a dose of several sieverts in tissue local to the 30 micron range of their decays. The review commissioned by the Committee concluded that the risk from particles of this kind was not significantly greater over the range of exposures studied than that assumed by the ICRP averaging model. We assert that this must be a result of a trading balance between cell killing close to the particle and an enhanced mutagenic effect in cells further away which are subject to lower doses. At the top end of the range cell killing would predominate and at the bottom end the effects would be indistinguishable from the effects of external radiation. In other words since a good proportion of the effect of the radiation from the particle is wasted in cell killing the mutagenic efficiency of the unwasted portion must be considerably greater than assumed by the ICRP model. If this is the case then particles of lower activity, where cell killing does not predominate, must represent an enhanced health risk. This may happen because on pure theoretical grounds it is possible to show that the mid range will be in the quadratic region of the well documented dose/response curve for radiation effects, in which double strand breaks occur. Alternatively (or in addition) second event processes may occur as a result of sequential decays, a probabilistic circumstance which is vanishingly rare for external radiation at natural background levels (see "Second Event" below). Finally, there is the question of multiple damage or damage amplification to the many cells which are inside the radius for bystander effects.

We believe that the range of specific exposures in terms of local dose makes it extremely likely that the two most common particulate hazards (Plutonium oxide and Uranium oxide) are serious contributors to cancer, leukaemia and lymphoma risks and that this view is supported by epidemiological evidence cited elsewhere in this report.

Sellafield/Seascale doses

A good example of the error that may be introduced into risk assessment by averaging dose into tissues is afforded by the NRPB / COMARE calculations of dose from Uranium and Plutonium particles to the tracheo-bronchial lymph nodes of children living in Seascale. These calculations formed part of official responses to the arguments that the 10 fold excess of childhood leukaemia in Seascale was causally related to exposure to materials from BNFL Sellafield. Calculations made by NRPB and used by COMARE indicated that the doses to the TBLNs were very much less than natural background doses and therefore could not be a cause of the illness. However, close examination of the calculations revealed that the lymphatic system in this case had been modelled as an 11 Kilogramme aggregate of many of the organs in the body, whilst in reality the radiation doses from the material in the TBLNs was restricted to the TBLNs whose combined mass was a few grammes. This in itself would have been sufficient to provide the required 300-fold enhancement factor necessary to explain the observed leukaemia. Note that the "natural" radiation dose to the same tissues would in fact be from the massive amount of Uranium released from Sellafield.

Secondary photo electron emission in particles of elements of high atomic number.

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At the workshop we advanced the hypothesis that metal particles of high atomic number will focus incident energy from the natural background radiation gamma field by factors of up to 1000 fold as a consequence of the well known 4th power law relation between atomic number and absorption coefficient. The overall result will be to cause increased ionisation due to gamma conversion to short range photoelectrons in the vicinity of particles of Plutonium, Uranium or even gold resulting in genetic damage to local tissue.

Dr. Howard suggested another mechanism for enhancement of cancer risk through the mediation of micro inflammation in tissue in the vicinity of hot particles. He argued that induction of damage in tissue local to the particle would result in an inflammatory response followed by cell replication which would lead to a higher incidence of genetic damage and cell co-operative effects.

At the workshop, Professor Bridges also pointed out that as a result of the Bragg effect there would be a shell of dead cells at a radial distance equal to the decay range of the alpha particle. This zone of dead cells would effectively insulate a community of potentially damaged cells preventing communication with healthy cells outside the range of the decays. The implications of this for the development of clonal damage may be significant and warrant further research.