Public Health England

Key points in response to Dr C Busby letter challenging justification decision on EPRs

1. Use of a linear non-threshold relationship to describe exposure dependent cancer risk in radiation exposed populations.

Dr Busby challenges the use of a linear relationship between radiation dose and risk to health and further the simple proportionality of risks of small additional exposures amongst a larger average exposure (p2, lines 1-14).

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 the International Commission on Radiological Protection (ICRP 103, 2007) and the National Council on Radiation Protection and Measurement (NCRP 136, 2001) and the US National Academy of Sciences (NRC, 2006.

2. Alleged evidence for the failure of Hiroshima Studies

(a) Selection of control groups

The decision to change the control groups used in the Japanese A-bomb survivor Life Span Study (LSS) that follows the health of those exposed to radiation due to atomic bombings of Hiroshima and Nagasaki has a robust and coherent basis. Epidemiological studies of radiation cancer risk are strongly affected by a variety of 'confounding factors' because radiation is not the sole, or even major cause of cancer, even in highly exposed populations. Where a control group is compared to an exposed group, any differences between their underlying disease risks will result in biased estimates of radiation risk from a comparison of the two groups. For example, differences in the age structure have a substantial impact on the incidence of cancers.

To overcome this issue, a common approach in many epidemiological studies, is to avoid the need for a separate control group by performing an internal comparison. In this form of analysis the disease patterns among those with the lowest exposure are compared to those with higher exposure. This approach was adopted for the analyses of the LSS as 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). In studies of nuclear workers this approach is adopted as such workers whose health is regularly monitored tend to exhibit significantly lower incidences of disease compared to the general population, even taking differences in the age structures of the two groups into account.

(b) LSS and internal contamination

Internal contamination has been considered in the LSS cohort in some detail. The *Brief Description* document produced by the Radiation Effects Research Foundation provides a useful summary of this and indeed all aspects of the Hiroshima and Nagasaki follow-up studies (http://www.rerf.jp/shared/briefdescript/briefdescript_e.pdf). The RERF DS86 report (http://www.rerf.jp/shared/ds86/ds86a.html) concluded that internal exposures in the LSS cohort are small compared to external (gamma) exposures.

A complex mixture of fission products were formed from the detonation of the atomic bombs in Japan, more than 300 different isotopes of 36 light elements have been identified among them. These emit beta and gamma radiation. Although the mixture is complex, with no single half-life, the decrease in the total radiation intensity from the fission products can be calculated approximately and is fairly rapid: activity after 7 hours is about 10% of activity after one hour (Glasstone and Dolan, 1980). Estimates of residual unfissioned uranium in the fallout from the Hiroshima bomb (a uranium bomb unlike the plutonium bomb used at Nagasaki), point to around 50kg of unfissioned ^{235}U in the fallout (Sakaguchi et al 2010). Recent surveys of uranium in fallout areas indicate that residual uranium from fallout is a minor part of the total, the majority attributable to global fallout from nuclear weapons testing elsewhere (Takada et al, 1983; Sakaguchi et al 2010).

(c) New evidence on genetic risk from uranium exposure

The potential increase in damage to DNA as a consequence of uranium binding to DNA has been considered in detail previously (Tanner et al 2012). The detailed modelling studies undertaken suggested that there was little enhancement to the deposition of energy and radiation-induced damage as a consequence of uranium binding to DNA. The binding of uranium, displacing calcium, was also judged to be a low frequency event on the basis of the substantially larger size of the uranium U^{2+} ion compared to Ca^{2+} ion. The Japanese atomic bombing survivor studies have addressed the potential impact on health of exposure to potentially contaminated rain post-blast; the most recently published analysis does not find a robust association between post-blast rain exposure and acute symptoms of radiation exposure amongst a group of over 93,000 survivors and post-blast rain exposure (Ozasa et al 2016). Longer term follow up of those exposed to post-blast rainfall found that in Hiroshima exposure to rain was not significantly associated with increased mortality or incidence for any end points: total mortality, cancer mortality, solid cancer incidence or leukemia incidence (Sakata et al 2014). In Nagasaki a marginal association between rain exposure and “death due to all causes” was found but was judged by the author to be a spurious finding (Sakata et al 2014), note also that the Nagasaki bomb used plutonium as a fissile material and thus uranium fallout would be very substantially lower that in Hiroshima. The Schmitz-Feuerhake, 2016 paper (Schmitz-Feuerhake et al 2016) claims to have identified increased levels of congenital malformations

amongst the children of parents exposed to radiation as a consequence of the Chernobyl accident. The paper does not make clear how the figure of a 1000-fold underestimation of heritable disease risk was derived. International consensus reviews of evidence developed by the United Nations Scientific Committee on the Effects of Atomic Radiation do not support the conclusion that there are increased congenital malformations or hereditary effects in the children of those exposed to radiation in the post-Chernobyl environment or from involvement in clean-up operations (UNSCEAR 2001 and 2008 annex D). Furthermore, direct evidence of the risk of hereditary diseases in humans is lacking (UNSCEAR 2010), including recent follow-up on the health of the offspring of Japanese A-bomb survivors for 62 years (Grant et al 2015).

3. Alleged evidence of genetic damage as a consequence of Chernobyl fallout

As noted above, international consensus reviews have not identified consistent health effects in the children of those exposed to Chernobyl fallout. The Schmitz-Feuerhake (2016) paper does not make clear how the claimed 1000-fold underestimation of hereditary risk was derived. Many studies of human populations, including Japanese A-bomb survivors, have not revealed hereditary effects of radiation exposure, risks are therefore inferred from experimental animal studies.

4. Ethical basis of ICRP recommendations

The ICRP recommended factor for the risk to human health (detriment adjusted nominal risk coefficient) is approximately 5% per Sievert (Sv) – or 0.05 per Sv (not 0.5 per Sv). The risk associated with 1 mSv is therefore 1 in 20,000. The ICRP (ICRP 103, 2007) estimate for risk of hereditary disease is 0.001 per Sv in a working adult population or 0.002 in the whole population. The risk associated with a 1mSv exposure is therefore (using the whole population estimate) 1 in 500,000 – it is unclear where the 1 in 500 figure has been obtained.

As noted we are unable to find evidence in the literature cited by Dr Busby for the claimed 1000 fold underestimation of hereditary disease risks and this is clearly out of agreement with widespread international consensus values.

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