LETTERS TO THE EDITOR

ICRP draft recommendations 2005 and radon exposure On the linear no-threshold model On effects of low doses Reply

To cite this article: 2004 J. Radiol. Prot. 24 423

View the article online for updates and enhancements.

Related content
- The bell should toll for the linear no-threshold model
  D J Higson
- Radiation protection principles
  John R Cooper
- The report of the French Academy of Science: "Problems associated with the effects of low doses of ionising radiation"
  M Tubiana
LETTERS TO THE EDITOR

ICRP draft recommendations 2005 and radon exposure

Dear Sir

The International Commission on Radiological Protection (ICRP) has recently published a consultation draft of its revised recommendations for a system of radiation protection [1]. This confirms annual effective dose constraints of 1 mSv for any controllable dose for the general public and 20 mSv for radiation workers, as recommended in ICRP Report 60 [2]. In the case of multiple dominant sources, the draft recommends a maximum public constraint for a single source of radiation of 0.3 mSv per annum.

Radon gas, which occurs naturally with a variable geographical concentration, and which can concentrate in domestic homes, overground workplaces, and caves and mines, is treated separately from all other controllable sources. Continuing the logic of Publication 65 [3], the policy is based on an annual effective dose from radon of 10 mSv, resulting in the proposal of maximum constraints of 600 Bq m$^{-3}$ for domestic properties, and 1500 Bq m$^{-3}$ for the workplace. National regulatory authorities are encouraged to establish their own lower constraints using the principle of optimisation, and many countries, including the UK [4], currently specify domestic and workplace Action Levels of 200 Bq m$^{-3}$ and 400 Bq m$^{-3}$ respectively.

Northamptonshire, UK, is one area where raised radon levels can be found in both domestic and workplace premises. We have studied the doses received by occupants of both classes of environment for some years, and our studies in residential properties [5] confirm the UK domestic Action Level of 200 Bq m$^{-3}$ to be responsible for an annual dose to occupants of around 10 mSv. This analysis, and all the other results quoted here, was calculated using the relation that 1 mSv is equivalent to 126 kBq m$^{-3}$ h, which is derived from an analysis by the National Radiological Protection Board (NRPB) [6], and the relation that 10 mSv is equal to 1 Working Level Month [7], assuming an Equilibrium Factor of 0.5. It is, however, apparent that the time spent by individuals in the home varies widely [8], with some population groups, such as invalids and Asian wives in purdah, having almost 100% occupancy. Taking the typical cyclical daily variation in radon levels into account [5], we estimate that, in such extreme cases, the annual dose could exceed the population average by as much as 50% at the Action Level.

In the Health Service workplace, we found that the dose from radon to staff was greater, in both magnitude and in numbers of staff affected, than the dose arising from occupational exposure to the medical use of x-rays and radioactive isotopes [9]. At the UK workplace Action Level of 400 Bq m$^{-3}$, the average annual radon dose to full-time staff was found to be 4.4 mSv, with the 75% quartile being 5 mSv [5]. In a number of cases, members of staff are exposed to both medical x-ray doses and to radon, as we located two dental surgeries with raised radon levels. In designing the safe use of dental x-rays, a dose constraint of 1 mSv should be used, yet the current UK workplace Action Level permits a dose of 5 mSv to be received from radon.

We have recently shown that remediation to reduce radon levels in the workplace preferentially reduces the higher levels of radon found at night, and therefore the benefits of remediation to daytime workers are significantly lower than the drop in 24-hour average
radon level would suggest [10]. As a result of our work, we proposed a post-remediation Action Level of 225 Bq m$^{-3}$ (relative to the 400 Bq m$^{-3}$ level) to ensure that occupants of 75% of the rooms would have doses lower than the pre-remediation acceptance criterion. Table 1 summarises the annual doses that our analysis predicts would match the proposed constraints.

<table>
<thead>
<tr>
<th>Proposed ICRP maximum constraint (Bq m$^{-3}$)</th>
<th>Domestic</th>
<th>Workplace</th>
<th>Remediated workplace</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average dose to occupant (mSv)</td>
<td>30</td>
<td>16.5</td>
<td>35</td>
</tr>
<tr>
<td>Maximum dose to occupant (mSv)</td>
<td>45</td>
<td>65</td>
<td>55</td>
</tr>
</tbody>
</table>

Our work has also shown that programmes to reduce radon levels in the workplace, schools and homes [11] in radon Affected Areas produce positive health benefits and can also be cost-effective. The higher the Action Level is set, the greater the number of residual (i.e. after remediation) lung cancers caused by radon are predicted to occur in the general public and staff [12]. Table 2 shows the effect of the choice of different Action Levels on the numbers of averted and residual lung cancers following a completed domestic radon remediation programme [11], using the NRPB estimate of $3.5 \times 10^{-4}$ lung cancers per Working Level Month [13].

<table>
<thead>
<tr>
<th>Domestic Action Level (Bq m$^{-3}$)</th>
<th>Cancers averted annually</th>
<th>Residual annual cancers</th>
</tr>
</thead>
<tbody>
<tr>
<td>200</td>
<td>23</td>
<td>42</td>
</tr>
<tr>
<td>600</td>
<td>9</td>
<td>59</td>
</tr>
</tbody>
</table>

In a radon Affected Area, there would be a small but significant risk that some people would be exposed to radon both at home and at work, or home and at school; and as noted above our study population had two workers exposed to radon and diagnostic x-rays. There is therefore an argument to treat each source of radon—at work or at home—as a single source, and to establish a constraint which is compatible with multiple dominant exposures.

In view of these issues, there appears to be no logic in setting far higher maximum constraints for radon than any for other single cause of radiation exposure. There is, moreover, a need to propose a post-remediation workplace constraint.

Yours faithfully,

**A R Denman, S Parkinson and C J Groves-Kirkby**

Medical Physics Department
Northampton General Hospital NHS Trust
Cliftonville
Northampton NN1 5BD
UK
(E-mail: chris.groves-kirkby@ngh.nhs.uk)
Dear Sir

The ‘opinion’ article by D J Higson [1], published in the previous issue of the journal, is to be warmly applauded. I had previously read Higson’s views in a specialist journal [2] a couple of years ago and, since then, had hoped that they would reach a wider readership. All of the bullet points he makes in section 2 [1] should be given the widest possible publicity.

The timing of Higson’s article [1] is also important as it provides a clear refutation of the arguments in the paper by Brenner et al [3] concerning the cancer risks attributable to low doses of ionising radiation. As Higson did not comment on that paper, I would appreciate being able to do so.

On the linear no-threshold model

Dear Sir

The ‘opinion’ article by D J Higson [1], published in the previous issue of the journal, is to be warmly applauded. I had previously read Higson’s views in a specialist journal [2] a couple of years ago and, since then, had hoped that they would reach a wider readership. All of the bullet points he makes in section 2 [1] should be given the widest possible publicity.

The timing of Higson’s article [1] is also important as it provides a clear refutation of the arguments in the paper by Brenner et al [3] concerning the cancer risks attributable to low doses of ionising radiation. As Higson did not comment on that paper, I would appreciate being able to do so.
A cornerstone of the arguments put forward by Brenner et al [3] is figure 2 in their paper. This purports to show the Excess Relative Risk (ERR) of mortality from cancer among groups of survivors of the atomic bombs. Each group is made up of persons exposed to a range of different doses, with upper limits of 50 mSv, 100 mSv, 150 mSv, etc. The individual points are taken as the mean dose for each group. A straight line is drawn through these points which, if extrapolated back, would pass through the origin.

The reference for these data is the recent paper by Preston et al [4] in which they update their studies of mortality of the atomic bomb survivors. However figure 2 in the Brenner et al paper [3] does not appear as such in the Preston et al paper [4]. It is actually derived from table 4 in the latter paper, in which an ERR/Sv is listed for each of the various dose ranges. Values of this vary inconsistently; leaving aside the extremely high value of nearly unity at the lowest doses, they still vary by a factor of 2 as the dose range is extended. Each ERR/Sv is then converted to an ERR using the average dose to the stated cohort. Thus an inconsistent set of data is used to create a straight line through the origin.

My next cause for concern is the fact that the Preston et al paper [4] does not contain mean doses for each cohort. Somehow Brenner et al [3] have performed some sort of averaging, but we are not told on what basis. Any simple average would assume linearity which, in showing linearity, becomes a circular argument.

Brenner et al [3] concede that the first two points on their plot are not statistically significant compared to those individuals who were exposed to <5 mSv. However even the next point (5–125 mSv) is of only marginal significance (0.74 ± 0.38) as is the one after that (5–150 mSv, 0.56 ± 0.32). All these four points appear to show approximately the same ERR of about 0.02. It is only for the cohort range 5–200 mSv that the ERR/Sv becomes significant and thus the ERR jumps to about 0.04. One’s conclusion is that the inclusion of the doses in the range 150–200 mSv has made the difference. In that case, what is the relevance of the fact that the average dose to the cohort 5–200 mSv is 47 mSv?

The view that there is no evidence for increased tumour rates below 200 mSv is not new. In a paper published several years ago Heidenreich et al [5] came to a similar conclusion. Using the data from an earlier study of the mortality of atomic bomb survivors they plotted the observed/expected (O/E) ratio both for mortality and for incidence as a function of a ‘class’. Each class consisted of a range of doses, but without overlap of doses between classes. Their plots clearly showed that the lowest class for which a significant increase in both mortality and incidence could be demonstrated was in the range 200–500 mSv. The Brenner et al paper [3] makes no reference to this conclusion.

Perhaps the most surprising aspect of the Brenner et al paper [3] is that it appears to ignore the findings of one of its own authors, Sir Richard Doll, who, with a group of his co-workers in Oxford, investigated the mortality from cancer and other causes in British radiologists over the 100-year period 1897–1997 [6]. The Standardised Mortality Ratio (SMR) was established using three control groups: (i) the general population, (ii) social class I males and (iii) medical practitioners. The radiologists were divided into four sub-groups according to their year of first registration with a radiological society, namely pre-1921, 1921–35, 1936–54 and 1955–79.

For mortality from all causes, it was found that, among radiologists aged less than 85 years, there were 752 deaths in those who first registered after 1920. This was significantly lower than the number expected from mortality rates in the general population (SMR 0.72, 95% CI 0.67–0.77). Compared with mortality rates in social class I males and with male medical practitioners, the number was also significantly reduced.

Looking at cancer mortality, they found that, among radiologists registering after 1920, there were significantly fewer deaths than expected from mortality rates in the general
population (SMR 0.63, 95% CI 0.54–0.74) and from mortality rates for social class I males (SMR 0.82, 95% CI 0.70–0.96). Compared with male medical practitioners the number of cancer deaths observed was slightly (but not significantly) greater than the number expected (SMR 1.04, 95% CI 0.89–1.21). When the data were sub-divided by year of first registration, the number of observed deaths from cancer exceeded the number of those expected for the first two of the sub-groups specified above, although the increase was not statistically significant in either case. For the radiologists who registered after 1954, there were fewer cancer deaths than expected in medical practitioners (SMR 0.70, 95% CI 0.49–1.03).

Individual dose information in the form of dosimetry or surrogate measurements was not available. However Braestrop [7] had estimated that radiologists in the 1920s and 1930s could have been exposed to as much as 1 Gy per year. This had been reduced to 0.1 Gy per year before the 1950s according to Smith and Doll [8], and perhaps to 0.05 Gy per year in the 1950s. Noting that even such relatively high doses did not lead to significant increases in the rate of cancer induction it is difficult to accept the assertion that, ‘the most reasonable assumption is that the cancer risk from low doses of x- or γ-rays decreases linearly with decreasing dose’.

One must be hesitant about challenging the conclusions of some of the most illustrious names in modern radiobiology and epidemiology. Nevertheless, for the reasons detailed above, these conclusions must be challenged before they pass into folklore and become holy writ.

Yours faithfully,

J A Simmons

References

J S, Ron E, Sacks R K, Samet J M, Setlow R B, and Zaiden M 2003 Cancer risks attributable to low doses of
ionising radiation: assessing what we really know Proc. Natl Acad. Sci. 100 13761–66
[5] Heidenreich W F, Paretzke H G and Jacob P 1997 No evidence for increased tumor rates below 200 mSv in the
atomic bomb survivors data Radiat. Environ. Biophys. 36 205–7
from cancer and other causes Br. J. Radiol. 74 507–19
54 187–94

On effects of low doses

Dear Sir

In his paper ‘Trivial risks and the new radiation protection system’ (J. Radiol. Prot. 24 (2004) 3–11) , Per Wikman concludes that individual effective doses less than 0.01 mSv in a year should not be excluded from the system of radiological protection. This conclusion is clearly
predicated on the assumption that the no-threshold (NT) model of dose–risk relationship is correct. Wikman recognises that NT is an assumption but he refers to risks inferred from it as being known to exist and treats them throughout his paper as being real.

Wikman also recognises that risks cannot be detected or measured at low levels of exposure to radiation but he does not mention that ‘low levels’, in this context, means:

• up to at least 10–50 mSv (depending whether \textit{in utero} or \textit{ex utero}) in the case of acute doses incurred at very high rates over short periods of time, as in an atomic bomb explosion or in some medical procedures; or
• up to at least 100 mSv/y in the case of chronic dose rates spread over a lifetime, \emph{viz}: lifetime doses much greater than 1000 mSv.

The assumption that a risk observed at (say) 100 mSv (acute) means that there must be a risk from less than 0.01 mSv/y (chronic) requires more justification than merely saying that there are ‘scientific reasons for the linear, no-threshold assumption’. What are these reasons? There are ‘scientific reasons’ against the linear extrapolation too. This is not just the matter of thresholds, although thresholds may well exist.

There is at least as much evidence of beneficial health effects (‘radiation hormesis’) as there is of harmful effects from exposures in this range. Without taking any position on the relative merits of the hormesis model and the linear-NT model, I have to say that they are mutually exclusive. Anyone who bases a conclusion on the linear-NT model needs to refute the evidence of hormesis or ignore it.

If there is a possibility of beneficial effects from exposure to radiation, then it is not a ‘precautionary approach to assume that there is a risk’. The application of the \textit{precautionary principle} to this matter can be stated in two ways, \emph{viz}:

• (P’) People should be protected from exposure to radiation unless it can be proved that such exposure is not harmful to their health.
• (P”) People should be exposed to radiation unless it can be proved that such exposure is not beneficial to their health.

These two propositions are equally plausible, but P” is unlikely to be an acceptable basis for public policy. Why should P’ be any more acceptable?

Yours faithfully,

D J Higson
260 Glenmore Road
Paddington NSW 2021
Australia

\textbf{Reply}

Dear Sir

The purpose of my paper was not to argue that very small doses should never be excluded from the radiation protection system. There might be good reasons for an exclusion, as pointed out in
my paper. Instead, the paper examines the arguments given by the ICRP for such an exclusion and finds these to be insufficient. Because the paper discusses the ICRP recommendations, which use the linear, no-threshold model, my conclusions are also based on this model.

Higson puts forward an interesting thesis in his response to my paper: ‘if there is a possibility of beneficial effects from exposure to radiation, then it is not a precautionary approach to assume that there is a risk’. The argument is based on the claim that in this case the ‘precautionary principle’ yields two contradictory results (P’ and P").

Higson’s argument shows that in a case of complete or severe ignorance we would have little or no grounds at all to be precautious. However, the thesis put forward by Higson is only valid if we actually believe it is equally or more likely that radiation is beneficial as it is harmful. If we think it is more plausible for low doses to be harmful than beneficial we may take the precautionary decision to protect people.

Higson claims that there is at least as much evidence of beneficial health effects as there is of harmful effects in the low-dose range. The fact that we cannot measure any increased (or decreased) risks at low levels is consistent with the lack of statistical power in the epidemiological studies at hand. At these levels we have to rely on biophysical models. This is not the place to go into lengthy discussions on this matter, but the prevailing scientific view, by the ICRP and the UNSCEAR, for example, is that the linear, no-threshold model is the best available approximation at low doses.

In conclusion, if we believe that small doses of radiation are harmful we could perfectly well make a ‘precautionary’ decision in order to protect people. If we instead believe that small doses of radiation are beneficial, we could take the ‘precautionary’ measure to irradiate people. If we find the latter hard to call ‘precautionary’ it may be because we do not really believe that small doses of radiation are beneficial.

Yours faithfully,

Per Wikman
Philosophy Unit, Teknikringen 78B
Royal Institute of Technology
SE-100 44 Stockholm
Sweden
(E-mail: perwi@infra.kth.se)