Response Statement

2003 Recommendations of the European Committee on Radiation Risk

The Recommendations of the International Commission on Radiological Protection (ICRP) form the basis for standards for protection of people against ionising radiation in almost every country in the world. These standards have recently been called into question in a lengthy report issued by the European Committee on Radiation Risk (ECRR), a self-styled organisation with no formal links to official bodies. The ECRR criticisms are largely about the risk to health from radionuclides that deliver doses after being taken into the body in food, water or air. These radionuclides are called 'internal emitters'. The ECRR argues that the risks to health from these radionuclides are underestimated by ICRP. In doing so, the ECRR provides limited evidence from epidemiological studies, and advances an alternative methodology for estimating radiation risks from internal emitters.

A critical examination of the ECRR report has been undertaken by NRPB staff. The cited epidemiological studies have been investigated in detail by NRPB staff and previously by other experts; their conclusions are generally different from those reached by ECRR. The methodology proposed by ECRR for estimating radiation risks from internal emitters is arbitrary and does not have a sound scientific basis. Furthermore, there are many misrepresentations of ICRP, misunderstandings, inconsistencies and unsubstantiated claims in the ECRR report. The ECRR report therefore provides no scientific basis for changing protection standards.

Overall, NRPB believes that the recommendations of ICRP provide a sound basis for radiological protection standards. In particular, risks from internal emitters are acceptably well understood and may, in some cases, be overestimated by ICRP.

Main Response

The scientific bases underpinning standards for protection should be transparent and robust. It is right and proper that these bases are from time to time rigorously questioned. However, any recommendation for fundamental changes to such standards, must be based on sound scientific reasoning.

The recommendations of the International Commission on Radiological Protection (ICRP) underpin standards of protection against ionising radiation for people in almost every country in the world. These recommendations are based on extensive knowledge of the health effects of ionising radiation which is reviewed and analysed by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). They have been incorporated in the European Council Directive on basic safety standards (EC, 1996) and in the International Basic Safety Standards (IAEA, 1996). These recommendations have recently been called into question in a lengthy report issued by the European Committee on Radiation Risk (ECRR).

(MORE)

R3/03
24 July 2003

National Radiological Protection Board,
Chilton, Didcot, Oxon, OX11 0RQ
Telephone: (01235) 822744 Fax: (01235) 822746
Email: pressoffice@nrpb.org

The ECRR is a self-styled organisation. In particular, it does not have formal advisory responsibilities to the European Commission or to the European Parliament. The ECRR report (Green Audit, 2003) is published by the special interest group, Green Audit. Dr Busby, of Green Audit, is secretary of the ECRR and lead author of the report. Many of the arguments contained in the report have appeared previously in Green Audit papers published on the Low Level Radiation Campaign's website (www.llrc.org/index.html).

The criticisms by ECRR cover two broad areas:

i) The ethical principles for standards of protection with ECRR advocating, in particular, application of the precautionary principle.

ii) The scientific bases underpinning ICRP recommendations.

Considering each of ECRR's criticisms in turn:

NRPB is not in a position to comment on the ethical aspects of standards. Two points, however, should be noted. Firstly, the effects of ionising radiation have been extensively studied over decades; the UNSCEAR 2000 report to the General Assembly of the United Nations totalled over 1000 pages (UNSCEAR, 2000). Arguably, ionising radiation may be one of the most extensively studied and well understood of any class of environmental agent. Secondly, the ECRR criticises the technique of 'cost benefit analysis' whereby benefits and detriments are assigned monetary values. ICRP has also done so in Publication 77 (ICRP, 1997). This is one issue, at least, on which ICRP and ECRR appear to agree.

The second broad area of criticism centres on the scientific basis for the protection standards against ionising radiation: this is the nub of the issue. The central tenet of the ECRR case is that ICRP bases its estimate of risks from ionising radiation on studies of the A-bomb survivors who were exposed to external irradiation, ie, from radioactive material outside the body, and that ICRP seriously underestimates the risk from artificial radionuclides that deliver doses following incorporation into body tissues, the so-called 'internal emitters'. In support of this position, ECRR cites a number of studies that purport to show that risks of cancer from internal emitters are substantially greater than assumed by ICRP. These studies characteristically identify a cluster of one or another form of cancer and then attempt to causally link it to exposure to releases of radionuclides from a nuclear site or sites. However, a number of the locations covered by these studies (Sellafield, Dounreay and Aldermaston/Burghfield) have been extensively investigated by the UK's Committee on Medical Aspects of Radiation in the Environment (COMARE), who concluded that releases of radionuclides from the sites did not appear to be able to account for the excess of cancer in the vicinity of these sites (COMARE, 1988, 1989, 1996, 1998). Some of the other cited studies were undertaken under the auspices of Green Audit, the organisation that published the ECRR report. One of these studies concluded that there was a significant excess of childhood leukaemia in North Wales associated with living close to the coast. Green Audit assumed that proximity to the coast was a surrogate for exposure to water-borne radionuclides released from the Sellafield reprocessing plant, so internal irradiation from these radionuclides was responsible. COMARE investigated this claim concluding that they 'have found no evidence to support the contention that there was an increased incidence of childhood leukaemia or other childhood cancers close to the North Wales coast' (COMARE, 1999). Green Audit has also claimed a significant excess of deaths from breast and prostate cancer in the immediate area around Bradwell Nuclear Power Station. COMARE investigated this allegation concluding that the data 'do not indicate any significant excess of cancer mortality around Bradwell' (COMARE, 2003). Thus, when the cited studies are investigated in detail, the conclusions are most frequently different from those reached by ECRR.

(MORE)

More generally, the ECRR report places particular emphasis on selective analyses of trends in disease rates over time or between different geographical areas. The interpretation of such descriptive studies is usually problematic, since they lack information on exposures and possible confounding factors for specific individuals. The ECRR report devotes less attention to more detailed studies, such as those described below, which have looked at groups of specific individuals. When the ECRR does cite such studies, it is again done on a selective basis and often with interpretations that run contrary both to those of the original investigators and to wider epidemiological knowledge. For example, the ECRR ascribes increases in mortality among radiation workers with increasing time since starting work to the effects of radiation exposure. However, the ECRR does not recognise that the "Healthy Worker Effect" (ie, workers in industry are healthier than the population as a whole) varies over time. In particular, studies of workers in many industries show that mortality rates are often very low soon after starting work and increase subsequently as the impact of selection into work diminishes.

The distinction made by ECRR between internal and external emitters has no foundation from a physics standpoint. A gamma ray entering the body from outside creates an electron internally and it is this electron that causes damage. An internal nuclide also creates an electron internally. There is scientifically no difference. A similar argument applies to attempts at making distinctions between the effects of internal natural and artificial alpha particle emitting radionuclides; there is no scientific reason why the effects should be different.

Furthermore, there are studies on humans exposed to internal emitters which, although not as comprehensive as the study on the A-bomb survivors, do provide reassurance that current ICRP risk estimates are not substantially in error. Included in these studies are patients with intakes of radium or Thorotrast (an alpha emitting thorium dioxide colloid), miners occupationally exposed to radon, and workers at former Soviet nuclear facilities exposed to plutonium-239. These studies have been the subject of a recent review (Harrison and Muirhead, 2003). Overall, taking account of the prevailing uncertainties, risks estimated for these internal emitters are consistent with risks estimated from data for external exposure of A-bomb survivors. There is good agreement for lung and liver cancers, whereas for leukaemia and bone cancers, it appears that the current ICRP approach could actually over-estimate risks from internal alpha-emitters. While it is true that the ICRP risk estimates for radiation induced cancers in general are based largely on studies of the A-bomb survivors, risks for liver and bone cancer are based primarily on the Thorotrast and radium data.

Having judged that risks from artificial internal emitters are underestimated, the ECRR develops a dose calculation methodology taking this judgement into account. ECRR proposes retention of the ICRP system of radiation weighting factors (w_R) and tissue weighting factors (w_T) (w_R accounts for the biological effectiveness of different radiations; w_T reflects differences in the radiosensitivity of different organs and tissues). However, ECRR weights the resultant effective dose by two additional factors: a 'biophysical factor', w_J, and a 'specific internal isotopic biochemical enhancement factor', w_K. The ECRR propose values of w_J ranging from 1 to 1000 and for w_K of between 2 and 1000. For any particular nuclide these two factors can be multiplied together depending on the particular exposure situation under consideration. The result for many artificial internal emitters would be a very substantial increase in effective dose.

ECRR's choice of values for w_J and w_K appears to be largely arbitrary with little or no supporting scientific evidence being provided. Thus, in only two cases is it possible to comment on ECRR's choice of value for w_J. A value for w_J of between 20 and 50 is proposed for 'Internal atomic second event types of exposure'. This proposed phenomenon has been the subject of previous calculations by Busby, but these have

(MORE)

been shown to be flawed (Edwards and Cox, 2000). Similarly, values for w_J of between 20 and 1000, depending upon activity, particle size and dose, are proposed by ECRR for internal insoluble particulate activity. Presumably, this is to account for the so-called 'hot particle' effect, whereby it is proposed that point sources within tissues somehow confer a greater risk to health than the same activity evenly distributed throughout the tissue (the assumption usually made by ICRP). The effect of hot particles is to increase the dose locally to a small part of the appropriate organ. In principle, this is no different from the high doses (3 or 4 Gy) received by some of the Hiroshima and Nagasaki A-bomb survivors, although for some hot particles the local doses can be much higher. Doses of around 3 Gy to the A-bomb survivors are a major determinant of the ICRP estimate of risk. For extrapolation to low doses and low dose rates, ICRP assumes direct proportionality between risk and dose but have chosen to divide the slope by a factor of 2 to account for dose and dose-rate effects (UNSCEAR, 2000). Even if hot particles of low linear energy transfer (LET) radiation were to confer extra risk, the increase above the ICRP estimate could be no more than a factor of a few. In a recent review of cancer risks from hot particles, Charles et al (2003) analysed data from *in vitro* and *in vivo* animal studies, as well as relevant human data on low LET and alpha particle effects, and concluded that ICRP dose averaging is likely to provide a reasonable estimate of carcinogenic risk, within a factor of 3 either way. It should be noted here that, although ICRP calculates average dose to tissues from incorporated radionuclides, account is taken of the distribution of target cells within tissues as well as the distribution of incorporated radionuclides. For example, the distribution of target cells in skeletal tissues is taken into account in estimates of risk of bone cancer and leukaemia. In other cases, the liver for example, the biologically reasonable assumption is made that target cells are distributed throughout the whole organ. Thus, the target tissue volume can vary from a single layer of cells to a whole organ.

Mechanisms that underpin ECRR judgements on w_K values for specific isotopes include 'transmutation and local dose', 'enzyme amplification', 'hydrogen bonding', 'interfacial ionic adsorption' and 'DNA binding'. How these mechanisms relate to health risks is largely unexplained and no reasons are given for the choices of w_K values.

Possible increased damage at a cellular level from internal artificial radionuclides, reflected in the choice of values for w_J and w_K, is relevant to another of the arguments in the ECRR report. ECRR cautions against attempting to gauge the effect of novel exposures, which include internal exposure to artificial radionuclides, by comparison to natural radiation. However, similar lung cancer risk estimates per unit dose are obtained for Russian workers exposed to plutonium-239, which would attract a large w_J or w_K or both, and uranium miners exposed to radon-222, a natural radioactive gas. Furthermore, animal studies comparing the effects of different radionuclides and external radiation show that the effects of radionuclides are consistent with their location and retention in the body and not their natural or artificial origin. For example, after allowing for differences in distribution, incidences of bone cancer in life-span studies using dogs are consistent with dose delivery near bone surfaces from plutonium-239 and americium-241 (artificial alpha emitters), thorium-228 and radium-226 (natural alpha emitters), and strontium-90/yttrium-90 (artificial beta emitters) (WHO, 2001).

ECRR also questions the value of the risk factor which is used by ICRP to convert from doses to risks. The risk factor recommended for members of the public by ICRP for fatal cancer at doses and dose rates generally encountered in the environment is $5\ 10^{-2}\ Sv^{-1}$. This value has been derived from studies on populations exposed to much higher dose rates by dividing by a DDREF (dose and dose rate effectiveness factor) of 2 as mentioned earlier. ECRR state that they will not employ a DDREF approach and, for no obvious scientific reason, adopt a risk factor appropriate for high doses and dose rates of $1\ 10^{-1}\ Sv^{-1}$ for all situations. This is despite the considerable body of scientific evidence from animal studies that risks per unit dose at low dose rates are less than those at high dose rates (NRPB, 1995).

(MORE)

ECRR also specifically criticises ICRP's methodology for assessing doses and risks when applied to exposure to radon, a natural radioactive gas that causes internal irradiation of the lung. In this case, ECRR considers that ICRP overstates the doses and that 'this misrepresentation has had the effect of minimising the contributions to human exposure from artificial radionuclides'. As mentioned earlier, risks from radon exposure are well understood, being studied extensively in workers exposed to radon in mines. The risk factor for lung cancer derived from these studies, $2.5\ 10^{-3}\ Sv^{-1}$, using the ICRP respiratory tract model to calculate doses from radon exposures, is similar to the value of $6.8\ 10^{-3}\ Sv^{-1}$ derived from the A-bomb survivor study. Thus, there is clear evidence that ICRP has not overstated or 'misrepresented' doses from radon and that, once again, ICRP's dose assessment methodology is acceptably robust. Furthermore, the discussion of radon dosimetry on pages 50-51 of the ECRR report includes a worrying misconception that an annual effective dose from radon inhalation of 1200 μSv results from an absorbed dose to the lungs of 60 μSv; in fact, the absorbed dose to the lungs is 500 μGy, as the authors have overlooked the 0.12 tissue weighting factor for the lung. (The calculation is as follows: 500 μGy absorbed dose from alpha emitters x 20 (w_R) gives 10,000 μSv equivalent dose x 0.12 (w_T for the lung) gives 1,200 μSv effective dose.)

The ECRR report also notes, but does not adequately explain, how data on poorly understood cellular phenomena such as post-irradiation induced genomic instability, bystander effects and biphasic dose-response has influenced judgements on cancer risk. Similarly, the ECRR claim that data on the rate of mutation induction in minisatellite DNA defines 'an error of between 700-fold and 2000-fold in the ICRP model for heritable damage', is wholly unsubstantiated. It ignores scientific uncertainty on these data and current knowledge on the molecular basis of heritable diseases (UNSCEAR, 2001).

In conclusion, the weight of evidence and considerations of biological plausibility argue against ECRR's thesis that ICRP's risk assessment methodology seriously underestimates risks from internal emitters. Furthermore, ECRR's proposed methodology is arbitrary, and does not have a sound scientific basis. There are many misrepresentations of ICRP, misunderstandings, inconsistencies and unsubstantiated claims in the ECRR report. It compares poorly with the detailed justification and referencing of published data characteristic of ICRP reports. In general, there is a good understanding of the risks associated with exposure to ionising radiation. Uncertainties in dose estimates for internal emitters are greater than for external radiation because of the necessity of estimating the time-course of exposure of different body tissues, but these uncertainties do not correspond to the large underestimates of risk claimed by ECRR. For these reasons the ECRR report cannot provide a basis for changing radiological protection standards.

(END)

References

Charles MW and Mill AJ (2003). Carcinogenic risk of hot particle exposures. *J Radiol Prot*, **23**, 5-28.

COMARE (1988). Second Report. Investigation of the possible increased incidence of leukaemia in young people near the Dounreay Nuclear Establishment, Caithness, Scotland. London, HMSO.

COMARE (1989). Third Report. Report on the incidence of childhood cancer in the West Berkshire and North Hampshire area, in which are situated the Atomic Weapons Research Establishment, Aldermaston and the Royal Ordnance Factory, Burghfield. London, HMSO.

COMARE (1996). Fourth Report. The incidence of cancer and leukaemia in young people in the vicinity of the Sellafield site, West Cumbria: Further studies and an update of the situation since the publication of the report of the Black Advisory Group in 1984. London, Dept of Health.

R3/03
24 July 2003

COMARE (1998). Fifth Report. The incidence of cancer and leukaemia in the area around the former Greenham Common Airbase. An investigation of a possible association with measured environmental radiation levels. Chilton, NRPB.

COMARE (1999). Committee on the Medical Aspects of Radiation in the Environment. Further Statement on the Incidence of Childhood Cancer in Wales. COMARE website: http://www.comare.org.uk/statements/comare_statement_wales.htm

COMARE (2003). Committee on the Medical Aspects of Radiation in the Environment. COMARE Statement: Cancer Mortality around the Bradwell Nuclear Power Station, Essex. COMARE website: http://www.comare.org.uk/statements/comare_statement_bradwell.htm

EC (1996). Council Directive 96/29EURATOM of 13 May 1996 laying down the basic safety standards for the protection of the health of workers and the general public against the dangers arising form ionising radiation. *Off J Eur Commun,* **39** (L159).

Edwards AA and Cox R (2000). Commentary on the second event theory of Busby. *Int J Radiat Biol*, **76** (1), 119-125.

Green Audit (2003). 2003 Recommendations of the European Committee on Radiation Risk. Editor Chris Busby with Rosalie Bertell, Inge Schmitz-Feuerhake, Molly Scott-Cato and Alexei Yablokov - http://www.euradcom.org/2003/ecrr2003.htm#press

Harrison JD and Muirhead CR (2003). Quantitative comparisons of cancer induction in humans by internally deposited radionuclides and external radiation. *Int J Radiat Biol*, **79**, 1-13.

IAEA (1996). International basic safety standards for protection against ionising radiation and for the safety of radioactive sources. Jointly sponsored by FAO, IAEA, ILO, NEA/OECD, PAHO and WHO. Vienna, IAEA, Safety Series No 115.

ICRP (1997). Radiological protection policy for the disposal of radioactive waste. ICRP Publication 77. *Ann ICRP*, **27** Supplement.

NRPB (1995). Risk of radiation-induced cancer at low doses and low dose rates for radiation protection purposes. *Doc NRPB*, **6** (1).

UNSCEAR (2000). United Nations Scientific Committee on the Effects of Atomic Radiation, 2000 Report to the General Assembly, with Scientific Annexes, Volumes 1 and 2. New York, United Nations.

UNSCEAR (2001). United Nations Scientific Committee on the Effects of Atomic Radiation, 2001 Report to the General Assembly, with Scientific Annex, Hereditary Effects of Radiation. New York, United Nations.

WHO (2001). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol 78 Ionizing Radiation, Part 2: Some Internally Deposited Radionuclides. World Health Organisation, International Agency for Research on Cancer. Lyon, IARC Press.

R3/03
24 July 2003

National Radiological Protection Board,
Chilton, Didcot, Oxon, OX11 0RQ
Telephone: (01235) 822744 Fax: (01235) 822746
Email: pressoffice@nrpb.org