Letter to the Editor:

Effects of ionising radiation on biota: Do we need more regulation?*

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1. Introduction

There has been much recent interest in protection of the environment from ionising radiation. The arguments concerning this issue are varied (e.g. IUR, 2003; Bréchignac, 2003; Pentreath, 2004; Strand and Borretzen, 2004). Firstly, the statement of the International Commission on Radiological Protection (ICRP, 1977) that

\[
\text{if man is adequately protected [from ionising radiation] then other living things are also likely to be sufficiently protected}
\]

has been criticised as “anthropocentric” and inadequate for protection of the environment from ionising radiation. The ICRP statement, usually quoted out of context, appears dismissive of the important issue of environmental protection. It is worth, therefore, reading the full paragraph in which this statement appears (ICRP, 1977):

\[
\text{Although the principal objective of radiation protection is the achievement and maintenance of appropriately safe conditions for activities involving human exposure, the level of safety required for the protection of all human individuals is thought likely to be adequate to protect other species, although not necessarily individual members of these species. The Commission therefore believes that if man is adequately protected then other living things are also likely to be sufficiently protected.}
\]

Though much criticised in recent years, in my view this statement has the considerable virtue of being a scientific hypothesis. In contrast to many subsequent comments made on this issue, the wording is clear and the words used can be defined in a scientifically meaningful way. It is therefore falsifiable, fulfilling the key criterion of a scientific hypothesis (Popper, 1963). It is not a purely “anthropocentric” approach. It uses humans as the most radio-sensitive “sentinel” species in order to protect all other species. If it works (and I believe that it does), then it can equally protect biota from ionising radiation. As I will discuss below, this hypothesis has been tested by empirical observations and has thus far protected the environment against damage from regulated releases of ionising radiation. It is, however, fair to criticise the statement because it only presents the hypothesis without stating the reasoning behind it.

It has been argued that as a method of environmental protection from ionising radiation this approach is inadequate for a number of reasons which can be summarised as follows:

1. there are many “data gaps” in current knowledge: more dose-effect studies are required of species other than man, bioaccumulation rates in non-human food chains are unknown for some radionuclides, and current models for bioaccumulation and dose calculation need to be improved to cover more organisms and radionuclides;
2. there are wildlife habitats where humans are not exposed to ionising radiation, for example, some aquatic environments and the Chernobyl Exclusion Zone;
3. the system of environmental protection from radiation is different to that for chemical toxins which explicitly considers environmental protection.
4. that the current system is not sufficient to demonstrate to decision-makers, pressure groups and the wider public that the environment is adequately protected.

In this letter I will expand the argument presented in Smith (2004) that these criticisms are not valid. Whilst the ICRP (1977) statement may need updating - a process currently underway within ICRP (ICRP, 2004) - I will argue that this simple hypothesis has not been falsified. In view of the insignificant effect of regulated releases of ionising radiation on wildlife, I believe that the simple and cost effective regulatory approach should be retained. This would allow more resources and regulatory effort to go to the many more pressing environmental problems we face.

1.1. Context

The damage humans have done (and continue to do) to the environment is so obvious that it seems unnecessary to re-state it here. But it is important to consider regulatory efforts for ionising radiation within the framework of overall environmental damage. Humankind’s current rate of environmental destruction is staggering. Approximately 14.6 million hectares (an area approximately the size of Greece) of forest are lost worldwide each year (WWF, 2002). A number of studies in recent years support the hypothesis that, because of human impacts such as this, “the biological world is approaching the sixth major extinction in history” (Thomas et al., 2004). In view of the ongoing environmental crisis, any sensible regulatory system should focus costs and resources on the most serious environmental problems and make regulation of minor or insignificant problems as simple as possible.

It is worth also considering more local environmental impacts which are allowed within the frame of current environmental legislation. In doing this I intend to counter some (but not all) of the current thinking in the on-going debate on radiological protection of the environment which appears to assume that no level of damage to individual organisms by ionising radiation is tolerable: i.e. that the regulatory limit should be set on a “no observable effects” basis. Current regulation of environmental damage (from impacts other than radiation) does not always attempt to reduce that damage to zero: this would be impossible. It rather attempts to weigh benefits from an action against risks in a manner which is acceptable to the majority of people. For example, the bed of the Irish Sea is subject to major damage from bottom trawling by commercial fisheries. In Norway, rotenone poison has been used to protect salmon fisheries (rotenone not only kills salmon and their parasites, but at the same time kills all other fish and most other aquatic animals).

I do not here make any ethical judgement on these activities: I simply wish to make the point that many actions which damage or alter the ecosystem are allowed because of their perceived benefits. Though I do not believe that regulated radioactive releases do
any significant damage to the ecosystem, I think that a “no observable effects” approach to their regulation would not necessarily be appropriate (though this is achievable, depending on how one defines “effect”).

2. Modelling

2.1. Environmental concentrations of anthropogenic radionuclides

In assessing the regulatory requirements for environmental protection against radiation, it is useful to consider how serious an environmental problem this is. Clearly, if regulated releases were (or were likely to become) significantly damaging to the environment, then improved regulation would be urgently needed. The simplest approach to assessing the impact of radioactive pollution of the environment is to calculate “screening” concentrations of radionuclides which could lead to a potentially significant dose. This is one of a number of approaches being considered by the ICRP (2004). A screening model study by Amiro (1997) estimated Dose Conversion Factors (DCFs) for biota based on total absorption of internal radiation by the organism and estimation of external dose using assumed conservative external exposure scenarios. A similar approach was taken by US DOE (2002), Jones et al. (2003) and Higley et al. (2003) in developing a model to screen environmental media. The Environmental Quality Standard (EQS) approach I have illustrated below is similar in principle to that previously presented by these workers.

The illustrative model I present below is similar to these previous approaches, but differs slightly in that it does not focus on any particular organism, environmental medium, or exposure scenario. Instead, it estimates a minimum concentration in any organism, tissue, or environmental medium which could lead to a dose rate to any organism above a given exposure limit. It does not explicitly calculate internal and external dose rates to an organism (or tissue), but implicitly determines these doses on the assumption that (for an equal activity concentration in the organism and the medium in which it resides), radiations escaping the organism (or tissue) are approximately balanced by the incoming radiations from the outside medium. I use it here as a simple way of determining concentration limits in organisms and environmental media.

I will here assume that the maximum allowable dose rate to any individual organism is 1 mGy d\(^{-1}\), as recommended by IAEA (1992). This compares with typical limits of 0.1 – 1.0 mSv y\(^{-1}\) for protection of the human population. For \(\beta-\) and \(\gamma-\)radiation, the limit to protect humans is therefore approximately one thousand times lower than that to protect individual biota. Note that FASSET (2004) found “few indications of readily observable effects [on biota] at dose rates less than 100 \(\mu\)Gy h\(^{-1}\) [2.4 mGy d\(^{-1}\)]”. Note that a relative biological effectiveness (RBE) is not applied here since the analysis is based on no effects at a given absorbed energy: i.e. the 1 mGy d\(^{-1}\) value. If \(\alpha\)-particles (or other radiations) were shown to be damaging at less than 1 mGy d\(^{-1}\), an RBE could easily be incorporated into the approach outlined here.

In an object which is infinite in extent and uniformly contaminated by a radionuclide, the average energy deposited (per unit mass or volume) at any point in that object is
equal to the average energy generated at any point (per unit mass or volume). (This approach was previously used by Amiro (1997) for internal exposures.) The rate of energy deposition (dose rate), \( E \) (Gy s\(^{-1}\)) in an object uniformly contaminated with concentration \( C \) (Bq kg\(^{-1}\), wet weight, w.w.) of any radionuclide is given by:

\[
E = C \times 1.6 \times 10^{-19} \sum \varepsilon_i
\]

where \( \varepsilon_i \) is the mean energy in electron Volts (1eV = 1.6 × 10\(^{-19}\) J), of the \( i \)th radiation emitted (weighted by intensity) when the radionuclide undergoes decay. The concentration, \( C_{\text{min}} \) (Bq kg\(^{-1}\) wet weight) of the given radionuclide required to give an average energy deposition of 1 mGy d\(^{-1}\) in this infinitely extended object is therefore:

\[
C_{\text{min}} = \frac{1}{60 \times 60 \times 24 \times 1000} \times \frac{1}{1.6 \times 10^{-19} \sum \varepsilon_i} = \frac{1}{1.38 \times 10^{-11} \sum \varepsilon_i}
\]

The assumption that the spatial extent of contamination is infinite means that the dose arising from a concentration, \( C_{\text{min}} \), will in practice usually be less than 1 mGy d\(^{-1}\). The extent to which the concentration required to give 1 mGy d\(^{-1}\) dose is under-estimated depends on the extent of contamination of a soil, sediment or water, the size of the organism and the type and energy of the emitted radiation. For \( \alpha \)-radiation, the path length in tissue is typically a few tens of \( \mu \)m (a few cells thickness), so a concentration, \( C_{\text{min}} \), of an alpha-emitting radionuclide in an organism or tissue will give rise to a dose very close to 1 mGy d\(^{-1}\) to that organism or tissue, except for extremely small organisms. For \( \beta \)-radiation, the typical path length in water, soil or tissue is approximately 2 mm for 0.5 MeV electrons rising to around 10 mm for 3 MeV electrons. Typically, mean \( \beta \) emission energies are of order 0.5 MeV or less, so for organisms of dimensions \( > 2 \) mm, \( C_{\text{min}} \) for a primarily \( \beta \)-emitter will again give rise to a dose close to 1 mGy d\(^{-1}\). For smaller organisms, \( C_{\text{min}} \) will give rise to a dose lower than 1 mGy d\(^{-1}\), unless the organism is residing in an environmental medium (soil, sediment or water) of activity concentration similar to \( C_{\text{min}} \).

The typical path length (for factor 10 reduction in intensity) of \( \gamma \)-radiation in water, soil or tissue is of order 12 cm for 0.1 MeV radiation and of order 30 cm for 1 MeV radiation. Therefore, an activity concentration \( C_{\text{min}} \) of a primarily \( \gamma \)-emitter in an organism will give rise to a dose significantly lower than 1 mGy d\(^{-1}\) in all but the largest organisms, unless the organism is residing in an environmental medium of activity concentration similar to \( C_{\text{min}} \). A very small organism living several cm deep within a soil or sediment of uniform concentration \( C_{\text{min}} \) would receive an external dose from \( \beta \) and \( \gamma \) radiation of approximately 1 mGy d\(^{-1}\).

Values of \( C_{\text{min}} \) are given in Table 1 for some (primarily) \( \alpha \)-emitting radionuclides and in Table 2, for some \( \beta \) and \( \gamma \)-emitting radionuclides. These values represent estimates of the minimum concentration in any object in a contaminated environment which could give rise to a dose at the chosen threshold of 1 mGy d\(^{-1}\). If organisms (for \( \alpha \), \( \beta \), \( \gamma \)) soil, sediments, and water (for \( \beta \), \( \gamma \)) contain radionuclide concentrations lower than this value.
then no organism is estimated to receive a dose of greater than 1 mGy d\(^{-1}\) (from external and/or internal radioactivity). Put another way, if every object in an environment (soil, sediment, water, organisms) were contaminated with uniform concentration \(C_{\text{min}}\), the dose rate arising in that environment would be approximately 1 mGy d\(^{-1}\). \(C_{\text{min}}\) values for radionuclides other than those presented in Tables 1 and 2 can be calculated from information on energy emitted per radioactive decay given in, for example, ICRP (1983), Delacroix et al. (1998) and Chu et al. (1999).

For contamination of an environment by \(N\) different radionuclides of activity concentration \(C^j\) (Bq kg\(^{-1}\)), the following criterion would be required:

\[
\sum_{j=1}^{N} \frac{C^j}{C_{\text{min}}^j} \leq 1
\]  

(3)

In this case, the concentration \(C^j\) is the maximum concentration of a given radionuclide in an organism, or the relevant environmental medium, whichever is the higher. For example, if mussels are contaminated with 30000 Bq kg\(^{-1}\) of \(^{99}\)Tc and 2500 Bq kg\(^{-1}\) of \(^{137}\)Cs and the sediment on which they live is contaminated with 1500 Bq kg\(^{-1}\) of \(^{99}\)Tc and 10 000 Bq kg\(^{-1}\) of \(^{137}\)Cs, then the calculation is:

\[
\frac{C^{Tc}}{C_{\text{min}}^{Tc}} + \frac{C^{Cs}}{C_{\text{min}}^{Cs}} = \frac{30000}{854000} + \frac{10000}{90000} = 0.146 \leq 1
\]

since the highest \(^{99}\)Tc activity concentration is in the mussels, whilst the highest \(^{137}\)Cs is in the sediment. Note that for primarily \(\alpha\)-emitters (i.e. the vast majority of the emitted energy is in \(\alpha\)-particles), activity concentrations in environmental media would not be included in the calculation since it is recommended that only internal doses are considered for these radionuclides.

Though this is a crude approach, it does provide an extremely simple model for screening a contaminated environment for potential effects on biota and is useful here to illustrate the levels of environmental contamination by single radionuclides required to potentially reach a significant dose to organisms. For multiple radionuclides, Equation 3 may need to be repeatedly applied to different biota (tissues) and environmental media to test whether any fail the criterion. An illustration of the model is given below. If any material failed simple screening models such as this, then more complex modelling may be required. However I think this would be extremely unlikely for current or future regulated releases, so (according to current knowledge of radiation exposures and effects) more complex modelling is unlikely to be required.

Note that the concentrations in soil, sediment and water are not intended to imply that organisms living in environmental media with these concentrations could not bio-accumulate radioactivity to a level above \(C_{\text{min}}\). For example, a fish living in water contaminated with 90 000 Bq kg\(^{-1}\) of \(^{137}\)Cs would certainly bio-accumulate radiocaesium to a level much greater than \(C_{\text{min}}\). The \(C_{\text{min}}\) values therefore only represent
a measure against which to assess maximum doses arising from measured (or predicted) concentrations in environmental media or organisms. They do not attempt to account for bioaccumulation of radionuclides.

2.2. Accounting for bioaccumulation in environmental media

A complementary system for assessing activity concentrations in environmental media is to determine activity concentrations in that medium which could lead to activity concentrations in biota which are above the $C_{\text{min}}$ values. An illustration of these concentrations for some $\beta$- and $\gamma$-emitting radionuclides in freshwaters is presented in Table 3. Such concentrations are analogous to Environmental Quality Standards (EQS) for chemical toxins in freshwaters. The activity concentration, $C_{\text{EQS}}$ in water (Bq kg$^{-1}$) which would lead to an activity concentration, $C_{\text{min}}$, in fish is given by:

$$C_{\text{EQS}} = \frac{C_{\text{min}}}{CF}$$

where $CF$ (dimensionless) is the Concentration Factor for freshwater fish. As is appropriate for regulated releases, I have here applied an equilibrium bio-accumulation model. The model presented here is similar to that in US DOE (2002), but the US DOE model accounts for uncertainty in $CF$ values whereas these illustrative values do not. Note also that the dose limits used in US DOE (2002) (10 mGy d$^{-1}$ for aquatic animals and terrestrial plants, 1 mGy d$^{-1}$ for terrestrial animals and an RBE for $\alpha$-particles) are different to the limit I have chosen here.

3. Discussion

It is important to note that the concentrations presented in Tables 1 - 3 are, in the context of regulated radionuclide releases, extremely high. To my knowledge, there is nowhere in Western Europe where radionuclides from current regulated releases reach anywhere near these levels (the coastal area near Sellafield, UK, is discussed below).

In the UK (for example) activity concentrations of radionuclides in the environment from regulated releases are typically a few bequerels per kg and usually less (RIFE, 2003). Some of the highest activity concentrations in biota in recent years have been observed around BNFL Sellafield (Table 4) and in the Severn Estuary from releases from Amersham plc, Cardiff (Table 5). Tables 4 and 5 show application of the illustrative screening model to lobsters near Sellafield and molluscs near Cardiff. In both cases, concentrations of radioactivity in these organisms/environmental media are around two orders of magnitude lower than the screening criterion (Equation 3). Note that in the Sellafield example, I have not included contamination of sediments since I wanted to illustrate doses from recent discharges, particularly of $^{99}$Tc, the discharges of which have been a topic of some controversy in recent years. Historic discharges from Sellafield are discussed below.
Even in the context of current contamination from past accidental releases, the $C_{\text{min}}$ concentrations are relatively high. The concentrations of Pu isotopes presented in Table 1 are higher than those expected to occur in soils of the most contaminated parts of the Chernobyl Exclusion Zone (estimated from data in Lux et al., 1995; Kashparov et al., 2003). Because bioaccumulation factors are low for these $\alpha$-emitters, their activity concentrations in organisms in this area are likely to be orders of magnitude lower than the values presented in Table 1 (for these primarily $\alpha$-emitting radionuclides, it is recommended that only internal exposure is considered). Activity concentrations of $^{90}\text{Sr}$ and $^{137}\text{Cs}$ in the Chernobyl Cooling Pond are currently lower than their $C_{\text{EQS}}$ values presented in Table 3 (Institute of Hydrobiology, Ukraine, unpubl. res.), though a few small lakes around Chernobyl have higher activity concentrations. Similarly, levels of $^{137}\text{Cs}$ and $^{90}\text{Sr}$ in soils and terrestrial biota in some of the most contaminated parts of the Exclusion Zone, and in the Cooling Pond sediments can exceed the $C_{\text{min}}$ values in Table 2. For illustration, assuming a wet bulk density of soil of 1700 kg m$^{-3}$ and uniform contamination of the soil to a depth of 10 cm, the $C_{\text{min}}$ values for $^{90}\text{Sr}$ and $^{137}\text{Cs}$ represent inventories per unit area of 10.9 MBq m$^{-2}$ and 15.3 MBq m$^{-2}$ respectively.

For some radionuclides, chemical toxicity is much more significant than any radio-toxic effects, as has been discussed by Jones et al. (2003). For example, the activity concentrations ($C_{\text{min}}$) of uranium isotopes in Table 1 represent concentrations of natural uranium in the body (or a particular tissue) of an animal or plant of 0.64 grammes per kg (to deliver a maximum dose of 1 mGy d$^{-1}$). This limiting concentration for radionuclides is significantly higher than levels expected to cause effects from the chemical toxicity of uranium. The LD50 (the dose at which 50% of the organisms are killed) for uranium orally administered as uranyl acetate is 0.242 g kg$^{-1}$ and 0.201 g kg$^{-1}$ in mice and rats respectively (Domingo et al., 1987). For $^{234}\text{U}$ (specific activity more than 1000 times higher than $^{235}\text{U}$ or $^{238}\text{U}$), Jones et al. (2003) argued that “chemical toxicity of $^{234}\text{U}$ is a potential concern at concentrations below the radiological benchmark”.

3.1. “Data gaps”

When considering the question of whether there are significant gaps in the data available to assess environmental impacts of radiation in biota, it is important to note that there are always an infinite number of “data gaps” in any regulatory system. All the proposed methods of environmental protection acknowledge the fact that it is impossible to collect information on radionuclide uptake to every tissue of every species of organism and to assess every effect on every species or combination of species: pragmatic approaches to this issue have been discussed by (for instance) Howard and Beresford (2004). The truism that it is impossible to have full information on all species lies behind proposals to regulate doses to biota using “reference organisms” (Pentreath, 2004; FASSET, 2004). The question, then, is not “Are there data gaps?”, because there are always data gaps, but “Is it worth spending money and effort to improve the data (and models) available?” Since there is never a point at which data is complete, this is a matter of judgement of where to focus resources to best protect the environment.
I believe that there are significant data gaps which should be addressed by radioecologists, but these relate primarily to the system of protection of humans from ionising radiation: for example, in biosphere transfers of some very long-lived radionuclides. If we can reliably ensure that doses to humans are less than permitted levels, I believe (as the ICRP hypothesis states) that this will ensure that wildlife are not significantly damaged. This does not assume that human food chains are the same as wildlife food chains. It merely assumes that the (highly diverse) human food chain covers a sufficiently large number of organisms and environments to adequately ensure that activity concentrations in biota will be below levels which would cause significant damage. If dose limits for humans were the same as those for biota, such an approach would not be adequate. But, dose limits to humans are much more conservative (by around 3 orders of magnitude) than those for biota.

3.2. “Harmonisation” with systems for chemical pollutants

A question related to “data gaps” concerns comparison between the system of radiation protection of biota and that of protection from chemical pollutants. In these discussions it is often implied that the system for chemical pollutants is superior to that for radioactive pollutants. I believe that there is more and better information available concerning the environmental transfers and effects of radioactivity on biota than on most chemical (particularly organic) contaminants. Chemical risk assessments commonly use data from one species to predict uptake and effects on another. For example, there is little or no information on uptake and effects of many organic pollutants in predatory birds, partly because experimentation on such species is morally unacceptable to many people. As would likely be the case for radiological assessments, data from rats or domestic birds are commonly used instead (e.g. EC, 2002).

There is a huge and growing literature on the effects of radiation on many species of biota, and modelling and monitoring of radioactivity in the environment is very well developed. In contrast, new pesticides (for example) require only relatively simple toxicity testing before being allowed onto the market. This usually begins with acute toxicity tests on selected organisms (e.g., for freshwaters, algae, daphnia and rainbow trout) to calculate the LC50 (the concentration at which 50% of the organisms are killed) or the EC50 (the concentration at which some response – e.g. number of daphnia immobilised – reaches 50%). This acute test is carried out over an exposure period of typically 24 – 96 hours. The likely exposure (“predicted environmental concentration”, PEC) of the organism is then estimated using simple environmental models. If: (LC50 or EC50) ÷ PEC > ~ 100 then the pesticide passes the test. If it fails, chronic toxicity tests may be required. These are carried out over a longer period (e.g. from 21 days up to the full life cycle of the organism). The chronic toxicity test is passed if (LC50 or EC50) ÷ PEC ≥ ~ 5 – 10, or PEC/NOEC < 1, NOEC being the “no observable effects” concentration.

The LC50 for exposures to radiation over a period of 30 days is more than 150 mGy d⁻¹ for even the most radio-sensitive of organisms (IAEA, 1991). The LC50 for shorter exposures is likely to be much higher. Exposures of biota from regulated releases of
radioactivity (given the stringent regulations to protect human health) are expected to be (and experience has shown to be) much less than 1 mGy d$^{-1}$. This value is directly analogous to the PEC for chemical toxins. Applying the acute toxicity test criterion: $\text{LC}_{50} \div \text{PEC} > 150$. This is greater than the value of 100 required to pass the acute toxicity test. For chronic exposures of fish (for example) “reduced reproductive success would be likely at dose rates in the range of 24-240 mGy d$^{-1}$” (IAEA, 1991), thus easily passing the chronic toxicity test assuming exposures (“PEC”) of 1 mGy d$^{-1}$ or less.

I do not here suggest that these toxicity tests are necessarily appropriate for ionising radiation. I intend only to illustrate that (a) the data available for radiation protection of the environment is analogous to that available for chemical pollutants (indeed it is better than that for most chemicals because many more species have been studied) and (b) regulated radionuclide releases would easily pass chemical toxicity testing criteria. In contrast to the relatively simple system for chemical toxicity where a few species are studied in laboratory tests, studies of radiation effects on biota cover many different effects at a large number of different exposures to dozens (maybe hundreds) of different species.

3.4. Testing the ICRP hypothesis at Savannah River and Sellafield

Whicker and Bedford (1995) examined the ICRP hypothesis in relation to radiation doses arising from radioactivity in Par Pond, a former reactor cooling reservoir at the Savannah River site in the USA. The partially drained pond was contaminated with around 1 MBq m$^{-2}$ of $^{137}$Cs. This paper (Whicker and Bedford, 1995) summarised the results of assessments of doses to humans and biota at Par Pond, as follows:

Site specific data and simple models were used to estimate both human health and ecological risks for hypothetical human residents subsisting on the lake bed and for resident aquatic and terrestrial organisms. Whereas the potential maximum lifetime risk of fatal cancer for humans was about $3 \times 10^{-3}$ (over one order of magnitude above that requiring action under the US Environmental Protection Agency’s “Superfund” Act), the risk to local biota was two to three orders of magnitude below the threshold for reproductive impairment. Actually, in this case, the criterion used for human health protection was more sensitive (by about three orders of magnitude) than the 1 mGy d$^{-1}$ criterion for plant and animal populations proposed by the IAEA. Clearly, in this case, and likely in nearly all cases, human health risk and not ecological risk is probably the limiting factor. Furthermore, [in a review of literature] no cases were found where radionuclide releases or contamination within allowable limits for human exposure have led to documented effects on other species or on environmental quality.

The Savannah River site did have a detrimental effect on the ecology of some local aquatic systems, but this was due to the release of cooling water at a temperature of around 70 °C, not to radioactive releases.
Releases of radioactivity from the Sellafield site in Cumbria (UK) provide another test of the ICRP hypothesis. Discharges of radioactivity from Sellafield to the Irish Sea during the 1970's resulted in exposures to a critical group of local people (with high consumption rates of locally caught fish, crustaceans and molluscs) of approximately 2-3 mSv y\(^{-1}\) (Kershaw et al., 1992). This is significantly higher than current guidelines for doses to members of the public arising from nuclear installations (in the UK these should be limited to < 0.3 mSv y\(^{-1}\)). In spite of the relatively high doses to the human population, biota were not predicted to be affected by releases from Sellafield. Kershaw et al. (1992) quote Woodhead (1980):

*The dose rates around Sellafield are at least an order of magnitude below those which would be expected to elicit any effect under controlled laboratory conditions, and about two orders of magnitude below those which might be expected to have an effect at the population level (Woodhead, 1980) during the period of maximum discharges.*

3.5. Accidentally contaminated environments

It has been argued that the ICRP hypothesis is limited because it does not apply to areas such as the Chernobyl Exclusion Zone where humans are not exposed to ionising radiation but organisms are. This argument does not, however, invalidate the ICRP hypothesis. The Chernobyl accident, obviously, was not a regulated release.

Studies of the area around Chernobyl appear to support the ICRP hypothesis rather than contradict it. It is well known (e.g. IAEA, 1991) that extremely high dose rates during and shortly after the accident led to serious damage to wildlife (to pine trees in particular) in some areas. There is evidence (though some is contradictory) that much lower chronic exposures in subsequent years have had a detrimental/measureable effect on organisms. There is some evidence of increased fluctuating asymmetry in wildlife, potentially indicating radiation stress (Moller, 1998). There is also evidence, for example, of increased microsatellite mutation rate in wheat grown on very contaminated soils (Kovalchuk et al., 2000) and damage to the DNA of catfish in the Cooling Pond (Sugg et al., 1996). Some studies of rodents have shown a negative impact of radiation (see studies quoted in Jackson et al., 2004), though a study showing high levels of genetic change in rodents (Baker et al., 1996) was subsequently retracted (Baker et al., 1997).

Physiological studies of small mammals at sites of varying contamination levels near to Chernobyl were carried out by Jackson et al. (2004). The most contaminated study site had \(3.5 \times 10^6\) Bq kg\(^{-1}\) of \(^{137}\)Cs and \(2.5 \times 10^6\) Bq kg\(^{-1}\) of \(^{90}\)Sr in the surface soil layer, and external gamma dose rates to humans of up to 3.4 mSv d\(^{-1}\) (Red Forest site). This study concluded that:

*Overall, the findings ... indicate that no gross physiological effects on body mass or organ size can be detected and there are no obvious effects on population diversity and abundance, or on age or sex distribution. These results*
support the general conclusions of previous commentators that little harmful effect is evident on small mammal populations within the exclusion zone.

In assessing the ecological consequences of the Chernobyl accident, the (potential) negative impact of radiation on the environment must be weighed against the positive impact the removal of humans from the area has had on wildlife habitats. On a macro-ecological (i.e. large-) scale, there has been a dramatic increase in populations of wild mammals and bird species (Williams, 1995; Baker and Chesser, 2000). Radiation levels in the 30 km Zone are still in many areas much higher than those considered safe for human habitation. But wildlife appears to have been relatively unaffected by the radiation and has therefore benefited from the absence of human disturbance and damage through agriculture, forestry, hunting and fishing. I do not wish in any way to downplay the tragic consequences of Chernobyl to the human population of the area. However, the experience from Chernobyl is a clear demonstration of the insignificance of chronic radiation effects on wildlife compared to the huge environmental impact of humankind’s everyday activities.

An earlier study at a site at Rocky Flats (Colorado, USA) contaminated with plutonium also demonstrated the resilience of wildlife to chronic radiation. This study is summarised in an IAEA (1991) report as follows:

Measurements included vegetation structure and biomass; litter mass; arthropod community structure and biomass, population density, biomass, reproduction, organ mass, pathology, and parasite occurrence in a small mammal species. No differences attributable to radiation exposure were found for any of the measurements, even though levels of $^{239}$Pu in the upper 3 cm of the soil were as high as $1.5 \times 10^7$ Bq m$^{-2}$ [$= 294 \, 000$ Bq kg$^{-1}$, assuming a soil density of 1700 kg m$^{-3}$].

3.6. Deep sea waste disposal

The deep sea is one wildlife habitat not utilised by humans and therefore the ICRP hypothesis may not apply. Past dumping of waste in the deep North Atlantic could have led to doses to marine organisms of up to $\sim 0.2$ mGy d$^{-1}$ (Woodhead, 1993) whilst exposures to humans were minimal. Deep sea disposal may therefore require specific assessment of doses to biota. This issue may, however, be somewhat academic since the London Convention on the Prevention of Marine Pollution by the Dumping of Wastes prohibits dumping of waste at sea.

3.7. What happens when biota are exposed?

Oughton (2003) has highlighted some ethical complexities in protecting biota from ionising radiation as exemplified by an incident at the Dounreay UK Atomic Energy Authority (UKAEA) site. In 2003, a Scottish Environment Agency (SEPA) inspector observed rabbits burrowing near to pits containing low-level waste encased in concrete. There was some concern that rabbits could become contaminated and pose a potential threat to the human food chain. The UKAEA took action to prevent wildlife from
entering the area, and arranged “a cull of rabbits on the site and [reviewed] what measures may be taken to curb other wildlife” (quoted in Oughton, 2003). The “Dounreay rabbit incident” may have been sparked by a previous incident at Sellafield: “Sellafield’s nuclear pigeons”. In 1998, elevated levels of radioactivity were found in pigeons in the area of the British Nuclear Fuels Limited (BNFL) Sellafield site in Cumbria (FSA, 2004). There was concern that local residents could be exposed, though no evidence was found of local people consuming pigeon meat. The response of BNFL was to cull the entire flock and bury the bodies at the Drigg low level waste dump. Possible effects of radiation on the pigeons were not discussed in reports.

Note, firstly, that in both of these incidents, perceived risks to human health triggered the response by regulatory authorities, not potential damage to wildlife (supporting the ICRP hypothesis). Secondly, the solution to the problem was, in both cases, to kill the animals then to take action to prevent wildlife from entering the contaminated areas. If the response to contamination is (at best) to prevent wildlife from entering an area (i.e. to take away their habitat) this makes a nonsense of specific environmental protection regulations. Habitat removal is very likely to destroy or seriously reduce a wildlife population: in other words it would be far more damaging to that population than the radiation from which it was being “protected”. In the US, Whicker et al. (2004) have argued against destructive remediation of US DOE sites (potentially required because of a possible radiation risk to humans) because of the damage this would do to wildlife habitats.

Of course, for potential future releases, the best solution would be to prevent wildlife from becoming contaminated in the first place. However, for reasons outlined in this letter, I do not think that regulated releases would ever in practice lead to significant exposures to wildlife because of the stringent limits to reduce human exposures. Neither of the above examples were regulated contamination pathways: they should not have occurred as they presented a potential risk to humans.

4. Conclusions

4.1. Public perception and “political” considerations

It would be naïve of me to think that the current focus on protecting the environment from ionising radiation is based on a genuine belief that this will prevent environmental damage. I know of no incidents of regulated releases of radioactivity in Western Europe which have caused any radiation damage to wildlife. Much of the work on this issue is being driven by environmental legislation. The fact that this is more a regulatory issue than a real environmental problem has previously been noted by some working in the “doses to biota” field (e.g. Pentreath, 2004; Higley and Alexakhin, 2004) and this appears to be the current position of the ICRP itself (ICRP, 2004). The primary intention appears to be to develop a system which more clearly demonstrates to pressure groups, politicians and the public (and in response to legislation) that the environment is being protected than the somewhat stark ICRP hypothesis. This is a worthwhile goal, and one which is currently being pursued by an ICRP working group.
In my opinion, however, some of the approaches currently being proposed and developed in the field of environmental protection from ionising radiation are not either the most efficient or effective way to achieve this goal. Some of the approaches currently being suggested consider application of new dynamic bioaccumulation models (Copplestone et al., 2004), complex dosimetric models and perceived “data gaps” (FASSET, 2004) and attempts to relate “damage” of the individual to population level effects (Bréchignac, 2003; Woodhead, 2003). For reasons given above, I do not believe that these more complex approaches are justified by the available empirical evidence.

4.2. A pragmatic approach

I believe that whilst empirical radioecological research to test the ICRP hypothesis should continue, this simple and cost-effective approach should be retained until there is clear evidence to show that it does not work. The statement could be re-written to better demonstrate that this is not simply an “anthropocentric” approach and to better highlight the wealth of empirical evidence to support it. If reference levels for environmental levels of radioactivity are required by regulatory authorities for assessment and to “demonstrate compliance” by industry, simple dosimetric models (which have been available for many years) are adequate to calculate limiting concentrations, as discussed above. Such calculations clearly demonstrate that regulated radioactive releases do not significantly damage ecosystems. If more complex approaches are proposed, they should be justified by demonstrating that they will lead to a significant environmental benefit.

I am not here advocating an end to scientific research on this issue. Empirical radioecological research to test our hypotheses concerning dose rate limits and effects is important. For example, well-conducted studies in radioactively contaminated environments such as the Chernobyl Exclusion Zone have had (and I hope will continue to have) enormous value in empirically quantifying these effects. Such studies make a vital contribution to scientific and public understanding of the environmental risks of radiation and to debates on the environmental costs and benefits of nuclear energy.

But, I believe that a clear and simple regulatory response to this issue is the best way to alleviate people’s natural concern over potential radiation damage to the environment. This would be a response in proper proportion to this insignificant environmental problem. Furthermore, it would benefit the environment by directing scarce resources to more significant and urgent problems.
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Tables

Table 1. Minimum concentration, $C_{\text{min}}$ (wet weight) of principally $\alpha$-emitting radionuclides in an organism or tissue which could result in a dose rate of 1 mGy d$^{-1}$.

<table>
<thead>
<tr>
<th>Radionuclide</th>
<th>Mean $\Sigma \varepsilon$ per Disintegration$^+$, MeV</th>
<th>Dose rate, mGy d$^{-1}$ per Bq kg$^{-1}$</th>
<th>Minimum conc., Bq kg$^{-1}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>U-234</td>
<td>4.77</td>
<td>$6.6 \times 10^3$</td>
<td>15 200</td>
</tr>
<tr>
<td>U-235</td>
<td>4.58</td>
<td>$6.3 \times 10^5$</td>
<td>15 800</td>
</tr>
<tr>
<td>U-238</td>
<td>4.18</td>
<td>$5.8 \times 10^5$</td>
<td>17 300</td>
</tr>
<tr>
<td>Pu-238</td>
<td>5.48</td>
<td>$7.6 \times 10^5$</td>
<td>13 200</td>
</tr>
<tr>
<td>Pu-239</td>
<td>5.14</td>
<td>$7.1 \times 10^5$</td>
<td>14 100</td>
</tr>
<tr>
<td>Pu-240</td>
<td>5.12</td>
<td>$7.1 \times 10^5$</td>
<td>14 000</td>
</tr>
<tr>
<td>Am-241</td>
<td>5.45</td>
<td>$7.5 \times 10^5$</td>
<td>13 300</td>
</tr>
</tbody>
</table>

* It is appropriate, for principally $\alpha$-emitting radionuclides to consider internal doses only so these values are not here intended to apply to environmental media (soil, sediment, water). If necessary, possible external exposures from these radionuclides could be assessed by calculating $C_{\text{min}}$ values for soil/sediment/water using only the much lower energy $\beta$ and $\gamma$ emissions of these radionuclides. These concentrations would be much higher than the ones shown above. Note also that the values given here do not include decay of daughter radionuclides. + From data in Delacroix et al. (1998) and Chu et al. (1999).

Table 2. Minimum concentration, $C_{\text{min}}$ (wet weight) of $\beta$ and $\gamma$-emitting radionuclides in an organism, tissue or environmental medium which could result in a dose rate of 1 mGy d$^{-1}$.

<table>
<thead>
<tr>
<th>Radionuclide</th>
<th>Mean $\Sigma \varepsilon$ per disintegration$^+$, MeV</th>
<th>Dose rate, mGy d$^{-1}$ per Bq kg$^{-1}$</th>
<th>Minimum conc., Bq kg$^{-1}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>H-3</td>
<td>0.0059</td>
<td>$8.17 \times 10^8$</td>
<td>12 300 000</td>
</tr>
<tr>
<td>C-14</td>
<td>0.051</td>
<td>$7.05 \times 10^7$</td>
<td>1 420 000</td>
</tr>
<tr>
<td>P-32</td>
<td>0.696</td>
<td>$9.6 \times 10^6$</td>
<td>104 000</td>
</tr>
<tr>
<td>Co-60</td>
<td>2.60</td>
<td>$3.59 \times 10^5$</td>
<td>27 900</td>
</tr>
<tr>
<td>Sr-90/Y-90</td>
<td>1.13</td>
<td>$1.56 \times 10^5$</td>
<td>64 000</td>
</tr>
<tr>
<td>Tc-99</td>
<td>0.085</td>
<td>$1.2 \times 10^6$</td>
<td>854 000</td>
</tr>
<tr>
<td>I-131</td>
<td>0.55</td>
<td>$7.6 \times 10^6$</td>
<td>131 500</td>
</tr>
<tr>
<td>Cs-134</td>
<td>1.7</td>
<td>$2.35 \times 10^5$</td>
<td>42 600</td>
</tr>
<tr>
<td>Cs-137/Ba-137</td>
<td>0.81</td>
<td>$1.12 \times 10^5$</td>
<td>90 000</td>
</tr>
</tbody>
</table>

+ From data in Delacroix et al. (1998) and Chu et al. (1999). These values include decay of daughter radionuclides where indicated.
Table 3. Illustrative concentrations, $C_{EQS}$, of $\beta$ and $\gamma$-emitting radionuclides in freshwater which could result in a concentration $C_{min}$ in freshwater fish. Concentration Factors are taken from IAEA (1994) except where specified.

<table>
<thead>
<tr>
<th>Radionuclide</th>
<th>Concentration Factor (w.w.)</th>
<th>Minimum concentration, $C_{min}$, Bq kg$^{-1}$</th>
<th>Concentration in freshwater, $C_{EQS}$, Bq kg$^{-1}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>H-3</td>
<td>1*</td>
<td>12 300 000</td>
<td>12 300 000</td>
</tr>
<tr>
<td>C-14</td>
<td>$5 \times 10^4$</td>
<td>1 420 000</td>
<td>28.4</td>
</tr>
<tr>
<td>P-32</td>
<td>$5 \times 10^4$</td>
<td>104 000</td>
<td>2.1</td>
</tr>
<tr>
<td>Co-60</td>
<td>300</td>
<td>27 900</td>
<td>93</td>
</tr>
<tr>
<td>Sr-90/Y-90</td>
<td>7500*</td>
<td>64 000</td>
<td>8.5</td>
</tr>
<tr>
<td>Te-99</td>
<td>20</td>
<td>854 000</td>
<td>42 700</td>
</tr>
<tr>
<td>I-131</td>
<td>40</td>
<td>131 500</td>
<td>3290</td>
</tr>
<tr>
<td>Cs-134</td>
<td>20 000**</td>
<td>42 600</td>
<td>2.1**</td>
</tr>
<tr>
<td>Cs-137/Ba-137</td>
<td>20 000**</td>
<td>90 000</td>
<td>4.5**</td>
</tr>
</tbody>
</table>

* Tritium as HTO. + Estimated concentration factor in bone in water with very low calcium concentration (ca. 2-3 mg l$^{-1}$; Blaylock, 1982). ** Value for water of very low potassium concentration (0.5 – 1.0 mg l$^{-1}$, Smith et al., 2002).

Table 4. Maximum activity concentrations of various radionuclides in lobsters near Sellafield during the period 1991-2002 (data taken from graphs in RIFE, 2003). The application of the simple screening model (Equations 2 and 3) is illustrated.

<table>
<thead>
<tr>
<th>Radionuclide</th>
<th>Activity concentration, Bq kg$^{-1}$ wet weight</th>
<th>Ratio $C/C_{min}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-14</td>
<td>275</td>
<td>$1.9 \times 10^{-4}$</td>
</tr>
<tr>
<td>Co-60</td>
<td>4.5</td>
<td>$1.6 \times 10^{-3}$</td>
</tr>
<tr>
<td>Te-99</td>
<td>16 000</td>
<td>0.019</td>
</tr>
<tr>
<td>Cs-137</td>
<td>10</td>
<td>$1.1 \times 10^{-4}$</td>
</tr>
<tr>
<td>Pu-239+240</td>
<td>0.57</td>
<td>$2.0 \times 10^{-5}$</td>
</tr>
<tr>
<td>Am-241</td>
<td>6.5</td>
<td>$4.9 \times 10^{-4}$</td>
</tr>
<tr>
<td>$\Sigma C/C_{min}$</td>
<td></td>
<td><strong>0.021</strong></td>
</tr>
</tbody>
</table>

Table 5. Maximum activity concentrations of $^3$H and $^{14}$C in molluscs and $^{137}$Cs in sediments in the Severn Estuary near Cardiff, Wales, as a result of discharges from Amersham plc during the period 1997-2002 (data taken from graphs in RIFE, 2003). The application of the simple screening model (Equations 2 and 3) is illustrated.

<table>
<thead>
<tr>
<th>Radionuclide</th>
<th>Biota/medium</th>
<th>Activity concentration, Bq kg$^{-1}$ wet weight</th>
<th>Ratio $C/C_{min}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>H-3</td>
<td>Mollusc</td>
<td>50 000</td>
<td>$4.1 \times 10^{-3}$</td>
</tr>
<tr>
<td>C-14</td>
<td>Mollusc</td>
<td>800</td>
<td>$5.6 \times 10^{-4}$</td>
</tr>
<tr>
<td>Cs-137</td>
<td>Sediment</td>
<td>30</td>
<td>$3.3 \times 10^{-4}$</td>
</tr>
<tr>
<td>$\Sigma C/C_{min}$</td>
<td></td>
<td><strong>0.005</strong></td>
<td></td>
</tr>
</tbody>
</table>