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## Ecological Risk Assessment for Radionuclides: Current Status and Critical Knowledge Gaps

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**ABSTRACT:** The debate on the need for a system of radiological protection of the environment drives regulators to urge scientists on conceptualisation of methods demonstrating explicitly that the environment is protected against radioactive contaminants. As regards the FASSET database on radiation effects to non-human biota, one of the major difficulties in the implementation of ecological risk assessment for radioactive pollutants is the lack of data for chronic low level exposure. A general way to deal with situations for which there are no relevant data as regards the actual situation where the risk is to be estimated is to use safety factors. The highest are their values, the highest is the uncertainty on the risk estimate, but, on the other hand, any safety factor could be reduced as more data become available. Going back to radionuclides for which data are sparse, both concerning fate and effects in ecosystems, derivation of ecologically relevant and scientifically defensible benchmarks become a critical issue in ERA. The scope of this paper is to illustrate the relevance of the development of a greater depth of understanding of radionuclide fate and biological effects at several hierarchical levels to support quantitative risk assessments with defined and acceptable uncertainty bounds.

### 1 CONTEXT AND GLOBAL SCOPE

At the present time, international debate on the need for a system of radiological protection of the environment [1,2] drives regulators to urge scientists on a rapid conceptualisation and implementation of methods demonstrating explicitly that the environment is protected against radioactive contaminants. This debate is obviously brought together with the preservation of resources, habitats and genetic and biological diversity; these major issues do not point out any particular stressors but pollution in general. The recent FASSET project funded by the European Commission, has provided a framework for the assessment of environmental impact of this category of pollutants, with, among others, an extensive database on radiation effects to non-human biota [3]. For chemicals, Ecological Risk Assessment approach constitutes the traditional methodology to help the demonstration of the providence of an appropriate level of protection for ecosystems [4]. ERA is a process that evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure

to one or more stressors. According to this methodology, in complementarity with health risk examination, any risk assessment to biota from exposure to radionuclides is to be associated with (1) different source-terms and environmental released scenario, (2) exposure pathways and potential biological effects at different organization level, (3) estimation of no-effects values and finally, (4) risk calculations as the ratio between predicted concentrations in the source of exposure and estimated no-effects concentration. One difficulty in the implementation of ecological risk assessment for radioactive pollutants is the lack of data for chronic low-level exposure. Indeed, most studies on the effects of radionuclides on non-human organisms have focused on acute exposure to high-doses of radionuclides, mainly by external  $\gamma$  irradiation. A general way to deal with situations for which there are no data (or no relevant data as regards the actual situation where the risk is to be estimated) and then to propose a method to manage with the unknown is to propose the use of assessment/safety factors. The use of these factors is proposed by the European Commission in its Technical Guidance Document [5] to accommodate uncertainty in risk assessment data for existing and new chemicals and to reduce the probability of causing deleterious effects on the ecosystem. An expected ecologically meaningful safe/acceptable levels (called Predicted no-effect value) is derived from ecotoxicological data from the so-called “base-set” organisms (e.g. for freshwaters, primary producers –*algae*; primary consumers –*daphnids*; predators –*fishes*). The weakness of the method relies on the need to make allowance for the various extrapolation issues from laboratory base-set to actual ecosystems (i.e. acute to chronic exposure conditions, single species to multiple species systems; laboratory to field). This extrapolating method consists in applying to the lowest observed effect values  $L(E)C_{x\%}$  from the base-set, a largely arbitrary conservative and protective factor ranging from 1 to 1000 according to the quality and relevance of existing effects data. Obviously, this pragmatic method corresponds to a precautionary approach to environmental management mainly applied during the screening stage. The highest are the safety factors, the highest is the uncertainty on the risk estimate, and flexibility of the method recommended for their determination is crucial as any safety factor could be reduced as more data become available. Going back to radioactive substances for which data are sparse, both concerning fate and effects of radionuclides in ecosystems, derivation of ecologically relevant and scientifically defensible benchmarks become a critical issue in the implementation of any ecological risk assessment methodology. Within this global framework, the scope of this extended abstract is to propose and illustrate the relevance of a research plan (1) to scientifically support the previously cited extrapolation issues, (2) to develop a greater depth of understanding of radionuclide biological effects at several hierarchical levels, (3) to fit with proper interpretation of biomonitoring data, and finally (4) to support quantitative risk assessments with defined and acceptable uncertainty bounds. In other words, at the present time, “we know what we do not know” and we are aware to which extent the knowledge gaps will be critical to go towards a robust methodology for ERA associated with radionuclides.

## **2 GAPS ON WILDLIFE CHRONIC INTERNAL EXPOSURE TO $\alpha$ OR $\beta$ EMITTERS ARE AMONG THE MOST CRITICAL FOR ERA**

### **2.1 Background**

According to the Fasset Radiation Effects Database examination [6], gaps are particularly crucial for chronic exposure of some taxonomic groups whatever the irradiation pathways and for internal contamination by  $\alpha$  and/or  $\beta$  emitters whatever the wildlife group. Concerning this latter, situations of chronic exposure at low levels are likely to cause toxic responses distinct from those observed after acute exposure at high doses because of the

bioaccumulation phenomena. Biochemical mechanisms can lead to a gradual accumulation of elements present at trace level in the external medium, inducing a highly localised deposit within tissues or cells. These highly localised accumulations of radio nuclides, coupling radiological and chemical toxicities, particularly for “heavy” elements such as actinids, may give rise to particular biological responses of a cell group, capable of causing functional or structural abnormalities. The assessment of these bioaccumulation phenomena investigated within the ENVIRHOM programme [7], is primordial with regard to internal exposure to radio nuclides since they increase locally both the radionuclide concentration and the biological effect of the delivered dose. Gaps of knowledge within this field constitute a strong limitation to our capability to make a reasonable risk estimate. Internal doses cannot be accurately calculated and potentially associated biological effects at any organisation level remain fairly unknown.

Throughout the following sections, only examples related to uranium on the element behaviour (whatever the considered isotope) are given. Experiments devoted to the establishment of dose (in terms of delivered energy)-effects relationship are in progress with several U isotopes such as U-233 to enhance the delivered dose and to contribute to the understanding on interaction between chemo toxicity and radio toxicity. In any case, issues discussed hereafter are generic to any radionuclide.

### *2.1.1 Radionuclide bioavailability is a key knowledge to obtain an accurate assessment of both exposure and effect: media quality criteria are needed*

The biogeochemical behavior of a radionuclide depends on the chemical properties of the corresponding element (redox states, ability to form complexes with inorganic and/or organic ligands...). It is now well established that the knowledge of the distribution of the element (radioactive or not) amongst its various physico-chemical forms (speciation), is needed to understand both the mobility (transport) and the biological reactivity (transfer and effect). It is commonly admitted (but a number of exception exists!) that the bioavailability and toxicity of dissolved metals are closely linked to the metals' chemical speciation in solution. Metal uptake, nutrition and toxicity normally vary as a function of the concentration of the free-metal ion in solution (Free-Ion Model; FIM), thus complexation of a metal normally leads to a decrease in its bioavailability. The prevailing paradigm for metal uptake by aquatic organisms, *i.e.* the Free Ion Model or its derivative the Biotic Ligand Model, assumes that metals enter living cells via facilitated cation transport. Antagonism with other cations is implicit. The following example of uranium and freshwater phytoplankton illustrates the relevancy of considering geochemical behavior (or media quality criteria) of the radioelement to better predict bioavailability for organisms. Whatever the considered radioactive isotopes,  $H^+$  /  $UO_2^{2+}$  antagonism explains large discrepancies observed under different pH exposure conditions and the FIM has been successfully applied to describe uranium – algae interactions in simple well-defined exposure media. Cellular uptake as a function of U concentration follows typical Michaelis-Menten saturation processes with a somewhat similar half-saturation but clearly contrasting maximum uptake rates. Uranium uptake increased markedly (up to ~4X) with pH (5  $\ddagger$  7) in spite of the substantial decrease (55  $\ddagger$  0.02 %) in the proportion of calculated free uranyl ion concentration in solution within this pH range [8]. Globally, these marked differences in uptake rates will lead on consequences in terms of delivered dose and obviously observed effects, pointing out the need for media quality criteria. Bioaccumulation studies carried out on a bivalve model (*Corbicula fluminea*) also revealed a significant pH effect on the bioaccumulation rate (x14 when pH 8.1  $\ddagger$  7) [9].

*2.1.2 Chronicity is a key exposure situation to an accurate assessment of both dose calculation and effect: exposure conditions (concentration and duration) strongly modify the radionuclide internal distribution at various biological scales*

For a given living organism, the chronicity of any exposure to a pollutant obviously leads to different bio kinetics but also to different toxicity mechanism than when the exposure is acute. For example, a set of experiments was performed with an invertebrate model (the bivalve *Corbicula fluminea*), to compare the bioaccumulation rates and tissue distributions after a short-duration exposure combined with a high contamination level, with a semi-chronic exposure conditions to a low concentration. A marked difference of U distribution in organs was observed as a function of exposure levels and duration. Gills were favoured in the case of high exposure levels (this organ contributes to 40% of the total internalised quantity when bivalves are exposed to 6.3  $\mu\text{M}$  during 7 days), whereas the visceral mass presented higher accumulation levels for environmentally-relevant representative U concentrations level (85% of the total internalised quantity when bivalves are exposed to 0.4  $\mu\text{M}$  during 42 days). These results suggested that the main primary toxicity of uranium should potentially take place in the gills for acute exposure and in the digestive gland for chronic exposure. Obviously acute to chronic extrapolation involved large uncertainties in terms of exposure assessment and delivered dose to the target organs. Concerning chemicals, acute-to-chronic ratios used in ERA vary over a wide range from 1 to 20000 depending on the species and the chemical; many are less than 50 [4,5].

*2.1.3 Considering different scales for biological effects (from early to delayed and from sub cellular to high level of organization) is crucial to evidence ecologically relevant indicators*

When pollutant exposure level increases (dose and duration), organisms counteract this stress with a wide range of physiological responses in the dose-effect continuum, from exposure to resultant disease. Effects at higher hierarchical level are always preceded by early changes in biological process, from subtle biochemical disturbances to impaired physiological functions (behaviour, growth, reproduction), increased susceptibility to any other added stresses, reduced life span. For example, laboratory experiments were carried out to analyze the first valve closure response of *Corbicula fluminea*, exposed to uranium during a 5-hours period and equipped with a non invasive method of valve recording. Minimal sensitivity threshold determined, expressed as uranium concentration in water inducing the valve closure of fifty percent of the bivalve, was 0.05  $\mu\text{mol/L}$  of total uranium at pH 5.5 after 300 minutes of exposure [9]. For the same organism, the exposure to uranium at 0.25  $\mu\text{mol/L}$  resulted in a significant decrease in ventilation rate (divided by 3) in comparison with the reference condition. For unicellular algae, growth inhibition experiments (pH=5,  $\text{EC}_{50}$ =340 nM) showed that toxicity appeared when a critical point of  $10^{-6}$  nmole/cell of internalised U was reached, and then was proportional to internalised uranium by the cells. Most of these data could be relevant as regards the population dynamics and as a first approach to bridge the gap between the (sub)individual level and the ecosystem level, simplified life-history models may be used to make ecological relevant links between test results on various effects endpoints and their implications for population dynamics (density, survival, recruitment, time for reproduction, capacity to produce offsprings).

### 3 CONCLUSION AND PERSPECTIVES

We are aware of missing knowledge and scientists can fill data gaps through further testing and investigations consistent in quality and quantity with the magnitude of the current uncertainties brought by these gaps. The previously cited unresolved issues in terms of risk assessment are to be investigated to propose in a rational and transparent process, scientifically defensible environmental criteria for radionuclides based on ecological relevant endpoints. From the point of view of the present discussion and illustration, derivation of the no-effect dose or dose rate is of main interest and is to be linked to the trend to integrate the behaviour of pollutants (bioavailability, bioaccumulation, biotransformation) to develop knowledge. Apart from introducing media quality criteria, another challenge would be to link the observed effects at infra or/and individual scale with the population dynamics in the ecosystem using ecological modelling approach. The set of experimental data needed will also contribute to answer the question of how and to which extent radionuclides and other stressors may affect different organisms and therefore change community structure, distinguishing direct (toxicity) or indirect (food-chain) effects.

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