Towards an ecological modelling approach for assessing ionising radiation impact on wildlife populations

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Abstract

The emphasis of the international system of radiological protection of the environment is to protect populations of flora and fauna. Throughout the MODARIA programmes, the United Nations' International Atomic Energy Agency (IAEA) has facilitated knowledge sharing, data gathering and model development on the effects of radiation on wildlife. We present a summary of the achievements of MODARIA I and II on wildlife dose effects modelling, extending to a new sensitivity analysis and model development to incorporate other stressors.

We reviewed evidence on historical doses and transgenerational effects in wildlife from radioactively contaminated areas. We also evaluated chemical population modelling approaches, discussing similarities and differences between chemical and radiological impact assessment in wildlife. We developed population modelling methodologies by sourcing life history and radiosensitivity data and evaluating the available models, leading to the formulation of an ecosystem-based mathematical approach. This resulted in an ecologically relevant conceptual population model which we used to produce advice on the evaluation of risk criteria used in radiological protection of the environment, and a proposed modelling extension for chemicals.

This work seeks to inform a stakeholder dialogue on factors influencing wildlife population responses to radiation, including discussions on the ecological relevance of current environmental protection criteria. The area of assessment of radiation effects in wildlife is still developing with underlying data and models continuing to be improved. IAEA's ongoing support to facilitate the sharing of new knowledge, models and approaches to Member States is highlighted, and we give suggestions for future developments in this regard.

Keywords: Ecological approach; Non-human biota; Population modelling; Radiation effects

1. Introduction

The International Commission on Radiological Protection's (ICRP) Publication 103 [1] recommends the explicit radiological protection of the environment from ionising radiation, with the target of protection being populations or ecosystems rather than individual organisms. The objectives of the ICRP's environmental radiation protection recommendations are species conservation, maintaining biodiversity and protecting natural habitats, communities and ecosystems.

To enable environmental radiation protection to be demonstrated, the ICRP introduced an assessment system based on the use of Reference Animals and Plants (RAPs) for relating exposure to dose and its effects. As it is unfeasible to consider all species of flora and fauna in assessments, RAPs are defined at the taxonomic level of Family and used to assess environmental impact under different exposure situations [2, 3]. The resultant dose rate estimates for the RAPs are compared with Derived Consideration Reference Levels (DCRLs), which are RAP-specific dose rate bands used to evaluate the potential for radiation impact. Within the dose rate range of each DCRL, some individuals may experience deleterious effects of ionising radiation but this will not necessarily lead to population level impacts. When considered together with other relevant information, the DCRLs can be used as a point of reference to optimise the level of protection [2].

The IAEA draws on the ICRP's Recommendations when developing practical regulatory guidance and IAEA's Safety Fundamentals state that people and the environment, both present and future, must be protected against radiation risks [4]. The IAEA has also included the ICRP's RAP approach in its 2018 GSG-10 Safety Guide [5]. The IAEA recommends to use lower bounds of the DCRL bands as criteria to use in excluding dose rates from further consideration but leaving the regulator free to decide what doses rates from within the band are acceptable. If the dose rate exceeds the upper bound of the band, GSG-10 recommends that the regulatory body should decide whether more control of the source or further protection are warranted. In 2004, the IAEA established a 'Biota Working Group' as part of its EMRAS (Environmental Modelling for Radiation Safety) programme [6, 7]. As part of this, activities on radiation effects to wildlife populations, and subsequently population modelling, were initiated [8]. This culminated in the MODARIA (Modelling and Data for Radiological Impact Assessments) I and II programmes, with working groups focusing on the development and application of population effects modelling for wildlife.

In MODARIA I, a "Working Group 9" was formed with a focus on modelling methods to link effects reported for laboratory or field organisms to consequences on populations. This goal stemmed from the fact that radiation effects on wildlife are poorly documented at the population level and for species in the field. The work covered population modelling methodologies and data to analyse the effects of ionising radiation in wildlife. The subsequent MODARIA II "Working Group 5" subgroup on effects in wildlife aimed at making the transition from the hitherto uncovered scientific knowledge to practical examples of population models, with emphasis on ascertaining whether population modelling can provide arguments to support the robustness of benchmarks for radiation protection of wildlife.

We report the modelling work and lessons learned from these IAEA working group activities, focussing on the development of an ecological modelling approach to assess the impact of ionising radiation on populations and incorporating aspects of the approaches utilised in chemicals risk assessment, paving the way for the possible study of mixed exposure situations. The overarching aim was to apply population models that accounted for spatial and ecological issues, identifying what new data are needed to test and validate models and increase the robustness in the models used for routine radiological impact assessments. Overall, the available science was examined in context of the regulatory commitments of the IAEA, taking advantage of the exchange of expertise that occurred during the MODARIA programmes.

Our main question was whether dose rate criteria established for the protection of wildlife based on effects in individuals (such as the ICRP DCRLs) are applicable to extrapolate conclusions on protection at the level of a population. This was addressed using a specific case study (a population of voles from the Chernobyl Red Forest [9]) and reviews of the available effects data for wildlife populations. Model outputs were compared with thresholds for effects at the level of individuals in experimentally controlled exposures (such as those provided within the FREDERICA database - www.frederica-online.org) to test whether existing benchmarks for wildlife population exposure are appropriate for the scenario considered, as well as to gain insight on whether the assessment approaches that are being routinely applied could include evaluations of how populations may grow and be affected by the presence of a contaminant. In addition to deriving conclusions from the aforesaid case study [9, 10], we give further sensitivity analysis of the model developed in respect of the key reproduction rate and migration parameters, and an extension of the method to cover the effects of radionuclides in combination with chemicals is proposed. We also present suggestions for further work related to IAEA's guidance on radiological environmental impact assessment, based on lessons learned from the MODARIA programme.

2. Review studies

The "biota effects modelling" working groups spanning the MODARIA I and II programmes reviewed the available literature and prior research by the participants. This covered (a) information on adaptive response, non-targeted effects and historical effect data for wildlife, (b) evaluation of population models developed within chemical assessment, leading to selected practical approaches to radiation dose effect modelling in wildlife populations, (c) identification of sources of information for population modelling such as life history and ecology data as well as radiation effects datasets, and (d) comparison of population modelling for chemical pollutants and radiation.

2.1. Review of radiation effects data for wildlife

The objective of this review was to identify parameters relevant for population modelling. This was challenging on two accounts: (i) difficulties in understanding transgenerational effects in populations of wildlife, given that population effects can manifest themselves as fewer/more or poorer/better quality offspring, and these phenomena are not easy to evaluate with the limited studies available; and (ii) difficulty in interpreting some of the mechanisms for effects caused by low dose rates of ionising radiation, of which the following have been suggested as potentially important: hormetic effects [11], adaptive responses [12], genomic instability [13] and hypersensitivity [14]. It is not currently possible to prove conclusively that there is a positive response from low levels of radiation in exposed species, or what transgenerational impacts may be induced by such low-level exposures on populations. It is also complex to discern what makes an effects study a truly population-relevant study different from short term radiation studies made for a sample of a population consisting of relatively few individuals.

The full details of our review of data on effects are reported elsewhere [10]. A significant review of the non-targeted effects of ionising radiation was identified [15], along with an additional review including wildlife considerations [16]. This information showed that hormetic effects and adaptive responses are rarely considered in species sensitivity distributions (SSD), probably because there is still not enough information on these types of responses in wildlife; however, the applicability of hormetic dose rate–effects relationships has been discussed [17]. There is significant research on genomic instability, bystander effects and adaptive responses, but the processes controlling these phenomena and their link to ionising radiation in the environment are not clearly understood.

Regulatory approaches (such as those based on ICRP recommendations) are based on benchmarks deriving from radiation effect studies which implicitly include some of these processes, with the notable exception of the study of transgenerational consequences. This is due to the difficulty of considering multiple generations on which the population level impacts can be assessed through the full assessment of reproductive impacts. However, there is a valid question of why transgenerational effects are not considered yet as there could be effects reported in the literature for some highly contaminated sites which are probably caused by historic exposures. An aim of the population modelling work described here was to explore these factors, beginning to test whether the benchmarks are protective when the few data available on transgenerational studies are applied, as an early test of robustness for the regulatory approaches being applied, facilitating further thinking/testing of benchmarks.

We also examined available data on radiation-induced inheritable (memory) effects to distant progenies of wildlife in chronic or acute low dose exposures. The main objective was to identify scientific reports linking current radiation effects to historic dose rates, leading to potential issues in the interpretation of the currently applied benchmarks for radiation protection. Reported findings range from claims of beneficial hormetic effects and adaptive responses to claims of low dose hypersensitivity, bystander effects and genomic instability increasing the relative effects of low doses [18, 19]. Specific studies are detailed in the European Radiobiological Archives (https://era.bfs.de), and in an article on the quantification of thresholds for lifetime health effects in wildlife [20].

Historic dose reconstruction exercises were identified, including one in which the yield of mutations in bird populations from the Chernobyl Exclusion Zone (CEZ) were compared against a new generation of mutation rates from cellular lines using lethal mutation assay [21]. That study suggested that there is a non-targeted type of effect (an effect that is not the direct consequence of radiation interacting with the DNA of a given cell but may be imported from neighbouring irradiated cells). This is because there are no signs of increase in effect with increasing dose, but rather a saturated response was seen. Another study on the Fukushima Dai-ichi Nuclear Power Plant accident suggests that historic acute exposure and its resulting non-targeted effects (NTEs) may be partially involved in the high mortality/abnormality rates seen across generations of pale grass blue butterflies around Fukushima, but continual accumulation of mutations over generations in their natural contaminated habitats remains a likely contributor to the observed outcome [22]. It is still being debated whether effects of radiation in wildlife populations reported in areas such as the CEZ are due to past acute exposure and subsequent transgenerational transfer of genetic damage, or due to current dose rates.

Adaptation can now begin to be explored in a modelling environment with the use of the existing transgenerational effects data, because a mathematical modelling approach is already available. A study [23] presents a set of dynamic equations considering (i) a memory mechanism in which successful repair leads to radio-adaptation and (ii) a double communication mechanism whereby sick organisms can induce healthy organisms to adapt or, additionally, adapted organisms can themselves induce protection into healthy organisms. The proposed dynamic equations were used in our ecological model (see Section 2.5).

The overall conclusion from the review of historical dose reconstruction exercises on effects is that the outcomes of acute radiation exposures are still controversial, but more quantitative information is becoming available. It appears possible that acute dose exposures from past accidents left a significant mutational load in animals that survived, leading to effects at the significantly lower chronic dose rates prevailing now, but this is unlikely to be the case in situations of chronic routine low level discharges. It remains important to continue reviewing and synthesising new information as it becomes available.

2.2. Review of population modelling methodologies

We identified modelling studies that apply population models of representative wildlife species to investigate population responses to ionising radiation. Overall, the source of information most directly relevant for the work to be performed in MODARIA programmes was the deliverable report D5.2 of the EC programme STAR[1] [24], covering (a) physiology-based matrix population models for aquatic invertebrates exposed to chronic gamma radiation [25], using matrix models known as Leslie matrices [26], and (b) ordinary differential equation (ODE models) for fish and mammals that capture population damage and recovery by means of recovery and fecundity pools, in a system of dynamic equations [27-31]. The ODE approach was tested in an inter-comparison of population models performed at the end of EMRAS II's WG6 activities [32].

We concluded that both matrix and ODE approaches are applicable for determining the sensitivity of different end points to chronic and acute doses of radiation in wildlife populations. Matrix models are more common in the literature and have been used to compare radiosensitivity between individual and population endpoints [33], whilst ODE models have been used to reproduce the range of radiation effects in fish and mice as reported in the FREDERICA database [31].

Methodological guidance for population modelling necessitates the definition of a "population" for modelling purposes in the context of radiological protection. This was defined as a self-sustaining group of individuals of the same species with common ecological characteristics (as defined by the ecological parameters of the population) and living within a defined territory. Hence, for our purposes, they are exposed together to a level of radiation stress that can be modelled through an ecological approach to simulate the collective response of the group. A population in a density dependent context is protected at a certain dose rate when, upon continuous application of that dose rate in the presence of all relevant natural stressors, the total size of the population tends to a stable level. The modelling problem consists of deducing healthy and radiation-impaired population abundances at various levels of dose rate.

The next methodological step was the formulation of a mathematical solution or conceptual model for specific animals and plants using effects data from both acute and chronic radiation exposures, focusing on how radiation dose rate impacts the fecundity and repair mechanisms leading to impacts on population abundance. This has been be achieved in the past by (i) first-order logistic ODE approaches [34, 35] and (ii) the Leslie matrix approach [36]. In the matrix approach, the population is represented as an age-structured vector with all age classes instantaneously advancing at discrete, equidistant time intervals. In the ODE population model, time and population are continuous variables and the age classes are mapped to compartments governed by differential equations. ODE model rate constants represent the survivorships of the different age classes and are equivalent to the inter-stage probabilities of the matrix model.

The Leslie matrix and ODE approaches are, in principle, equivalent; however, ODE models are more amenable to the amalgamation of radiation effects modelling with ecological processes which are traditionally expressed as differential equations, leading to a compact mathematical equation system covering both acute and chronic exposure situations, operating at the community level and integrating eco-physiological factors such as food limitation, density-dependence, self-limiting growth and animal mobility. Consequently, the ODE approach was adopted as a basis for our model development, though we do not necessarily recommend one approach above the other.

[1] Strategy for Allied Radioecology, the EC-funded Network of Excellence in Radioecology - https://www.ceh.ac.uk/our-science/projects/strategy-allied-radioecology-star

The MODARIA I WG9 adopted the ODE "radiation damages recovery and repair" approach [28] for further development, in which the population consists initially of healthy individuals, some of which can become sick due to the effect of radiation. A radiation-sensitive 'repair pool' regulates the proportion of sick individuals that recover or die. The model also includes a radiation-sensitive 'fecundity pool'. Both repair and fecundity pools can auto-recover, and the model represents this by means of logistic functions. The mathematical treatment, including how the previous equations can be generalised to various species, exposure situations and age groups, is fully described elsewhere [24] and a dual-age class version of the model developed during IAEA's EMRAS programme [31] was used to prove that (i) population survival under the effect of radiation is higher for short-lived, fast reproducing species than for long-lived, slow reproducing animals, and (ii) dose rates that cause extinction are in the order of magnitude of 10^{-2} Gy d^{-1} and above. The MODARIA programmes allowed us to increase the ecological realism of ODE population models, including accounting for the effect of natural immigration and the influence of a limiting resource in an ecosystem on population response to ionising radiation [37].

2.3. Review of population model parameter data

We reviewed relevant sources of population model parameters which can enhance the ecological realism of the models and their applicability to chronic dose rate exposure situations. For ecological parameters, the focus was on life history information for species representative of the ICRP RAPs taxonomic groups [2] (including different life stages) for which there are well-studied radiation effects. The AnAge curated database of ageing and life history in animals (https://genomics.senescence.info/species/) provided an accessible and extensive collection of information, including average lifespan, average mortality rate, reproduction rate, survivorship of young and adult organisms, carrying capacity and, for vegetation, biomass (and loss rate) and germination rate. These data are suitable for the parameterisation of standard ecological processes in many types of models, including those developed in the MODARIA studies.

The primary sources of radiation effects parameters for wildlife are the FREDERICA database [38-40] and a significant study on species radiosensitivity [41]. In these sources, data are given for three relevant endpoints: mortality, morbidity and reproduction. Therefore, we identified the need to use these key endpoints in population modelling aiming at assessing protection levels for populations. These sources also allowed the identification of the relevant species (and endpoints) that have been studied in field experiments. From the foregoing, it was concluded that there are sufficient life history and radiosensitivity data to parameterise population models for most of the ICRP RAPs, but mainly for single age class models, with multiple age class model data only available in a limited number of cases.

2.4. Review of population modelling of chemical pollutants and analogies with radiation

The purpose of this review was to examine population models applied to the chemical pollutants domain, establishing a point of comparison with how far population modelling has been developed to assess similar issues to those in radioecology. Typically, to estimate the potential effects of chemical contaminants on wildlife, assessors extrapolate chemical toxicity from laboratory tests to natural field conditions with the application of safety factors. We identified a review of chemical toxicity population models for chemical risk assessment [42]. Some 90 models are described, of which 27 deal with ecological processes, consisting mainly of age-structured population models. The endpoints of models are similar to those of the population models used in MODARIA I and II for radiation, namely, growth, mortality within population, repairable damage and fecundity.

A particularly useful approach for ecological risk assessment of mixtures of radiological and chemical stressors has been proposed [43], which involves the use of species sensitivity distributions for ionising

radiation and chemicals such as Cr, Cu, Ni, Pb, Zn, B, chlorides and sulphates. This is based on applying species sensitivity distributions in combination with both concentration addition and independent action mixture models, in order to calculate an msPAF (multi-substance potentially affected fraction of species) as a way to represent the combined ecological impact of radiation and chemical substances. This is an extension of the species sensitivity distribution approach for radiation used in the ERICA project [44] which derived a screening value of 10 $\mu Gy\ h^{-1}$ from species sensitivity distributions [45-47], defined at the population level but ultimately protective of the function and structure of the ecosystem.

A subgroup of models identified in our review are dynamic energy budget (DEB) models, which follow the energy budget of an individual organism's state (structure, reproductive effort and amount of reserves). These models can be integrated with bioenergetics modelling (BEMs), considering energy expenditure, losses, gains and efficiencies of transformations in the body. A relevant example of a BEM-type model applied to radioecology is AQUATRIT [48, 49], developed for tritium transfer in aquatic food chains and considering both organically bound and dissolved organic tritium. Application of DEB modelling to ecotoxicological problems leads to DEBTox models, used to mathematically link toxicant concentrations to the effects on life-history traits (survival, growth and reproduction) over time in what is essentially a toxicokinetic-toxicodynamic modelling approach. Such models already have a history in the chemical field and are being adapted to model radiological exposure of wildlife [50, 51], providing a mechanism for investigating links between exposure level, molecular responses and effects in organisms. A curated database of DEBTox model parameters with some 1000 species available (containing data for most animal ICRP RAPs but including many more species besides) provides a valuable resource for parameterising these models (https://www.bio.vu.nl/thb/deb/deblab/add_my_pet/index.html).

Another subset of models used in ecotoxicology are Individual-Based Models (IBMs), where individuals are modelled and population effects are obtained as emergent properties [52, 53]. IBMs can capture the dynamics of the populations in a realistic way when the environmental parameters are fluctuating. However, this type of model is complex and computationally demanding in comparison with the ODE and matrix models developed in our study, where the key processes are captured in a few equations that can be solved by an iterative integration algorithm and, in some cases, an analytical solution can be found.

Ultimately, the fields of ecotoxicology and radioecology share a trait in common in that population models are not routinely used for regulatory assessments because of their perceived complexity and inherent uncertainties. This fits into a broader general mismatch between the speed at which scientific insights and policy align. The bridging of the individual-population gap with models is one of the areas where this mismatch is very pronounced. However, as in the case of radiation, population models need to be applied to test that currently used regulatory approaches are fit for purpose, by verifying whether benchmarks are robust and uncertainties are well addressed, identifying potential counterexamples. In this way, population models can be a tool to demonstrate by means of a different method that the approach used in regulation is serviceable.

3. Modelling studies

We used the above information to derive and parameterise equations for modelling the radiation effects on a population of Chernobyl voles in an ecological context, and we proposed a mathematical extension of the ODE approach to model chemicals and radiation in a mixed contaminant situation.

3.1. Population model equations for the Chernobyl voles scenario

The information described in previous sections was synthesised into a model for voles inhabiting the Chernobyl Red Forest area at the time of the Chernobyl reactor accident in 1986. Full details are available in our previous publication [9] so the results are only summarised here. The model considers an inner contaminated region, an intermediate partly contaminated region and a non-contaminated outer region, labelled i = 1 to 3. Each region has a "carrying capacity" limiting its population size, following logistic equations [34, 35]. Animal mobility is determined by migration fluxes proportional to population density. The following governing equations are cited from our previous publication [9]:

$$\frac{dX_i}{dt} = r_i \frac{X_i}{L_i} \mathrm{F}_i \left(1 - \frac{L_i}{\mathrm{K}_i}\right) + \frac{M_i}{L_i} X_i + \eta_i W_i - (\mathrm{d}_i + \alpha_i \mathrm{DR}_i) X_i + \mathrm{p}_i \kappa_i Y_i \mathrm{R}_i$$

$$\frac{dY_i}{dt} = r_i \frac{Y_i}{L_i} \mathrm{F}_i \left(1 - \frac{L_i}{\mathrm{K}_i}\right) + \frac{M_i}{L_i} \mathrm{Y}_i - (\mathrm{d}_i + \varepsilon_i) Y_i + \alpha_i \mathrm{DR}_i X_i - \kappa_i Y_i \mathrm{R}_i$$

$$\frac{dW_i}{dt} = r_i \frac{W_i}{L_i} F_i \left(1 - \frac{L_i}{\mathrm{K}_i}\right) + \frac{M_i}{L_i} \mathrm{W}_i - \left(\mathrm{d}_i + \eta_i\right) W_i + (1 - \mathrm{p}_i) \kappa_i Y_i \mathrm{R}_i$$

$$\frac{dF_i}{dt} = r_i \mathrm{F}_i \left(1 - \frac{F_i}{\mathrm{K}_i}\right) - r_i \mathrm{F}_i \left(1 - \frac{L_i}{\mathrm{K}_i}\right) - \alpha_{fi} DR_i F_i + M_i^F$$

$$\frac{dR_i}{dt} = \mu_{ri} R_i \left(1 - \frac{R_i}{\mathrm{K}_i}\right) - \kappa_{ri} Y_i \mathrm{R}_i - \alpha_{ri} DR_i R_i + M_i^R$$

$$\frac{dK_i}{dt} = K_i^0 \sigma_i \left(1 - \frac{K_i}{K_i^0}\right) - \delta_i DR_i$$

Where X_i, Y_i and W_i are the healthy, sick and radiation-adapted individuals; K_i are the regions' carrying capacities; $L_i = X_i + Y_i + W_i$ is the total number of living individuals (used to compare with K_i) and F_i and R_i are dose-dependent repairing "pools" for fecundity and radiation repair, respectively. The terms M_i , M_i^F and M_i^R represent spatial displacement fluxes for population, fecundity and recovery pools, respectively. M_i is equal to $\sum_{j=1}^{3} \left(\mu_{ji} \frac{L_j}{S_j} - \mu_{ij} \frac{L_i}{S_i}\right) + \phi_0 \delta_{i3}$ for the living population L_i, with equations defined similarly for the fecundity and the recovery pools by changing L_i to F_i and R_i, respectively. The elements of the migration rate matrix μ_{ij} depend on a set surface migration rate and the surface area of each zone. The additional term $\phi_0 \delta_{i3}$ uses a Kronecker delta function δ_ij to signify immigration from Region 3 to the inner regions.

The parameters of this model are defined as follows: r_i and d_i are the reproduction and death rates (d^{-1}); $\alpha_{\square}$, α_ri and α_fi are parameters controlling the rate of damage to the population, its repairing pool and its fecundity, respectively (Gy^{-1}); κ_i and κ_ri are rate constants for radiation repair and non-lethal damages recovery, respectively (d^{-1}); ε_i is the lethality rate for damaged individuals (d^{-1}); μ_{ri} is the damaged individuals repair rate (d^{-1}) and p_i and η_i are the probability of successful repair without adaptation (unitless) and the rate constant for return of adapted organisms to full healthy state (d^{-1}), respectively. Lastly, K_i^0, σ_i and δ_i are the optimum carrying capacity (unitless) and the rate constants for vegetation recovery and damage (d^{-1}), respectively.

The derivation of the equations and associate parameter values cited here is given in detail in our previous publication [9]. The model was run with a realistic time-dependent dose profile for small mammals in the region, represented by the equation $DR(t) = 144e^{-\frac{1.1t}{365}} + 7.20 \times 10^{-3} e^{-\frac{0.05t}{365}}$ ($\mathrm{Gy\ d}^{-1}$) [54].

The key conclusions of the study [9] were that (a) migration from outer regions was the key agent for the recovery of the vole population in Region 1 after the initial exposure; (b) Region 3 has to be at least 20 km^2 to stabilise Regions 1 and 2 with a fresh vole supply; (c) a small peak of adapted voles forms at 100 days in Region 1; (d) for a constant dose rate scenario, the model predicts severe population effects at 0.01 Gy d^{-1} without migration, and 0.1 Gy d^{-1} with migration, exceeding the DCRL for small mammal (rat) [55]; (e) therefore, the rat DCRL would be sufficiently protective in this case and (f) the model is most sensitive to migration and adaptation parameters, as well as to the spatial configuration of the system. For this reason, in the present article we perform a sensitivity analysis of these parameters (see Section 4).

3.2. Derivation of new population model equations combining chemicals and radiation

In the present article, we propose a new set of candidate equations that combine radiological and toxicological processes with population-level ecological factors. We use as a basis the logistic population model for radiation effects in wildlife with dose-dependent fecundity and a recovery pool, collapsing it to a single spatial domain with constant carrying capacity and no species migration, with extension to two types of stressor. The equations for healthy, sick and adapted populations, as well as fecundity and repair, are as follows:

$$\frac{dX}{dt} = \mathrm{r}\frac{\mathrm{X}}{\mathrm{L}}F\left(1-\frac{\mathrm{L}}{\mathrm{K}}\right) + \eta W - (d + \alpha\mathrm{DR} + \beta c)X + p\kappa\mathrm{YR}$$

$$\frac{dY}{dt} = \mathrm{r}\frac{\mathrm{Y}}{\mathrm{L}}F\left(1-\frac{\mathrm{L}}{\mathrm{K}}\right) - (d+\varepsilon)\mathrm{Y} + (\alpha\mathrm{DR} + \beta c)X - \kappa\mathrm{YR}$$

$$\frac{dW}{dt} = \mathrm{r}\frac{\mathrm{W}}{\mathrm{L}}F\left(1-\frac{\mathrm{L}}{\mathrm{K}}\right) - (d+\eta)W + (1-p)\kappa\mathrm{YR}$$

$$\frac{dF}{dt} = \mu_f F\left(1-\frac{\mathrm{F}}{K}\right) - \mathrm{rF}\left(1-\frac{\mathrm{L}}{\mathrm{K}}\right) - \left(\alpha_f\mathrm{DR} + \beta_f c\right)\mathrm{F}$$

$$\frac{dR}{dt} = \mu_r\mathrm{R}\left(1-\frac{\mathrm{R}}{\mathrm{K}}\right) - \kappa_r\mathrm{YR} - (\alpha_r DR + \beta_r c)R$$

Where C_e is the concentration of chemical in the environment (mg kg^{-1}), c is the concentration of chemical in the organism c and β, β_f and β_r are proportionality constants equivalent to α, α_f and α_r in units of mg^{-1}. The remaining model constants are defined in the same way as in Section 3.1.

Only the linear combination $\alpha DR + \beta c$ appears in these equations. Thus, the model assumes that the chemical and radiation can be treated as additive, effective stressors. Other modes of interaction, e.g. multiplicative or sub-multiplicative synergism could also be considered by expanding the combination function to $\alpha DR + \beta c + \gamma cDR$. In future work we will explore the significant similarities and differences in model prediction by means of a sensitivity analysis of the parameters α, β and γ of such a generalised synergistic function, exploring cases of antagonism and synergism.

In addition, the model equations make the following assumptions: (a) The same organic system repairs damages caused by radiation and other environmental stressors, (b) the equations include deterministic effects only and (c) chemical concentrations in the wildlife are at equilibrium with the surrounding environment, implying that contaminant uptake and elimination processes are in balance (a more refined approach would be to include a biokinetic model, introducing the intake rate, the absorption efficiency and the biological half-life of elimination as additional parameters [56]).

The model can be parameterised as follows. In the absence of natural death, repair, adaptation or reproduction (i.e. by making $\kappa = \eta = p = 0$), the equation for initial damages becomes simply $\frac{dX}{dt} \approx -$

$(\alpha DR + \beta c)X$ giving $X = X_0 e^{-(\alpha DR+\beta c)t}$. The time for 50% damages (T_{50}) is calculated as follows: $X_0 e^{-(\alpha DR+\beta c)T_{50}} = \frac{1}{2}X_0$, so $T_{50} = \frac{ln2}{\alpha DR+\beta c}$. Setting T_{50} = 30 d would kill 50% of the population if the radiation dose received is the $LD_{50/30}$ for radiation; hence $30DR = LD_{50/30} = \frac{ln2}{\alpha}$ and therefore $\alpha = \frac{ln2}{LD_{50/30}}$.

For a chemical toxicity, the same approach is followed but the relevant magnitude is now the $LC_{50/30}$ (mg kg^{-1}). Hence, a concentration of $LC_{50/30}$ applied to the organism over 30 days would kill 50% of the population. By the same argument as above, $\beta LC_{30/50} \times 30 = ln2$ and therefore $\beta = \frac{ln2}{30LC_{30/50}}$ kg mg^{-1} d^{-1}.

The pollutant concentration in the organism can be calculated as a function of the pollutant concentration in the environment (water, soil or air), C_e, as this is more readily known in environmental impact studies. For the simple case of a steady state situation, this is done simply by using the concentration ratio, defined as $CR = \frac{c}{C_e}$. If c and C_e are not in equilibrium, a biokinetic model can be used.

The model is able to calculate effects at low concentrations based on acute data, owing to the dose-dependent recovery pool; recent studies on transforming acute ecotoxicity data into chronic data will allow us in future to improve upon this method [17]. By analogy with radiation [9], initial chemical-damage effects are assumed to appear at lower concentrations than reproduction effects, which in turn appear at lower concentrations than mortality effects. So, the condition $\beta << \beta_f < \beta_r$ can be imposed, and we conjecturally propose $\beta_f = 10 \times \beta$ and $\beta_r = 2 \times \beta_f = 20 \times \beta$, in order to allow an early testing of the model's behaviour. Ultimately, the $LC_{50/30}$ is the governing parameter for the model. Where such data are not available for the required chemicals and biological species, estimates could be made by comparison to similar toxicants and/or organisms, and safety factors can be added to account for conceptual or data uncertainties.

Other parametric relationships used are $\kappa_r \approx 1.5\kappa$ [30] and $\mu_R \approx 1.5\mu$ so that processes occurring within the repairing pool are somewhat faster than those occurring at the population level [28]. However, if other values exist for a specific species as part of a recent review, the latter are to be preferred [37]. It is also necessary to adapt the parametric saturation equation for the probability of forsaking adaptation and instead going into successful full repair (p_R), which is a function of cumulative dose as $p_R = \frac{p_0+p_1\int_{T-L}^{T} DR(t)dt}{1+p_0+p_1\int_{T-L}^{T} DR(t)dt}$, where p_0 and p_1 are experimentally derived coefficients [23]. We assumed that the chemical stressor can also induce an adaptive response with a non-adaptation probability $p_C(c) = \frac{q_0+q_1 c}{1+q_0+q_1 c}$; whereupon the combined probability for adaptation either by radiation or a hazardous chemical (which are not mutually exclusive events) is $p = p_R(DR) + p_C(c) - p_R(DR)p_C(c)$. It is quite evident that such a model can give a small "priming" effect whereupon a small chemical concentration can trigger adaptation that protects from radiation, and vice-versa.

4. New modelling results

4.1. Population model for Chernobyl voles in the Red Forest

We performed a sensitivity analysis of the migration rates by setting up in the model a multiplier parameter k for the migration rate and varying it between 8×10^{-4} and 10 in ten logarithmically equidistant intervals, using ModelMaker 3's sensitivity analysis tool. The case of k = 1 corresponds to the default

migration rate multiplied by area set in the model, or 3.65×10^5 m^2 d^{-1}. The significance of $k = 8 \times 10^{-4}$ is that, for the scenario considered, this gives the minimum migration rate below which the population in the most contaminated area is tipped into extinction due to lack of the compensating influx of healthy animals from less contaminated areas. The results are shown in Fig. 1 (top).

All simulations for $k > 8 \times 10^{-4}$ restore the population, equilibrating to the same value, but the time of onset of X_1 decreases with increasing k. From this simulation, we extracted the recovery onset time T_R (defined as the time at which the healthy population in the most contaminated area begins to increase above a minimum of one individual) for different values of k. Since there is a relationship between time and dose rate for the modelled dose profile, it is possible to link k with dose rate, leading to the best fit curve $DR = 148\left(1 - e^{-0.894k}\right)$ with $R^2 = 0.996$ which, together with the calibration point $k = 1$ for a migration rate of 3.65 x 10^5 m^2 s^{-1}, allows for a "broad-brush" indication of what dose rate would make a population with a different migration rate sustainable (a more accurate calculation requires re-parameterising the model with species-specific LD_{50} and life history parameters).

We also performed a sensitivity analysis of the reproduction rate, given that the number of offspring per year is variously reported to be between 12 and 25 and the sex ratio can vary between 0.52 and 0.62 as previously reviewed by us [9]; hence the reproduction rate can vary between 0.017 and the model's "optimum reproduction" value of 0.059 d^{-1}. We set up a multiplier parameter $0.1 \leq k \leq 1$ to cover this interval. Results are shown in Fig. 1 (bottom), revealing that the model is not strongly sensitive to this parameter as equilibration of the population occurs always, and the effect of sex ratio variation is minimal.

We also made a sensitivity analysis of the relevant adaptation parameters. The parameter η signifies the average duration of the adaptation during which the presence of adapted animals reduces the effect of radiation on population as a whole, and this was reasonably well determined in our vole model, based on comparison with field data. The largest uncertainty lies in the parameters p_0 and p_1 which were set to 0.11 ± 0.10 and 0.023 ± 0.017, respectively, following a previous study [23]. Given that p_1 is small, for sufficiently low radiation dose rates, the probability of no adaptation is, approximately, $p_R(0) \approx \frac{p_0}{1+p_0}$; hence, $p_0 = 0$ signifies that all repaired organisms would undergo adaptation whereas, for large values of p_0 (> 10), more than 90% would be restored to healthy status without undergoing adaptation. We tested the effect of varying p_0 by modifying it with a multiplier $0 \leq k \leq 10$ to see the effect on producing an adapted population over this parameter range. The results in Fig. 2 (top) show that the model is relatively insensitive to variation in p_0; hence p_1 contributes the most to the overall model sensitivity. For $p_0 = 0$ this gave a conservative estimation of the adapted population W. As shown in Fig. 2 (bottom), a low value of p_1 maximises W_1 but reduces the onset time, whereas a higher p_1 causes W to peak at 1000 – 3000 s.

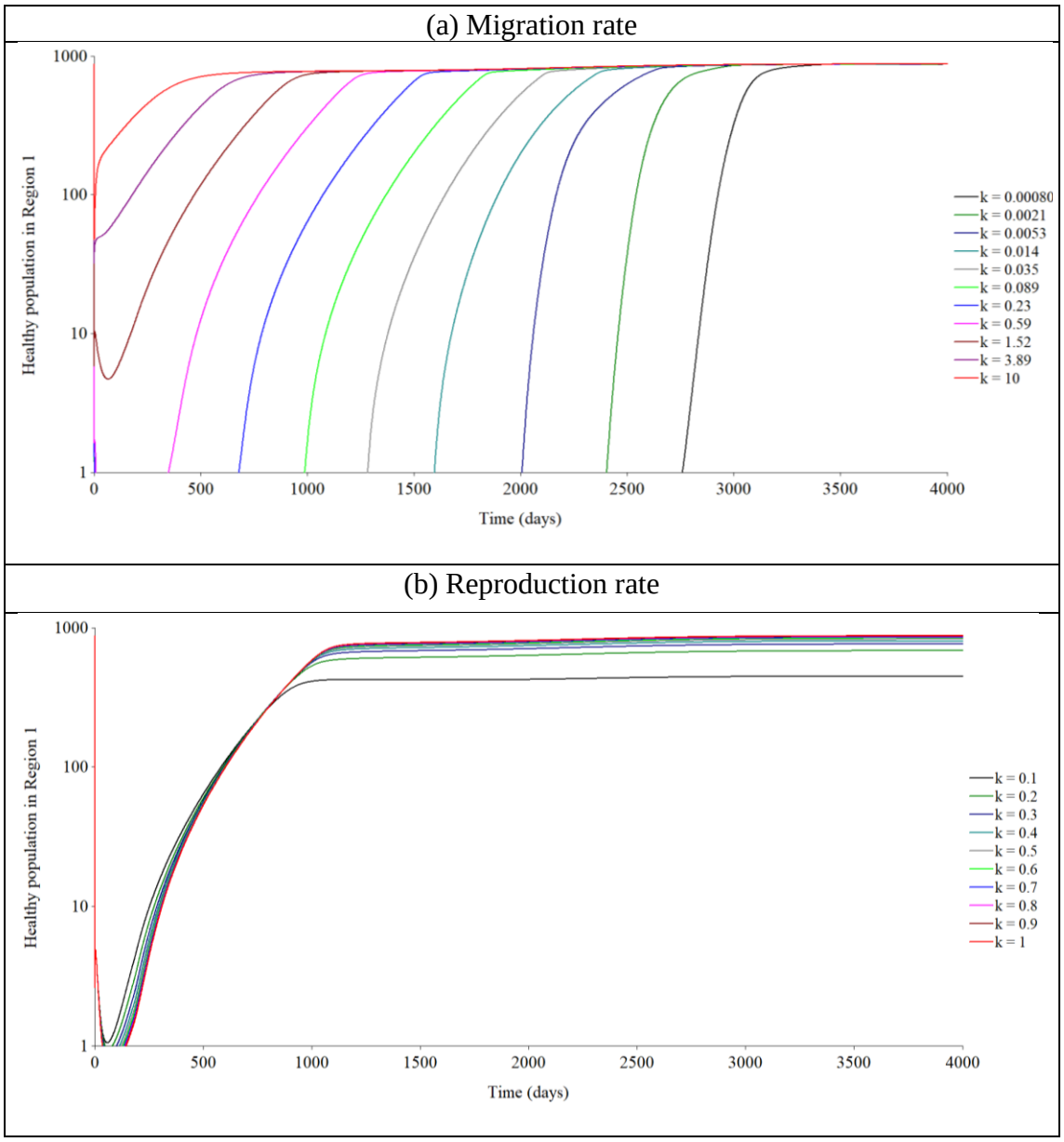


Figure 1: Sensitivity analysis of healthy voles in Region 1 as a function of a multiplier *k* acting upon the Rates of migration (top) and reproduction (bottom)

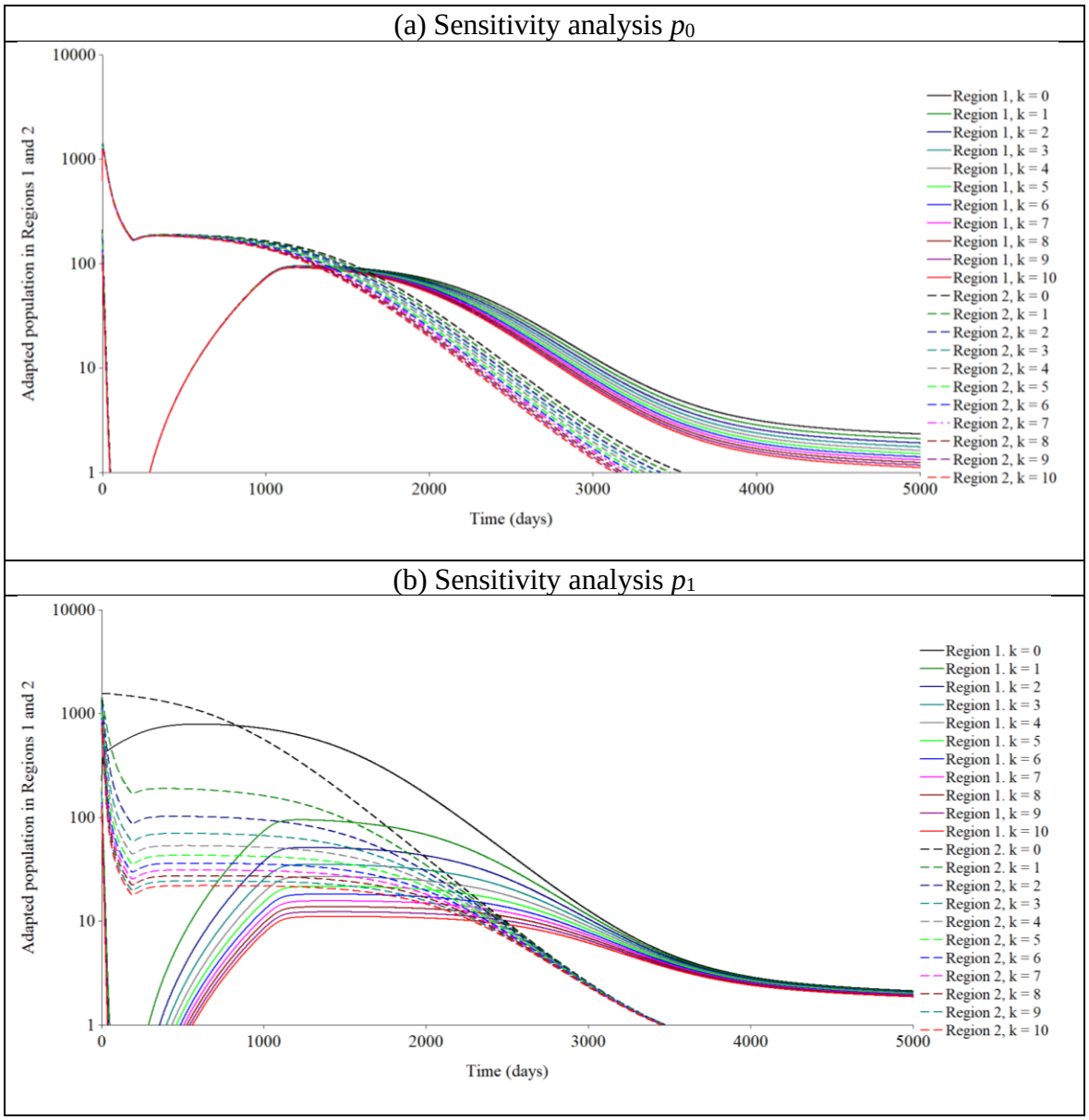


Figure 2: Sensitivity analysis for a population of adapted voles in Regions 1 and 2 as a function of a multiplier *k* acting upon p_0 (top) and p_1 (bottom)

4.2. Simulation of population response to radiation dose rate and chemical concentrations

The chemical and radiological model described in Section 2.6 was set up in Cherwell Scientific's software ModelMaker 3 [57], with equations being solved by the Runge-Kutta method with an accuracy of 10^{-5}, a minimum value of 10^{-11} and error scaling proportional to a constant value of 10. The ModelMaker set-up is shown in Fig. 3.

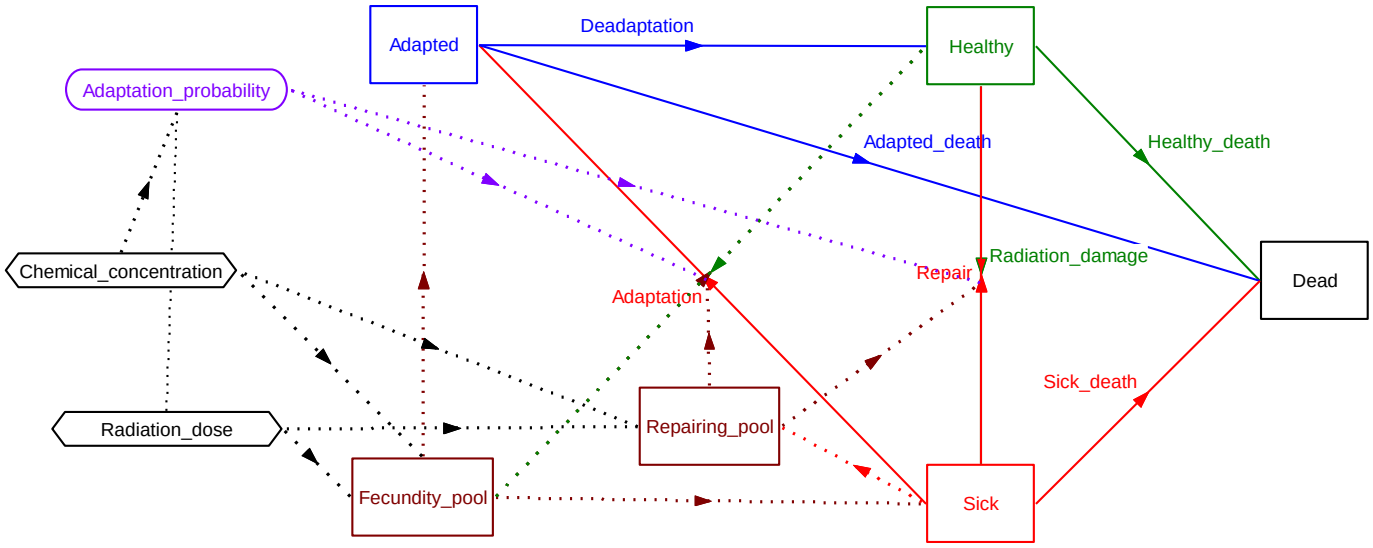


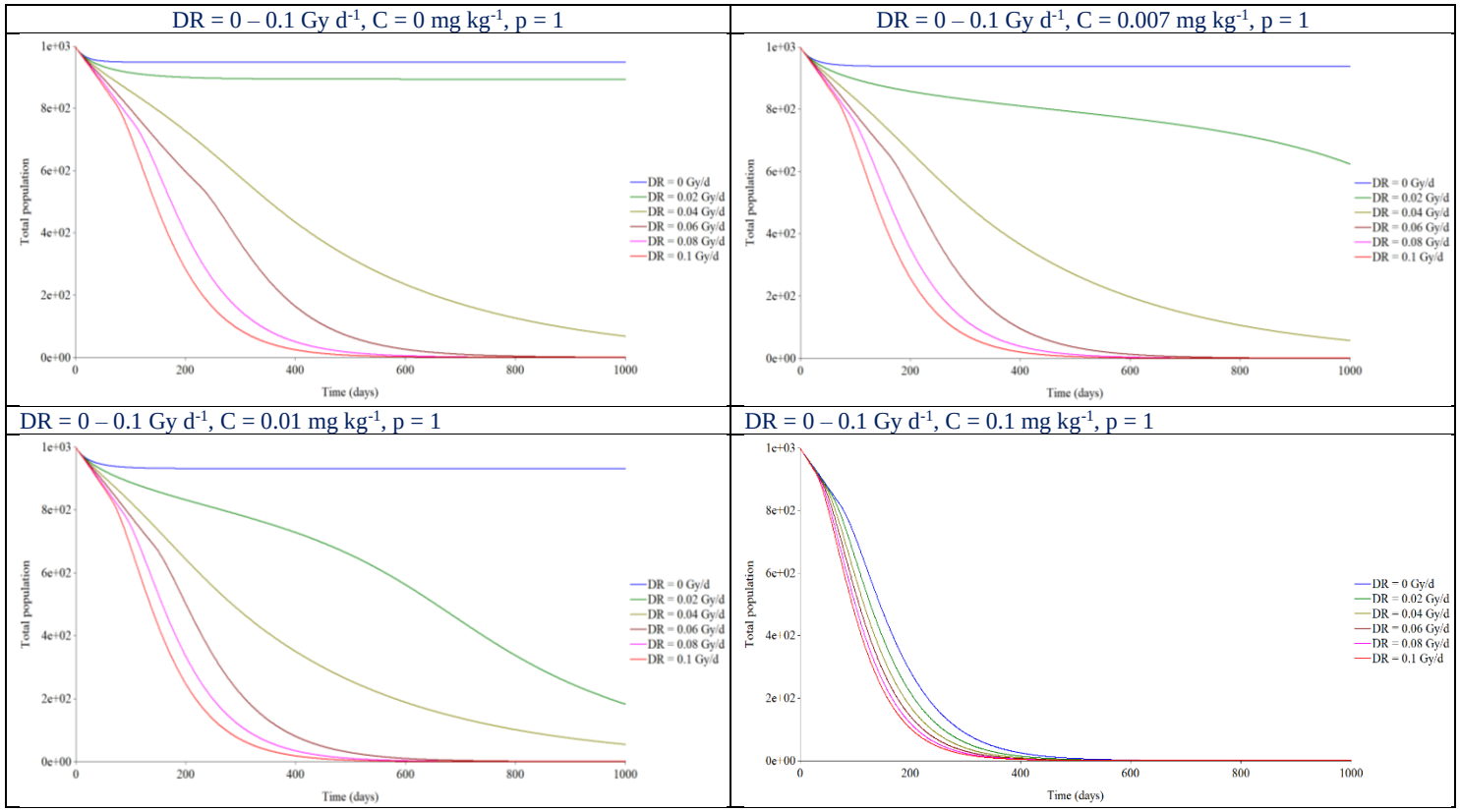
Figure 3: ModelMaker 3 setup. Rectangles represent model compartments, rounded rectangles are variables, hexagonal rectangles are definitions, arrows are flows and dotted arrows are influences.

The model features a switch that, when set to zero, fixes the probability of no-adaptation p to 1, thereby eliminating the adaptation process in the simulations, given that we found no data with which to parameterise q_0 and q_1. A future task then is to demonstrate synergistic and antagonistic phenomena that may emerge when radiation and chemicals are combined.

The exercise was carried out for a hypothetical contamination scenario involving gamma radiation in combination with sodium arsenate in the soil, inhabited by a population of small mammals (for which the parameters of the vole model were used). The LD_{50} for sodium arsenate is 112 mg kg^{-1} (or 40 mg kg^{-1} in terms of elemental arsenic) [58]. Fig. 4 demonstrates the effect of adding a progressively higher chemical dose for a range of radiation dose rates between 0 and 10^{-1} Gy d^{-1}. For zero chemical concentration, the results are consistent with the vole model without migration in that extinction is predicted only for dose rates > 10^{-2} Gy d^{-1}. Chemical concentrations above 0.007 mg kg^{-1} led to a population becoming extinct that would have remained otherwise stable at 0.02 Gy d^{-1}, and at 0.1 mg kg^{-1} the chemical concentration tipped into extinction a population that would have been stable in the absence of radiation. For elements of different toxicity, the tipping points were different, allowing us, in principle, to produce a comparative assessment of mixed chemical and radiological impact for an existing situation.

We expect that future research will enable us to draw conclusions about the most restrictive mixed exposure situations in terms of effects to the population, once the existing uncertainty in the toxicity model parameters has been addressed. It is difficult to include all the stages of the damage and repair process for chemical substances in a relatively simple and practical approach, leading to unavoidable conceptual uncertainties. It is therefore essential to improve the model with lessons learned from well-established approaches for chemical risk assessment [43, 59, 60], signalling the direction of future investigations.

Figure 4: Effect of adding a progressively higher chemical dose for a range of radiation dose rates



5. Conclusions

This paper has contributed to the work of the IAEA related to the production and application of international Safety Standards for the protection of the environment from ionising radiation, addressing the interest to test whether current benchmarks for risk assessment are appropriate when considering higher organisational levels of populations of species. Our study can inform the stakeholder dialogue on factors influencing population responses to radiation in the environment.

The project started by analysing the acquired knowledge on radiation effects and ecological interactions and applied it to radiological protection of wildlife populations, whereupon we defined the concept of population for the purposes of radiation protection modelling and proceeded to demonstrate a population model for voles in the CEZ to assess the historical exposure situation from a population perspective [9]. We further developed the vole model by carrying out sensitivity analysis for key factors influencing model output: reproduction rate, migration rate and adaptation probability. This allowed us to discover (a) that the model is more sensitive to changes in the migration rate than the reproduction rate, (b) that there is a mathematical link between dose rate, migration rate and population sustainability and (c) that the model is relatively insensitive to the adaptation model probability at zero dose.

We also introduced a simple candidate model to compare chemical and radiation responses, but it remains for the future to test and evaluate this theoretical model against mixture study data and to explore different forms of the generalised additive-multiplicative synergistic function by means of a sensitivity analysis. The development of models of this type provides an appropriate framework for formulating questions about how the effects of mixed stressors on populations are induced and expressed that would otherwise be difficult to articulate.

The main lessons learned from this project are (a) the need to define a population both spatially and temporally, (b) the need to consider the purpose of the population modelling, e.g. testing benchmarks that are not demonstrably set using population type effects, (c) the identification of key sources of data for population dose effects modelling such as the D5.2 of the EC programme STAR, the European Radiobiological Archives and the AnAge, AddMyPet and FREDERICA databases (d) the interest to test whether benchmarks are protective when transgenerational effects are included, as a test of robustness for the regulatory approach, (e) the need to improve our understanding on the mechanisms for effects caused by low dose rates of ionising radiation, (f) the need to advance further with population models to improve ecological realism and, particularly, as species mobility is a key factor, further questions arise as to the viability of fragmented habitats, even when the fragments are connected by migration corridors, (g) the need to extend to other species representing RAPs, (h) that developments in chemical risk assessment population modelling such as DebTox and individual-based modelling are relevant for radioecology, with need for consistency of approach between the two, and (i) that ODE population modelling is nevertheless a good practical approach for the purposes of radiation protection, given the ease with which it can be coupled to ecological processes and multi-stressor situations, serving as an appropriate framework for question formulation.

Our suggestions for the future are given in Table 1, where a distinction is made between points that are most important for radiological assessment (applying most directly to assessors and regulators), and points which are detailed research topics to be explored in scientific studies (applying most directly to researchers and modellers), although there is an inevitable degree of overlap between the two categories. Table 1 seeks to inform the dialogue on factors influencing wildlife population responses to radiation, including discussions on the ecological relevance of current environmental criteria and potential applications in a wider ecological risk assessment context.

The key general points for regulatory purposes, linking to the objective of aiding the evaluation of risk criteria used in radiological protection, are items 1 on population model applicability, 8 on model generality and 9 on assessment complexity. It is also important for regulators to foster dialogue between experts, the end users and themselves, as indicated in points 21 – 23. Moreover, it is advisable to seek consistency of approach between environmental impact assessment in ecotoxicology and in radiological protection, as indicated in point 20. Finally, advice items 5, 10 and 17 have a research focus, but it is also important for regulators to be involved if further use of population models is anticipated.

1 Table 1: Suggestions for the future applying most directly to (a) for assessors and regulators and (b) researchers and modellers

Nº	Criteria	Target	Description	Justification
General suggestions				
1	Applicability of models	Assessors/regulators	Population models should not be used as a new method for regulatory assessments, nor to change the structure of the existing radiation protection system, but as a tool to answer regulatory relevant questions, such as how robust are the existing benchmarks for exposure to wildlife populations in different exposure situations.	The reference approach provided by ICRP is sufficiently practical for the radiological impact assessment of releases to the environment, but there is need for model-aided discussions about populations in certain radiological scenarios.
2	Scope of modelling	Researchers/modellers	Widen the scope of modelling to additional scenarios/case studies covering different exposure situations.	It is necessary to cover more situations to underpin discussions with stakeholders on what scenarios and species are more sensitive.
3	Level of protection	Researchers/modellers	Identify sensitive protection targets within an assessment in terms of ecology for different exposure situations, particularly regarding life history and habitat occupation.	Need to consider large and/or long-lived species due to higher population vulnerability in chronic exposures. It is also needed to consider the tipping points where the most exposed organisms are sufficiently numerous to put the overall population at risk in a heterogeneously contaminated zone.
Experimental and field data availability				
4	Increasing robustness of data sets	Researchers/modellers	Obtain robust datasets on life-history for wildlife and biological effects (mortality, morbidity and reproduction) in chronic life-time exposures.	The information is still rather limited and extrapolation from acute to chronic effects has significant uncertainties; hence a critical evaluation is needed to increase model reliability.
5	Radiation effects in regulatory context	All	Continue reviewing studies indicating non-targeted effects, genomic instability, hormesis and transgenerational effects as potential factors for historical effects on populations.	Critical evaluation of new evidence is needed to reach a synthesis of the information, potentially helping to resolve the controversies reported in some field studies.
6	Exposure scenarios	Researchers/modellers	Obtain more effects data and dose scenarios for planned exposures, which cover most potential applications.	The Chernobyl Red Forest vole dose scenario is an existing exposure situation.
7	Species mobility data	Researchers/modellers	Review field data on animal mobility for different species and environments.	Since migration appears to be the most effective population recovery strategy, this is a key factor to follow up in an ecological impact assessment.
Population modelling				
8	Model generality	Assessors/regulators	Seek models as generic and applicable to many different species as possible, whilst preserving sufficient realism in terms of exposure situation, population size and range and survival areas.	Model complexity must be limited, reaching a balance between fitness for purpose and radioecological realism, with open and clear communication of the assessment uncertainties, bridging the gap between science and regulation.

9	Assessment complexity	Assessors/regulators	Avoid models that are too complicated by focussing on monospecific population responses of representative species (e.g. RAPs) and basic ecological interactions.	Using a minimum number of models that can cover a maximum of situations (transferable models) is a more practical approach but this needs to be balanced with model realism.
Model application				
10	Benchmark testing	All	Verify if benchmark testing results are robust when evaluated using different modelling approaches, comparing Matrix and ODE with IBM and DEBTox models	Inter-comparison between modelling approaches is a well tried and tested approach to establish the robustness of model predictions.
Increase ecological model realism				
11	Evolving approach	Researchers/modellers	Use the models developed in MODARIA as a stable base to add multiple stressors and indirect effects, making models more process-based.	Some of the concerns voiced by stakeholders are about multiple stressors and indirect effects.
12	Spatial issues	Researchers/modellers	Consider a more complex connectivity pattern between patches with different levels of contamination.	In real cases, the spatial dose rate pattern is heterogeneous and anisotropic rather than radial.
13	Habitats	Researchers/modellers	Improve the representation of habitat degradation and restoration in the equations representing habitat occupation, focussing on carrying capacity.	Habitability of contaminated regions depends on topography, changes in ecosystem resource and area recognition of immigrating populations, which are seldom included in models.
14	Sex ratio	Researchers/modellers	Include sex differentiation in populations (sex ratio, different home ranges/mobility and behaviour).	For many species, the sex ratio is 1.0 at birth but it becomes biased at maturity, due to differential dispersal of the sexes [9].
15	Predator-prey interactions	Researchers/modellers	Incorporate the Lotka-Volterra predator-prey equations [61, 62] to better consider ecosystem level effects.	The death rate is a strong function of predation pressure, leading to a radiation-dependent predator/prey balance [63, 64].
16	Radio-adaptation	Researchers/modellers	Adaptation of wildlife to chronic levels of ionising radiation requires more detailed modelling consideration.	Adaptation involves biological changes in a spatially heterogeneous and slowly declining dose profile whose importance is not wholly clear. Information on adaptation probability for multicellular animals is needed.
17	Model validation	All	Further population model validation is needed, requiring (a) experimental studies using laboratory ecosystems and (b) field studies in contaminated ecosystems.	Further model validation is precluded by insufficient long-term studies on the response of wildlife to chronic radiation stress.
Assessment of radiation impact in context of chemicals				
18	Model parameter uncertainties	Researchers/modellers	Reduce uncertainty in model parameters for the chemical ODE model by using dose response data and species sensitivity distributions to transform acute ecotoxicity data into chronic data.	Need to reduce uncertainties when extrapolating from $LC_{50/30}$ to derive toxicity test endpoints (e.g. EC_{10}, NOEC) for chronic situations.
19	Model conceptual uncertainties	Researchers/modellers	Reduce the conceptual uncertainty in the ODE model for chemical contaminants by making the damage and repair process for chemical substances more process based and consistent other approaches for chemical risk assessment.	The chemical assessment field has well developed approaches that should be reviewed and incorporated in future projects [43, 59, 60].

20	Consistency of approach	Assessors/regulators	Foster consistency of approach between environmental impact assessment in ecotoxicology and in radiological protection, using proof-of-concept models.	Many NORM and legacy sites where protection of wildlife needs to be assessed have a mix of radioactive and chemical contamination.
21	Implementing projects	Assessors/regulators	Develop in international programmes promoting sharing of new knowledge/approaches, model testing and inter-comparison, and training for member states (e.g. IAEA MODARIA and successors).	This type of project can be helpful to improve the rate at which science is integrated into regulation and guidance on radiological protection.
22	Interaction between experts	Assessors/regulators	Stimulate exchanges between the radioecological and ecotoxicological fields by encouraging expert meetings and cross-pollination of concepts.	Consistency of approach is desirable in producing technical and guidance documents as required by the United Nations Environment Programme.
Interaction with stakeholders				
23	Stakeholder dialogue	Assessors/regulators	Maintain a stakeholder dialogue on factors influencing wildlife population responses to radiation exposure in the environment and how this affects the validity of the benchmarks used for radiological protection of wildlife.	Models combining radiological and ecological impact can be used as a viable tool to inform this stakeholder dialogue, recognising that further validation is required, so the models are not yet usable for regulatory assessment.
24	Uncertainties	Researchers and modellers	Improve communication of modelling uncertainties	Population modelling is not more complex than existing assessment models but there is a general issue with model complexity in terms of their transparency and openness and the uncertainties built into them in terms of communication. An approach that is as practical and simple as possible in this regard requires a less substantial investment to foster stakeholder acceptance and understanding.

2

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