

Modelling the effects of ionising radiation on a vole population from the Chernobyl Red forest in an ecological context

Vives i Batlle, J. (✉)^{1*}, Sazykina, T.², Kryshev, A.², Wood, M.D.³, Smith, K.⁴, Copplestone, D.⁵ and Biermans, G.⁶

¹Belgian Nuclear Research Centre (SCK CEN), Boeretang 200, 2400 Mol, Belgium. Tel: +32 (0)14 33 88 05, Fax: +32 (0)14 32 10 56, e-mail: jordi.vives.i.batlle@sckcen.be

²Research and Production Association “Typhoon”, 4 Pobedy Str., Obninsk, Kaluga Region 249038, Russia

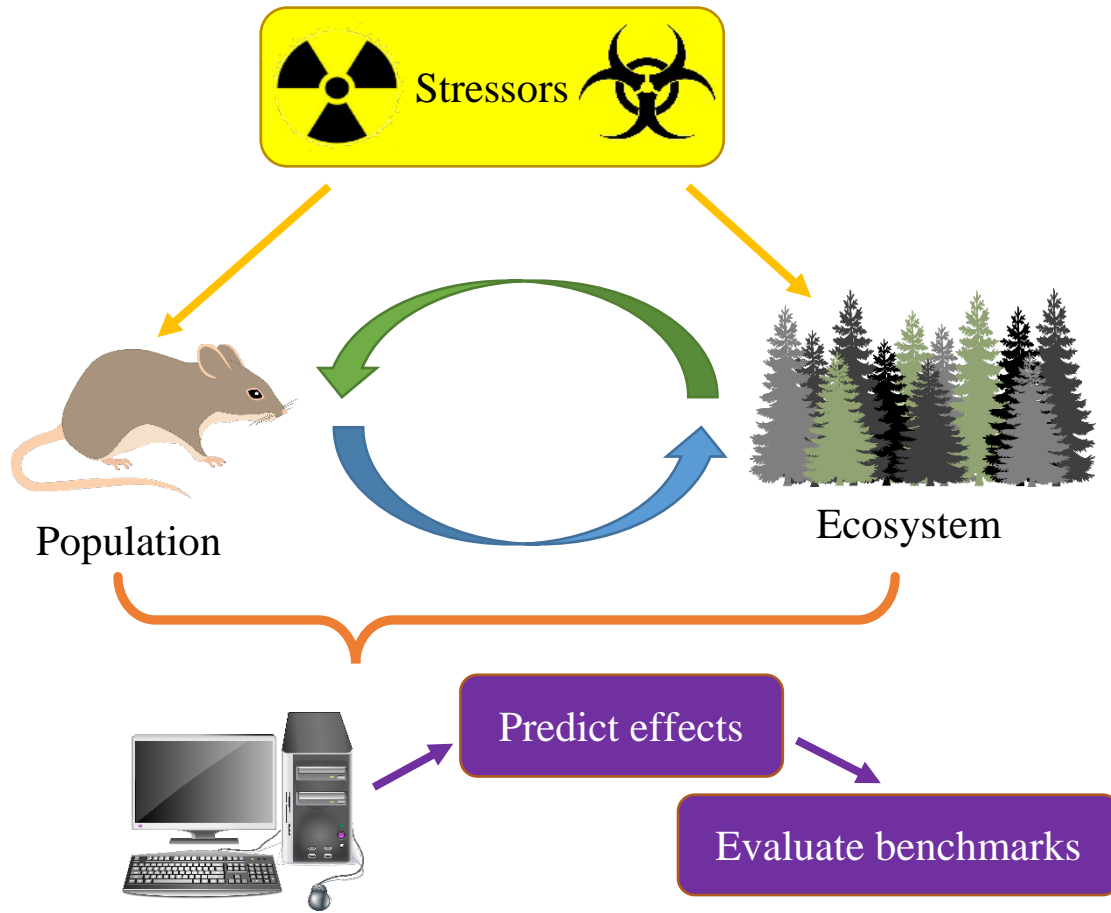
³School of Science, Engineering & Environment, University of Salford, Manchester, M5 4WT, United Kingdom

⁴RadEcol Consulting Ltd., 5 The Chambers, Vineyard, Abingdon, OX14 3PX, United Kingdom

⁵Faculty of Natural Sciences, University of Stirling, Stirling, FK9 4LA, United Kingdom

⁶Federal Agency for Nuclear Control, Rue Ravensteinstraat 36, 1000 Brussels.

*The corresponding author dedicates this paper to the memory of his parents.



Highlights

Migration is effective in compensating for vole deaths at high levels of radiation exposure.

Long term effects simulated include a small historic dose component.

Adaptation can account for low dose radio-hypersensitivity and increased radio-resistance.

Current radiation dose assessment benchmarks are protective **for the modelled** vole population.

1 **Abstract**

2 A novel mathematical model was developed to study the historical effects of ionising radiation from
3 the 1986 Chernobyl accident on a vole population. The model uses an ecosystem approach combining
4 radiation damages and repair, life history and ecological interactions. The influence of reproduction,
5 mortality and factors such as ecosystem resource, spatial heterogeneity and migration are included.
6 Radiation-induced damages are represented by a radiosensitive ‘repairing pool’ mediating between
7 healthy, damaged and radio-adapted animals. The endpoints of the model are repairable radiation
8 damage (morbidity), impairment of reproductive ability and mortality.

9
10 The focus of the model is the Red Forest, an area some 3 km west of the Chernobyl Nuclear Power
11 Plant. We simulated ecosystem effects of both current exposures and historical doses, including
12 transgenerational effects and adaptation. The results highlight the primary role of animal mobility in
13 stabilising the vole population after the accident, the importance of ecosystem recovery, the time
14 evolution of the repairing and fecundity pools and the impact of adaptation on population
15 sustainability. Using this model, we found dose rate tipping points for mortality and morbidity, along
16 with a limiting migration rate for population survival and a limiting size of the most contaminated
17 region needed not entailing loss of survival.

18
19 Our ecosystem approach to radioecological modelling enables an exploration of the impact of
20 radiation in an ecological context, consistent with the available observations. Model predictions
21 indicate that population sensitivity in this exposure scenario does not contradict the benchmarks
22 currently considered in risk assessments for wildlife. The model can be used to support advice on the
23 extent to which historical doses and other ecological factors may influence different exposure
24 modelling scenarios. The approach could easily be adapted to accommodate other stressors, thereby
25 contributing to the evaluation of other regulatory benchmarks used in non-radiological risk
26 assessment.

27 1. Introduction

28 Population modelling is well known in classical ecology (Lotka, 1925; Verhulst, 1838; Verhulst,
29 1845) and the potential role of these models in ecological risk assessment has been recognised (Galic
30 et al., 2010). They have been used to investigate the impacts of chemical contaminants on wildlife
31 (Forbes and Calow, 2002; Hanson and Stark, 2011; Ibrahim et al., 2014; Stark et al., 2004). Recently,
32 population modelling has been introduced in the field of radioecology (Alonzo et al., 2016; Monte,
33 2009; Sazykina, 2018; Vives i Batlle et al., 2012). These models help researchers to explore the
34 potential population level consequences of ionising radiation, but they are insufficient for informing
35 regulatory decision making where an ecosystem approach is increasingly advocated (Bradshaw et al.,
36 2014; Brechignac, 2009). We present here a new type of radioecological model considering the impact
37 of radiation in an ecological context (e.g. resource availability, migration, spatial heterogeneity and the
38 impact of historical doses) on a population of voles living in a radioactively contaminated area close to
39 the Chernobyl nuclear power plant (ChNPP) in Ukraine, such as the field vole *Microtus agrestis*, the
40 bank vole *Myodes glareolus* and other vole species.

41 1.1. The 1986 Chernobyl NPP accident

42 The ChNPP accident of 26 April 1986 remains the worst and most significant nuclear accident in
43 history. Some 2600 km² of Ukrainian territory around the ChNPP are officially designated as the
44 Chernobyl Exclusion Zone (CEZ). Due to the levels of radioactivity in the CEZ soils (mainly ¹³⁷Cs but
45 also ⁹⁰Sr, ²⁴¹Am and Pu- isotopes), the entire area is closed to the public. As a result of human
46 removal, abundant fauna and vegetation have inhabited the region and the CEZ has become an area of
47 high scientific interest for the study of radiation effects in wildlife.

48
49 An area of special interest near the epicentre of the accident is the Red Forest (Fig. 1), where the
50 radioactive cloud killed Scots pines (*Pinus sylvestris*). Although there was clearing and burial of top
51 soil in the Red Forest following the accident, it remains the world's most radioactively contaminated
52 terrestrial ecosystem. Deciduous trees, which are more radio-resistant than pine trees, now provide the
53 dominant tree cover in the Red Forest and the area supports a diversity of animal populations (Bird
54 and Little, 2013). One of these is the vole, a herbivorous mammal approximately 10 cm in length, of
55 the order *Rodentia* (Wood et al., 2009). Commonly found throughout the European continent, field
56 voles inhabit humid grass environments such as woodlands, marshes and river margins (Kryštufek et
57 al., 2008). Voles are an ideal bio-indicator to study the effects of environmental radiation in the Red
58 Forest due to their ubiquitousness, proximity to ground (the main source of radioactive contamination)
59 and relatively limited home range (Borowski, 2003).

60
61 It has been estimated that the absorbed dose rate to small mammals in the Red Forest decreased from
62 an initial maximum of 6 Gy h⁻¹ in 1986 to around 150 μGy h⁻¹ in 2005 (Gaschak et al., 2011). As of
63 2018, dose rates to wildlife from the Red Forest, estimated from measured organism and soil activity
64 concentrations, were in the order of 20 – 150 μGy h⁻¹, specifically 48 μGy h⁻¹ for vole species
65 (Beresford et al., 2019). A double exponential representation of the dose profile in Gy d⁻¹ can be
66 inferred, as $d_r(t) = 144e^{-\frac{1.1t}{365}} + 7.2 \times 10^{-3}e^{-\frac{0.05t}{365}}$ where t is the number of days since the accident.
67 In this representation, 4500 days is the transition point T_S where the short lived radionuclides have

68 decayed sufficiently, so 95% of the dose rate is accounted for by the slow decaying term,

69 $7.2 \times 10^{-3} e^{-\frac{0.05t}{365}}$.

70

71 Such dose rates exceed even now the dose rates below which populations are unlikely to be
72 significantly harmed based on current knowledge (known as benchmarks). One such benchmark is the
73 set of International Commission on Radiological Protection (ICRP) derived consideration reference
74 levels, or DCRLs. DCRLs are bands of dose rate within which there is likelihood of deleterious effects
75 of ionising radiation for Reference Animals or Plants (RAPs). These bands are currently set at 4-40
76 $\mu\text{Gy h}^{-1}$ for mammal, bird and pine tree RAPs (ICRP, 2008). Another type of benchmark is the ERICA
77 default screening dose rate, applicable to incremental (above background) exposures. This screening
78 dose rate, as derived from species sensitivity distributions, is $10 \mu\text{Gy h}^{-1}$ (Brown et al., 2008).

79

80 Published data on actual radiation effects from the CEZ are controversial, with significant
81 disagreement between researchers as to the extent to which wildlife has been affected (Beresford et al.,
82 2020a). There are reports of increases in total wildlife abundance over time as humans left the area
83 (Deryabina et al., 2015), and even reports of “beneficial” physiological effects in voles, such as
84 increased resistance of fibroblasts from against oxidative and DNA stresses in bank voles (Mustonen
85 et al., 2018). However, there is a consensus that, at the high dose rates present in 1986 (with monthly
86 doses of 22 Gy for γ -irradiation and 860 Gy for β -), animals would have been negatively impacted.

87

88 Population declines in mammals (including voles) by the autumn of 1986 have been documented, with
89 relatively quick recoveries by 1987 (Geraskin et al., 2008; Kryshev et al., 2005; Meeks et al., 2007;
90 Testov and Taskaev, 1990). There are also reports of voles showing slight but significant increases in
91 chromosomal aberrations, mitochondrial DNA mutations and cataracts, yet other work found no signs
92 of genotoxic stress (Baker R et al., 2017; Lehmann et al., 2016; Rodgers and Baker, 2000; Ryabokon
93 and Goncharova, 2006). In an attempt to address data deficiencies and discrepancies, projects COMET
94 (EU) and TREE (UK)¹ performed new field studies (Beresford et al., 2020b), compared results with
95 biota dose assessment benchmarks (Brown et al., 2016; Brown et al., 2008; ICRP, 2008) and showed
96 that external dose rates for radiocaesium often exceeds internal (Chesser et al., 2000), with effects
97 occurring in the dose rate ranges expected.

98 **2. Objectives and hypotheses of the study**

99 The objective of this study was to model the effects of ionising radiation in voles from the Red Forest
100 in their ecological context and to use this model for evaluating risk criteria (benchmarks) used in
101 regulation. For this, we considered spatial influences (e.g. migration, inhomogeneity of contamination)
102 and historical doses (higher exposure of previous generations), although we did not consider seasonal
103 variations due to the large uncertainties induced in model parameterisation.

104

105 The emphasis of methodologies for the protection of the environment from radiation is to protect
106 populations (rather than individuals) of flora and fauna, from the deterministic effects of radiation.
107 Presently, population modelling is not included in regulatory assessments, so our intent was to

¹ <https://radioecology-exchange.org/content/comet>; <https://tree.ceh.ac.uk/>

108 introduce the concept in the stakeholder dialogue on factors influencing wildlife population responses
109 to radiation. The model presented here is therefore a simplified representation fit for that purpose. We
110 wanted to capture an ecosystem approach in a compact set of equations that is as simple and practical
111 as possible, requiring a less substantial investment for acceptance by regulators and stakeholders who
112 wish to understand the key problem variables. Therefore, we intend this study to be used as a stable
113 base for future development in the process of bridging the gap between science and application.

114

115 We focused this study on two questions: (a) what are the key ecological factors that, in combination
116 with radiation sensitivity, determine the voles' vulnerability to radiation? and (b) what is the impact of
117 previous 'acute' exposures on organisms? We hypothesised that animal movement between differently
118 contaminated areas is a major influence on population stability at high doses of radiation, and that
119 transgenerational effects such as adaptation are not so influential, but they can help to understand
120 discrepancies between effects observed and predicted at low doses. An additional hypothesis was that
121 the existing protection benchmarks for small mammals are fit for purpose in an ecological context.

122 **3. Model description**

123 The model is a semi-realistic representation of the voles' habitat comprising three abstract regions: the
124 middle of the Chernobyl Red Forest (characterised by high dose rates), with an estimated surface area
125 of $2.5 \times 10^5 \text{ m}^2$, a surrounding patch of $5 \times 10^5 \text{ m}^2$ with 10% of the dose of the previous region, and an
126 external area with zero exposure. Region 3 could be very large, but for our purposes we define it
127 arbitrarily as equal to Region 2, or $5 \times 10^5 \text{ m}^2$, but connected to the external environment so voles
128 from outside can migrate into the inner regions. Migration between patches are a function of
129 differences in population density. Animals moving both ways between Regions 1 and 3 must pass
130 through Region 2.

131

132 Voles can be in four states: healthy (X), sick (Y), adapted (W) or dead (Z). Radiation-sensitive, logistic
133 auto-recoverable functions F and R exist for fecundity and radiation damage repair (Kryshev et al.,
134 2006; Kryshev et al., 2008). Due to current lack of the necessary systematic knowledge, it is not
135 possible to model each stage of the complex repair process, but this is not really necessary for our type
136 of model, as we only aim to reproduce the qualitative behaviour of the system. Evidence for an
137 adaptive response to chronic low-dose radiation at Chernobyl is somewhat equivocal (Møller and
138 Mousseau, 2016), but there is some indication of such effect, as mentioned previously (Mustonen et
139 al., 2018). Hence, the mechanism was introduced in our model, represented by a 'memory effect' in
140 which successful repair occurs with a given probability (Section 3.5).

141

142 A summary of the model parameters as determined by literature review, best judgement and/or field
143 observations, are shown in Table 1. This table gives the parametrisation of the model at the level of
144 vole, as a complete set of parameters for a specific vole species is not available.

145 **3.1. Representation of the ecology**

146 Suitable literature was identified on vole range sizes. Range size is variable depending on habitat (as
147 we know for other species) and that males range further than females (Borowski, 2003). As a first
148 attempt, since there is no sex differentiation in the model, we assume that the range size is the average
149 of the male and female range sizes for Birch woodland, or 510 m^2 .

150

151 There are various studies for voles and similar rodents reporting population densities between 6 and
152 100 individuals ha⁻¹ (Aulak, 1973; Borowski, 2003; Hutterer et al., 2016; Spitzenberger, 1999; Torre
153 and Arrizabalaga, 2008), averaging to 34 individuals ha⁻¹. Seasonal variation is large, namely 10¹–10²
154 individuals ha⁻¹ (Wereszczyńska et al., 2007). Studies performed in bank voles and mice show
155 fluctuations between lower values of 6–15 individuals/ha in spring (Jedrzejewska and Jedrzejewski,
156 1998) and 11–155 individuals/ha in autumn (Stenseth et al., 2002). However, we did not consider
157 seasonal variations in density, given the large spread of the reported data and consequent uncertainty,
158 and the fact that, although densities fluctuate from year to year, the long-term trend appears stable
159 (Hutterer et al., 2016). Hence, we adopted an annual average of 37 individuals ha⁻¹, or 3.7×10^{-3} voles
160 m² (Aulak, 1973; Borowski, 2003) for a deciduous woodland study is a reasonable starting point for
161 our modelling. Region 3 is really pine forest and Region 2 has a mixed vegetation, but for simplicity
162 we assume that all patches have the same maximum number of animals per unit area. Therefore, the
163 maximum capacity of each patch would be 925, 1850 and 1850 voles for Regions 1 – 3, respectively.
164

165 Regions of finite sizes cannot sustain an infinite growth of the population; population growth is
166 therefore modelled according to Verhulst's logistic equation (Verhulst, 1838; Verhulst, 1845), which
167 in essence predicts that the rate of growth of a population of N individuals follows the governing
168 equation $\frac{dN}{dt} = rN \left(1 - \frac{N}{K}\right) - dN$, where r is the reproduction rate constant, K is the carrying capacity,
169 representing the maximum number of individuals (sum of X , Y or W) that the ecosystem can support
170 (and to which the model tends asymptotically with time) and d is the natural death rate constant. In
171 optimum conditions, K is the surface area multiplied by the population density allowed by habitat
172 quality in the absence of radiation, but in reality there are subtractive terms in above equation to
173 account for mortality, predation, radiation damage etc., so the population grows to an asymptotic value
174 below K .

175

176 This model could be improved by introducing a minimum number γ below which the population
177 cannot recover (for example 2, i.e. a single couple of male and female), and introducing an additional
178 term to the growth part of the equation, such as $\frac{dN}{dt} = rN \left(1 - \frac{N}{K}\right) \left(1 - \frac{\gamma}{N}\right)$. Our model equations
179 include this term, but we found no data on the minimum vole population that is genetically viable;
180 hence we set γ to 0 until more information is available.

181

182 The reproduction rate constant was calculated as follows. Voles have a high reproductive potential of
183 between 4 and 5 litters per year, each one consisting of 3-5 (EOL, 2020), 4-7 (Glorvigen, 2012) and 5
184 (Sundell, 2002) young. Voles grow quickly with females maturing at 2-3 weeks and males maturing at
185 6-8 weeks ((MacDonald, 2001), cited in (EOL, 2020)). In order to approach the optimum reproduction
186 rate, we selected the upper value of 35 voles per year (0.10 days⁻¹). However, each newly born vole
187 originates from a pair of breeding voles, so the rate constant should be corrected in order to obtain the
188 mean *per capita* reproduction rate constant. The fraction of females in the population at birth is close
189 to 0.5, but it becomes weighted to females as maturity approaches (Myllymäki, 1977). This is
190 especially true at peak abundance, due to differential dispersal of the sexes. We derived a female
191 fraction of 0.57 ± 0.05 for *Microtus agrestis* in Sweden in 1973, a year in which there was a

192 population peak and so the effect is discerned with higher statistical significance (Hansson, 1978).
193 Therefore, we adopted a population averaged reproduction rate constant of 0.06 days⁻¹.

194

195 The natural death rate constant was deduced from a 2-year study of a population released on an island
196 in which the proportion of individuals surviving averaged to 0.54 (Boratyński and P., 2009). From
197 this, a natural death rate constant of 6.3×10^{-4} day⁻¹ could be assumed. However, this value does not
198 include predation. A natural death rate constant (combining natural death and predation) of 0.0031
199 day⁻¹ (Sazykina and Kryshev, 2016) was therefore adopted, based on data from the AnAge database
200 (AnAge, 2020). This means that some 80% of deaths are due to predation, and the death rate is a
201 strong function of predation pressure (voles are an important part of the diet of barn owls and they are
202 also preyed on by kestrels, other owls, weasels, stoats, foxes and snakes).

203

204 Whereas the reproduction rate changes with increasing population density following the logistic model
205 (Verhulst, 1838; Verhulst, 1845), the death rate (and hence the predation rate) remains unaltered in our
206 formulation. A formulation such as the Lotka-Volterra predator-prey model (Monte, 2009) is not used,
207 because the model's intended purpose is to compare model output with the DCRL band of dose rate
208 for a small mammal defined for the ICRP RAPs (ICRP, 2008), as seen in Section 4 on tipping points
209 and the testing of benchmarks. Presently, the international system of radiological protection does not
210 incorporate explicitly predator-prey interactions. Hence, we are making a compromise between
211 factorising predation in a sufficiently simple model by means of a compound parameter (the total
212 death rate constant) and more sophisticated models that would require many more site specific
213 parameters and would not be necessary be fitter for purpose.

214

215 We introduced an additional layer of ecological realism in the model by making K for the most
216 contaminated patch variable, given that the resource in the ecosystem (vegetation) was initially
217 damaged and recovered subsequently. To do this, we adopted a simple logistic equation for the
218 carrying capacities of the three regions of the model, K_i ($i = 1 \dots 3$):

219

$$220 \quad \frac{dK_i}{dt} = K_i^{max} \sigma_i \left(1 - \frac{K_i}{K_i^{max}} \right) - \nu_i d_{ri} \quad [1]$$

221

222 The term $\delta_i DR_i$ in Eq. 1 is a linear dose–response relationship without threshold for the ecosystem.
223 The parameter K_i^{max} is the maximum carrying capacity (equal to surface area SA_i multiplied by the
224 optimum population density ρ_i). We used the same optimum density for all the patches. d_{ri} is the dose
225 rate constant and the σ_i and ν_i are the rate constants for vegetation recovery and damage, respectively.

226

227 We parameterised Eq. 1 as follows. For σ_i (taken to be the same in all regions) we assumed that a
228 certain fraction f of the vegetation has recovered exponentially after a time τ : $f = 1 - e^{-\sigma\tau}$, and so
229 $\sigma = \frac{1}{\tau} \ln \left(\frac{1}{1-f} \right)$. Assuming a 95% recovery after half a year, $\lambda = \frac{2}{365} \ln \left(\frac{1}{1-0.95} \right) = 0.0164 \text{ d}^{-1}$. For ν_i
230 (which has units of Gy⁻¹, and is also assumed to be equal for all regions) we require the dose rate d_r
231 that kills a certain fraction f of the population in a short exposure time τ , so $\nu = \frac{\ln f}{d_r \tau}$. If the dose kills
232 50% of the population in 30 days, then ν must be of the order of $\frac{\ln 2}{LD_{50/30}}$. Since the grass understory is

233 the relevant vegetation for voles, we used the mean LD_{50} s of 16, 20, and 22 Gy for barley, wheat, and
 234 oats (all wild grasses), respectively (Real et al., 2004). This gives the conservative value $\delta = \frac{\ln 2}{19.33} =$
 235 $0.036 \pm 0.006 \text{ Gy}^{-1}$.

236

237 There is further information on impacts of radiation on terrestrial biomass (Monte, 2009; Real et al.,
 238 2004; Sazykina and Kryshev, 2006; Sazykina and Kryshev, 2003; Whicker and Schultz, 1982a;
 239 Whicker and Schultz, 1982b). This should allow, in future work, to include additional refinements,
 240 such as that thinning-out of trees would have led to an increase in ground vegetation and hence
 241 potential food resource availability for voles.

242

243 It must be emphasised that Eq. 1 does not include vole avoidance reactions, that is, whether incoming
 244 voles have recognition of any problems with the area, such as by observing unoccupied and degraded
 245 nests and remains of their predecessors. This kind of effect is very complex to model, with no specific
 246 data available as yet for voles in the region considered.

247 **3.2. Representation of animal migration**

248 We adopted a simplified matrix-based representation in which migration rates from Region i to Region
 249 j are assumed to be proportional to the gradient of population density between regions. We also
 250 assumed that healthy, sick and adapted have equal mobility:

$$251 \quad M_1 = \text{Migration}_{2 \rightarrow 1} - \text{Migration}_{1 \rightarrow 2} = \mu_{21} \frac{T_2}{S_2} - \mu_{12} \frac{T_1}{S_1}$$

$$252 \quad M_2 = \text{Migration}_{1 \rightarrow 2} + \text{Migration}_{3 \rightarrow 2} - \text{Migration}_{2 \rightarrow 1} - \text{Migration}_{2 \rightarrow 3}$$

$$253 \quad = \mu_{12} \frac{T_1}{S_1} + \mu_{32} \frac{T_3}{S_3} - \mu_{21} \frac{T_2}{S_2} - \mu_{23} \frac{T_2}{S_2}$$

$$254 \quad M_3 = \text{Migration}_{2 \rightarrow 3} - \text{Migration}_{3 \rightarrow 2} = \mu_{23} \frac{T_2}{S_2} - \mu_{32} \frac{T_3}{S_3} + \phi_0$$

255 Where μ_{ij} are the elements of the migration matrix for a patch of a specific surface area representing
 256 animal movement from Region i to Region j ($i = 1$ to 3 - in units of $\text{m}^2 \text{ d}^{-1}$) and $T_i = X_i + Y_i + W_i$.
 257 Consequently:

$$258 \quad M_i = \sum_{j=1}^3 \left(\mu_{ji} \frac{T_j}{S_j} - \mu_{ij} \frac{T_i}{S_i} \right) + \phi_0 \delta_{i3} \quad [2]$$

259 If we assume that there is no preferential direction of travel, then the migration matrix is symmetrical
 260 ($\mu_{ij} = \mu_{ji}$). All diagonal matrix elements μ_{ii} have zero value.

261 The term $\phi_0 \delta_{i3}$ (where $\delta_{i3} = 1$ if $i = 3$ and 0 if $i \neq 3$) is introduced to signify that Region 3 is an
 262 unlimited source of animals, being connected to the outside world, so any loss or supply of individuals
 263 between region 3 and its neighbour Region 2 is by definition balanced by a supply or loss of
 264 individuals from outside. In our results, we made additional simulations for a Region 3 isolated from
 265 the outside world in order to investigate the impact of varying surface area in that region, as a form of
 266 sensitivity analysis.

267 Mathematically, $\phi_0 = -\sum_{j=1}^3 \left(\mu_{j3} \frac{T_j}{S_j} - \mu_{3j} \frac{T_3}{S_3} \right) = \frac{T_3}{S_3} \sum_{j=1}^3 \mu_{3j} - \sum_{j=1}^3 \mu_{j3} \frac{T_j}{S_j}$ and $M_i =$
 268 $(1 - \delta_{i3}) \sum_{j=1}^3 \left(\mu_{ji} \frac{T_j}{S_j} - \mu_{ij} \frac{T_i}{S_i} \right)$. This means that, for Region 3, migration applies to X , Y and W
 269 whereupon it is apportioned to each category by a weighting factor: $M_i^X = \frac{X_i}{T_i} M_i$, $aM_i^Y = \frac{Y_i}{T_i} M_i$ and
 270 $M_i^W = \frac{W_i}{T_i} M_i$, respectively. This weighting is important: without it, mathematical asymmetries would
 271 be introduced in the model equations; migration must not depend on class of individual but on
 272 differences between total numbers of voles present in adjacent regions.

273 Eq. 2 above makes a simplification for Region 3, because migration from Region 2 to Region 3 gives
 274 rise to dilution of Y and W into an infinite pool and the migration back from Region 3 to Region 2 is
 275 considered to be solely of individuals of type X . The justification is that the proportion of Y and W in
 276 region 3 would be extremely low, given that it represents an infinitely large region where the dose rate
 277 is assumed to be zero. Hence, individuals born in that region are overwhelmingly of type X .
 278 Ultimately, calculation of population in region 3 is not directly relevant to our study, as this region
 279 acts merely as a reservoir.

280 Migration applies also to the quantities F and R , since they are pools that “move” along with their
 281 carriers. It is assumed that they intermix with migrations M_{Ri} and M_{Fi} as function of fecundity and
 282 recovery, respectively, with the same migration matrix coefficients as for M_i ; hence $M_{Ri} =$

$$283 \sum_{j=1}^3 \left(\mu_{ji} \frac{R_j}{S_j} - \mu_{ij} \frac{R_i}{S_i} \right) + \phi_0 \delta_{i3} \text{ and } M_{Fi} = \sum_{j=1}^3 \left(\mu_{ji} \frac{F_j}{S_j} - \mu_{ij} \frac{F_i}{S_i} \right) + \phi_0 \delta_{i3}.$$

284 the migration matrix elements μ_{ij} in the above equation could not be found directly in the literature,
 285 nor could be measured, so we resorted to derive them indirectly by means of an additional, purposely
 286 developed random walk model. We assumed for simplicity that voles wander in a randomised walk
 287 pattern, starting with a population of 925 voles (indexed $i = 1$ to 925) inhabiting a square patch of
 288 surface area $S_i = 2.5 \times 10^5 \text{ m}^2$, and a carrying capacity of 925 inhabitants, in order to give the required
 289 density of 37 voles ha^{-1} . We set up a separate algorithm that calculated the initial coordinates of a
 290 random distribution of voles:

$$291 x_{i,0} = W \left(\rho - \frac{1}{2} \right), y_{i,0} = L \left(\rho' - \frac{1}{2} \right) \quad [3]$$

292 Where ρ and ρ' are random numbers between 0 and 1. Note that this equation defines the origin of
 293 coordinates at the centre of the patch, so that the extremes of the calculated coordinates are $\left\{ \pm \frac{W}{2}, \pm \frac{L}{2} \right\}$.

294 At each time step $j = 0$ to $\frac{T}{\Delta t}$, defined in increments of $\Delta t = 0.01$ days for a total simulation of $T = 1$
 295 day, the algorithm updates these coordinates:

$$296 x_{i,j} = x_{i,j-1} + \left(\rho - \frac{1}{2} \right) \sqrt{2} v \Delta t, y_{i,j} = y_{i,j-1} + \left(\rho' - \frac{1}{2} \right) \sqrt{2} v \Delta t \quad [4]$$

297 Where v is the velocity of the vole. Note that this equation is defined such that the maximum distance
 298 walked by the vole (the modulus of the displacement vector) at each time step is, in Cartesian
 299 coordinates:

$$300 d_i = \sqrt{(x_{i,j} - x_{i,j-1})^2 + (y_{i,j} - y_{i,j-1})^2} \leq v \Delta t \quad [5]$$

301 During each time step, the voles that cross a region border to emigrate are calculated as those which
302 fulfil the following conditions: (a) that they were inside the patch at the previous time step, so
303 $|x_{i,j-1}| \leq \frac{W}{\sqrt{2}}$ and $|y_{i,j-1}| \leq \frac{L}{\sqrt{2}}$, and (b) that they step out of the patch at the present time step, hence
304 $|x_{i,j}| > \frac{W}{\sqrt{2}}$ or $|y_{i,j}| > \frac{L}{\sqrt{2}}$. The algorithm then counts the number of voles that remain in the region at each
305 time step $t_i = i\Delta t$. The migration rate constant was then calculated by least-squares fitting of the
306 function $m_i = \beta_0 e^{-\beta_1 t_i}$ where β_0 and β_1 are least squares best-fit parameters calculated by the
307 algorithm, conform to the definition $\mu = \beta_1 S_A$. The process was repeated 10 times and the results were
308 averaged.

309
310 The key parameter in the above calculation is the average velocity v , which could not be found directly
311 from literature. A study reports a mean daily-range size of field voles of 600 m², and an interfix
312 distance (the mean distance that the vole had moved between two consecutive fixes, used as a daily
313 mobility index of voles) of about 10 m (Borowski and Owadowska, 2010). A previous study gives a
314 daily interfix distance of 16 ± 4 m (Koivula and Korpimäki, 2001), noting that this varies greatly, from
315 1 to 65 m per day. This is considerably lower than the mobility of larger mammals such as weasels and
316 stoats, found to have daily ranges of 300 m and 1000 m, respectively (Klemola et al., 1999).

317
318 Conversion of the interfix distance into a mean velocity was performed by extending our algorithm to
319 calculate 100 vole trajectories starting from the same position at $T = 0$, allowing the animals to wander
320 for 1 day. We obtained $v = 200$ m d⁻¹ as the velocity required to obtain an average drift of 10 m after
321 one day. This result was fed into Eqs. 4 and 5. The two-step stochastic algorithm approach to calculate
322 the migration rates was implemented on an Excel VBA (Visual Basic for Applications) scripting code.
323 The model as set up calculated, for 10 independent simulations, a mean μ_{ij} of $(3.7 \pm 0.5) \times 10^5$ m² d⁻¹.

324 **3.3. Approach for radiation damage and recovery**

325 The approach for assessing radiation damage and recovery is based on our previously published dual
326 age class population model with radiation damage repair (Vives i Batlle, 2012). This model assumes a
327 dynamic exchange between X and Y voles regulated by repairing and fecundity pools R and F ,
328 respectively (Kryshev et al., 2006; Kryshev et al., 2008). R represents the capacity to repair radiation
329 damage of Y to become X again. Radiation dose causes a detriment in R , but R can recover as a logistic
330 function. If R is depleted, more voles die. F controls the reproduction of X and is also affected by
331 radiation, as well as being able to auto-recover logistically. The governing equations for radiation
332 damage and recovery are given in Fig. 2.

333
334 The key parameters were determined by a deduction process (Kryshev and Ryabov, 2000; Kryshev
335 and Sazykina, 2015; Kryshev et al., 2006; Kryshev et al., 2008; Sazykina and Kryshev, 2016;
336 Sazykina and Kryshev, 2012), which we have incorporated into a previous model (Vives i Batlle,
337 2012). The parameter $\alpha = \ln(2)/LD_{50/30}$ controls the initial radiation damages (mGy⁻¹), and α_f describes
338 damages to the reproductive system (mGy⁻¹). The time-dependence for mortality is due to damage to
339 the haemopoietic system and consequential suppression of cell division leading to profound
340 immunodeficiency, whereas damage to the reproductive system is mainly due to the sterilisation of
341 stem cells for sperm production (oocytes tend to be more resistant). Stem cells for reproduction are
342 more sensitive than stem cells that produce diverse lines lymphocytes and platelets, so it is generally

343 assumed that $\alpha_f = 10 \times \alpha$ (Kryshev et al., 2006; Kryshev et al., 2008). However, in our case, the
344 parameter can be calculated directly from a reported dose threshold of $> 4\text{Gy}$ causing $> 90\%$ organism
345 sterility (Sazykina and Kryshev, 2016).

346
347 The rate constant for damages to repairing pool α_r (mGy^{-1}) is derived from the same source. The
348 parameters κ and κ_r (signifying recovery induced by the repairing pool) are assumed by the same
349 study to be $\kappa_r = 1.5 \times \kappa = 1.5/R_{max}$. The Parameter μ_r is a conversion rate constant for sick individuals
350 returning to a repaired state, and we gave it a value of 0.032 days^{-1} , as seen in Table 1 of a publication
351 giving population sensitivities of animals to chronic ionising radiation (Sazykina, 2018). We chose the
352 recommended parameter for mouse (*Mus musculus*) because this is a warm-blooded animal with a
353 mass of 30 g, similar to the vole, and this parameter value is thought to reflect best the fast metabolism
354 of small mammals. A timescale of this order is plausible at least for males, due to cell division and
355 repopulation of the spermatogonial stem cell pool. Lastly, ε is derived from dose data for total lethality
356 (Sazykina and Kryshev, 2016).

357
358 In the original model by Kryshev et al. (Kryshev et al., 2006; Kryshev et al., 2008) and our previous
359 dual age-class model (Vives i Batlle, 2012), it was assumed that Y do not reproduce. Here, we adopted
360 a more realistic stance allowing reproduction of Y . We used a common F and R for all the categories
361 of population, and a single overall carrying capacity K .

362
363 We avoided separate fecundities F_x and F_y for X and Y respectively, and hence $\mu_{x/y}$ and $r_{x/y}$ due to lack
364 of realistic parameter data. Values of F and R for sick voles at different radiation dose rates are not
365 readily available. With this simplification, the model cannot distinguish which organism is healthy and
366 has full capacity to reproduce or self-repair, and which organism is sick and has these faculties
367 depleted. In other words, the model captures these processes at the overall population level and not at
368 the individual level. With this simplification, we can still represent at a phenomenological level
369 generational damages whilst keeping at a minimum the number of model parameters.

370
371 A possible question is whether offspring from sick are themselves sick or are healthy, and whether the
372 reproductive rate of the sick is lower. Our model is not an individual-based model, hence it cannot
373 represent these features directly. However, these phenomena are indirectly captured because radiation
374 depletes the repair pool, the fecundity pool is also depleted, so in practice populations with sick
375 members have a correspondingly calculated lower fecundity.

376 **3.4. Characterisation of the radiological exposure**

377 We adopted a dual exponential fitting for the dose rate received by the voles vs. time in Gy d^{-1} :

378 $d_r(t) = 144e^{-\frac{1.1t}{365}} + 7.20 \times 10^{-3}e^{-\frac{0.05t}{365}}$ where t is the time in days since the accident, based on
379 (Gaschak et al., 2011). This function has two features: (i) a rapidly decreasing exponential term for the
380 short-lived radionuclides that were significant contributors to dose in the initial period following the
381 accident, and (ii) a slowly decreasing term to represent long-lived radionuclides (^{137}Cs and ^{90}Sr)
382 (Monte, 2009). This approximation is valid because ^{40}K , ^{60}Co , ^{134}Cs , ^{154}Eu , $^{238,239,240}\text{Pu}$ and ^{241}Am
383 concentrations in soil were lower by 2-3 orders of magnitude than ^{90}Sr and ^{137}Cs (Gaschak et al.,
384 2011). The bioavailability of actinides to small mammals is low (Beresford et al., 2016) and, at

385 present, ^{90}Sr and ^{137}Cs are the main contributors to the total dose rate experienced by small mammals
386 in the Red Forest (Beresford et al., 2019).

387

388 The model has an option to use a step function at the border between regions of high and low dose
389 rate, and this was used in additional simulations with a constant dose rate over time (Section 3).

390 **3.5. Approach for modelling adaptation**

391 Effects studies indicating non-targeted effects such as adaptation as possible influences on the
392 historical effects of radiation are still being critically evaluated. Consideration of adaptation in this
393 model was introduced for exploratory purposes as a relatively simple phenomenological model, given
394 that a population approach for the dynamics of cellular responses to radiation is already available
395 (Wodarz et al., 2014). This model has a memory mechanism in which successful repair occurs with a
396 probability $1 - p$ and leads to adaptation with an average duration $1/\eta$, and a communication
397 mechanism under which an organism Y can induce an organism X to adapt with a rate proportional to
398 β_0 . An organism W can also induce protection in an organism X with a rate β_1 , but this happens mainly
399 in single cells rather than whole animals; hence we can assume $\beta_0 = \beta_1 = 0$.

400

401 We adapted Wodarz's equations as such: $\frac{dX}{dt} = -\alpha d_r X + \eta W$, $\frac{dY}{dt} = \alpha d_r X - cY$, $\frac{dW}{dt} = (1 - p)cY -$
402 ηW and $\frac{dZ}{dt} = pcY$, combining them with our general model by assuming that the outcome of the
403 repair (κYR) undergoes a branching between the formation of healthy voles, κYRp , and of adapted
404 voles, $\kappa YR(1 - p)$, where p is the probability of non-adaptation. Adapted individuals can in turn
405 become healthy at a rate equal to ηW . We also assumed that the repairing function is always 1 for W
406 organisms, since they are adapted to the radiation.

407

408 The parameterisation of the adaptation sub-model for voles remains conjectural for now due to the
409 lack of observations. However, we used indirect information to infer some of the parameters.
410 Intuitively, the adaptation rate constant should be slower than a fraction f of the repairing pool
411 recovery rate constant κ , hence $\kappa YR(1 - p) < f\kappa YR$ so $p > 1 - f$. Taking arbitrarily $f = 0.5$ gives $p > 0.5$.

412

413 The referenced adaptation study (Wodarz et al., 2014) states that at very low radiation doses (<0.3 Gy)
414 there is a hyper-radiosensitivity (HRS) phase. At slightly higher doses (0.5–1 Gy), an increased radio-
415 resistance (IRR) phase occurs. At higher doses ($\gg 1$ Gy), the mechanism loses its effectiveness. This
416 means that p is dependent on cumulative dose, tending to diminish at high doses. Therefore, adaptation
417 is more effective when a phase of low dose radiation (i.e. a 'priming' phase) occurs prior to a phase of
418 higher dose radiation, somewhat reducing the overall susceptibility of the population or (in the case of
419 an accident) when radiation levels have decreased enough for adaptation to begin.

420

421 We used a parametric saturation equation for p as a function of dose, following the previous study
422 (Wodarz et al., 2014) as $p(D) = \frac{p_0 + p_1 D}{1 + p_0 + p_1 D}$, where $D = \int_{T-L}^T d_r(t) dt$, and L is the relevant time of
423 accumulation of the dose. It makes sense for L to be, at least, a small number of generations (e.g. two).
424 According to one study, the average lifespan for voles is 0.5-2 years, with most individuals not lasting
425 more than one breeding season (MacDonald, 2001); we therefore took 1 year as a reasonable estimate.

426 Since the animals produce between 4 and 5 litters in the course of their lifespan, we assume that 0.5
427 years, rather than the full lifetime dose, is the relevant dose accumulation period L . The inevitable
428 uncertainty in L is not critical for short-lived animals (1 year) compared with the simulation timescale
429 of some 35 years, for the most part giving lifetime doses far exceeding 1 Gy.

430

431 In the study by Wodarz et al., the parameters p_0 and p_1 were set to 0.11 ± 0.10 and 0.023 ± 0.017 ,
432 respectively, based on averages ($n = 8$) of best fittings to previously reported dose-response curves for
433 HT29, HGL21, MR4, T98G and U138 cell lines (Krueger et al., 2007; Short et al., 1999a; Short et al.,
434 1999b). It is debatable whether the values assigned to the parameters p_0 and p_1 are applicable to
435 multicellular organisms, since they are for glioblastoma tumour cells, posing an unavoidable
436 parametric uncertainty in the adaptation part of the model. The key difference is that whole organisms
437 are longer-lived cell conglomerates and, as such, they are prone to absorb a higher dose during their
438 lifespan compared with cells considered in isolation. Therefore, we assign values to the parameters p_0
439 and p_1 conjecturally, since they are (after all) probabilities for a mechanism operating at the cellular
440 level, and we must await future experimental research to resolve the issue. The value of making this
441 assumption now is that we can at least provide the mathematical mechanism to model adaptation and
442 integrate it into our ecological population model, and although simulations are illustrative rather than
443 predictive, this enables to set guidance for future model development.

444

445 For the rate constant of conversion of adapted to healthy cells (η), the Wodarz et al. study uses a value
446 of $0.01 \text{ min}^{-1} = 14 \text{ d}^{-1}$. This is unlikely to be the same for whole animals, because in this case, one
447 would expect animals to have complex defence systems that would slow down the loss of adaptation,
448 in order to counteract their lower radio-resistance. Therefore, we expect $\eta < 14 \text{ d}^{-1}$ to be an upper
449 limit.

450

451 A very high dose of radiation would eliminate rapidly any of the few adapted individuals that formed
452 very early (before the accumulated dose was too great). This means that, in our model, η must exceed
453 0.08 days^{-1} , or else the number of W would be sustained for the first 3 years after the accident, unlike
454 observed. Our model gives a lower limit of $\eta = 0.15 \text{ days}^{-1}$ for 90% reduction of adapted organisms in
455 the first 30 days. This value is intuitively correct, satisfying the conditions for a sharp drop in W
456 followed by a subsequent peak of 100 individuals after 900 – 2100 days. This is consistent with
457 reported observations of increased radio-resistance to super-lethal acute doses of γ -radiation in animals
458 from the Kyshtym accident area for the 40th generation of mice of mice living in radiation
459 biogeocenosis (Sazykina and Kryshev, 2006), because for voles, 40 generations is equivalent to 2550
460 days.

461 ***3.6. Model equations and parameters***

462 The completed model was implemented in ModelMaker 4 (Adamatzky, 2001; Rigas, 2000), as shown
463 in Fig. 3. The ordinary differential equations describing the development of the vole population over
464 time are:

465

$$466 \quad \frac{dX_i}{dt} = -(d_i + \alpha_i d_{r_i})X_i + p_i \kappa_i Y_i R_i + \frac{r_i X_i}{X_i + Y_i + W_i} F_i \left(1 - \frac{X_i + Y_i + W_i}{K_i}\right) \left(1 - \frac{\gamma_i}{X_i + Y_i + W_i}\right) + \eta_i W_i - (\beta_{0i} Y_i +$$

$$467 \quad \beta_{1i} W_i) X_i + \frac{M_i}{T_i} X_i \quad [7]$$

$$468 \quad \frac{dY_i}{dt} = -(d_i + \varepsilon_i)Y_i + \alpha_i d_{r_i} X_i - \kappa_i Y_i R_i + \frac{r_i Y_i}{X_i + Y_i + W_i} F_i \left(1 - \frac{X_i + Y_i + W_i}{K_i}\right) \left(1 - \frac{\gamma_i}{X_i + Y_i + W_i}\right) + \frac{M_i}{T_i} Y_i \quad [8]$$

$$469 \quad \frac{dW_i}{dt} = -d_i W_i + \frac{r_i W_i}{X_i + Y_i + W_i} F_i \left(1 - \frac{X_i + Y_i + W_i}{K_i}\right) \left(1 - \frac{\gamma_i}{X_i + Y_i + W_i}\right) - \eta_i W_i + (\beta_{0i} Y_i + \beta_{1i} W_i) X_i +$$

$$470 \quad (1 - p_i) \kappa_i Y_i R_i + \frac{M_i}{T_i} W_i \quad [9]$$

$$471 \quad \frac{dZ_i}{dt} = d_i (X_i + Y_i + W_i) + \varepsilon_i Y_i \quad [10]$$

$$472 \quad \frac{dF_i}{dt} = r_i F_i \left(1 - \frac{F_i}{K_i}\right) - r_i F_i \left(1 - \frac{X_i + Y_i + W_i}{K_i}\right) - \alpha_{f_i} d_{r_i} F_i + M_{F_i} \quad [11]$$

473

$$474 \quad \frac{dR_i}{dt} = \mu_{r_i} R_i \left(1 - \frac{R_i}{K_i}\right) - \kappa_{r_i} Y_i R_i - \alpha_{r_i} d_{r_i} R_i + M_{R_i} \quad [12]$$

475

476 Where X_i , Y_i , W_i , Z_i are the healthy, sick, radiation-adapted and dead individuals at Regions $i=1, 2$ and
477 3; F_i and R_i are the (dose-dependent) fecundity and radiation damage repairing functions, the
478 migration fluxes M_i are given by Eq. 2, $d_r(t)$ is the dual exponential fitting to the dose rate, and the
479 time-dependent carrying capacity K_i is given by Eq. 1, with initial value $K_i^{max} = SA_i \omega_i$ with SA_i and ω_i
480 being the surface area and the ideal population density of the three geographical patches, respectively.
481 It is easy to check that if reproduction ceases, the model is mathematically in mass balance, since the
482 sum $X_i + Y_i + W_i + Z_i$ is constant.

483

484 A stable solution for the model for the case of no radiation or migration can be calculated by setting all
485 the derivatives and all d_{r_i} to zero, whereupon $Y_i = W_i = 0$ and $X_i = T_i$ are constant. If we further
486 simplify by assuming $\gamma_i \ll X_i$ then we have $X_i = F_i = K_i \left(1 - \frac{d_i}{r_i}\right)$ and $R_i = K_i$. which can be
487 retrofitted to the model as an approximation to initial conditions along with $\gamma_i = SA_i \omega_{min}$ where $\omega_{min} \geq 2$
488 and the initial conditions $Y_i(0) = W_i(0) = 0$. As for the total population, assuming that $\gamma_i \ll X_i$, $\frac{dT_i}{dt} =$
489 $-d_i T_i - \varepsilon_i Y_i + r_i F_i \left(1 - \frac{T_i}{K_i}\right) + M_i$. This can be used to show that, if $d_i \ll r_i$, the model solution is
490 relatively insensitive to variability in r_i . In our model, for example, $d_i/r_i = 3.1 \times 10^{-3}/6 \times 10^{-2} = 5 \times 10^{-2}$,
491 whereupon $X_i = 0.95 K_i$. Reducing r_i by 50% would give $X_i = 0.90 K_i$, causing only a 5% difference in
492 model output for the case considered.

493 4. Results and discussion

494 The model equations were solved numerically from within the ModelMaker 4 software. The time step
495 was set to 1 day. The simulation length was set to 10^4 days, covering the initial phase of the accident
496 and its long-term aftermath, given the time passed since the event. We used the Runge-Kutta solving
497 algorithm with an accuracy of 10^{-5} and a minimum value of 10^{-11} . The model output was checked for
498 mass balance and found to be correct in this respect. The solution accuracy was checked explicitly
499 against algebraically-derived approximate solutions.

500

501 Healthy organisms: Fig. 4a shows an initial fall of X in Region 1. At this point, there are limited
502 resources available for the voles. Migration from patch 2 into 1 begins to occur with new arrivals

503 becoming sick. As radiation decreases, more voles move into Patch 1 from 3, which acts as a donor
504 compartment, through 2, which acts as a transit compartment. Fig. 5 confirms this increased inward
505 migration, peaking at 15 voles day⁻¹ in Region 1 and 10 voles day⁻¹ in Region 2, respectively. The
506 cause of this is a steep gradient of population density after the initial irradiation. After 3000 d, net
507 immigration declines sharply as the dose rate diminishes and population gradients tend to zero.
508

509 The initial dose rate (about 6 Gy h⁻¹) is so high that it reduces X in Region 1 to less than 3 individuals
510 in just one day, turning them into Y . The original population from this Region would have rapidly
511 disappeared (allometrically derived LD_{50} for voles ≈ 6 Gy (Bytwerk, 2006)) but for immigration from
512 the adjacent regions, which causes a build-up to begin after 150 days. Initially, newly immigrated
513 individuals become sick as soon as they enter Region 1. By 800 days, X begins to increase as radiation
514 decreases, overtaking Y after 900 days. In Region 2, Fig. 4b, after an initial drop of healthy voles to
515 300 in 4 days, they begin to recover reaching 1000 voles at $t = 90$ days. Henceforth, X in Regions 1
516 and 2 recover steadily. Region 2 reaches 90% of the initial model value of 1750 voles after 1000 days.
517 Region 1 recovers more slowly, reaching 90% of the initial model value of 877 voles after 1670 days.
518 Thus, from 1700 days, X is restored, sustained by immigration. Fig. 4b shows that, by $T = 10000$ days,
519 adaptation does not significantly influence the results; hence, immigration from less contaminated
520 areas is the responsible agent for recovery in our model, offsetting losses by radiation damage.
521

522 Ecologically speaking, voles would only move into a contaminated area if there was a food or habitat
523 resource that they could utilise, so it depends on a suitable habitat existing throughout in Region 1.
524 The vole diet consists of leaves, seeds, grains nuts and fruit, some of which would have been available
525 as understory vegetation began to regrow. Incoming voles would still be exposed to harmful dose
526 rates, so population recovery by immigration would have been slow to begin with. Our model can
527 capture this implicitly with the very simple Eq. 1.
528

529 We performed sensitivity analysis on the immigration rate, as shown in Fig. 6. By parameter variation,
530 we obtained that if the migration matrix elements are reduced by a factor of 8×10^{-4} (equivalent to 290
531 m² d⁻¹), then X in Region 1, Fig. 6a, are still able to recover after 2800 days, when dose rates in Region
532 1 reach 0.035 Gy d⁻¹. At 255 m² d⁻¹ at 0.035 Gy d⁻¹ however, there is a tipping point for X in Region 2
533 (Fig. 6b). In region 2 which is receiving 10% of the dose rate, there are no observable differences
534 between the two migration rates, and X is able to recover in both cases (Fig. 6c).
535

536 Sick organisms: As stated previously, most X in Region 1 (and to a lesser extent in Region 2) become
537 sick in the days after the accident. In region 1, Fig. 4a, this takes the form of a shallow peak with a
538 maximum of 850 individuals in 56 days. Y remains high (> 800) until $T = 800$ days. By 900 days, Y
539 are overtaken by X , and collapse in 1600 days. In Region 2, Fig. 4b, a peak of 400 sick voles is
540 reached at 90 days, decreasing by 50% at 200 days and becoming exhausted after 960 days. This
541 behaviour is explained by the time evolution of R as shown in Fig. 7a: Initially, R is zero. Whilst it
542 increases quickly for Region 2, in the case of Region 1 there is no significant restoration of R until
543 after 1000 days, at which point recovery in Region 2 has already risen to 15% of its full value. Thus,
544 animals in region 2 are already self-repairing whilst they cannot do so in Region 1. A dose rate of > 7
545 Gy d⁻¹ induces the collapse of R in Region 1 at $T = 1000$ days, and a dose rate of 10 Gy d⁻¹ at $T = 90$
546 days causes a minimum of the recovery pool at 0.8% of its initial value. Therefore, a dose rate of 7 –

547 10 Gy d⁻¹ (5 – 7% of the initial dose rate at T = 0 days), typical of conditions in Region 1 some 900 -
548 1000 days after the accident or of Region 2 at about 120 - 240 days, is a tipping point for *R* in both
549 compartments.

550

551 At 4000 days, the dose rate to the voles is around 0.005 Gy d⁻¹ = 200 μGy h⁻¹ and the dose profile
552 begins to settle into a slower exponential decrease. The population in Region 1 has approached
553 stability and *R* for Regions 1 and 2 are very close to equilibrium (see below). By the end of the
554 simulation, with a dose rate of 40 μGy h⁻¹ = 0.001 Gy d⁻¹, there is no further redistribution between *X*
555 and *Y*. For comparison purposes, 200 μGy h⁻¹ corresponds to the middle of the intermediate DCRL
556 band for amphibians and grass, and 40 μGy h⁻¹ is the upper DCRL for mammal, bird and, pine trees
557 (ICRP, 2008).

558

559 Our model simulations show no difference in repair (and fecundity) when considering or not
560 considering adaptation in Region 1, and the same for Region 2, simply because the equation of
561 recoveries for *R* (and *F*) are independent from adaptation processes, hence *F* and *R* for the case of no
562 adaptation are not shown in Fig. 7. Although not shown in the figures, for the case of no migration, we
563 found that *R* is at optimum value for Region 3 but is obliterated for Regions 1 and 2. The situation for
564 zero radiation is a trivial case, with all pools at carrying capacity.

565

566 Fecundity: The time evolution of *F* is also given in Fig. 7. Initially, *F* in Region 1 collapses (Fig. 7a)
567 but it begins to recover almost immediately, whereas *R* stays impaired for 1000 days. This is because
568 our model assumes that *Y* are able to reproduce, and the balance of high exposure and immigration
569 from Region 2 into 1 conspire to maintain a reproducing sick population. In our previous modelling
570 studies we showed that, for isolated populations, fecundity is a more sensitive endpoint than morbidity
571 (Vives i Batlle, 2012; Vives i Batlle et al., 2012). For Region 2, Fig. 7b, there is an initial 90% loss of
572 fecundity which recovers by T = 4000 days. Henceforth, fecundity in both regions stabilises. In other
573 words, after 5 years voles in Region 1 reproduce near to optimum levels, coinciding with dose rates of
574 0.1 Gy d⁻¹ = 4000 μGy h⁻¹.

575

576 Adaptation: *W* form shortly after the beginning of the simulation, as soon as organisms begin to
577 accumulate enough dose to trigger it, but the effect is short-lived due to the immediate build-up of
578 cumulative dose (see Section 2.5). For Regions 1 and 2, Fig. 4a and 4c, the fraction of *W* at T < 1 day
579 was 22% and 50%, respectively. Then, *W* in Region 1 reach a minimum at 150 days, from whence
580 they increase to form a broad peak with a maximum of 95 voles at T = 1225 days. *W* remain at > 50%
581 of this value from 875 to 2300 days, coinciding with cumulative doses of 2500 – 35 and 250 – 3.5 Gy,
582 respectively, in Regions 1 and 2. The probabilities of adaptation at these levels of cumulative dose are
583 0.017 - 0.52 and 0.14 – 0.84, respectively. Taken together, this implies that most *W* in Region 1 at that
584 time come from Region 2, which has reached the IRR phase (see Section 2.5). Fig. 4c shows that *W* in
585 Region 2 also form a broad peak at this significant time interval (maximum of 10% of initial voles
586 adapted at T = 385 days).

587

588 Both peaks of *W* in Regions 1 and 2 subsequently fall, even though the probability of adaptation for
589 Regions 1 and 2 is 0.9 after 4000 days (Fig. 8a), coinciding with dose rates of 5 × 10⁻³ Gy d⁻¹ (200
590 μGy h⁻¹) in Region 1 and 10% of that in Region 2, and cumulative doses of 1 and 0.1 Gy, respectively.

591 The cause for the fall in W is that the proportion of Y decreases as dose rate decreases, and our model
592 does not consider adaptation from X . In addition, W gradually return to their normal healthy state. We
593 found no information to deduce if adaptation from healthy is ecologically significant at low doses
594 (presently the model has $\beta_0 = \beta_1 = 0$ due to lack of data).

595

596 Fig. 4b shows minor impact of adaptation on population sustainability. Some 10% of Y become
597 adapted in 875 - 2300 days, reducing the overall population morbidity, but these voles would be in the
598 healthy group if adaptation were not considered. We cannot draw strong conclusions on whether
599 adaptation can protect a population from extinction for the current scenario since the effect is masked
600 by migration. As we refine adaptation modelling with data from planned field experiments, its
601 significance may be further assessed. However, the example from Fig. 6a suggests that for species
602 with a low migration capacity compared with voles, adaptation may be more significant. Examples
603 would be ground-dwelling invertebrates and vegetation for which models are needed.

604 Effect of area size: We considered the consequences of the finite size of a Region 3 open to unlimited
605 exchange with the outside world. This assumption is mathematically equivalent to a large, closed
606 Region 3, provided its area exceeds 40 times that of the current Region 3 (equivalent to 25 times that
607 of Regions 1 and 2 combined), because then the population can be sustained by migration of voles
608 born in that outer area. A small and relatively uncontaminated area of 20 km² with an autochthonous
609 population of healthy voles is therefore enough to sustain the populations in the inner regions at the
610 radiation levels considered. There is more than enough vegetation around the Chernobyl Red Forest to
611 justify this assumption, since the area is surrounded by several kilometres of countryside comprising
612 coniferous plantation, deciduous forest, abandoned farm lands and even some wetlands. Another way
613 to interpret the above information is that, for the scenario considered, the contaminated part of a
614 heterogeneously contaminated patch should be in a ratio of 1:25 or less with respect to the total area.

615 Tipping points and the testing of benchmarks: As shown in our previous studies (Vives i Batlle, 2012;
616 Vives i Batlle et al., 2012), this type of radiation damage and repair model has tipping points around
617 which benchmarks can be verified for protection of a population. Even if migration is considered, the
618 model has tipping points, albeit at higher radiation levels. We performed a series of model simulations
619 with a constant dose rate over time, varying that dose rate in order to find general tipping points for
620 population at different levels of exposure. The results are shown in Figs. 9 (population) and 10 (repair
621 pool and fecundity). We discuss only Region 1, as Region 2 is the situation for 10% of the Region 1
622 dose rate.

623

624 At dose rates coinciding with the lower and upper limits of the DCRL for small mammal (rat), namely
625 10^{-4} and 10^{-3} Gy d⁻¹ (ICRP, 2008), no effects on population or recovery are predicted, irrespective of
626 migration, and both fecundity and the repair pool are at optimum levels. Thus, for our simulated vole
627 population, both the ICRP DCRL for small mammal - rat (4-40 mGy d⁻¹) and the IAEA's maximum
628 allowable dose rate for populations of wild mammals of 1 mGy d⁻¹ (IAEA, 1992) are found to be
629 protective of the population.

630

631 At an order of magnitude higher in dose rate (10 mGy d⁻¹) minor effects are predicted as a very small
632 number (ca. 5) of adapted organisms are formed. At this dose rate, species survival is not

633 compromised however. The pools R and F are not significantly altered if migration is considered, but
634 the first indications of morbidity and reproductive effects appear if migration is set to zero (some 15%
635 loss of repairing ability and 10% loss of fecundity in Region 1). Previous studies report that radiation
636 doses exceeding 10 mGy d^{-1} can begin to disrupt reproductive functions of animals (Gaychenko, 1995;
637 Suschenya et al., 1995; Suschenya et al., 1990).

638
639 At 100 mGy d^{-1} , the overall population is still unaffected if migration is considered, but the proportion
640 of W increases further to 20 animals in Region 1. Detriments of 5% and 10% in R and F , respectively,
641 appear at this dose rate. This is in line with previous investigations that, from above-background to
642 100 mGy d^{-1} , genetic effects in sexual and somatic cells of small mammals have been recorded
643 (Goncharova et al., 1999; Pomerantseva et al., 2006). If migration is excluded then X collapse and Y
644 peak at 230 individuals at 135 days, overtaking X at 170 days. Finally, the population in Region 1
645 collapses by $T = 650$ days. Both R and F fall precipitously; R is virtually extinguished by $T = 50$ days
646 and F follows suit at 300 days. This aligns with data that only chronic doses exceeding 100 mGy d^{-1}
647 are capable of causing a significant increase in the mortality rate of small mammals (Chesser et al.,
648 2000; Pryakhin et al., 2002; Sokolov et al., 1994; Suschenya et al., 1995). However, migration can offset
649 effects at this level of dose. In general, migration delays the onset of effects by an order of magnitude
650 of the dose rate.

651
652 The finding that the population collapses at 0.1 Gy d^{-1} without migration is not surprising, given that
653 the model uses $\alpha = \ln(2)/LD_{50/30}$ with an $LD_{50/30}$ of 6.2 Gy for mouse (Sazykina and Kryshev, 2016). At
654 0.1 Gy d^{-1} , this dose would be delivered in 62 days. The population model predicts a collapse at 135
655 days (cumulative dose 13.5 Gy). Protraction of dose over timescales sufficient for multiple cycles of
656 cellular reproduction therefore tends to increase the “apparent” value of the $LD_{50/30}$.

657
658 At 1000 mGy d^{-1} and with migration, the population is visibly compromised. More than 100
659 individuals (12% of the initial population) are sick, half of R is lost and there is a 40% loss of F . If
660 migration is excluded, the healthy disappear to $< 10\%$ of its initial size over 70 days, and the sick in
661 390 days. R collapses in 5 days and fecundity in 15 days. Lastly (not shown) the population disappears
662 between $10 - 100 \text{ Gy d}^{-1}$ even with migration (this is equivalent to the initial dose rate to the voles in
663 our simulation).

664
665 Comparison with published results indicates that our model gives sensible answers, providing a point
666 of validation. We also compared our findings with a previous model to model inter-comparison of
667 radiation effects in populations (Vives i Batlle et al., 2012). For mice, the previous study reported
668 population survival at $10^{-2} \text{ Gy d}^{-1}$, followed by a sharp decrease in survivors between 0.02 and 0.03 Gy
669 d^{-1} over a simulation period of 5 years. With migration disabled (to better compare with the
670 simulations of the previous study), our model predicts population extinction in 1300 days (3.6 years)
671 at 0.06 Gy d^{-1} , within < 2 of the inter-comparison result but in the same interval of $10^{-2} - 10^{-1} \text{ Gy d}^{-1}$,
672 providing an additional degree of validation for our approach.

673
674 Historical dose effect: Several phenomena reported here occur with a time delay with respect to
675 exposure: (a) nearly all X become Y initially, and Y die out fast, but after the dose rate has decreased
676 sufficiently, Y and W begin to form in Region 1 and they peak at 56 and 1221 days, respectively; (b) In

677 Region 2, *Y* and *W* peak at 90 and 390 days, respectively (although this is not easily visible in Fig. 6, it
678 can be seen in the data). These are examples of historical effects, appearing as they do months and, in
679 some cases, years after peak exposures. The historical effects predicted are relatively small in
680 comparison with current total population levels, but in a field situation, depending on the type of
681 sampling (particularly if sick animals are oversampled, as they may be easier to catch) they point at
682 potential miss-association of effects from the initial exposure to current (and lower) exposures.

683
684 To illustrate this, we conducted a theoretical simulation with the same starting population size but
685 using a step-function of the dose rate, set to be equal to 25 Gy d⁻¹ over 30 days, and 10 Gy d⁻¹
686 thereafter. Results are shown in Fig. 11. In this abstract scenario, after transition to the lower dose rate,
687 *Y* actually increase from 320 at 30 days to a peak of 420 at 180 days, with *X* reduced to 44% and a
688 shallow secondary peak of 70 adapted voles forming after 225 days. If radiation had been maintained
689 at 10 Gy d⁻¹, then the model does not predict the peaking of *Y* and *W* but instead it predicts that these
690 sub-populations change monotonically. The two simulations stabilise to the same end level.

691
692 This result shows that phenomena that depend on achieving a certain cumulative dose rather than dose
693 rate, like adaptation, will generally manifest with a time delay. The peak of *Y* also manifests as a
694 “memory” effect in the system.

695 **5. Future model development**

696 The model presented here is a simplified representation fit for the purpose of exploring issues relevant
697 to the current environmental radiological protection system. The equations provided are relatively
698 simple and practical. They can be solved numerically with relatively simple computational resource,
699 and partial analytical solutions can be explored for certain specific cases. Based on the present study,
700 we can already foresee improvements to add more realism to the model.

701
702 Our model presumes that the migration rates for the various populations *X*, *Y* and *W* are equal. A more
703 general type of model could be developed, in which the distinct populations of *X*, *Y* and *W* have
704 different mobility. This would have the benefit of generality, so that cases could be explored in which
705 these rates are varied. However, this would require knowledge of the differential migration rate
706 constants of sick and adapted individuals, which is not provided for by current field studies.

707
708 Although the random walk model presented in this paper is a useful first step, the Monte Carlo
709 approach could be extended to encompass a system in which females disperse more slowly than males.
710 It should be kept in mind that male voles maintain a territory and defend it by expelling other males
711 from it, whilst females just have a home range which may overlap with that of a neighbour. After
712 leaving the nest, young female voles remain in or near their mother's home range, but young males are
713 forced to disperse by the aggressiveness of the adult males. Female voles sometimes spontaneously
714 move in the time gap between weaning one litter and producing the next, a phenomenon typical of this
715 species. Hence, a modified algorithm that takes this into account would give an improved
716 representation of the dispersion.

717
718 Adaptation of animals to radiation may require more detailed consideration in future modelling
719 investigations than given here. This is because adaptation as a phenomenon may represent various

720 processes, such as stimulation of DNA repair or partial synchronisation of the cell cycle. It is possible
721 that the effect may depend more on dose rate than it is assumed here. It is still not fully clear how
722 important this process is in a slowly declining spatially heterogeneous dose field, whether adapted
723 individuals would revert to the healthy phenotype or whether the adapted state would persist.
724 Additionally, it is necessary to determine empirically the probability of adaptation as a function of
725 radiation dose for multicellular organisms.

726
727 Similarly, the spatial element of our model could be further developed. Our current model calculates
728 overall population movement, but it cannot predict a reduction of the overall population drift due to
729 the amount of time individuals are not moving when feeding/sleeping/mating, etc. It is mainly the
730 younger voles that will migrate in order to find new territories and this is different to the general
731 movement to find food within the home range. Moreover, in general, the dose rate pattern in any
732 geographical region is heterogeneous and anisotropic rather than a simple set of radially
733 interconnected regions. Therefore, it would be useful to explore a more complex pattern of
734 connectivity between the differently contaminated regions, although this would tend to make the
735 model less generic and more case specific.

736
737 The model could be further developed to add more ecological realism. Possible extensions include
738 considering in detail seasonality, sex ratio and predator-prey interactions as explored in our previous
739 work (Doi et al., 2005; Wilson et al., 2010). This would enable a more explicit behaviour of
740 populations of voles and their predators in the wild, both generally and at Chernobyl.

741
742 A more realistic and detailed treatment would eventually require a Monte Carlo simulation of the
743 whole system, facilitating further the model's application in evaluations of radiation exposure in
744 heterogeneously contaminated landscapes (Aramrun et al., 2019). It is also possible to obtain, within
745 an individually-based (IBM) type of model, population effects arising as emergent properties from
746 what happens to individuals. Such a model would allow individuals of different ages coexisting,
747 aging-related death to be accounted for and predation to be modelled as chance encounters between
748 predators and their prey. Sex differences could be factorised, and inherited effects could be tracked
749 over different generations. In addition, it would be possible to distinguish between individual animals
750 that have received a potentially fatal dose but may recover, and animals that have received a sub-fatal
751 dose and are likely to recover, but with reduced or completely suppressed capability to breed. This
752 distinction would be useful because sterilised animals can continue to compete for fertile mates,
753 whereas animals that have received a fatal dose are soon eliminated from the population. An IBM type
754 of model would have the additional advantage of explicitly considering the fecundity and repair state
755 of all individuals over time.

756
757 However, IBM modelling is complex and computationally demanding, in comparison with the ODE
758 model developed here. Our approach is fit for current purposes, because the stated protection goal of
759 radiation protection of the environment is the protection at the population level, and it needs to be
760 based on a criteria of "sufficiently complex to be realistic but sufficiently simple to be practical", a
761 decision that all modellers must make.

762

763 Lastly, our model could be adapted to investigate, at the level of R and F , the impact of multiple
764 stressors. For example, in 2016, wildfires burnt approximately 80% of the Red Forest. There would
765 have been, according to the dose profile used in this model, some 66 mGy h^{-1} in Zone 1 at the time. A
766 future development of our model could therefore be the introduction of fire as a stressor. The
767 recurrence of fires in the region presents an opportunity to revisit the area and make ecosystem
768 restoration observations, improving the model parameterisation for this case.
769

770 **6. Conclusions**

771 A conceptual population model for a vole population has been developed, parameterised and applied
772 to a Chernobyl Red Forest scenario to analyse the radiological impact of the accident at the population
773 level. The model suggests that increased inward migration in the early phase of the accident was the
774 main driver to restore population lost by the impact of the high levels of radiation. Newly immigrated
775 individuals became sick but the population of healthy voles recovered steadily over about 3 years,
776 sustained by immigration. In this situation, the repairing pool recovers more slowly than the fecundity
777 pool. The impact of adaptation was also modelled and its effect seems to be small, but it could be a
778 more important effect in less mobile species.
779

780 For the scenario considered, our model estimates that a migration rate constant of $255 \text{ m}^2 \text{ d}^{-1}$ at 0.035
781 Gy d^{-1} is a tipping point for vole population survival. A dose rate of $7 - 10 \text{ Gy d}^{-1}$ is an additional
782 tipping point for vole morbidity. The model predicts that a small and relatively uncontaminated area of
783 20 km^2 with an autochthonous population of healthy voles would be able to sustain the population. We
784 also found a tipping point for population survival if an area ratio of 1:25 or more is reached between
785 the most contaminated patch and the total area. Historical effects of radiation are predicted, with a
786 time delay of 1 year or more since exposure. Lastly, population level radiation effects predicted by our
787 model are in reasonable agreement with previous field observations, migration appears to delay the
788 onset of effects appearing at high dose rates by an order of magnitude and our model suggests that
789 benchmark values such as the ICRP DCRLs are sufficiently protective for this case study.
790

791 This study can inform stakeholder dialogue on factors influencing population responses to radiation in
792 the environment. Our model has the potential to aid evaluation of radiation benchmarks in multiple
793 case studies, the effects of multiple stressors and the influence of historic doses. As such, this model is
794 a valuable addition to the suite of modelling tools currently available to support both radioecological
795 research and radiation protection. Furthermore, the model that we have developed has potential
796 application in other ecological risk assessment contexts. The model allows consideration of the
797 sensitivity of the population's key biological functions, including survival and reproduction, in the
798 presence of ecological factors such as migration, ecosystem resource, biological adaptation and the
799 spatial scale of a stressor (radiation in the case study that we present). The model could easily be
800 adapted to accommodate other stressors, thereby contributing to the evaluation of other regulatory
801 benchmarks used in non-radiological risk assessment.

802 **CRedit author statement**

803 **Jordi Vives i Batlle:** Conceptualisation, Methodology, Software, Validation, Formal analysis, Writing
804 - Original Draft. Writing - Review & Editing, Supervision; **Tatiana Sazykina:** Conceptualisation,
805 Methodology, Validation, Writing - Review & Editing; **Alexander Kryshev:** Conceptualisation,
806 Methodology, Validation, Writing - Review & Editing; **Michael D Wood:** Conceptualisation,
807 Investigation, Writing - Review & Editing, Visualisation; **Karen Smith:** Conceptualisation,
808 Investigation, Writing - Review & Editing; **David Copplestone:** Conceptualisation, Writing - Review
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822

823

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Figure 1: The Red Forest in April 2016, just before the 2016 fire, showing the remains of dead pine trees lying on the ground, the deciduous trees that replaced them and some pines attempting to grow in the foreground. The grass, moss and lichen ground cover are also visible.

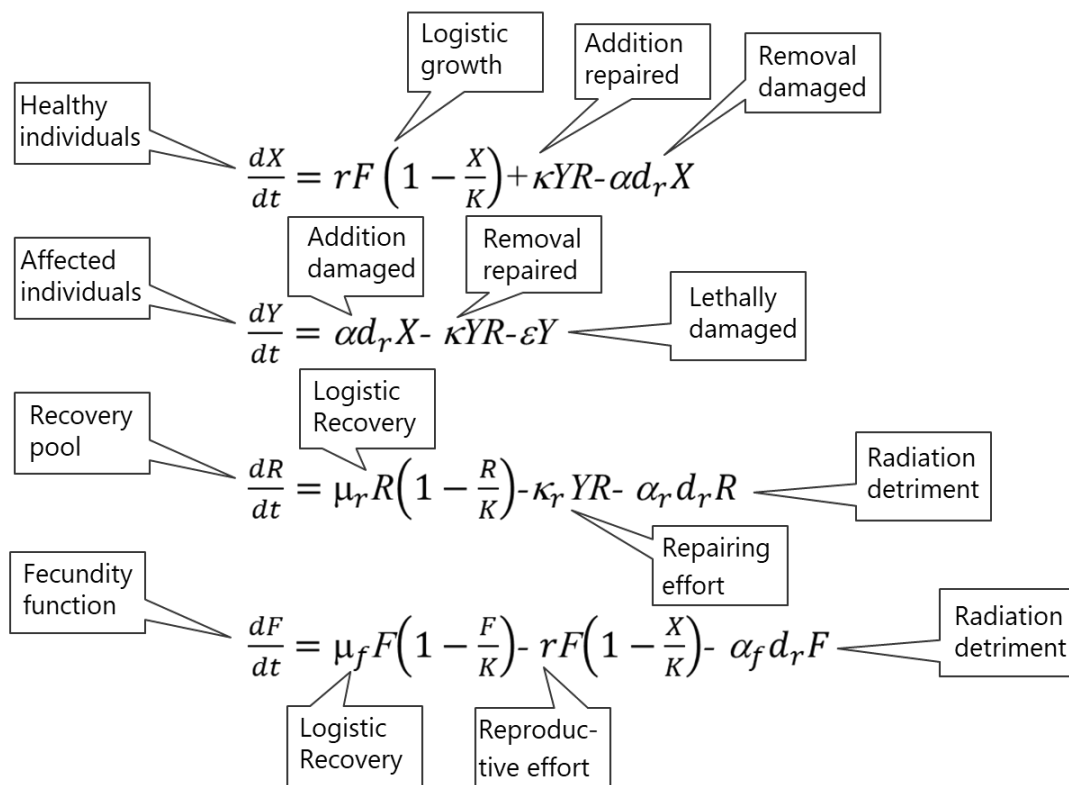


Figure 2: Equations governing the exchange between healthy (X) and sick (Y) members of the population, their recovery pool R and the fecundity F . Symmetry considerations demand that $\mu_f = r$ (Vives i Batlle, 2012).

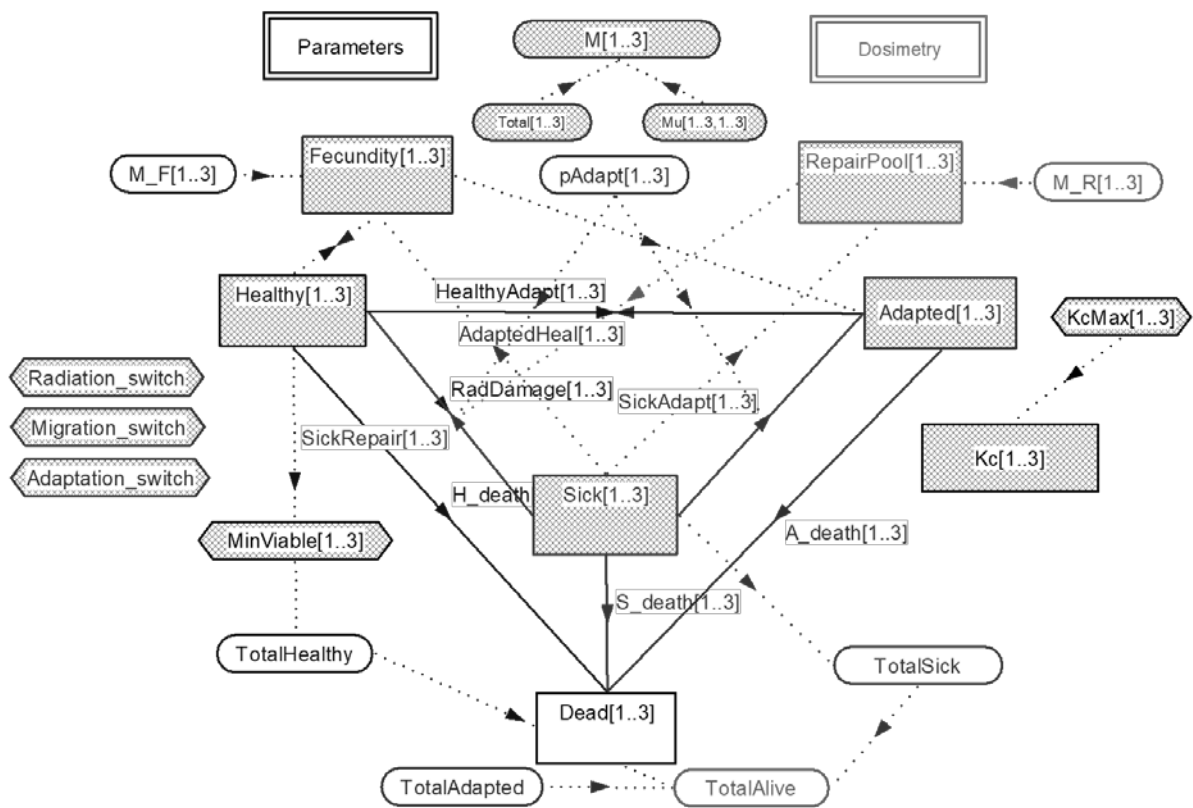
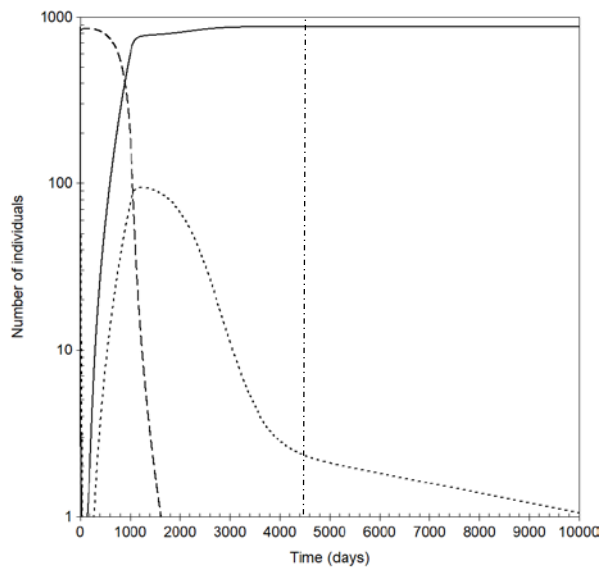
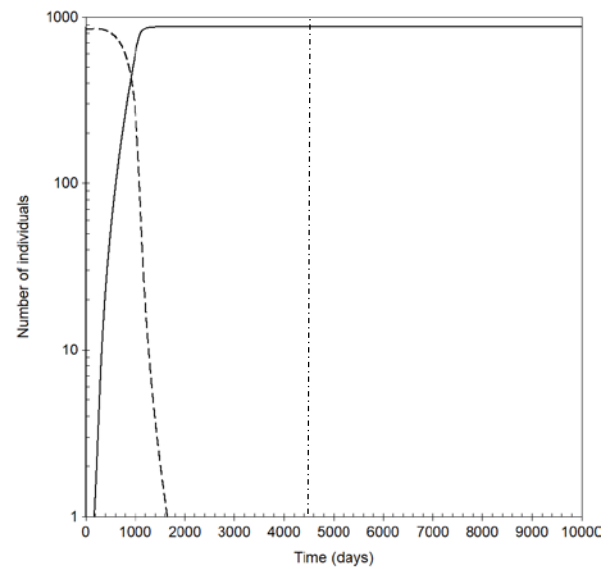


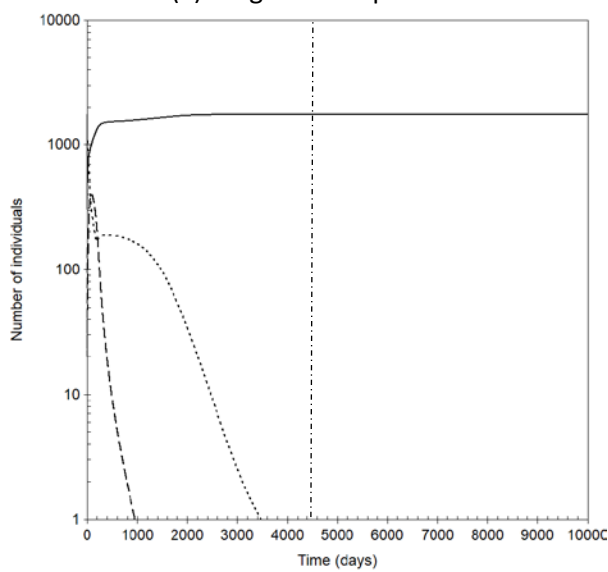
Figure 3: Model in ModelMaker 4. The indices 1-3 describe the three patches. Notation: Rectangles are compartments, rounded rectangles are variables, hexagonal rectangles are definitions, arrows are flows and dotted arrows are influences.



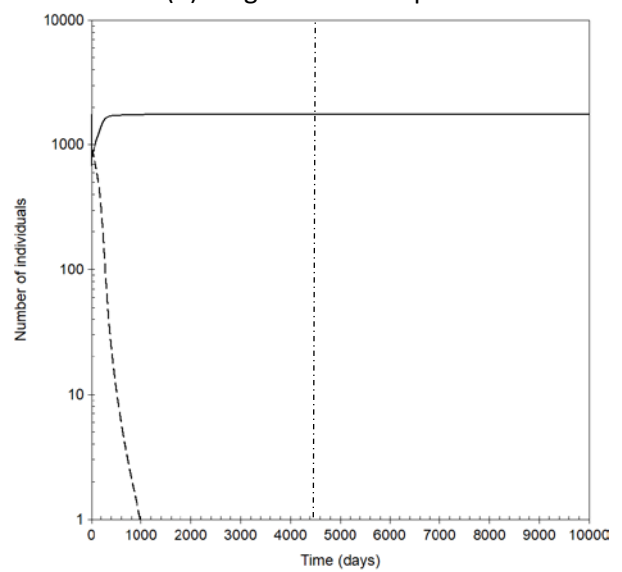
(a) Region 1- all processes



(b) Region 1- no adaptation



(c) Region 2 - all processes



(d) Region 2 - no adaptation

Figure 4: Model simulations of X (solid line), Y (dashed line) and W (dotted line) voles. The vertical line at 4500 d in this and subsequent figures is the transition point T_S where long-lived radionuclides dominate the dose.

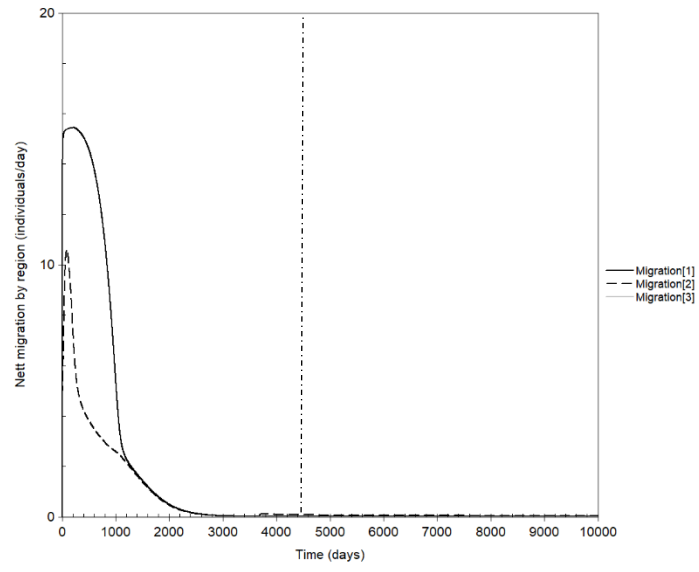
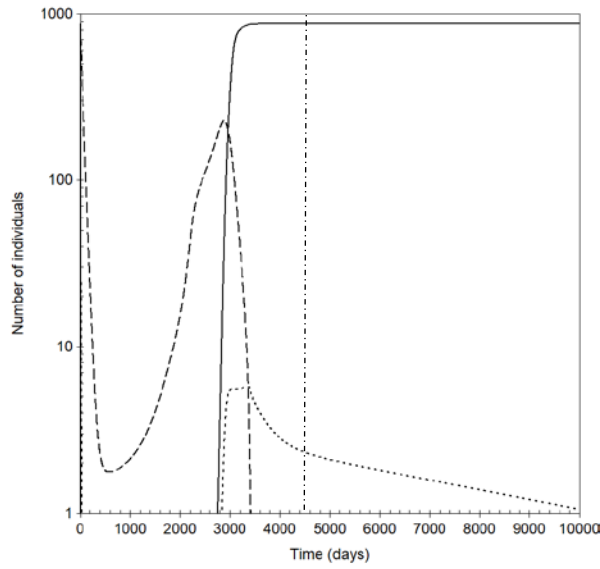
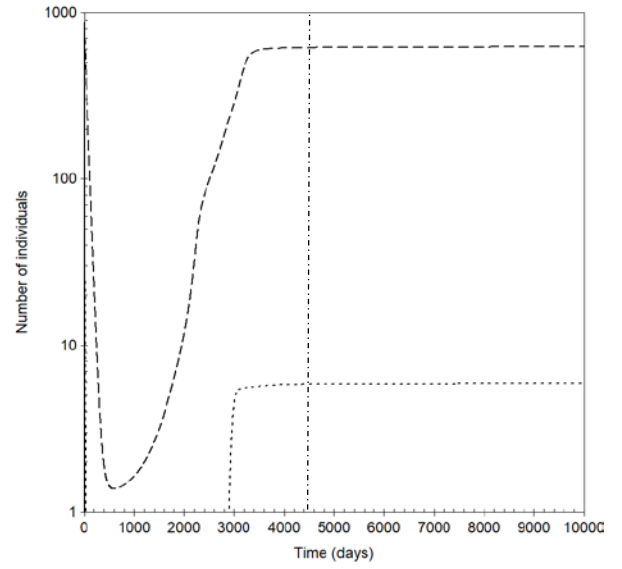


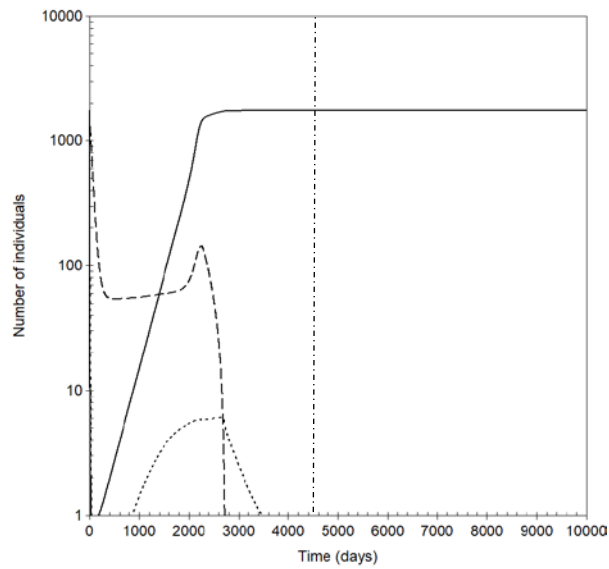
Figure 5: Migration fluxes for the different patches



(a) Region 1, migration rate = $290 \text{ m}^2 \text{ day}^{-1}$

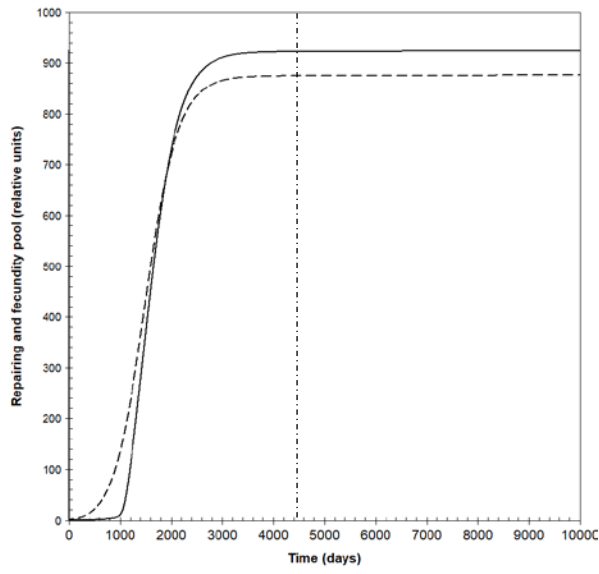


(b) Region 1, migration rate = $255 \text{ m}^2 \text{ day}^{-1}$

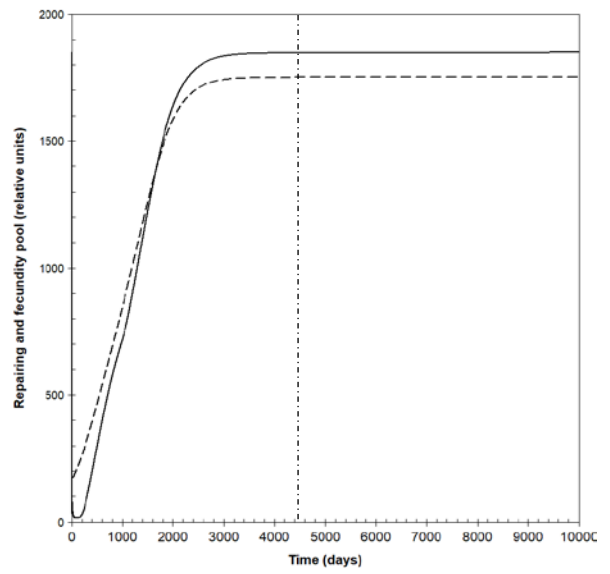


(c) Region 2, migration rate = $290 \text{ m}^2 \text{ day}^{-1}$

Figure 6: Model simulations of X (solid line), Y (dashed line) and W (dotted line) voles, illustrating the result of reducing the migration rate from $290 \text{ m}^2 \text{ day}^{-1}$ to $255 \text{ m}^2 \text{ day}^{-1}$, chosen for being close to the point at which X disappears.



(a) Region 1



(b) Region 2

Figure 7: Model simulation of R (solid line) and F (dashed line), with all processes included

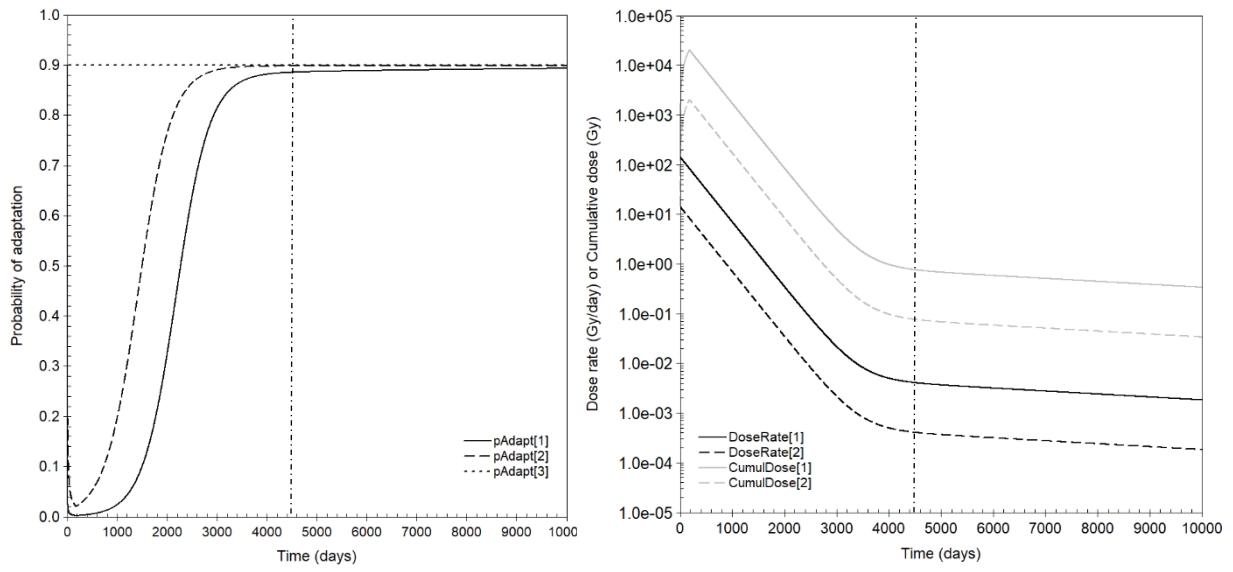


Figure 8: Probability of adaptation (left) and dose rates (black) plus cumulative doses (grey) (right). The data correspond to Regions 1 (solid), 2 (dashed) and 3 (dotted), respectively.

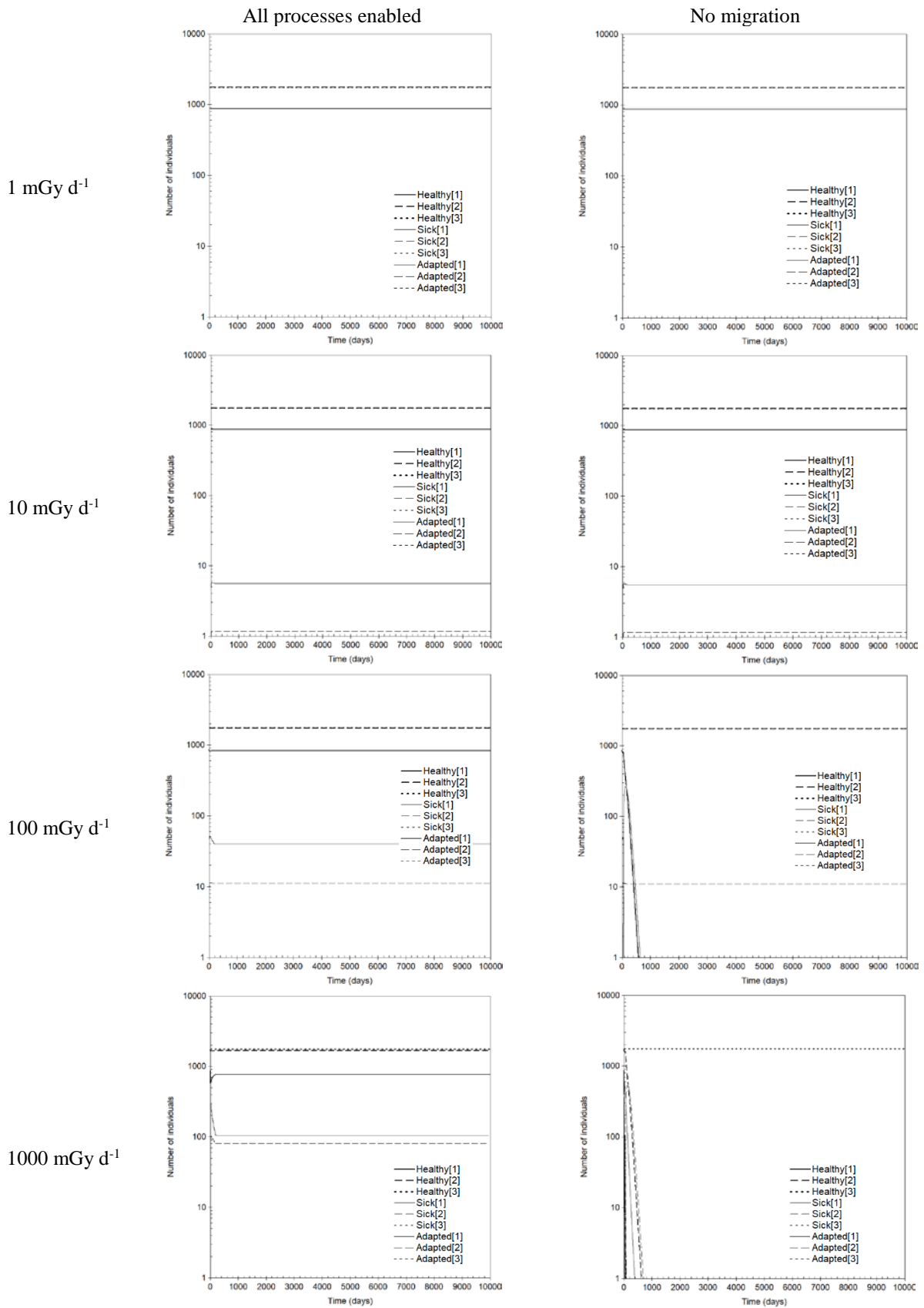


Figure 9: Model simulations of population with varying levels of dose rate in Region 1, assumed constant in time

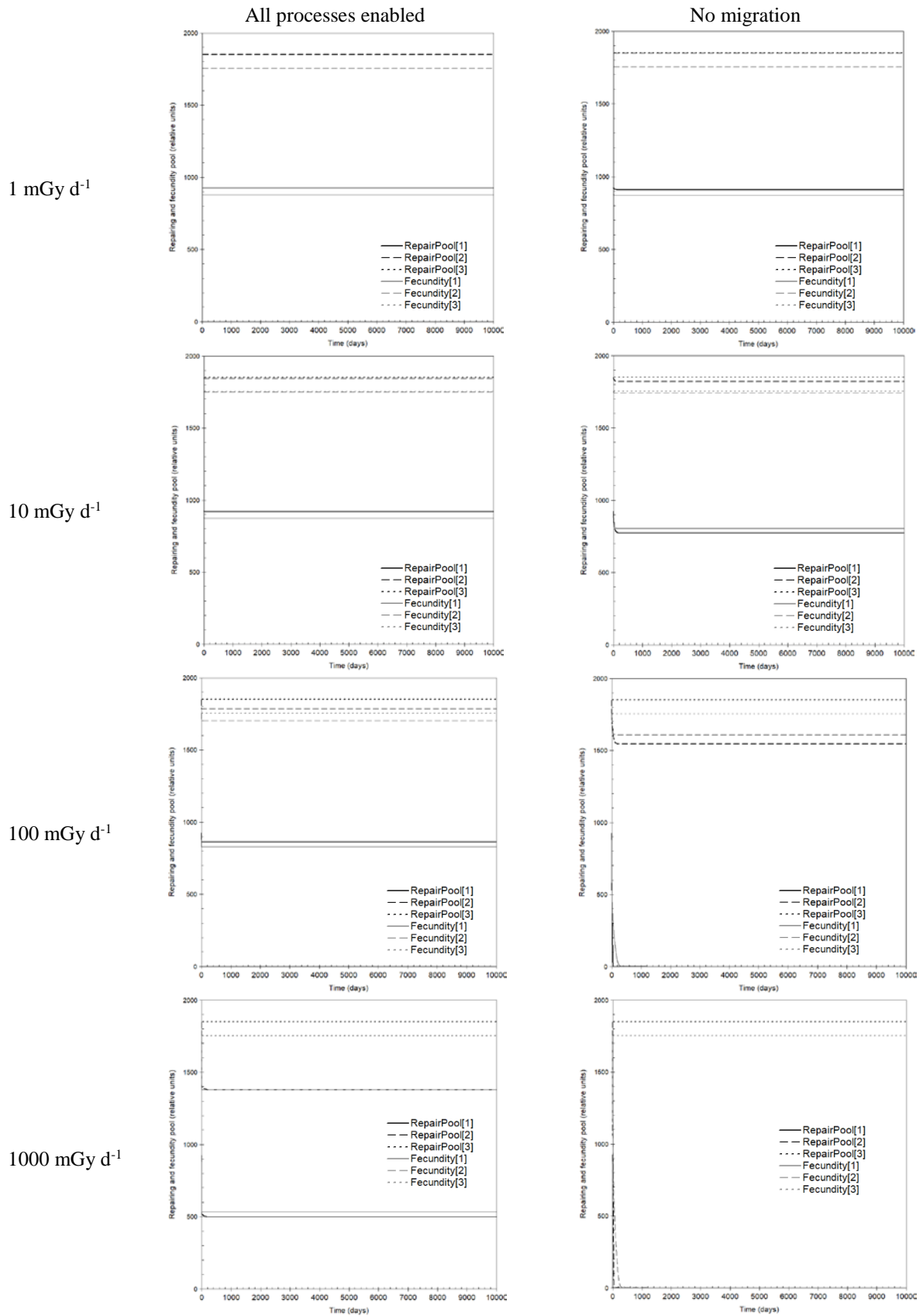


Figure 10: Model simulations of repairing pool and fecundity with varying levels of dose rate in Region 1, assumed constant in time

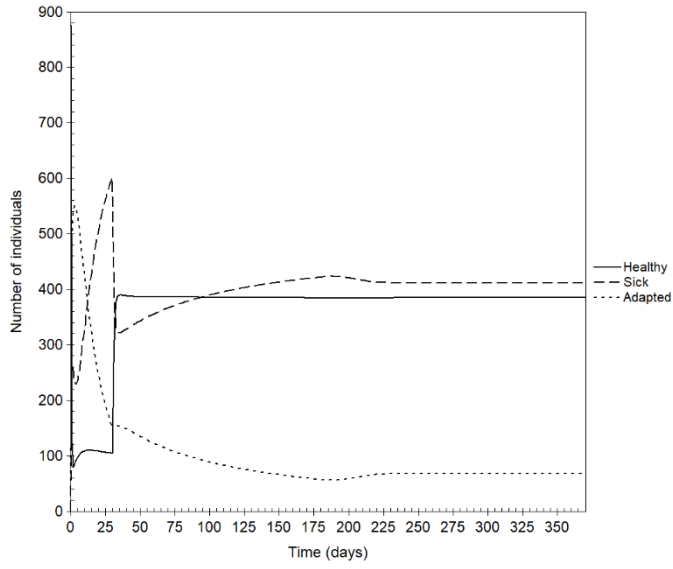


Figure 11: Simulation with dose rate step function of 25 Gy d^{-1} for $T < 30 \text{ d}$ and 10 Gy d^{-1} for $T \geq 30 \text{ d}$

Table 1: Parameter values for the model

Parameter	Description	Units	Value	Reference
<u>Radiobiological parameters</u>				
α	Radiation damage	Gy ⁻¹	0.11	Calculated from $LD_{50/30}$ of 6.2Gy for mouse (Sazykina and Kryshev, 2016)
α_r	Repairing pool damage	Gy ⁻¹	0.4	(Sazykina and Kryshev, 2016)
α_f	Fecundity pool damage	Gy ⁻¹	0.45	Based on dose for sterility > 4Gy (Sazykina and Kryshev, 2016)
ε	Lethality rate	day ⁻¹	0.015	(Sazykina and Kryshev, 2016) (from dose data for total lethality)
κ	Output of repairing process rate ($\kappa/\kappa_r < 1$)	day ⁻¹	0.2	Derived from metabolic rate (Sazykina and Kryshev, 2016)
κ_r	Non-lethal damages recovery	day ⁻¹	0.21	Derived from metabolic rate (Sazykina and Kryshev, 2016)
μ_r	Damaged individuals repair (repair pool auto-recovery rate constant)	day ⁻¹	0.032	From published information (Sazykina, 2018)
p_0	Coefficient for saturation function controlling adaptation probability	Unit-less	0.11	From published information (Wodarz et al., 2014)
p_1	Coefficient for saturation function controlling adaptation probability	Gy ⁻¹	0.023	From published information (Wodarz et al., 2014)
η	Conversion of adapted organisms to healthy	day ⁻¹	0.15	Model calibration
<u>Ecological parameters</u>				
L_v	Vole lifespan	day	180.5	From published information (MacDonald, 2001)
d	Death (combining natural death and predation)	day ⁻¹	0.0031	AnAge database (see (Sazykina and Kryshev, 2016))
r	Reproduction (fecundity pool auto-recovery)	day ⁻¹	0.06	From published information (Glorvigen, 2012)
ρ	Vole population density	m ⁻²	0.037	From published information (Aulak, 1973)
v	Vegetation damage	day ⁻¹	0.036	See Section 3.1.
σ	Vegetation recovery	day ⁻¹	0.0164	See Section 3.1.
μ_{ij}	Elements of the migration rate matrix	m ² d ⁻¹	3.7×10^5	See Section 3.2.