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Effects of contaminants and trophic cascade regulation on food chain stability: application to cadmium soil pollution on small mammals - raptor systems

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Abstract

Within the current context of global change, ecosystems face simultaneously multiple stressors such as environmental pollution, excessive enrichment, and additional disturbances affecting population dynamics. Numerous theoretical and empirical studies explored the transfer of contaminants by food ingestion between trophic levels, and how contaminant effects on survival and fecundity may change regime shifts of classical predator-prey dynamics. However, the extent to which those contaminants may influence the cascade effect of external stressors, as a change in resources (bottom-up cascade) or a variation in predator abundance (top-down cascade) is still poorly understood. We develop a data-driven model to explore how soil contamination modulate the food chain stability and resilience to changes in prey nutrient and in the apex predator mortality rate. We particularly focus on the ecotoxicological impact of the trace metal, cadmium, on a widespread raptor, the barn owl (*Tyto alba*), feeding on several prey distinct by their trophic positions: herbivores (*Microtus* spp), omnivores (wood mouse *Apodemus sylvaticus* and bank vole *Myodes glareolus*) and insectivores (shrews: *Crocidura* spp and *Sorex* spp). Our model reveals the alternative steady states in population dynamics and the occurrence and position of regime shifts where a subtle change in conditions causes a sudden shift in the ecological system. Based on mathematical modelling and bifurcation analysis, the results show for instance that under toxicity threshold, where no population decline is observed, the contaminant weakens food-chain resilience. Then at higher contamination, the toxic effects on predator releases the top-down control over prey that may increase. This range of chemical stress overturns the paradox of enrichment, a central concept in trophic cascade theory. The transition phase at the highest contamination, where the whole community collapse, exhibits multiple patterns, from smooth to abrupt, depending on external stressors and the prey population. Thus, this work provides a methodology to identify ecological traits of preys that are critical for transferring adverse effects of contaminants across the whole community.

1 Introduction

Across the globe, anthropogenic activities increase the input of contaminants in ecosystems, disturbing their regime and potentially threaten their health. One of the most widely observable impact is the lost of apex predators (Estes et al., 2011; Heath et al., 2014). As a consequence, there is a surge in attention concerning the effect of pollution at the food web scale. The response of a food-chain to external disturbances may be gradual (i.e., from a fixed state to another or to an oscillating pattern), or remains inert until a sudden regime shift, a critical transition, where the food-chain structure and composition are drastically altered (Scheffer, 2009). These sudden changes often correspond to abrupt degradation of ecosystem services which are difficult, if not impossible, to recover (Rockström et al., 2009). Due to their socio-economical importances, analysis of discontinuous transitions (i.e., regime shift) have been widely studied (Scheffer et al., 2015), but very few theoretical works have considered terrestrial food-chain impacted by the combination of

39 a chemical contaminant and external disturbances (i.e., trophic cascades). This lack of a mathe-
40 matical description on how polluted ecosystems respond to external stressors (e.g., eutrophication,
41 climate change, diseases) is due to the combination of many direct and indirect effects (Fleeger
42 et al., 2003; Rohr et al., 2006; Relyea and Hoverman, 2006; De Laender et al., 2015)

43 Direct effects of severe contaminant exposure as population declines (by increasing mortality or
44 reproductive failure) are possibly predictable from controlled laboratory toxicity tests. Those tests
45 are the classical approach to assess the potential risk of contaminants in ecosystems, and most
46 of them use one single species and one chemical compound, and quantify the toxicological effect
47 through several threshold of exposure concentration (i.e., NOAEL, LOAEL, LC₅₀) (Villeneuve
48 and Garcia-Reyero, 2011). However, there is a difficulty in predicting the ecological effects of
49 an environmental pollution from those laboratory results (Mineau, 2005; Villeneuve and Garcia-
50 Reyero, 2011).

51 Indeed, the exposure of vertebrate wildlife to environmental contaminants integrates complex
52 interactions between the routes of exposure (inhalation, ingestion, dermal absorption and maternal
53 transfer), the intensity (concentration in the vector agent and bioavailability), the frequency and
54 duration of the exposure, and the life history traits of exposed organisms (Smith et al., 2007). For
55 instance, among factors affecting metals body burden in small mammals, several field studies en-
56 lightened the critical role of the feeding behavior and the habitat use (Veltman et al., 2007b; Fritsch
57 et al., 2010a; Van Den Brink et al., 2011). In addition to this great variability in the exposure,
58 the fate of a contaminant within the organism (i.e., toxicokinetics) and its toxicological effect (i.e.,
59 toxicodynamics) differ greatly between and within species (Rainbow, 2002; Burger, 2008; Fritsch
60 et al., 2010a). This complexity in exposure and in the response of organisms induces a dispro-
61 portional impact on the constituent species of a community and therefore many indirect effects
62 (e.g., changes in the diversity of resources and/or consumers) (Rohr et al., 2006). For instance, a
63 non-sensitive species may still be affected when one of its resource, predator, mutualistic species
64 or competitor is affected by the contaminant (Fleeger et al., 2003; Clements and Rohr, 2009).

65 One of the most known type of propagation of indirect effects in food web is the “trophic
66 cascade” where the food chain is disturbed by either a change in the abundance of the highest
67 trophic level (i.e., top-down cascade) or by a change in the resources (i.e., bottom-up cascade)
68 (Carpenter et al., 1985; Heath et al., 2014). For instance, a top-down cascade initiated by a
69 decrease in predator abundance releases the pressure on its prey which consequently increase
70 in density and subsequently induce more pressure on prey resources. The reverse process of a
71 bottom-up cascade in a three-level food chain, may start by an increase/decrease of prey resources
72 favoring/constraining in first prey density and then its predator (Heath et al., 2014). Another
73 well-known bottom-up effect is the “paradox of enrichment” where the increase of prey resources
74 (i.e., the carrying capacity) has a destabilizing effect where population dynamics of prey and
75 predators shift from a fixed stable state to an oscillating behavior (Rosenzweig, 1971). However,
76 recent studies in aquatic systems showed that exposure to contaminants may inhibit the paradox

77 of enrichment by reducing oscillations and driving the system back to a fixed equilibrium (Prosnier
78 et al., 2015; Huang et al., 2015).

79 Many individual-based models for exposure of terrestrial wildlife to contaminants have been
80 proposed (Loos et al., 2010b; Schipper et al., 2012; Schmitt et al., 2015). Those highly detailed
81 models are very efficient for the management of a specific ecosystem (Schmitt et al., 2015; DeAngelis
82 and Grimm, 2014), but the wide variety of components and mechanisms induces a black box effect
83 hiding more or less the possibility of an analytical description of critical transitions (Gómez-Mourelo
84 and Ginovart, 2009). Therefore, in order to achieve a stability analysis of steady state(s) (i.e.,
85 namely a bifurcation analysis) (Kooi et al., 2008; Huang et al., 2015), we develop an ODE model
86 including the ecological dynamics of a two-level food chain with a soil compartment, small mammals
87 (rodents and shrews) as preys of the raptor barn owl (*Tyto alba*) contaminated by the trace metal
88 cadmium (Cd). Presence of Cd occurs in the Earth’s crust at relatively low concentration (0.1–0.5
89 ppm), and Cd is well-known to be highly toxic since non-biodegradable and because of its ability
90 to bind with many organic molecules that distribute it in all or part of the organism (Hopkin et
91 al., 2012). The main anthropogenic sources are non-ferrous metal mining and refining, application
92 of phosphate fertilizers and byproducts of battery manufacturer (Burger, 2008; Faroon et al., 2012).
93 Mammals are mainly contaminated through oral consumption of contaminated water and food
94 items (Smith et al., 2007). Moreover, there is a great variety of small mammal diet composition
95 with a gradient from herbivores to carnivores by including omnivores species which can induce
96 various patterns of exposure and responses to environmental contaminants within this mammal
97 group and shape the exposure of their predators. Similarly, while birds may be contaminated
98 by direct unintentional ingestion of the contaminant (human poisoning, chemically treated seeds,
99 polluted water), secondary poisoning through the consumption of contaminated food items is
100 more common (Smith et al., 2007). Due to their high trophic positions, their scavenging activities,
101 their large spatial living area and long lifespan over which to accumulate contaminants raptor
102 species are highly exposed to persistent and/or bioaccumulative contaminants (Gómez-Mourelo
103 and Ginovart, 2009; Bustnes et al., 2013; Espín et al., 2016).

104 Based on empirical scenarios and data, our aims are to provide a theoretical model on how
105 external stressors distributed in food chains by trophic cascades modulate the occurrence and
106 strength of regime shifts emerging in polluted ecosystems. We first explore different exposure sce-
107 narios of the predator according to trophic position of preys (herbivores, carnivores or omnivores).
108 Then, we analyze the occurrence and position of regime shifts in the predator-prey dynamics with
109 variations in Cd concentration in soils. Finally, we test the sensitivity of the system to external
110 disturbance inducing trophic cascade effects: variations in prey resources and in intrinsic predator
111 mortality rate.

2 Methods

2.1 Formulation of the general model

Contaminants are commonly measured in concentration per biomass of an individual, [$\mu\text{g}\cdot\text{g}^{-1}$] denoted ppm. In the terrestrial ecotoxicological system, we consider a soil with contaminant concentration denoted by C_s in [$\mu\text{g}\cdot\text{g}^{-1}$]. Cadmium being a persistent contaminant (neither degradation nor metabolization), we assumed C_s to be constant since the transfer of contaminant to prey species cannot significantly change the soil concentration (i.e., negligible over the considered period owing to the quantities in respectively the soil and biomass compartments, and included in a balanced uptake/release cycle), and no external inputs of Cd in soils can be expected since the main point source is no longer emitting cadmium. Also, in the system under consideration, several publications showed the same order of magnitude of soil contamination in samplings from 1995 to 2006 (while not exactly on the same locations) (Sterckeman et al., 2000; Sterckeman et al., 2002; Pruvot et al., 2006; Douay et al., 2009).

Then, the contaminant is transferred to a barn owl's preys (i.e., small mammal species), which are exposed to the environmental contaminant mainly through ingestion of contaminated foodstuffs (i.e., trophic transfer), and Cd accumulates in their tissues. We denote x the density of the prey species and B_x the mean biomass of an individual of that species. The notation C_x holds for the mean concentration of the contaminant in one individual, commonly called the body burden of the prey. The growth function of the prey population is a function $g_x(x, C_x)$ in [day^{-1}] depending on the population density x and the concentration of the contaminant C_x . The additional mortality due to pollution is a function $\mu_x(C_x)$ in [day^{-1}]. The dose-response curve $\mu_x(C_x)$ is defined by a log-normal cumulative distribution function as in Loos et al. (2010a) (see detailed parameterization hereafter, section 2.2.2 and Supporting Information). We denote $\kappa(C_s)$ the rate of transfer of the contaminant from soil to prey which follow a log-log linear regression (i.e., $\ln(C_x) = a + b \ln(C_s)$ defined from data, see section 2.2.1). The rate of excretion of the contaminant in the prey population is a constant denoted by $k_{out,x}$.

The highest level of the food chain is the predator with density z . The predator feeds on prey at a rate $\Phi(x)$, which is called the functional response. We assume that predator is only contaminated through this trophic interaction with an absorption coefficient η (i.e., bioavailability). The excretion rate of the contaminant by the predator is denoted $k_{out,z}$. Previous ecotoxicological model based on ODEs were considered in aquatic system, where every prey species, whatever their trophic level, are exposed to the contaminant present in the water compartment (Kooi et al., 2008; Bontje et al., 2009; Huang et al., 2015). In our terrestrial ecotoxicological system, we assume the predator not to be exposed directly to soil contaminant. We call $\psi(C_z)$ the stepwise linear function, equation (4), of reproduction efficiency of the predator depending on contaminant concentration within the predator denoted C_z , m the natural death rate of the predator, and $\mu_z(C_z)$ the linear function, equation (5), standing for the additional mortality rate due to the contaminant.

149 With all the previous assumptions, the model for the transfer of contaminant is given in
 150 Supporting Informations S1.1. Also, a classical growth function for rodents is the logistic type
 151 growth rate. We then assume that the contaminant affects the intrinsic growth rate: $g_x(x, C_x) =$
 152 $\pi(C_x)(1 - x/K)$, with $\pi(C_x)$ following a log-normal cumulative distribution function (Loos et
 153 al., 2010a) (see parameterization in section 2.2.2 and Supporting Information). The carrying ca-
 154 pacity K represents the resource of prey and is used as the parameter for bottom-up cascade in the
 155 bifurcation analysis. For top-down cascade effect, we used the natural mortality rate of the preda-
 156 tor, m . Prey-dependent functional response satisfies the properties: $\Phi(0) = 0$ and $\Phi'(x) > 0, x > 0$.
 157 Thus, it is straightforward to express the dynamics as:

$$\left\{ \begin{array}{l}
 \frac{dx}{dt} = \overbrace{x\pi(C_x)\left(1 - \frac{x}{K}\right)}^{\text{growth rate}} - \overbrace{\mu_x(C_x)x}^{\text{additional mortality}} - \overbrace{\Phi(x)z}^{\text{predation}} \\
 \frac{dC_x}{dt} = \overbrace{\kappa(C_s)}^{\text{exposure}} - C_x \left(\overbrace{k_{out,x}}^{\text{excretion}} + \overbrace{\pi(C_x)\left(1 - \frac{x}{K}\right)}^{\text{release rate}} \right) \\
 \frac{dz}{dt} = \overbrace{z\psi(C_z)\Phi(x)}^{\text{growth rate}} - \overbrace{z(\mu_z(C_z) + m)}^{\text{mortality rate}} \\
 \frac{dC_z}{dt} = \eta \overbrace{\frac{B_x}{B_z}\Phi(x)C_x}^{\text{intake rate}} - C_z \left(\overbrace{k_{out,z}}^{\text{excretion}} + \overbrace{\psi(C_z)\Phi(x)}^{\text{release rate}} \right)
 \end{array} \right. \quad (1)$$

158 2.2 Parameterization of transfers and effects of cadmium in a soil-small 159 mammals-raptor food chain

160 For parameterization, we consider the case of cadmium (Cd) transfer in a trophic chain from soil
 161 to small mammals and finally to a raptor feeding on these preys (see Fig. 1). Cadmium does
 162 not appear to have biological function in small mammals and birds, but is well-known to be one
 163 of the most mobile toxic metals (Cooke et al., 2011; Wayland et al., 2011). In this section, we
 164 parameterize model (1) to obtain five food chains corresponding to five different small mammal
 165 prey species (herbivores: *Microtus* spp ; omnivores: *Apodemus sylvaticus* and *Myodes glareolus* ;
 166 carnivores: *Sorex* spp and *Crocidura* spp) and one raptor predator, the barn owl (*Tyto alba*).

167 2.2.1 Prey exposure to the contaminant

168 When Cd is ingested through the diet, the absorption is around 1 to 5 % (Andersen et al., 1994;
 169 Schipper et al., 2008; Cooke et al., 2011). Many study use a fixed contaminant absorption efficiency
 170 at 2% which is also used in the present study (Schipper et al., 2008; Loos et al., 2010a). Exposure
 171 to cadmium is mainly chronic (a gradual accumulation from diet). The excretion rate of cadmium
 172 is long because of the binding to metallothionein (MT) (Fritsch et al., 2010a; Cooke et al., 2011).
 173 The half-lives of Cd is in the range of [100 – 300] days in rats (Cooke et al., 2011) what would give
 174 an excretion rate of: $-\ln(1/2)/[100 - 300] \approx [0.0023, 0.0069] \text{ day}^{-1}$. Most of the studies (Gorree

175 et al., 1995; Veltman et al., 2007a; Van Den Brink et al., 2010) use an excretion rate based on
 176 Friberg et al. (1986) which estimates the half-time for the whole body to be around 200 days,
 177 which gives an excretion rate of $k_{out,x} = 0.0035 \text{ day}^{-1}$.

178 To parameterize the function of transfer from soil to prey, denoted $\kappa(C_s)$, we use data collected
 179 from 2006 to 2010 in a 40 km^2 area surrounding the former smelter “Metaleurop-Nord”, north of
 180 France (see publications Fritsch et al. (2010b); Fritsch et al. (2011); Tête et al. (2013); Fritsch
 181 et al. (2010a)). Soil Cd level ranges from 0.1 ppm to 2402.1 ppm of dry matter based on more
 182 than 260 samples of composite 0 – 25 cm soil (Douay et al., 2009; Fritsch et al., 2010a). Small
 183 mammals were captured in sub-samples of 500×500 squares selected along a soil pollution gradient.
 184 Small mammals were identified at the species level using morphometric, skull and teeth criteria.
 185 Total body concentration in the different small mammal species were calculated from kidney and
 186 liver concentrations measured in the animals Veltman et al. (2007a). From those data, we define
 187 the function $\kappa(C_s)$ by a log-log linear regression (i.e., $\ln(C_x) = a + b \ln(C_s)$) as previously used
 188 (Veltman et al., 2007a; Fritsch et al., 2010a) (see Table S1 in Supplementary Information and
 189 Fig. 2.A).

190 2.2.2 Toxicity effect of Cd on reproduction and mortality of prey

191 The effect of Cd on the small mammals organism are, among others, a reduction of food and water
 192 intake, growth depression, renal dysfunction, hypertension, anemia and cancer (Cooke et al., 2011).
 193 According to Świergosz-Kowalewska (2001), the reproductive organs are more sensitive than other
 194 organs and a low doses may affect spermatogenesis and ovulation. In the report of US-EPA (2005),
 195 the NOAEL for reproduction and growth is a diet with a concentration of $1.86 \mu\text{g.g}^{-1} \cdot \text{day}^{-1}$, that
 196 is $10.63 \mu\text{g.g}^{-1}$ in the whole body. Using data and methodology of the same report, the LOAEL
 197 (Lowest Observed Adverse Effect Level) for reproduction is estimated at $6.27 \mu\text{g.g}^{-1} \cdot \text{day}^{-1}$, that
 198 is $35.83 \mu\text{g.g}^{-1}$ which is in the range estimated previously from Shore and Douben (1994). Again,
 199 with the data provided by US-EPA (2005) we obtain a NOAEL (Non Observed Adverse Effect
 200 Level) for mortality of $6.72 \mu\text{g.g}^{-1} \cdot \text{day}^{-1}$ (i.e., $38.4 \mu\text{g.g}^{-1}$), and from the same report, a LOAEL
 201 of $9.72 \mu\text{g.g}^{-1} \cdot \text{day}^{-1}$ (i.e., $55.54 \mu\text{g.g}^{-1}$).

202 We derive dose-response curves using log-normal law (Loos et al., 2010a) defined by:

$$\pi(C_x) = r \left(1 - \frac{1}{2} \left(1 + \operatorname{erf} \left[\frac{\ln(C_x) - \mu_r}{\sigma_r \sqrt{2}} \right] \right) \right) \quad (2)$$

203 where the function $\operatorname{erf}()$ is the Gauss error function defined as: $\operatorname{erf}(x) = \frac{2}{\sqrt{\pi}} \int_0^x e^{-t^2} dt$.

204 For parameterization, we use the equivalence $\text{NOAEL} = EC_{10}$ and $\text{LOAEL} = EC_{30}$ (Greenberg
 205 et al., 2014), and then a least square regression to estimate parameters of the log-normal curve.
 206 For reproduction, we estimate $EC_{50} = 82.91 \mu\text{g.g}^{-1}$, the parameter of the log-normal curve, equa-
 207 tion (3) are $\mu_r = 4.418$ and $\sigma_r = 1.601$ (see Fig. 2.A in Supporting Information). Combination
 208 of soil-prey contamination (i.e., $\kappa(C_s)$) described in Table S1 in Supporting Information) with the

209 dose-response function (i.e., $\pi(C_x)$) gives the probability of prey to reproduce in function of con-
 210 centration in soil (see curves plotted in Fig. 2.B in Supporting Information). The equation of the
 211 dose-response curve for prey reproduction is given by the cumulative distribution function of a
 212 log-normal law: The reproduction rate of small mammals are well-known to be high. From Quéré
 213 and Le Louarn (2011), we estimate the maximal growth rate of preys r around $[1/365 - 25/365]$
 214 per day, and fixed it at $r = 20/365$ for every prey species.

215 For mortality, we infer on a log-normal cumulative function, equation (3), with $\mu_{m_x} = 4.272$
 216 and $\sigma_{m_x} = 0.486$ (see Fig. 2.A in Supporting Information).

$$\mu_x(C_x) = m_x \frac{1}{2} \left(1 + \operatorname{erf} \left[\frac{\ln(C_x) - \mu_{m_x}}{\sigma_{m_x} \sqrt{2}} \right] \right) \quad (3)$$

217 The combination of soil-prey transfer with dose-response curve for mortality, $\mu_x(C_x)$ gives the
 218 probability of prey mortality as a function of concentration in soil (see curves plotted in Fig. 2.B
 219 in Supporting Information when assuming the maximum of mortality rate due to Cd at $m_x = 1$).
 220 Compared to rodents, the insectivores shrews have often highest exposure to cadmium but they are
 221 able to tolerate high concentrations of Cd in their kidney and liver without adverse effect (Cooke
 222 et al., 2011), what is confirmed with the data of US-EPA (2005) where shrews have highest NOAEL
 223 and/or LOAEL for growth and survival than other species. However the set of data was too small
 224 to give specifics NOAEL and LOAEL for shrews. Therefore, we gave different maximum rate of
 225 mortality due to contamination: $m_x = 2/365$ for rodents (*Microtus*, *Apodemus* and *Myodes*), and
 226 $m_x = 0.5/365$ for shrews (*Crocidura* and *Sorex*).

227 2.2.3 Exposure of raptors to cadmium

228 As for small mammals, birds accumulate Cd mainly through their diet (Wayland et al., 2011;
 229 Burger, 2008). While there are some works on avian toxicity of Cd, few concern raptors. In the
 230 review of US-EPA (2005), the species are mainly herbivores and insectivores. From these species,
 231 US-EPA (2005) estimates the NOAEL for reproduction and growth at $1.47 \mu\text{g}.g^{-1}.\text{day}^{-1}$ and the
 232 LOAEL at $6.35 \mu\text{g}.g^{-1}.\text{day}^{-1}$. For mortality, the NOAEL is estimated at $10.33 \mu\text{g}.g^{-1}.\text{day}^{-1}$ and
 233 the LOAEL around $22.97 \mu\text{g}.g^{-1}.\text{day}^{-1}$.

234 Also, from Gorree et al. (1995) (on kestrel and barn owl) we assume an excretion rate for bird
 235 $k_{out,z} = 0.0035 \text{ day}^{-1}$. From Wayland et al. (2011), the absorption rate, denoted η in the model, is
 236 in the range of $[0.01 - 0.07]$, that we fixed at 2% (Schipper et al., 2008; Loos et al., 2010a). Therefore,
 237 to estimate the whole body concentration, we use the same equation as for small mammals, and
 238 we obtain for reproduction: NOAEL= $8.4 \mu\text{g}.g^{-1}$ and LOAEL= $36.28 \mu\text{g}.g^{-1}$, and for mortality,
 239 we have: NOAEL= $59.03 \mu\text{g}.g^{-1}$ and LOAEL= $131.26 \mu\text{g}.g^{-1}$.

2.2.4 Toxicity effect of Cd on reproduction and mortality in raptors

The natural mortality rate of the barn owl, denoted m , is based on the usual life span which is about 4 years, so $m = 1/(365 \times 4)$ in day^{-1} . For barn owl dynamics, the bifurcation analysis constraints the use of a log-normal distribution for the energy conversion and the predator's mortality rate. As a consequence, for the function of energy conversion in predator, we therefore use the following stepwise linear function:

$$\psi(C_z) = \varepsilon \max\{\alpha C_z + 1, 0\} \quad (4)$$

We estimate α in function of NOAEL and LOAEL. Using a classical linear regression model, we get $\alpha = -0.008$, and therefore, reproduction is not possible for the threshold concentration $C_z > T_R = 118.287 \mu\text{g.g}^{-1}$, where T_R is the threshold concentration such as $\alpha T_R + 1 = 0$. We set the conversion efficiency to $\varepsilon = 0.001$. Indeed, if we assume that 4 to 8 prey are eaten every day (around 70 to 100 grams of prey see Table S1 in Supplementary Information), we obtain a reproduction rate of $0.001 \times [4 - 10] \times 365 = [1.46 - 3.65]$ on an annual basis.

252

For mortality function, we compute a linear regression based on NOAEL and LOAEL giving:

$$\mu_z(C_z) = m_z \gamma C_z, \quad \text{with } \gamma = 0.002 \quad (5)$$

3 Results

3.1 Bifurcation diagram on soil concentration

A classical summary of the long-term behavior of populations dynamics is with bifurcation diagram. Fig. 3 represents the steady points of the prey populations for Cd concentration in soil ranging from 0.4 to 2402.1 ppm with carrying capacity fixed at $K = 1121.8$ (see also Fig. S2.A in Supplementary Information), and the natural mortality rate of predator at $m = 10/(4 \times 365)$. When external changes occur, here Cd concentration in soil, C_s , ecosystems changes can be in a continuous way or exhibit a sudden discontinuity. On a certain domain of parameters, ecosystems may have several stable points (i.e., bistability for 2 stable points) where stochastic disturbances may drive the system toward a different steady state.

For prey densities (see Fig. 3), we observe three situations: at low concentration, depending on initial condition, prey tends to a prey-only equilibrium E_1 (predator is extirpated), a prey-predator coexistence equilibrium E_* (with limit cycle) or a no-species steady point E_0 . Prey-only steady point starts at the carrying capacity and decreases faster and faster with the increase in C_s . For the prey-predator coexistence, prey point starts lower and increases slightly with the increase of soil contamination. This point of coexistence is unstable but with a limit cycle, and the amplitude of the limit cycle decreases until the Hopf-bifurcation point, denoted HB. Simultaneously, predator population is either extirpated or decreases slightly (see Fig. 3.A-B). Again, predator oscillates

272 around the coexistence point decreasing with the increase in contaminant. At the coexistence
 273 point, oscillation stops at HB but prey still increases and predator decreases until the transcritical
 274 bifurcation (TB) at concentration $C_{s,2}$ where predator are extirpated and only prey-only steady
 275 point is feasible. Finally, prey is also extirpated by a TB at concentration $C_{s,1}$ (see Fig. 3.C)
 276 where no species can survive. For some range of parameter values, we may observe a homoclinic
 277 bifurcation when the periodic orbit collides with the prey-only saddle steady point.

278 To better understand the resilience of the system, the “marble-in-a-cup” diagram in Fig. 3.C
 279 represents the stability landscape where valleys are stable zone and top of hills are unstable points.
 280 In this graph, the concentration of Hopf bifurcation, $C_{s,H}$, and transcritical bifurcation $C_{s,1}$ and
 281 $C_{s,2}$ are represented.

282 3.2 Cascade effects with different small mammals species

283 In order to study at the bottom-up and top-down cascade effects on the steady points for the five
 284 groups of small mammals, we analyse and compute 2D bifurcation digrams in Fig. 4 and Fig. 5.
 285 Fig. 4 represents bottom-up cascade effect by an increase in prey resources parameterized with the
 286 carrying capacity K ranging from 0 to 5000. The predator mortality rate is fixed at $m = 1/(4 \times 365)$.
 287 Fig. 5 represents top-down cascade effect by an increase in the predator mortality rates, m , ranging
 288 from 0 to $20/(4 \times 365)$. The carrying capacity is fixed at $K = 2500$. In both Fig. 4 and Fig. 5, panels
 289 A are the null models where the concentration in the prey is the concentration is the soil $C_x = C_s$
 290 with a maximal intrinsic prey mortality rate fixed at $m_x = 2/365$. For a better distinction of
 291 bifurcation zones, for each panel of Fig. 4 and Fig. 5 the y-axes (i.e., carrying capacity or predator
 292 death rate) are square-root scaled and the x-axis (i.e., concentration in soil) are log-transformed
 293 in the range 0.4 to 2402.1 ppm as for data representation in Fig. 2.A.

294 3.2.1 Bottom-up cascade effects

295 The null model of Fig. 4 shows that at a very low carrying capacity, the increase in contaminant
 296 changes the system from prey-only to no-species steady point through a transcritical bifurcation.
 297 The resources of preys are sufficient to maintain the prey population but without predation. In
 298 this situation, there is no limit cycle since the predator is extirpated. Limit cycle can only appears
 299 with coexistence. When the carrying capacity increases, coexistence is possible (prey-only and no-
 300 species are unstable points). Then, prey-only equilibrium is stable, and the predator is extirpated.
 301 Finally, after ≈ 80 ppm, no-species can survive, the soil contamination is too high. The two changes
 302 in steady states stability are called transcritical bifurcations (green lines). When the carrying
 303 capacity increases, the coexistence point becomes unstable with a limit cycle. This represents
 304 the famous “paradox of enrichment” showed by Rosenzweig (1971). However, the increase of
 305 contaminant stabilizes the system since the oscillation of the coexistence points disappears at
 306 the Hopf bifurcation (red lines). And then, as previsouly, the system undergoes two transcritical
 307 bifurcations. The fourth dynamic is the one described previously in Fig. 3 with bistability of

308 coexistence and prey-only steady point. When carrying capacity is low, the ecosystem changes
309 are continuous between steady points. But at high carrying capacity bistability occurs, where
310 discontinuous changes happen at the tipping points represented by fold bifurcations (blue lines).
311 For other panels of Fig. 4, for every species, the increase in K has a destabilizing effect. At low
312 carrying capacity, there is no limit cycle, and a limit cycle appears in every chain when K increases.
313 Also, the increase in Cd has a stabilizing effect since we observe a change from oscillations to stable
314 coexistence, prey-only steady point and finally extinction of species. At high carrying capacity,
315 we observe a zone of alternative stable (or limit cycle) states of coexistence and prey-only. This
316 illustrates that the enrichment in prey resources favors the occurrence of regime shifts.

317 **3.2.2 Top-down cascade effects**

318 In Fig. 5, we observe that m has a stabilizing effect. At low predator mortality, every system
319 exhibits oscillation when soil concentration is low. The oscillations disappears with the increase
320 in m . As for bottom-up bifurcation diagrams (Fig. 4), the increase in Cd in soil has a stabilizing
321 effect with the decline of oscillations until their disappearances. When the predator mortality
322 rate is high, the transition from coexistence to prey-only are smoother. Under this condition,
323 the predator fitness is weak and a slight increase in contaminant reducing the fitness drives the
324 predator to extinction. Then at intermediate predator mortality rate, there are tipping points due
325 to bistable zone (see Fig. 5.A-B,F). Finally, at low predator mortality rate, there is no transition
326 from coexistence to prey-only but directly from coexistence to no-species. This last pattern means
327 that the predator survival is not driven by the contaminant but by the presence of prey.

328 **3.2.3 Predator and prey extirpation**

329 The transcritical bifurcation from prey-only to no-species happens at the threshold value $C_{s,1}$
330 where the mortality rate of a prey population is greater than its reproduction rate leading to
331 the extinction of prey. Since the prey is extincted, predator cannot survive. For the null model
332 where concentration in prey is the same that the Cd concentration in the soil $C_x = C_s$, the
333 threshold is $C_{s,Null Model,1} \approx 74.1$ ppm. For the herbivores *Microtus* spp, $C_{s,Mi,1} \approx 79.5$ ppm.
334 This transcritical bifurcation for omnivores and for *Sorex* spp is not in the range considered for
335 Cd soil concentration [0.4 – 2402.1] ppm. For *Crocidura* spp $C_{s,Cr,1} \approx 62.9$ ppm.

336 **4 Discussion**

337 This work provides a set of conditions on stressors (e.g., environmental pollution, excessive enrich-
338 ment, predator depletion) that may introduce regime shifts in small mammals-raptor dynamics
339 taking place in metal-polluted ecosystems. Along the soil contaminant concentration gradient,
340 the food chain exhibits four types of outcomes: (i) predator-prey coexistence, (ii) prey-only, (iii)
341 population extinction and (iv) a bistable state where a coexistence point and a prey-only steady

342 point form two stable valleys separated by an unstable coexistence area. This last case means that
343 a slight change in populations densities, due to stochastic variations, may lead the system to a
344 totally different state (i.e., from coexisting predator-prey state to a prey-only state where predators
345 are extirpated). Also, we observed oscillations when prey and predator coexist even in a bistable
346 context. These results provide a conceptual framework, with its limitations discussed hereinafter,
347 to modelize and analyze the effect of chemical pollution in the classical trophic cascade theory, in
348 term of stability (i.e., resilience and oscillations) and strength of transition between states (i.e.,
349 smooth transition or abrupt change due to tipping point). For environmental risk management,
350 predicting alternative stable states and system sensitivity to external variations is essential to
351 anticipate or prevent abrupt changes in ecosystem (Scheffer, 2009).

352 **4.1 Gradient of soil contamination**

353 Within the range of low contamination of soil, the observation of population densities reveals no
354 impact of Cd pollution at the ecosystem level, but the bifurcation diagrams shows that when the
355 contamination increases, the resilience of the system decreases as the system moves closer to prey-
356 only or no-species stable states. Also, when the Cd concentration increases, there is an increase in
357 prey densities simultaneous with a decrease in predator population. The reason is that the decrease
358 in predator due to the contaminant releases the pressure on prey population. The prey population
359 is also impacted by the contaminant but the balance between the release of predation and the stress
360 of contamination is beneficial for the prey. Then, at a higher level of contaminant concentration
361 in the soil, predator population goes extinct. This leads the system to a prey-only steady point
362 where the contaminant concentration is the only external factor affecting prey survival until its
363 extinction. If prey is more sensitive and/or more exposed to the contaminant than the predator,
364 then, a transition from coexisting predator-prey steady state to a total extinction without the
365 transition at a prey-only equilibrium may occur.

366 **4.2 Combined effects of trophic cascades and contamination**

367 The transition from predator-prey coexistence to prey-only state is different according to bottom-
368 up and top-down pressures: it may change smoothly, strongly around a threshold concentration or
369 exhibits tipping points with two alternative stable states.

370 Several theoretical and empirical studies predicted that enrichment (i.e., increase in resources)
371 leads to oscillation of population dynamics, what is known as the “paradox of enrichment” (Rosen-
372 zweig, 1971; Fussmann et al., 2000; Roy and Chattopadhyay, 2007b). The system we consider
373 shows also an increase in oscillation amplitude with the increase in resource enrichment (i.e.,
374 carrying capacity of prey). But we also show that the increase in soil contamination overturns
375 oscillating dynamics. Indeed, the transfer of contaminant in the food-chain, affecting the repro-
376 duction and mortality of the species involved a stabilizing effect. In the same way, considering a
377 one predator-two preys model, where one prey is toxic while the other is palatable, Roy and Chat-

378 topadhyay (2007a) showed that the introduction of a toxic prey reduces oscillations. In aquatic
379 ecosystem, Kooi et al. (2008); Prosnier et al. (2015); Huang et al. (2015) made the same ob-
380 servations for respectively persistent toxicant-bacterium-ciliate, copper-*Scenedesmus-Daphnia* and
381 methylmercury-aquatic insects-rainbow trout systems. These models, showing that contaminant
382 may contribute to reduce oscillations in the community dynamics, concern chronic exposure to con-
383 taminant in bi-trophic food chain. However, the theoretical work of Garay-Narvez et al. (2013)
384 showed that more complex systems (i.e., with a greater species richness) are destabilized in terms
385 of reduction of food-web connectance, by the pulsed introduction of a contaminant into the envi-
386 ronment. Therefore, further works are necessary to understand the different effects between pulsed
387 and chronic exposure, from low to high species richness including variations in both structure and
388 composition of the food web.

389 We show that a decrease in predator mortality rate, that is a decrease in top-down regulation,
390 has also a destabilizing effect on the predator-prey dynamics. This result means that when top-
391 down pressure effect is high (i.e., high intrinsic predator mortality rate), the predator survival is
392 very sensitive to contaminant variation until a point where contaminant concentration drives the
393 predator population toward extinction. While with a low intrinsic mortality rate of the predator,
394 it is the density of prey that drives predator to extinction, and no more the contaminant. Also,
395 similarly to the paradox of enrichment, at low top-down regulation, contaminant has a stabilizing
396 effect. To our knowledge, the observation of this pattern is new and could be important for envi-
397 ronmental management. Indeed, if empirical studies confirm our prediction, we could argue that
398 to prevent the extinction of apex predators, when the ecosystem contamination is high, manager
399 should focus on reducing exposure to contamination first, and then, when contaminant level is
400 lower, care has to be taken on prey population. Our model also shows that the strength of steady
401 state transitions increases with the decrease of the top-down regulation. Again, this illustrates
402 that predator survival is more dependent on prey density and less on its intrinsic mortality rate.

403 **4.3 Trophic level of prey**

404 In this study, we consider five groups of prey pooled according to their diet (i.e., herbivores,
405 omnivores and insectivores). We compare these five models to a null model using a null-prey
406 having the same body burden as the concentration in the soil. From field data, the five groups can
407 be clearly distinguished by their responses along the pollution gradient in terms of bioaccumulation.
408 At low concentration of the contaminant in soil, the herbivores (i.e. *Microtus* spp) are the least
409 contaminated, and the insectivores (i.e., shrews) are the most contaminated. Cd body burden
410 in the omnivores are in between. This ranking follows previous studies (Veltman et al., 2007a;
411 Fritsch et al., 2010a) suggesting a biomagnification of Cd in food chain, where the species at the
412 highest trophic level is the most contaminated. However, when Cd increases in soil, responses of
413 the various groups are different: *Microtus* spp are the most sensitive, with a positive response
414 which is accentuated with an increase in soil contamination. As a consequence, for bottom-up and

415 top-down cascades, the bifurcation diagram of *Microtus* spp is similar with the null model. This
416 similarity is due to the elasticity of exposure close to one (1.56, see Table S1 in Supplementary
417 Information), meaning that the Cd body burden in *Microtus* spp varies with the similar amplitude
418 that soil concentration. Omnivores rodents, *A. sylvaticus* and *M. glareolus*, are the least sensitive
419 to soil contamination since they have also a positive response, but this response diminishes with
420 the increase of Cd in soil. Therefore, at the food-chain level, omnivores preys have a buffer effect.
421 Those results reflects previous observations that the body burden of Cd is more related to diet
422 preferences for mobile species as *A. sylvaticus* rather than soil concentration, and is the opposite
423 for a nonmobile species as *Microtus* spp (Van Den Brink et al., 2011). Finally, as expected,
424 insectivore have the highest level of contamination but the response to contaminant level variation
425 are not the same between the two groups. While *Sorex* spp present a high Cd body burden at low
426 concentration in soil, field data did not exhibit an increase of Cd body burden along the pollution
427 gradient. So, as for omnivores, this buffers the impact of contamination when its level increases.
428 For the other insectivores, *Crocidura* spp, the response to contaminant level variations is similar to
429 that of *Microtus* spp species, but the body burden is higher. Therefore, *Crocidura* spp are the most
430 risky species to consume for a predator. This knowledge about critical prey species is important to
431 assess the exposure of apex predators to environmental contaminants (Schipper et al., 2008; Loos
432 et al., 2010a). Also, the position of raptor at the top of food webs informs on the bioavailability of
433 a wide range of contaminants and on the exposure of species at lower trophic levels and is therefore
434 an important challenge for environmental risk assessment (Bustnes et al., 2013).

435 **4.4 Limitations and perspectives**

436 Our model explore the changes in the balance between the necessity for a predator to feed on
437 prey and the damaging consequences of predation in terms of exposure to contaminants. This
438 modeling of prey-predator relationships under a panel of pollution stress scenarios allows not
439 only predicting long-term direct and indirect effects of contaminants on populations, but also,
440 it shows how extinctions may occur within communities free-living in polluted sites. The model
441 reveals the occurrences and the positions of bifurcation points where a small change in parameters
442 (i.e., soil contaminant concentration, prey carrying capacity, predator mortality rate) induces a
443 sudden change in the food chain dynamics. The analysis of these bifurcations helps to identify
444 which prey ecological traits are critical for propagating adverse effects across trophic levels, how
445 a whole community is sensitive to contaminant. It also provides inputs for concepts about how
446 contaminants modulate trophic cascades, and further how multiple stressors can shape population
447 dynamics and tipping points in ecosystem functioning.

448 However, in this simplified model, the complexity of trophic interactions is reduced to only
449 one prey-one predator system even if different functional responses have been considered. Yet, a
450 variation in the diet of a species is likely to change the exposure of that species to contaminants.
451 The direct next step is therefore the inclusion of multi-prey predation to explore the influence of

452 the adaptive foraging behavior of the predator (Baudrot et al., 2016) on similar ecotoxicological
453 systems, and the role of biodiversity in prey community on shaping the responses of food-webs to
454 environmental disturbances such as environmental pollution. Among many complexification of the
455 models, since collection of data have been thought into a landscape approach, we suggest to take
456 this spatial heterogeneity of habitats into account (Fritsch et al., 2012).

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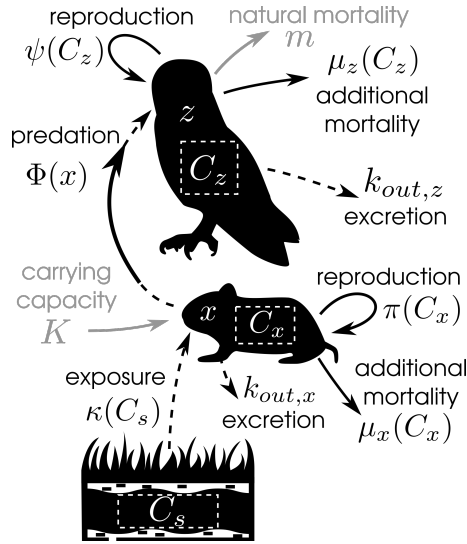


Figure 1: Flow diagram of the transmission of a contaminant along a trophic chain. The plain line is the trophic interaction, dashed lines are transfers of the contaminant and dotted lines are population intrinsic dynamics. Grey elements are bottom-up and top-down regulation parameterized with respectively the carrying capacity K and the predator mortality rate m .

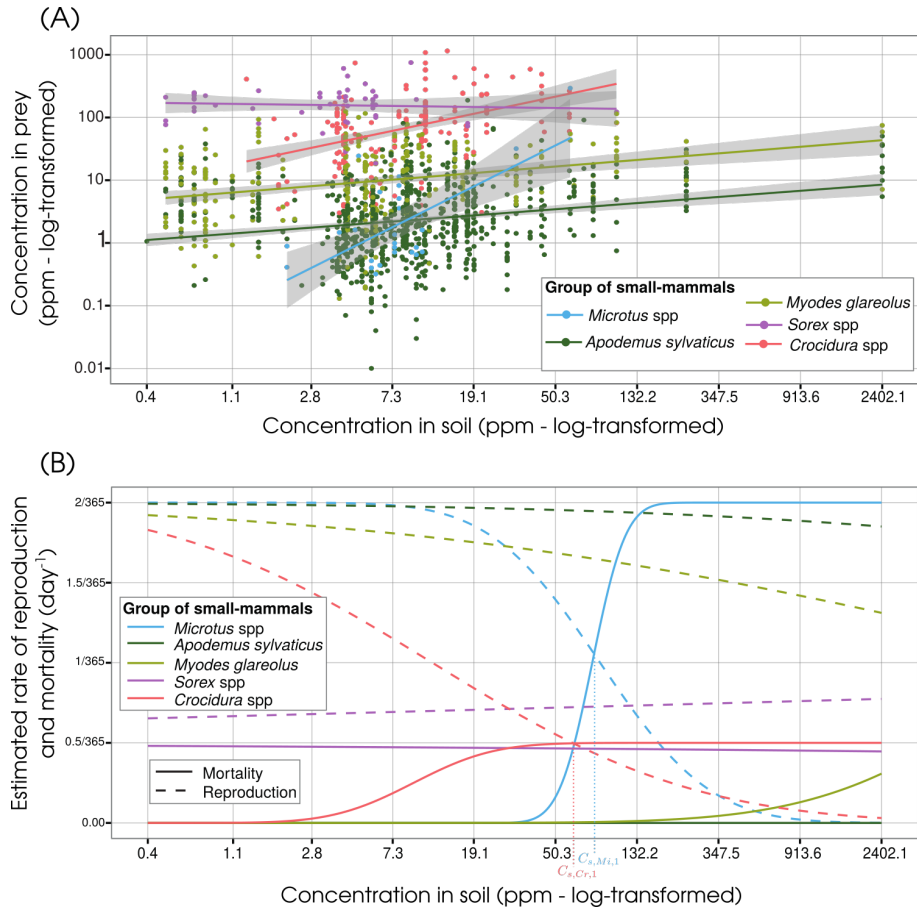


Figure 2: (A) Log-log linear model with 95% credible interval from data from Metaleurop. Whole body concentration in fresh weight and concentration in soil (dry weight). See equation in Table S1 in Supplementary Information. (B) Rate of reproduction with $r = 2/365 \text{ day}^{-1}$, and rate of mortality with $m_{Mi,Ap,My} = 2/365$ (maximal reproduction rate r) and $m_{Cr,So} = 0.5/365$ (1/4 of maximal reproduction rate to have reproduction greater than mortality at $C_s = 0$ for *Sorex* spp).

Symbols	Definitions	Values [References]
$\pi(C_x)$	Contaminant effect on prey reproduction	equation (2)
r	Maximal reproduction rate of prey	2/365 [a]
$\mu_x(C_x)$	Mortality in prey due to the contaminant	equation (3)
m_x	Maximal prey mortality due to contaminant	2/365 rodents, 0.5/365 shrews
K	Carrying capacity in prey	[0 – 5000]
$\kappa(C_s)$	Function of prey exposure	data in Table S1 in Sup. Inf. [b]
$k_{out,x}$	Excretion rate of contaminant in prey	0.0035 [c]
B_x	Mean biomass of prey individual	data in Table S1 in Sup. Inf.
$\Phi(x)$	Ingestion rate of prey: Holling III	$a = 0.003$ and $h = 0.3$
$\psi(C_z)$	Contaminant effect on predator reproduction	see text
ε	Maximal predator reproduction efficiency	0.001
$\mu_z(C_z)$	Contaminant effect on predator mortality	1/100
η	Percentage of absorption of ingested Cd	0.02 [d]
$k_{out,z}$	Excretion rate of contaminant in predator	0.0035 [c]
m	Natural mortality rate of predator	1/(4 × 365)
B_z	Mean biomass of individual predator	350 grams

Table 1: Definition of parameters and their values. References are [a] Quéré and Le Louarn (2011); [b] Fritsch et al. (2010a) ; [c] Friberg et al. (1986) ; [d] Schipper et al. (2008)

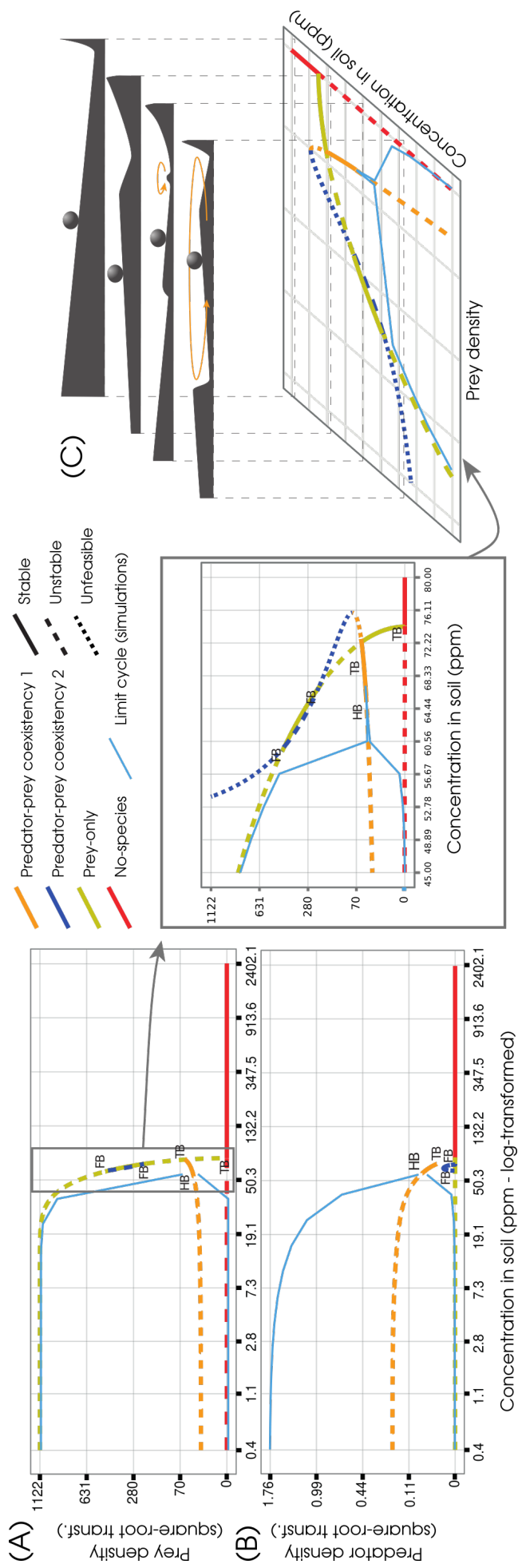


Figure 3: 1D-Bifurcation diagram illustrating smoothly changes and tipping points in the null model (prey Cd-concentration is equal to soil Cd-concentration $C_x = C_s$). TB: transcritical bifurcation. HB: Hopf Bifurcation. FB: Fold bifurcation. (A-B) Bifurcation diagram of predator and prey densities with respect to contaminant level in soil C_s . (C) "Marble-in-a-cup" representation of bifurcation diagram in the range [45 – 80] ppm. The marble rolls towards the bottom of valleys (stable steady points) moving away from top of hills (unstable steady points).

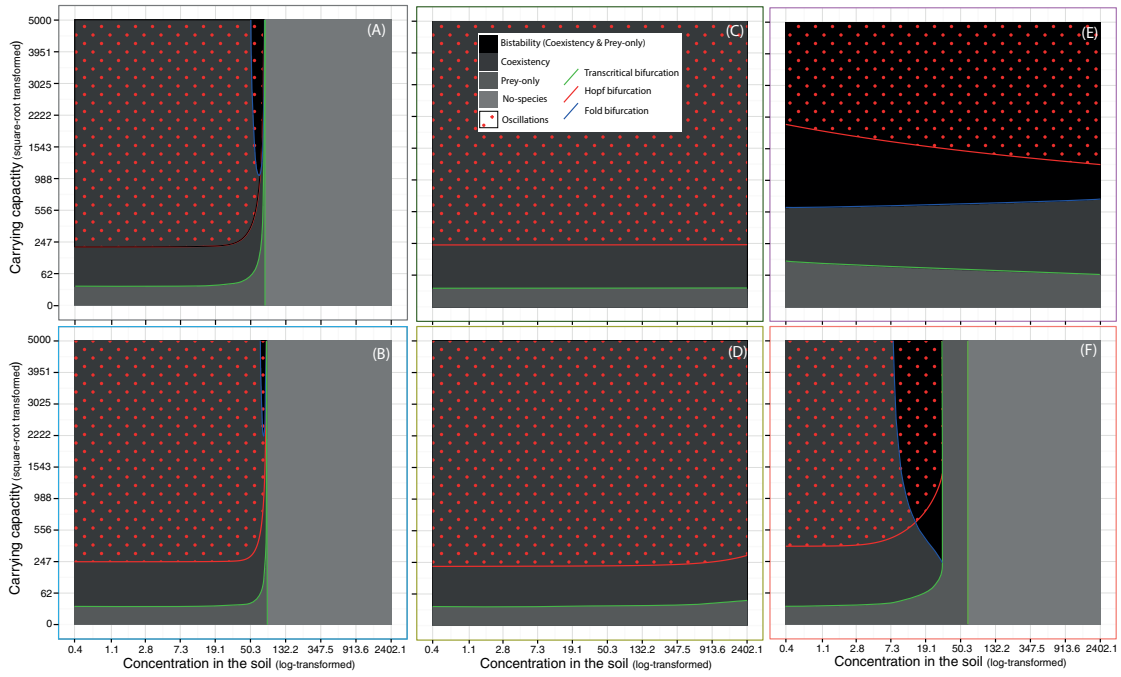


Figure 4: 2D-Bifurcation diagram of bottom-up forcing for the five preys. (A) Null model (B) *Microtus* spp, (C) *Apodemus sylvaticus*, (D) *Myodes glareolus*, (E) *Sorex* spp and (F) *Crocidura* spp.

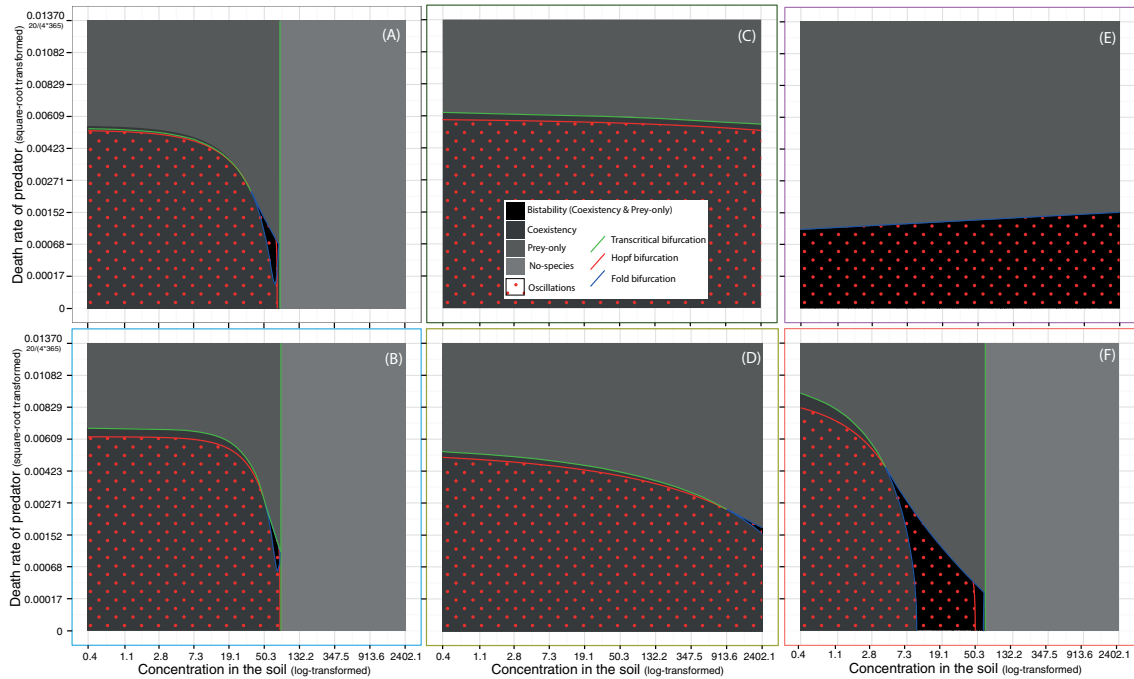


Figure 5: 2D-Bifurcation diagram of top-down forcing for the five preys. (A) Null model (B) *Microtus* spp, (C) *Apodemus sylvaticus*, (D) *Myodes glareolus*, (E) *Sorex* spp and (F) *Crocidura* spp.

632 S1 Supporting Information

633 Supporting Information provides all mathematical developments done in the present study.

634 S1.1 General model

With all the assumptions made in the manuscript (see also description of notations in the manuscript), especially the assumption that individuals of a same population have the same biomass and the same amount of contaminant in their bodies, the model for the transfer of a contaminant is given by:

$$\left\{ \begin{array}{l} \frac{dB_{Tx}}{dt} = B_{Tx}g_x(x, C_x) - \mu_x(C_x)B_{Tx} - B_x\Phi(x)z \quad (S1a) \\ \frac{dw_x}{dt} = \kappa(C_s)B_{Tx} - k_{out,x}w_x - w_x\mu_x(C_x) - C_xB_x\Phi(x)z + \gamma_xw_xg_x(x, C_x) \quad (S1b) \\ \frac{dB_{Tz}}{dt} = B_{Tz}(\psi(C_z)\Phi(x) - m - \mu_z(C_z)) \quad (S1c) \\ \frac{dw_z}{dt} = -k_{out,z}w_z - w_z(m + \mu_z(C_z)) + \eta C_xB_x\Phi(x)z + \gamma_zw_z\psi(C_z)\Phi(x) \quad (S1d) \\ w_x = C_xB_{Tx} \quad \text{and} \quad C_z = w_z/B_{Tz} \quad (S1e) \\ x = B_{Tx}/B_x \quad \text{and} \quad z = B_{Tz}/B_z \quad (S1f) \end{array} \right.$$

635

636 In the general model (S1), the equations (S1a) and (S1c) present the dynamic of the total
637 biomass of prey and the predator respectively. The equations (S1b) and (S1d) stand for the dy-
638 namics of contaminant concentrations within individuals for respectively the prey and the predator,
639 where the death of individuals (prey and predator) implies a decrease of amounts of the contam-
640 inant, respectively $-w_x\mu_x(C_x)$ and $-w_z(m + \mu_z(C_z))$. The transfer of the contaminant through
641 the predation is express by the term $C_xB_x\Phi(x)z$, which is removed in prey (S1b), and added in
642 the predator (with a factor of transfer efficiency η) (S1d).

643 While we could consider the population dynamic in term of biomasses, it is more convenient
644 to use density of population as suggested by the change of variable presented in equations (S1f)
645 which give directly the following equalities:

$$\frac{dx}{dt} = \frac{1}{B_x} \frac{dB_{Tx}}{dt} \quad \text{and} \quad \frac{dz}{dt} = \frac{1}{B_z} \frac{dB_{Tz}}{dt} \quad (S2)$$

646 S1.2 Dimensional transformation of variables

647 From the change of variables in equations (S1e), it is straightforward to give the contaminant
648 dynamics for the prey:

$$\begin{aligned}
\frac{dC_x}{dt} &= \frac{dw_x/B_{Tx}}{dt} = \frac{1}{B_{Tx}^2} \left(\frac{dw_x}{dt} B_{Tx} - w_x \frac{dB_{Tx}}{dt} \right) \\
&= \frac{1}{B_{Tx}^2} (B_{Tx}(\kappa(C_s)B_{Tx} + w_x(w_x\gamma_x g_x(x, C_x) - k_{out,x} - \\
&\quad \mu_x(C_x)) - C_x\Phi(x)z) - w_z B_{Tz} (\psi(C_z)\Phi(x) - m - \mu_z(C_z))) \\
&= \kappa(C_s) - k_{out,x}C_x - C_x\mu_x(C_x) - \frac{C_x B_x}{B_{Tx}} \Phi(x)z - \\
&\quad C_x g_x(x, C_x) + C_x\mu_x(C_x) + \frac{C_x B_x}{B_{Tx}} \Phi(x)z \\
&= \kappa(C_s) - C_x (k_{out,x} + g_x(x, C_x))
\end{aligned} \tag{S3}$$

649 and for the predator:

$$\begin{aligned}
\frac{dC_z}{dt} &= \frac{1}{B_{Tz}^2} \left(\frac{dw_z}{dt} B_{Tz} - w_z \frac{dB_{Tz}}{dt} \right) \\
&= \frac{1}{B_{Tz}^2} (B_{Tz}(-k_{out,z}w_z + w_z(\psi(C_z)\Phi(x)\gamma_z - m - \mu_z(C_z)) + \\
&\quad \eta C_x B_x \Phi(x)z) - w_z B_{Tz} (\psi(C_z)\Phi(x) - m - \mu_z(C_z))) \\
&= -k_{out,z}C_z - C_z(m + \mu_z(C_z)) + \eta C_x \Phi(x) \frac{B_x}{B_z} - \\
&\quad C_z (\psi(C_z)\Phi(x) - m - \mu_z(C_z)) \\
&= \eta C_x \Phi(x) \frac{B_x}{B_z} - C_z(k_{out,z} + \psi(C_z)\Phi(x))
\end{aligned} \tag{S4}$$

650 S1.3 Reduction of system using slow-fast dynamics

651 With ξ the low parameter, system (1) becomes:

$$\left\{ \begin{array}{l} \frac{dx}{dt} = x \left(\pi(C_x) \left(1 - \frac{x}{K} \right) - \mu_x(C_x) \right) - \Phi(x)z \\ \xi \frac{dC_x}{dt} = \kappa(C_s) - C_x \left(k_{out,x} + \xi \pi(C_x) \left(1 - \frac{x}{K} \right) \right) \\ \frac{dz}{dt} = z (\psi(C_z)\Phi(x) - \mu_z(C_z) - m) \\ \xi \frac{dC_z}{dt} = \eta \frac{B_x}{B_z} \Phi(x) C_x - C_z (k_{out,z} + \xi \psi(C_z)\Phi(x)) \end{array} \right. \tag{S5}$$

652 and letting $\xi \rightarrow 0$, we obtain:

$$\Rightarrow \left\{ \begin{array}{l} \frac{dx}{dt} = x \left(\pi(C_x) \left(1 - \frac{x}{K} \right) - \mu_x(C_x) \right) - \Phi(x)z \\ C_x = \frac{\kappa(C_s)}{k_{out,x}} \\ \frac{dz}{dt} = z (\psi(C_z)\Phi(x) - \mu_z(C_z) - m) \\ C_z = \frac{\eta B_x}{k_{out,z} B_z} \Phi(x) C_x \end{array} \right. \tag{S6}$$

653 **S1.4 Elasticity of the regression**

654 Note that we have:

$$\ln(C_x) = b \ln(C_s) + a \quad \Leftrightarrow \quad C_x = e^a C_s^b \quad \Leftrightarrow \quad \frac{\partial C_x}{\partial C_s} = e^a b C_s^{b-1} \quad \Leftrightarrow \quad b = \frac{C_s}{C_x} \times \frac{\partial C_x}{\partial C_s} \quad (S7)$$

655 Therefore, the parameter b is the elasticity of our model, that is a measure of the proportional
656 change in C_x in response to a proportional change in C_s .

657 **Biomagnification:** The reduction formulation of system (S9) gives the following coefficient of
658 biomagnification (BMF):

$$BMF = \frac{\eta B_x}{k_{out,z} B_z} \Phi(x) = \frac{0.02 \times [70 - 100]}{0.0035 \times 350} = [1.14 - 1.63], \quad \text{for 70 to 100 grams ingested daily} \quad (S8)$$

659 **S1.5 Parameterization for probability of reproduction and mortality**

Species	Mean individual biomass B_x (Std. Error)	Function of $\ln(C_x)$: Estimate (Std. Error)
<i>Microtus</i> spp	16.33 (1.03)	$1.56 (0.33) \times \ln(C_s) - 2.52 (0.72)$
<i>Apodemus sylvaticus</i>	17.28 (0.13)	$0.23 (0.03) \times \ln(C_s) + 0.32 (0.08)$
<i>Myodes glareolus</i>	19.29 (0.27)	$0.25 (0.04) \times \ln(C_s) + 1.83 (0.10)$
<i>Sorex</i> spp	7.06 (0.25)	$-0.03 (0.09) \times \ln(C_s) + 5.11 (0.14)$
<i>Crocidura</i> spp	9.81 (0.12)	$0.65 (0.10) \times \ln(C_s) + 2.82 (0.23)$

Table S1: Log-log linear model of internal concentration in small mammals depending on soil concentrations (see Figure 2). In the log-log linear regression, $\ln(C_x) = a + b \ln(C_s)$, a is the intercept and b is the elasticity of the model (see Supporting Informations S1.4), which is a measure of proportional change in prey concentration, C_x , in response to a proportional change in soil concentration C_s .

660 **S1.6 Description of a 2D bifurcation diagram**

661 **S1.7 Mathematical analysis**

662 The ecotoxicological model (1) is a 4-dimensional system resulting from the coupling of contaminant
663 and population dynamics. This complexity involving a great number of variables and parameters
664 challenges the mathematical stability analysis. We therefore take advantage of the two time scales
665 included in the system, to reduce the model (1) to a 2-dimensional system where fast events are
666 neglected (Auger and De La Parra, 2000). In our context, we consider the common assumption
667 (Kooi et al., 2008; Huang et al., 2015) that the contaminant dynamics are faster than the dynamic
668 of population growth. This assumption means that the time scale of population growth and the
669 efficiency to convert ingested prey into new-born are much slower than the intake and elimination of
670 contaminants and which approaches a quasi-steady state. Due to this slow-fast dynamics process,
671 we assume a constant concentration rate in populations (Kooi et al., 2008; Huang et al., 2015).

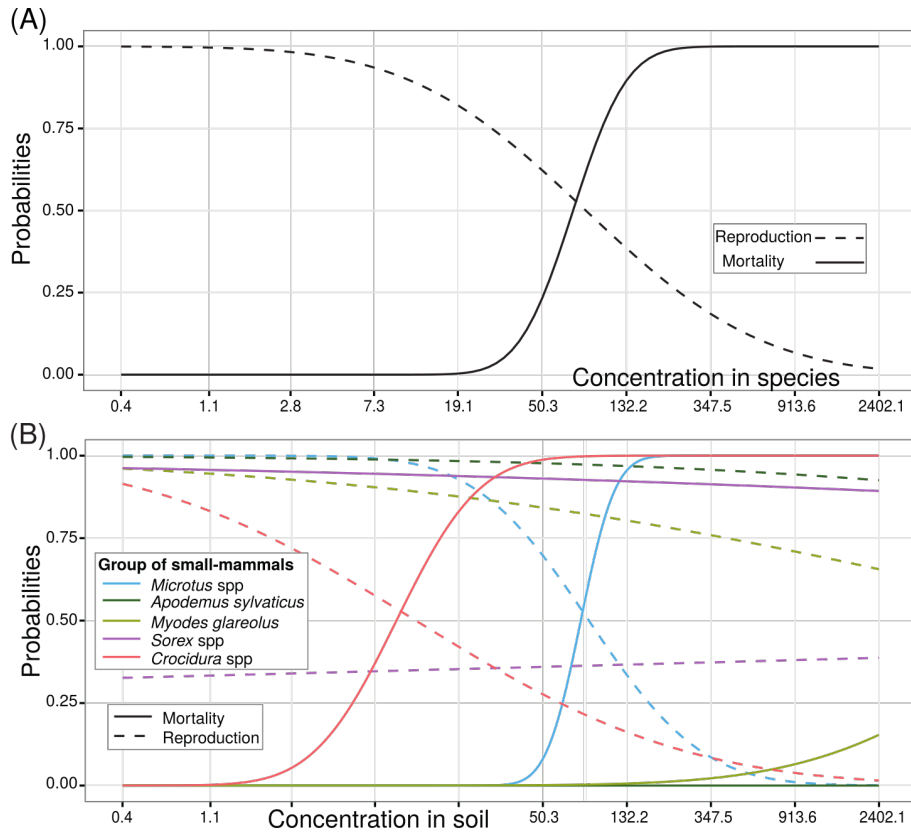


Figure S1: (A) Log-normal probabilities of reproduction and mortality for prey depending on the concentration in the species individuals. (B) Log-normal probabilities of reproduction and mortality depending on the concentration in the soil. We use same parametrization than in graph A.

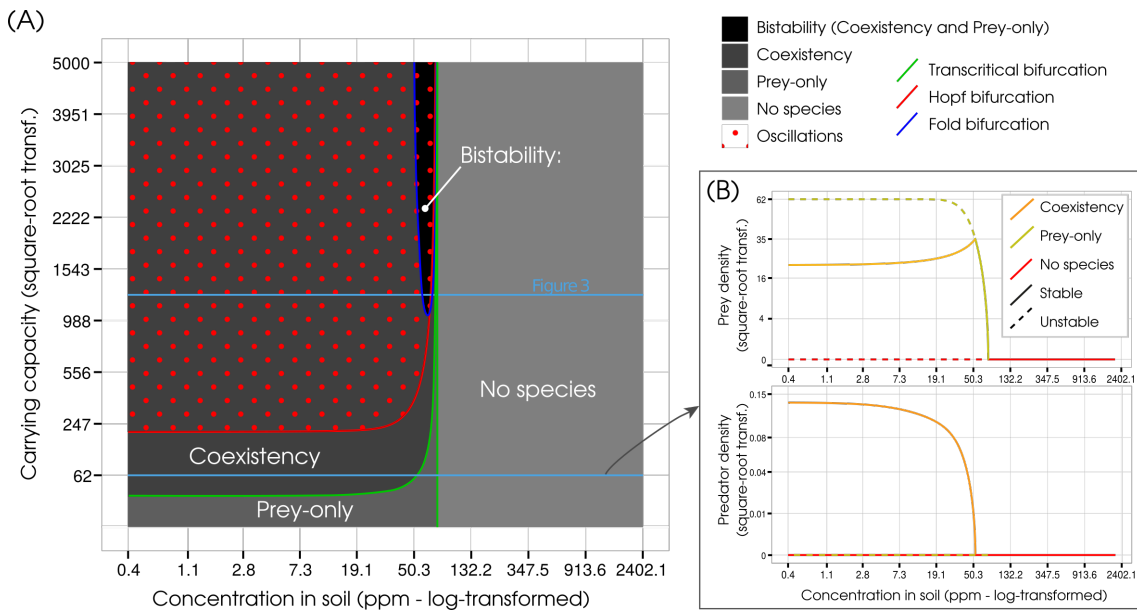


Figure S2: (A) 2D-Bifurcation diagram illustrating smoothly changes and tipping points in the null model (prey concentration is equal to soil concentration $C_x = C_s$). This diagram results from 500 1D-bifurcation diagram along a range of carrying capacity $K \in [0, 5000]$. (B) 1D-Bifurcation diagram showing a continuous change in ecosystem when the concentration in the soil increase.

672 Therefore, with ξ the low parameter, and letting $\xi \rightarrow 0$, system (1) becomes (see appendix S1.3
673 for details):

$$\left\{ \begin{array}{l} \frac{dx}{dt} = \overbrace{x\pi(\overline{C}_x)\left(1 - \frac{x}{K}\right)}^{\text{growth rate}} - \overbrace{\mu_x(\overline{C}_x)x}^{\text{additional mortality}} - \overbrace{\Phi(x)z}^{\text{predation}} \\ \frac{dz}{dt} = \overbrace{z\psi(\overline{C}_z)\Phi(x)}^{\text{growth rate}} - \overbrace{z(\mu_z(\overline{C}_z) + m)}^{\text{mortality rate}} \\ \overline{C}_x = \frac{\text{intake rate}}{\text{excretion}} = \frac{\kappa(C_s)}{k_{out,x}} \\ \overline{C}_z = \frac{\text{intake rate}}{\text{excretion}} = \frac{\eta B_x}{k_{out,z} B_z} \Phi(x) \overline{C}_x \end{array} \right. \quad (\text{S9})$$

674 Cadmium excretion is very low compared to other pollutant ($k_{out,\{x,z\}} = 0.0035$). Also, from
675 the parameterization done in the previous section (see Table 1), we have $\pi(C_x) \leq r = 2/365 \approx$
676 0.0055 and $\psi(C_z) \leq \varepsilon = 0.001$, and since reproduction rates are decreasing with the increase in
677 contaminant, there is a range of contamination where the slow-fast process is relevant. Also, the
678 prey reproduction is reduced with the saturating part of the logistic growth rate, $(1 - x/K)$.

679 S1.8 Steady states and their stability

680 The contamination of prey population and the transmission of contaminating substance through
681 the predation is described in model (S9). It is important to mention here that the properties of π ,
682 ψ , μ_x and μ_z can not be prescribed beforehand since it varies from case to case. From now and
683 onwards, we use the notation $\overline{\pi}(C_s)$, $\overline{\mu}_x(C_s)$, $\overline{\psi}(C_s, x)$ and $\overline{\mu}_z(C_s, x)$ instead of respectively $\pi(\overline{C}_x)$,
684 $\mu_x(\overline{C}_x)$, $\psi(\overline{C}_z)$ and $\mu_z(\overline{C}_z)$. Hence, the change in the system dynamics can be studied with C_s as
685 bifurcation parameter (see part S1.1):

$$\left\{ \begin{array}{l} \frac{dx}{dt} = x \left(\overline{\pi}(C_s) \left(1 - \frac{x}{K} \right) - \overline{\mu}_x(C_s) \right) - \Phi(x)z \\ \frac{dz}{dt} = z \left(\overline{\psi}(C_s, x) \Phi(x) - \overline{\mu}_z(C_s, x) - m \right) \end{array} \right. \quad (\text{S10})$$

686 S1.8.1 Existence of steady states

Above system possesses two axial equilibrium points, $E_0(0,0)$ and $E_1\left(K\left(1 - \frac{\overline{\mu}_x(C_s)}{\overline{\pi}(C_s)}\right), 0\right)$. The
second one is feasible if $\overline{\mu}_x(C_s) < \overline{\pi}(C_s)$. At the critical magnitude $C_s = C_{s,1}$, system undergoes
a transcritical bifurcation and the threshold of this bifurcation is given by:

$$\overline{\mu}_x(C_{s,1}) = \overline{\pi}(C_{s,1}) \quad (\text{S11})$$

687 Graphical representation of $C_{s,1}$ is given by Fig. 2.B. An example of the bifurcation behavior
688 of the prey-only steady point $(x_1, 0)$ is given by the bifurcation diagram Fig. 3.

689 We denote $E_*(x_*, z_*)$ the non-trivial steady point where prey and predator coexist. The interior
690 equilibrium point E_* bifurcates from the axial equilibrium point E_1 at the bifurcation threshold

691 $C_s = C_{s,2}$ where $C_{s,2}$ is a positive root of the equation $K\left(1 - \frac{\overline{\mu}_x(C_s)}{\overline{\pi}(C_s)}\right) = x_*$, that is when
692 $x_1 = x_*$ (see appendix S1.10 for details).

Steady points	Conditions of existence and stability
$E_0 = (0, 0)$	Always feasible. Stable when $C_s > C_{s,1}$, that is when only E_0 is feasible. Saddle for $C_s < C_{s,1}$, and transcritical bifurcation with E_1 at $C_{s,1}$.
$E_1 = (x_1, 0)$	Feasible for $C_s < C_{s,1}$. Unstable when E_* is feasible, that is for $C_s < C_{s,2}$. Stable for $C_{s,2} < C_s < C_{s,1}$.
$E_* = (x_*, z_*)$	Feasible for $C_s < C_{s,2}$. See text.

Table S2: Steady points with conditions of existence and stability.

693 S1.8.2 Stability of the steady states

694 The analysis of stability of the steady points E_0 , E_1 and E_* is totally given in the appendix S1.11.
695 Here, we describe the mains results.

696 From the Jacobian matrix for the system (S10) around the trivial equilibrium point E_0 , one
697 eigenvalue is always negative and E_0 is stable if $\overline{\pi}(C_s) < \overline{\mu}_x(C_s)$ and is a saddle-point if $\overline{\pi}(C_s) >$
698 $\overline{\mu}_x(C_s)$. The first eigenvalue becomes zero when $C_s = C_{s,1}$ and the model under consideration
699 undergoes first transcritical bifurcation. The transversality condition has to be satisfied, that is
700 the real part of the eigenvalues have to be non-zero.

701 For the second steady point E_1 where only the predator is extirpated, we know this point
702 is feasible when $\overline{\mu}_x(C_s) < \overline{\pi}(C_s)$ and hence one eigenvalue is always negative. Then, using the
703 condition of feasibility of E_* , the point E_1 is locally asymptotically stable whenever E_* is not
704 feasible, that is when $C_s > C_{s,2}$. Conversely, E_1 is a saddle-point when E_* is feasible.

705 For the coexisting point E_* , since the determinant of the Jacobian matrix of system (S10),
706 denoted J_* , is positive at this point, the stability of E_* is determined by the trace of the matrix as
707 whenever E_* is feasible. Consequently, E_* is stable if $Tr(J_*) < 0$, is unstable if $Tr(J_*) > 0$ and
708 undergoes Hopf-bifurcation if $Tr(J_*) = 0$. We call $C_{s,H}$ the Hopf-bifurcation threshold. With
709 Holling type II functional response, the sign of $Tr(J_*)$ is the same as:

$$-2ahx_*\overline{\pi}(C_s) + ahK(\overline{\pi}(C_s) - \overline{\mu}_x(C_s)) - \overline{\pi}(C_s) \quad (\text{S12})$$

710 S1.9 Explicit formulation of threshold concentration for predator-prey 711 coexistence

712 For the predator dynamics, substituting equations (4) and (5) into system (S10), we obtain:

$$\frac{dz}{dt} = z \left(\varepsilon \alpha \frac{\eta B_x}{k_{out,z} B_z} \Phi(x)^2 C_x + \varepsilon \Phi(x) - m_z \gamma \frac{\eta B_x}{k_{out,z} B_z} \Phi(x) C_x - m \right) \quad (\text{S13})$$

713 Then, as demonstrated in appendix S1.12, E_* has two solutions for $C_s < C_{s,3}$, denoted $E_{*,a}$
714 and $E_{*,b}$, one solution for $C_s = C_{s,3}$ and no solution otherwise. The threshold contamination $C_{s,3}$
715 is defined by:

$$C_{s,3} = e^{-a/b} \times \left[\frac{k_{out,z} B_z \varepsilon}{\eta B_x (m_z \gamma)^2} \left(m_z \gamma - 2\alpha m \pm 2\sqrt{\alpha m (\alpha m - m_z \gamma)} \right) \right]^{1/b} \quad (\text{S14})$$

716 If $C_s > C_{s,3}$, there is no equilibrium. If $C_s \leq C_{s,3}$, then there is two (or one if equal) possible
 717 steady points E_* appearing at $C_{s,3}$. If $C_{s,3} > C_{s,2}$ then the two points E_* appears when crossing
 718 the point E_1 at different level of contamination (case of the Fig. 3). When $C_{s,3} \leq C_{s,2}$, the point
 719 coexisting E_* appears at $C_{s,3}$ and then is divided into the two possible points.

720 However, the last condition required for the feasibility of $E_{*,\{a,b\}}$ is that $x_{i,*,\{a,b\}} < x_1$. There-
 721 fore, under the previous necessary conditions for coexistence, there are three possible situations: (1)
 722 If $x_{i,*,\{a,b\}} < x_1$, then there is two coexistence points (i.e., $E_{*,a}$ and $E_{*,b}$). (2) If $x_{i,*,a} < x_1 < x_{i,*,b}$,
 723 then there is only one coexistence point (i.e., $E_{*,a}$ or $E_{*,b}$). (3) Finally when $x_{i,*,\{a,b\}} > x_1$, then
 724 there is no coexistence point.

725 S1.10 The coexisting point E_*

726 Since $E_*(x_*, z_*)$ is the coexisting equilibrium point, then x_* is the root of:

$$\bar{\psi}(C_s, x_*) \Phi(x_*) - \bar{\mu}_z(C_s, x_*) - m = 0 \quad (\text{S15})$$

727 and z_* is:

$$z_* = \frac{x_*}{\Phi(x_*)} \left(\bar{\pi}(C_s) \left(1 - \frac{x_*}{K} \right) - \bar{\mu}_x(C_s) \right) \quad (\text{S16})$$

728 Therefore, if x_* is a positive root of (S15), then $z_* > 0$ is positive when $x_* < K \left(1 - \frac{\bar{\mu}_x(C_s)}{\bar{\pi}(C_s)} \right)$.

729 Feasibility condition for E_* is given by

$$0 < x_* < K \left(1 - \frac{\bar{\mu}_x(C_s)}{\bar{\pi}(C_s)} \right) := x_1 \quad \text{with} \quad \bar{\mu}_x(C_x) < \bar{\pi}(C_s) \quad (\text{S17})$$

730 The interior equilibrium point E_* bifurcates from the axial equilibrium point E_1 at the bifur-
 731 cation threshold $C_s = C_{s,2}$ where $C_{s,2}$ is a positive root of the equation $K \left(1 - \frac{\bar{\mu}_x(C_s)}{\bar{\pi}(C_s)} \right) = x_*$,
 732 that is when $x_1 = x_*$. Note also that $x_* < x_1$ when $C_s < C_{s,2}$, otherwise, $x_* > x_1$ but in this
 733 situation E_* is no more feasible. One requires $x_* > 0$ and functions $\bar{\pi}(C_s)$ is a decreasing function
 734 and $\bar{\mu}_x(C_s)$ is an increasing function. Therefore, we have $C_{s,2} < C_{s,1}$. Now we can find the local
 735 asymptotic stability conditions for various equilibrium of the system (S10).

736 S1.11 Stability of equilibria

737 **Stability conditions for point $E_0 = (0, 0)$**

738

739 First we calculate the Jacobian matrix for the system (S10) around the trivial equilibrium point

740 E_0 and find:

$$J_0 = \begin{bmatrix} \bar{\pi}(C_s) - \bar{\mu}_x(C_s) & 0 \\ 0 & -m \end{bmatrix} \quad (\text{S18})$$

741 One eigenvalue is always negative and E_0 is stable if $\bar{\pi}(C_s) < \bar{\mu}_x(C_s)$ and is a saddle-point if
 742 $\bar{\pi}(C_s) > \bar{\mu}_x(C_s)$. The first eigenvalue becomes zero when $C_s = C_{s,1}$ and the model under consid-
 743 eration undergoes first transcritical bifurcation. The transversality condition has to be satisfied,
 744 that is the real part of the eigenvalues have to be non-zero.

745 **Stability conditions for point $E_1 = (x_1, 0)$**

746

747 Now we evaluate the Jacobian matrix for the system (S10) around E_1 to obtain:

$$J_1 = \begin{bmatrix} -\bar{\pi}(C_s) + \bar{\mu}_x(C_s) & -\Phi(x_1) = -\Phi\left(K\left(1 - \frac{\bar{\mu}_x(C_s)}{\bar{\pi}(C_s)}\right)\right) \\ 0 & \bar{\psi}(C_s, x_1)\Phi(x_1) - \bar{\mu}_z(C_s, x_1) - m \end{bmatrix} \quad (\text{S19})$$

748 The eigenvalues of this matrix are given by the diagonal elements of the above matrix. For feasible
 749 existence of E_1 , we need $\bar{\mu}_x(C_s) < \bar{\pi}(C_s)$ and hence the first eigenvalue is negative.

750 Next we know that E_* is not feasible for $x_* > K\left(1 - \frac{\bar{\mu}_x(C_s)}{\bar{\pi}(C_s)}\right)$. Then using the relation
 751 that x_* is the positive root of the equation $\bar{\psi}(C_s, x_*)\Phi(x_*) - \bar{\mu}_z(C_s, x_*) - m = 0$, then we have
 752 $x_* = \Phi^{-1}\left(\frac{\bar{\mu}_z(C_s, x_*) + d}{\bar{\psi}(C_s, x_*)}\right)$, and we can derive that E_* is not feasible for:

$$\Phi^{-1}\left(\frac{\bar{\mu}_z(C_s, x_*) + d}{\bar{\psi}(C_s, x_*)}\right) > K\left(1 - \frac{\bar{\mu}_x(C_s)}{\bar{\pi}(C_s)}\right) \quad (\text{S20})$$

753 Since $\Phi(\cdot)$ is an increasing function and $\bar{\psi}(\cdot) > 0$, the last inequality is equivalent to:

$$\bar{\mu}_z(C_s, x_*) + m > \bar{\psi}(C_s, x_*) \times \Phi\left(K\left(1 - \frac{\bar{\mu}_x(C_s)}{\bar{\pi}(C_s)}\right)\right) \quad (\text{S21})$$

754 And therefore, we can derive that $\bar{\psi}(C_s, x_*)\Phi(x_*) - \bar{\mu}_z(C_s, x_*) - m < 0$ whenever E_* is not feasible.

755 Hence E_1 is locally asymptotically stable whenever E_* is not feasible, when $C_s > C_{s,2}$.

756 If the condition $\bar{\psi}(C_s, x_1)\Phi(x_1) = \bar{\mu}_z(C_s, x_*) + m$ is satisfied then one eigenvalue of J_1 is
 757 zero and E_1 loses stability through another Transcritical bifurcation. Proceeding with the similar
 758 argument we can prove that E_1 is a saddle-point if E_* is feasible.

759 **Stability conditions for point $E_* = (x_*, z_*)$**

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761 Finally we consider the local asymptotic stability of the coexisting equilibrium point E_* . Eval-
 762 uating the Jacobian matrix around E_* we find:

$$J_* = \begin{bmatrix} \bar{\pi}(C_s) - \frac{2\bar{\pi}(C_s)x_*}{K} - \bar{\mu}_x(C_s) - z_* \frac{\partial\Phi(x)}{\partial x} \Big|_{x=x_*} & -\Phi(x_*) \\ z_* \left(-\frac{\partial\bar{\mu}_z(C_s, x)}{\partial x} \Big|_{x=x_*} + \bar{\psi}(C_s, x_*) \times \frac{\partial\Phi(x)}{\partial x} \Big|_{x=x_*} + \Phi(x_*) \frac{\partial\bar{\psi}(C_s, x)}{\partial x} \Big|_{x=x_*} \right) & 0 \end{bmatrix} \quad (\text{S22})$$

763 Since the determinant is positive (i.e., $Det(J_*) > 0$), the stability of E_* is determined by the
764 trace of the matrix J_* as whenever E_* is feasible. Consequently, E_* is stable if $Tr(J_*) < 0$,
765 is unstable if $Tr(J_*) > 0$ and undergoes Hopf-bifurcation if $Tr(J_*) = 0$. Solving the equation
766 $Tr(J_*) = 0$ for C_s , we find the Hopf-bifurcation threshold $C_{s,H}$. Then satisfaction of the condition
767 $\left. \frac{d}{dC_s} Tr(J_*) \right|_{C_s=C_{s,H}} \neq 0$ ensures that system undergoes Hopf-bifurcation at $C_s = C_{s,H}$. We need
768 to keep in mind that the occurrence of Hopf-bifurcation is related to the existence of a positive root
769 of the equation $Tr(J_*) = 0$ in terms of C_s , apart from the satisfaction of transversality condition.

770 **Threshold for Hopf-Bifurcation:** $C_{i,H}$

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772 Let $F_i(x) = x \left(\bar{\pi}(C_s) \left(1 - \frac{x}{K} \right) - \bar{\mu}_x(C_s) \right)$. At E_* , we have $z_{i*} = \frac{F_i(x_*)}{\Phi(x_*)}$. Therefore, the trace
773 is equivalent to:

$$Tr(J_*) = \frac{\partial F_i}{\partial x} - z_{i*} \frac{\partial \Phi(x)}{\partial x} = \frac{\partial F_i}{\partial x} - \frac{F_i(x_*)}{\Phi(x_*)} \times \frac{\partial \Phi(x)}{\partial x} = \Phi(x_*) \times \frac{\partial}{\partial x} \left(\frac{F_i(x)}{\Phi(x)} \right) \quad (\text{S23})$$

774 Since $\Phi(x_*) > 0$ when $E_* = (x_*, z_*)$ is feasible, then the sign of $Tr(J_*)$ is the sign of

$$\frac{\partial}{\partial x} \left(\frac{F_i(x)}{\Phi(x)} \right) \quad (\text{S24})$$

775 The case where $\frac{\partial}{\partial x} \left(\frac{F_i(x)}{\Phi(x)} \right) < 0$ means that if we move a little from the point E_* , then the
776 number of prey eaten is greater than the number of prey produced, and therefore, the system goes
777 back to the steady point E_* . However, after a little change, if $\frac{\partial}{\partial x} \left(\frac{F_i(x)}{\Phi(x)} \right) > 0$, then the number
778 of prey produce will be greater than the number of prey eaten, and the system moves away from
779 the steady point E_* . With Holling's type functional response, the sign of $Tr(J_*)$ is the sign of:

$$\begin{aligned} \text{Holling type I:} &= -\bar{\pi}(C_s) \leq 0 \\ \text{Holling type II:} &= -2ahx_*\bar{\pi}(C_s) + ahK(\bar{\pi}(C_s) - \bar{\mu}_x(C_s)) - \bar{\pi}(C_s) \\ \text{Holling type III:} &= -2ahx_*^3\bar{\pi}(C_s) + (K - ahKx_*^2)(\bar{\mu}_x(C_s) - \bar{\pi}(C_s)) \end{aligned} \quad (\text{S25})$$

780 **S1.12 Explicit formulation of threshold concentration:** $C_{s,3}$

781 For the predator dynamic, we have:

$$\begin{aligned}
\frac{dz}{dt} &= z(\psi(C_s)\Phi(x) - \mu_z(C_z) - m) \\
&= z(\varepsilon(\alpha C_z + 1)\Phi(x) - m_z\gamma C_z - m) \\
&= z\left(\varepsilon\left(\alpha\frac{\eta B_x}{k_{out,z}B_z}\Phi(x)C_x + 1\right)\Phi(x) - m_z\gamma\frac{\eta B_x}{k_{out,z}B_z}\Phi(x)C_x - m\right) \\
&= z\left(\varepsilon\alpha\frac{\eta B_x}{k_{out,z}B_z}\Phi(x)^2C_x + \varepsilon\Phi(x) - m_z\gamma\frac{\eta B_x}{k_{out,z}B_z}\Phi(x)C_x - m\right) := zG_i(x, C_x)
\end{aligned} \tag{S26}$$

782 We define ϕ the roots of the polynomial $G_i(x, C_x)$ giving:

$$\varepsilon\alpha\frac{\eta B_x}{k_{out,z}B_z}C_x\phi^2 + \left(\varepsilon - m_z\gamma\frac{\eta B_x}{k_{out,z}B_z}C_x\right)\phi - m = 0 \tag{S27}$$

783 Since $\varepsilon\alpha\frac{\eta B_x}{k_{out,z}B_z}C_x < 0$, to have solution for the functional response, ϕ , we have to satisfy the
784 positivity of the discriminant we denote $\Delta \geq 0$ with:

$$\Delta := \left(\varepsilon - m_z\gamma\frac{\eta B_x}{k_{out,z}B_z}C_x\right)^2 + 4\varepsilon\alpha\frac{\eta B_x}{k_{out,z}B_z}C_xm > 0 \tag{S28}$$

785 The roots of $\Delta = 0$ according to C_x are:

$$C_{i,a}^* = \frac{\varepsilon k_{out,z}B_z}{\eta B_x(m_z\gamma)^2} \left(m_z\gamma - 2\alpha m - 2\sqrt{\alpha m(\alpha m - m_z\gamma)}\right) \tag{S29}$$

786 and

$$C_{i,b}^* = \frac{\varepsilon k_{out,z}B_z}{\eta B_x(m_z\gamma)^2} \left(m_z\gamma - 2\alpha m + 2\sqrt{\alpha m(\alpha m - m_z\gamma)}\right) \tag{S30}$$

787 Note that the part under the square root is positive since $\alpha < 0$.

788 Now, since $\left(m\gamma\frac{\eta B_x}{k_{out,z}B_z}\right)^2 > 0$, then $\Delta > 0$ for $C_x \notin]C_{i,a}^*, C_{i,b}^*[$.

790 The solution of the equation (S27) are:

$$\phi_{i,1} = \frac{k_{out,z}B_z}{2\varepsilon\alpha C_x\eta B_x} \left(\frac{\gamma m_z C_x \eta B_x}{k_{out,z}B_z} - \varepsilon - \sqrt{\Delta}\right) \quad \text{and} \quad \phi_{i,2} = \frac{k_{out,z}B_z}{2\varepsilon\alpha C_x\eta B_x} \left(\frac{\gamma m_z C_x \eta B_x}{k_{out,z}B_z} - \varepsilon + \sqrt{\Delta}\right) \tag{S31}$$

791 In the expression of Δ , we have $\varepsilon\alpha D_i C_x m < 0$, so there are always two positive solutions, and
792 we have:

$$\begin{aligned}
\varepsilon - \frac{\gamma m_z C_x \eta B_x}{k_{out,z}B_z} - \sqrt{\left(\varepsilon - m_z\gamma\frac{\eta B_x}{k_{out,z}B_z}C_x\right)^2 + 4\varepsilon\alpha\frac{\eta B_x}{k_{out,z}B_z}C_xm} &> 0 \\
\Leftrightarrow \varepsilon - \frac{\gamma m_z C_x \eta B_x}{k_{out,z}B_z} - 2\sqrt{-\varepsilon\alpha\frac{\eta B_x}{k_{out,z}B_z}C_xm} &> 0
\end{aligned} \tag{S32}$$

793 So the previous inequality (S32) implies that:

$$\varepsilon - \frac{\gamma m_z C_x \eta B_x}{k_{out,z} B_z} > 0 \quad \Leftrightarrow \quad C_x < \frac{k_{out,z} B_z \varepsilon}{m_z \gamma \eta B_x} \quad (\text{S33})$$

794 Also, let us define the equation $P(C_x) := \varepsilon - \frac{\gamma m_z C_x \eta B_x}{k_{out,z} B_z} - 2\sqrt{-\varepsilon \alpha \frac{\eta B_x}{k_{out,z} B_z} C_x m}$. The function
795 P is decreasing with C_x . Then, solving $P(C_{i,c}^*) = 0$ we get:

$$C_{i,c}^* = \frac{k_{out,z} B_z \varepsilon}{(m_z \gamma)^2 \eta B_x} \left(\gamma m_z - 2\alpha m \pm 2\sqrt{\alpha d(\alpha m - \gamma m_z)} \right) \quad (\text{S34})$$

796 Therefore, for $C_x < \frac{k_{out,z} B_z \varepsilon}{m_z \gamma \eta B_x}$, we get:

$$C_{i,c}^* = \frac{k_{out,z} B_z \varepsilon}{(m_z \gamma)^2 \eta B_x} \left(\gamma m_z - 2\alpha m - 2\sqrt{\alpha d(\alpha m - \gamma m_z)} \right) \quad (\text{S35})$$

797 Also, we previously found the threshold values:

$$C_{i,a}^* = \frac{\varepsilon}{D(m_z \gamma)^2} \left(m_z \gamma - 2\alpha m - 2\sqrt{\alpha m(\alpha m - m_z \gamma)} \right) \quad (\text{S36})$$

798 and

$$C_{i,b}^* = \frac{\varepsilon}{D(m_z \gamma)^2} \left(m_z \gamma - 2\alpha m + 2\sqrt{\alpha m(\alpha m - m_z \gamma)} \right) \quad (\text{S37})$$

799 As a consequence, we define $C_{i,3} = \min\{C_{i,a}, C_{i,b}, C_{i,c}\} = C_{i,c} = C_{i,a}$, which is the threshold
800 value of prey concentration for the existence of E_* .

801 We defined the soil concentration threshold $C_{s,3}$ the point:

$$C_{i,3}^* = e^a \times C_{s,3}^b \quad \Leftrightarrow \quad C_{s,3} = e^{-a/b} (C_{i,3}^*)^{1/b} \quad (\text{S38})$$

802 As a consequence, if $C_s < C_{s,3}$, then $G_i(x, C_x) > 0$ has two positive solutions on $\Phi(x)$ denoted
803 $\phi_{i,1}$ and $\phi_{i,2}$. And if $C_s > C_{s,3}$, then $G_i(x, C_x)$ has no solution.

804 S1.13 Positivity of the determinant

805 Determinant of the Jacobian matrix at the coexistence steady point E_* is:

$$\det(J_*) = \Phi(x_*) z_* \left(-\frac{\partial \bar{\mu}_z(C_s, x)}{\partial x} \Big|_{x=x_*} + \bar{\psi}(C_s, x_*) \times \frac{\partial \Phi(x)}{\partial x} \Big|_{x=x_*} + \Phi(x_*) \frac{\partial \bar{\psi}(C_s, x)}{\partial x} \Big|_{x=x_*} \right) \quad (\text{S39})$$

806 We have:

$$\frac{\partial \Phi(x)}{\partial x} > 0 \quad (\text{S40})$$

807 and since $\psi(\cdot)$ is an increasing function:

$$\frac{\partial \bar{\psi}(C_s, x)}{\partial x} = \frac{\partial \psi \left(\frac{\eta B_x}{k_{out,z} B_z} \Phi(x) C_x \right)}{\partial x} = \frac{\eta B_x}{k_{out,z} B_z} C_x \frac{\partial \psi(\Phi(x))}{\partial x} > 0 \quad (\text{S41})$$

Finally, since $\mu(\cdot)$ is a decreasing function:

$$\frac{\partial \bar{\mu}_z(C_s, x)}{\partial x} = \frac{\eta B_x}{k_{out,z} B_z} C^x \frac{\partial \mu_z(\Phi(x))}{\partial x} < 0 \quad (\text{S42})$$