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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 modulates 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 importance, 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 a chemical contaminant and external disturbances (i.e., trophic cascades). This lack of a mathematical description on how polluted ecosystems respond to external stressors (e.g., eutrophication, climate change, diseases) is due to the combination of many direct and indirect effects (Fleeger et al., 2003; Rohr et al.,

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2006; Relyea and Hoverman, 2006; De Laender et al., 2015).

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

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

One of the most known type of propagation of indirect effects in food web is the “trophic cascade” where the food chain is disturbed by either a change in the abundance of the highest trophic level (i.e., top-down cascade) or by a change in the resources (i.e., bottom-up cascade) (Carpenter et al., 1985; Heath et al., 2014). For instance, a top-down cascade initiated by a decrease in predator abundance releases the pressure on its prey which consequently increase in density and subsequently induce more pressure on prey resources. The reverse process of a bottom-up cascade in a three-level food chain, may start by an increase/decrease of prey resources favoring/constraining in first prey density and then its predator (Heath et al., 2014). Another well-known bottom-up effect is the “paradox of enrichment” where the increase of prey resources (i.e., the carrying capacity) has a destabilizing effect where population dynamics of prey and predators shift from a fixed stable state to an oscillating behavior (Rosenzweig et al., 1971). However, recent studies in aquatic systems showed that exposure to contaminants may inhibit the paradox of enrichment by reducing oscillations and driving the system back to a fixed equilibrium (Prosnier et al., 2015; Huang et al., 2015).

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

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

2. Methods

2.1. Formulation of the general model

Contaminants are commonly measured in concentration per biomass of an individual, $[\mu g\, g^{-1}]$ denoted ppm. In the terrestrial ecotoxicological system, we consider a soil with contaminant concentration denoted by C_s in $[\mu g\, g^{-1}]$. 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, 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