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Stochastic density-dependent matrix model for extrapolating individual-level effects of chemicals to the population: Case study on effects of Cd on *Folsomia candida*

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ABSTRACT

The extrapolation of individual-level effects observed in standard bioassays to the population level is one of the major challenges in ecotoxicology and ecological risk assessment of chemicals. Most of the information about ecotoxicity of chemicals is currently derived from laboratory ecotoxicological tests in which individual-level effects (e.g., the effects on individual life span, fertility, and body growth) of toxic chemicals are measured. However, the risk of chemicals at the population level may be strongly affected by stochastic events that can arise from population structure, density dependence, timing of exposure and environmental fluctuations which are not considered in laboratory experiments. In this study, we present a general matrix population model framework for assessing the ecological risks of chemicals on populations in the field by accounting for density dependence and environmental stochasticity arising from seasonal environmental fluctuations. As an illustration, we present a case study that predicts effects of cadmium (Cd) on *Folsomia candida* populations by considering environmental stochasticity arising from temporal and/or spatial temperature variation. The model results show that extrapolating individual-level effects of Cd to a higher level of biological organization without considering environmental variability underestimates effects of chemicals on population growth rate. Chemicals can also reduce the population size at its carrying capacity. The latter phenomenon arises from different effects of toxicants on different age/stage classes combined with natural demographic processes. One important and useful population-level endpoint that can combine all these effects (i.e. environmental variability, chemical-specific toxicity, and density dependence) is the mean time to extinction. Our model simulation results show that the mean time to extinction of the *F. candida* population exposed to 1000 mg Cd/kg soil can be reduced by 40% compared to the control population. Nevertheless, this significant reduction will not lead to fast extinction of the population as the estimated mean time to extinction is over 3000 years. Based on these results, a population of *F. candida* exposed to this concentration of Cd may not be considered to be endangered. However, other stresses (like habitat fragmentation, food limitation, etc.) can additionally influence the mean time to extinction.

1. Introduction

In standard ecotoxicological tests effects of chemicals are measured for selected species at the individual level. The most frequent endpoints are changes in body size, reproduction rate and short-term survival. These state variables are easy to measure and the data are inexpensive to produce, hence the bioassays are widely used for chemical risk assessments. The relevance of these short-term bioassays in view of the functioning of populations and ecosystems has been, however, questioned by a number of authors (cf Akçakaya et al., 2008; Barnthouse et al., 2008; Forbes et al., 2001a,b, 2008; Laskowski, 2001). Moreover, the use of individual-level endpoints, which are derived from these individual-level measurements, like the No-Observed Effect Concentration (NOEC), as an index for ecological risk assessment of chemicals, has also been criticized (e.g. Jager, 2011; Landis and Chapman, 2011; Laskowski, 1995). Regulations, policies, directives and guidance documents frequently discuss the need for ecological risk assessments to consider risks to populations, and not simply to individual organisms or organism-level life-history traits. Therefore,

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ecological impacts of toxicants have been increasingly assessed with the aid of population-level methods (Thorbek et al., 2010; Barnthouse et al., 2008; Akçakaya et al., 2008).

This shift from studying effects on individuals to populations is gaining momentum with increasing emphasis on population-level assessments (Akçakaya et al., 2008; Forbes et al., 2011). Furthermore, directives and guidelines request risk assessment at higher levels of biological organization (European Commission, 2002a,b). Nevertheless, information on the ecotoxicity for population-level risk assessments is still derived from laboratory individual-level bioassays, which are conducted under constant and, presumably, favorable conditions. However, in their natural environment organisms rarely experience optimal conditions, and these conditions are rarely constant. On the contrary, organisms are forced to cope with either suboptimal or severe environmental stress for most of their life spans (Holmstrup et al., 2010; Heugens et al., 2003). These added environmental stressors may or may not alter the effects of chemical contaminants in comparison to laboratory tests performed under controlled and optimal conditions. Risk assessment procedures therefore typically include safety (or uncertainty) factors in order to ensure conservative estimates of environmental concentrations that minimize risks posed by chemicals to organisms in the natural environment (Chapman et al., 1998). These uncertainty factors usually lack a scientific basis and may underestimate or overestimate actual environmental effects (Forbes et al., 2008; Hanson and Stark, 2012). On the other hand, it is technically as well as economically impossible to experimentally assess all possible interactions between different environmental conditions and chemicals.

This lack of realism in standardized bioassays makes the interpretation and extrapolation of observed individual-level effects to the population level uncertain. For example, Forbes and Calow (1999) evaluated effects of toxicants at both individual and population levels and found an inconsistent and unpredictable relationship between the two. The authors also found that effects of a toxicant on population growth rate (r) may be smaller, equal to or larger than effects on individual life-history traits contributing to it.

In a recent meta-analysis, Laskowski et al. (2010) reviewed and analyzed a range of significant effects of natural environmental conditions on toxicant effects in animals. Heugens et al. (2003) reported the influence of variable and suboptimal environmental conditions on the outcome of toxicity tests. Besides effects of natural physico-chemical stressors, interactions between toxicants and density dependence and their influence on population dynamics have gained interest among ecotoxicologists (Forbes et al., 2001a,b, 2003; Gui and Grant, 2008; Grant, 1998; Sibly et al., 2000; Hayashi et al., 2008; Noel et al., 2006).

Therefore, understanding and including intra-population and extra-population ecological factors that can modify effects of toxicants at individual and population levels is a key step in developing tools for extrapolating the results of laboratory toxicity tests to populations living in natural environments. Matrix population models can be used to investigate the dynamics of age/stage classified populations (Caswell, 2001; Tuljapurkar, 1997) and can estimate many characteristic population endpoints, such as asymptotic population growth rate, generation time, stable population structure, and reproductive values (Charles et al., 2009).

In this work, we present a general matrix population model framework for assessing the ecological risks of chemicals on populations in the field by accounting for density-dependence and environmental stochasticity arising from seasonal environmental fluctuations. As an illustration, we provide a case study that predicts the effect of cadmium (Cd) on *Folsomia candida* populations by considering environmental stochasticity arising from temporal and/or spatial temperature variation.

2. Materials and methods

2.1. General model structure

The general structure of the model is based on an age/stage based matrix. In the matrix model the state of a population is given by a vector $N(t) = [N_1(t), \ldots, N_n(t)]^T$, where $N(t)$ is the total population size (number of individuals) at time t, $N_i(t)$ represents the number of individuals in age class i at time t and the superscript T indicates the transpose of the vector (Tuljapurkar, 1997). This vector is projected from time t to $t+1$ by a projection matrix $\boldsymbol{A}$:

$$\boldsymbol{A} * N_t = N_{t+1} \tag{1}$$

where $\boldsymbol{A}$ is constructed from individual vital rates a_{ij}, while N_t and N_{t+1} represent the total number of individuals at time t and $t+1$ respectively.

This general age/stage based matrix model can be extended to model population-level effects of environmental stressors by including measured effects of a stressor on specific vital rates and by incorporating key ecological processes that can affect population dynamics (e.g. seasonal environmental variation and density-dependence in our case).

The framework of our model comprises five main elements: formulating the projection matrix, modelling effects of toxicants at the individual-level, estimating environmental stochasticity, modelling effects of density-dependence and eventually estimating overall population-level effects. The brief description of the methodological approach we applied to address these elements of the model is presented in the subsequent sections by considering the effect of Cd on *F. candida* populations.

2.2. F. candida as a standard bioassay species

F. candida is the most widely studied springtail species, both in terms of its ecotoxicology and life history, and it has been extensively used for toxicity testing (Fountain and Hopkin, 2005). They are easy to culture in the laboratory, and due to parthenogenetic reproduction, populations of one particular genotype can be obtained. Furthermore, an extensive amount of literature is available about the individual-level effects of toxicants and environmental stressors on *F. candida*. The species is used as a model organism in both OECD and ISO (OECD, 2007 and ISO, 1999) as a representative organism for risk assessment of chemicals to soil invertebrates.

2.3. Stage-based matrix model

The first step in formulating a matrix model is to select the type and structure of the projection matrix (age, stage, or size classified) and to determine an appropriate number of age/stage classes. The life history of *F. candida* consists of three distinct stages: egg (embryo), juvenile, and adult, and therefore we used a three-stage based matrix model (Eq. (2)):

$$\begin{bmatrix} P_{s1} & 0 & F_3 \\ G_1 & P_{s2} & 0 \\ 0 & G_2 & P_{s3} \end{bmatrix} \times \begin{bmatrix} N_{1,t} \\ N_{2,t} \\ N_{3,t} \end{bmatrix} = \begin{bmatrix} N_{1,t+1} \\ N_{2,t+1} \\ N_{3,t+1} \end{bmatrix} \tag{2}$$

where P_{si} is the probability of remaining in the same stage, G_i is the probability of moving to the next stage, F_i is the number of eggs produced per individual, and $N_{i,t}$ and $N_{i,t+1}$ represent the number of individuals in the different developmental stages at time t and $t+1$ respectively. As suggested by Caswell (2001), P_i and G_i can be estimated as follows:

$$P_{s1} = \sigma_1(1 - \gamma_1) \tag{3a}$$