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Coupling different mechanistic effect models for capturing individual- and population-level effects of chemicals: Lessons from a case where standard risk assessment failed☆

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ABSTRACT

Current environmental risk assessment (ERA) of chemicals for aquatic invertebrates relies on standardized laboratory tests in which toxicity effects on individual survival, growth and reproduction are measured. Such tests determine the threshold concentration of a chemical below which no population-level effects are expected. How well this procedure captures effects on individuals and populations, however, remains an open question. Here we used mechanistic effect models, combining individual-level reproduction and survival models with an individual-based population model (IBM), to understand the individuals' responses and extrapolate them to the population level. We used a toxicant (Dispersogen A) for which adverse effects on laboratory populations were detected at the determined threshold concentration and thus challenged the conservatism of the current risk assessment method. Multiple toxicity effects on reproduction and survival were reported, in addition to effects on the F1 generation. We extrapolated commonly tested individual toxicity endpoints, reproduction and survival, to the population level using the IBM. Effects on reproduction were described via regression models. To select the most appropriate survival model, the IBM was run assuming either stochastic death (SD) or individual tolerance (IT). Simulations were run for different scenarios regarding the toxicant's effects: survival toxicity, reproductive toxicity, or survival and reproductive toxicity. As population-level endpoints, we used population size and structure and extinction risk. We found that survival represented as SD explained population dynamics better than IT. Integrating toxicity effects on both reproduction and survival yielded more accurate predictions of population effects than considering isolated effects. To fully capture population effects observed at high toxicant concentrations, toxicity effects transmitted to the F1 generation had to be integrated. Predicted extinction risk was highly sensitive to the assumptions about individual-level effects. Our results demonstrate that the endpoints used in current standard tests may not be sufficient for assessing the risk of adverse effects on populations. A combination of laboratory population experiments with mechanistic effect models is a powerful tool to better understand and predict effects on both individuals and populations. Mechanistic effect modelling thus holds great potential to improve the accuracy of ERA of chemicals in the future.

1. Introduction

Environmental risk assessment (ERA) involves determining the adverse effects that chemicals and other stressors exert on ecological systems. Because it is impossible to eliminate all environmental effects of human activities, decisions were made to define protection goals which strike a compromise between the benefits of using the chemicals and costs in terms of acceptable effects. Protection goals vary among different biological levels of organization. In contrast to vertebrates, where the visible mortality of individuals has

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to be prevented, the target entity for aquatic invertebrates is the population rather than the individual (Brock et al., 2006; EFSA, 2010; Hommen et al., 2010), which implies that lethal and sublethal effects on individuals are accepted if they do not impair the functioning of the population.

Nevertheless, the standard ERA procedure for aquatic invertebrates still relies on laboratory tests at the individual level (Forbes et al., 2008), testing for effects of chemicals on simple endpoints like survival, growth or reproduction. One of the commonly used approaches in estimating the risk posed by chemicals relies on applying safety factors to the measured EC_x (the x% effective concentration) or NOECs (the no observed effect concentration) of tests with acute or chronic exposure to the chemical, to calculate the PNEC, the predicted no effect concentration (European Commission, 2003).

Such measures of risk have been criticized as they might not always be sufficient to ensure that protection goals are reached, thus limiting the application of risk assessment as a tool for managing environmental resources (Forbes et al., 2010). One example of the limitations of the current standard approach are the laboratory population test results for daphnid populations exposed to Dispersogen A (Hammers-Wirtz and Ratte, 2000), a substance used as an additive in several pesticide formulations as well as in industries such as textile printing (Kromm, 1995) Dispersogen A has been shown to spread into the aquatic environment (Karl, 1998; Schoenberger and Kaps, 1994).

Dispersogen A has a low acute toxicity for *Daphnia magna* (EC_{50} = 167 mg L^{-1}, 48 h) and a NOEC for reproduction of 10.2 mg L^{-1} (derived from 21-day reproduction tests, Hammers-Wirtz and Ratte, 2000). The PNEC value derived from standard reproduction tests, calculated as the ratio of NOEC to a safety factor of 50 (European Commission Technical Guidance Document, 2003), turned out to be not protective even for laboratory populations (conducted under controlled conditions) as it led to a reduction of the population size by almost 20% (Hammers-Wirtz and Ratte, 2000).

This suggests that, in this case, changes in population properties following exposure did not emerge solely from toxicity effects on the survival of individuals and on the number of living offspring, which are the endpoints considered in the classical risk assessment methodology, but that additional effects of the toxicant were important as well.

In addition to the measured toxicity effects on reproduction and survival, Dispersogen A has further complex effects on individual daphnids. First, a stimulatory (hormetic) response of the reproductive output accompanied by a decrease in the body length of neonates was reported (Hammers-Wirtz and Ratte, 2000).

Secondly, the same study showed significant effects on several endpoints in daphnid individuals born in the F1 generation (for details, see Section 2). Neither the stimulatory effects on the individual reproductive output nor the effects on the F1 generation are currently adressed in the risk assessment.

Therefore, here we explored the hypotheses that the risk assessment failed to be protective for populations in the case of Dispersogen A due to ignoring either the stimulatory toxicity effects on reproduction, or the observed effects on the F1 generation, or to ignoring both of these effects. To explore these different hypotheses and identify the most likely real drivers of effects observed at the population level, we need a tool that enables us to independently capture the toxicant's modes of action at the individual level and to test their effects at the population level. Mechanistic effect models, and particularly individual-based population models (IBMs), are used to overcome the limitations of standard tests. They allow us to test different assumptions about the organism level effects of chemicals (Forbes et al., 2008; Grimm et al., 2009; Preuss et al., 2009) and to explore which of these organism-level endpoints are most predictive of population-level effects (Preston and Snell, 2001). Moreover, IBMs allow the integration of different toxicokinetic/toxicodynamic (TK/TD) models, which dynamically simulate the processes that lead to toxicity within an organism, and its corresponding effects on survival (Ashauer et al., 2011; Jager et al., 2011).

In this study, we used an existing IBM of daphnids (IDamP, Preuss et al., 2009) combined with a TK/TD model for survival (GUTS, Jager et al., 2011) to extrapolate the effects of Dispersogen A from daphnid individuals to the population level. We contrasted different assumptions about individual-level effects of the toxicant and tested how well the resulting population models explained observations from two laboratory population experiments (Hammers-Wirtz and Ratte, 2000). Our main aim was to identify the modes of action triggering the population-level effects in daphnids exposed to Dispersogen A, which were the reason behind the failure of the current risk assessment to be protective at the population level.

2. Materials and methods

2.1. Dispersogen A: properties and modes of action

Dispersogen A is a condensation product of Naphthalene sulphonic acid with formaldehyde (Kromm, 1995). According to *Daphnia* reproduction tests (Coors et al., 2004; Hammers-Wirtz and Ratte, 2000), adverse effects of Dispersogen A were reported on the reproductive output of daphnids at as low as 0.1 mg L^{-1}. However, and contrary to classical toxicants which induce a reduction in the clutch size, exposure to Dispersogen A increases the clutch size (by as much as 53% compared to the control) up to a concentration of 10.2 mg L^{-1}, at the expense of decreasing neonate body length (fitness) (lowest observed effect concentration, LOEC = 0.1 mg L^{-1}). It is only at higher concentrations (25.6 mg L^{-1}) that the clutch size is reduced, and at concentrations exceeding 64 mg L^{-1}, reproduction is completely inhibited (Hammers-Wirtz and Ratte, 2000). In addition to effects on reproduction, Dispersogen A causes significant mortality (EC_{50} = 16.5 mg L^{-1}, Hammers-Wirtz and Ratte, 2000) at the individual level.

Furthermore, experiments with neonates from exposed mothers that were grown individually in uncontaminated medium showed that toxic effects of Dispersogen A transmit to the next generation (F1) where they cause even stronger negative effects than in the original generation. Examples include significant decreases in the body and clutch sizes observed at even very low concentrations, e.g. 1.64 mg L^{-1} in the F1 generation, compared to effects observed at a concentration of 25.6 mg L^{-1} in the original generation, or the decrease in neonate survival observed at 0.001 mg L^{-1} in the F1 generation test compared to 1.64 mg L^{-1} in the original generation (Hammers-Wirtz and Ratte, 2000).

2.2. The models

2.2.1. The Daphnia population model IDamP

We used the individual-based population model IDamP for *D. magna* (Preuss et al., 2009) to simulate the effects of Dispersogen A. IDamP predicts the population dynamics of *D. magna* based on individual life cycles, including the feeding, growth, development, reproduction and survival processes. The main drivers of these processes are the food conditions and, via crowding effects, the density of the population. The model is applicable at a laboratory scale, with the algae *Desmodesmus subspicatus* as a food source. All submodels of IDamP representing the life cycle process are descriptive regression models, which were based on a large dataset from different life cycle tests. Predictions of the full model regarding population size