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## Marine Pollution Bulletin

journal homepage: [www.elsevier.com/locate/marpolbul](http://www.elsevier.com/locate/marpolbul)

## Viewpoint

## What are the roadblocks to using population models in ecotoxicology studies?

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## ARTICLE INFO

**Keywords:**  
Ecotoxicology  
Population  
Modelling  
Polychaete  
Toxicity

## ABSTRACT

Understanding how pollution affects populations is critical for targeted environmental risk assessments and adequate protection of the environment. However, the vast majority of ecotoxicology studies still have a traditional focus of identifying effects on individual organisms and do not measure the effects at the population-level. Modelling tools that measure population effects of pollution are available and would add value to current ecotoxicology studies by aligning outcomes more closely to what needs to be protected. In this paper I outline three possible reasons why this knowledge gap still exists and consider how they could be adopted more broadly, including better considerations about what endpoints should be measured at the initial study design phase. The purpose of highlighting this knowledge gap is to assist in facilitating the integration of population-level endpoints into routine pollution monitoring programs and progress of ecologically relevant ecotoxicology research.

## 1. Introduction

Assessing the effects of pollution and subsequent environmental impacts are among the principal goals of environmental protection agencies worldwide (Carter and Howe, 2006; EPA Victoria, 2003; US EPA, 2015). However, there is a continuing disconnect between the individual-level measures that are used to assess the effects of pollution and management at the population-level (Forbes and Galic, 2016). Population endpoints are thought to be a better estimate of risk as regulatory decisions are most often defined at the population-level (Beyer and Heinz, 2000), and also because they account for important ecological processes, such as density dependence, resource limitation and life-history strategies (Stark and Banks, 2003).

Ecotoxicology approaches to assessing the risk of adverse impacts usually start with individual-level tests in a controlled laboratory setting. Individual end-points include biomarkers and other sub-organism metrics (e.g. genetic, pathological or behavioral), and commonly used LC50 or LD50 values, which estimate the lethal concentration (LC) or lethal dose (LD) for 50% of the test organisms (Malaj et al., 2016). Measuring individual-level endpoints have traditionally been favoured as they are relatively inexpensive to generate and data is often available for a wide range of species and/or toxicants combinations. Individual-level endpoints can provide clear links between a source of pollution and whole-organism biological responses (ANZECC, 2000; Simpson et al., 2013) and is often used as a trigger for further investigations (Lam and Gray, 2003; Stark et al., 2007). They are useful in conjunction with multiple lines of evidence that combine chemical testing,

laboratory toxicity tests and field community assessments to make a decision about the level of pollution and possible biological impacts (e.g. Chapman and Anderson, 2005; Kellar et al., 2014). However, individual endpoints cannot provide useful predictions of ecological effects on populations, communities or ecosystems (Forbes et al., 2006) unless they are explicitly tested or modelled (O'Brien and Keough, 2014).

Population processes, such as life-history strategies, population density and structure, can all influence the effects of pollution, and consequently alter (or not alter) risk assessments and management decisions (Forbes et al., 2001; Van Straalen et al., 1989). Ideally, individual-level endpoints would either act as an early warning indicator of population-level effects or explain the patterns observed in populations and higher levels (Forbes et al., 2006). However, there are few examples that specifically test the effects of pollution at multiple levels of biological organization (O'Brien and Keough, 2014) and so there is limited understanding of how pollution impacts are affected by population processes and how individual-level responses can be used to predict the effects on populations and beyond (Hommen et al., 2010; Pedersen et al., 2013).

Responses at the individual-level can be different from responses at the population-level (Hayashi et al., 2009). Population growth rates could be relatively stable when exposed to pollution; despite obvious toxic effects of the individual-level (Van Straalen et al., 1989) or have significant long-term population declines even with low level exposure that could not otherwise be detected with individual-based toxicity tests (de los Santos et al., 2015). A simple example using data extracted

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<http://dx.doi.org/10.1016/j.marpolbul.2017.08.038>

Received 3 April 2017; Received in revised form 31 July 2017; Accepted 16 August 2017  
0025-326X/ © 2017 Published by Elsevier Ltd.

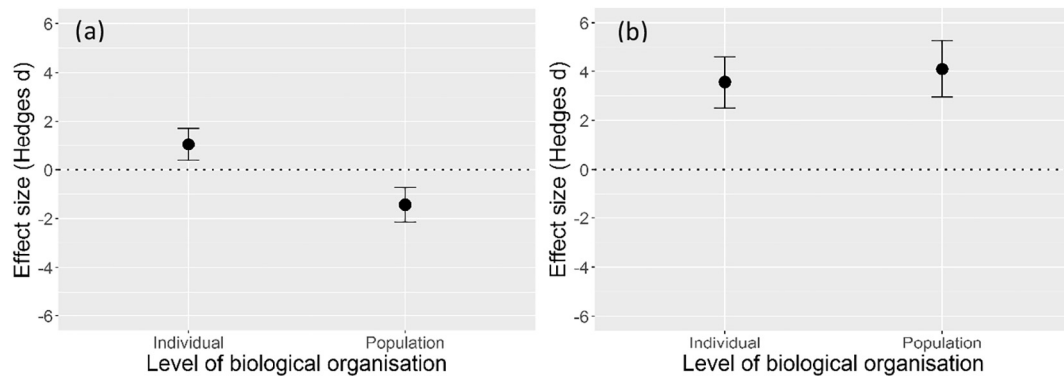


Fig. 1. Individual (age at first reproduction) and population (population growth rate) responses to sediment dosed hydrocarbons for the polychaetes (a) *Capitella* sp. I, and (b) *Streblospio benedicti* (raw data extracted from Levin et al., 1996, Table 2). Effect size (Hedges  $d$ ) is the mean difference between control and dose treatments ( $\pm$  95% confidence intervals).

from a study that reported both individual and population endpoints (Levin et al., 1996) shows a positive response at the individual level (age of first reproduction), for a polychaete (*Capitella* sp. I) exposed to hydrocarbons, but a negative response at the population level (population growth, Fig. 1a). However, endpoints for another polychaete species used in the same study (*Streblospio benedicti*) had similar magnitude and directions of change for both individual and population-level measurements (Fig. 1b).

Effect sizes and 95% confidence intervals were calculated based on methods used for meta-analysis (Gurevitch et al., 2000) using R-script provided in Hale et al. (2017) supporting material. Data from Levin et al. (1996) used in the calculations was age at first reproduction (weeks  $\pm$  standard error) and population growth rate ( $\lambda$ /week) with 95% confidence intervals based on an age-classified population models.

The importance of population endpoints in ecotoxicology and problems associated with extrapolating between individual and population-level endpoints are not new concepts, but ones that have been discussed in the literature since the late 1990s (Ferson et al., 1996; Forbes and Calow, 1999; Forbes et al., 2008). Although individual endpoints have proven to be useful in risk assessments using surrogate species (Banks et al., 2010), species distribution models (Awkerman et al., 2016) and evolutionary approaches (Malaj et al., 2016). There still appears to be a lack of general dissemination and uptake of these ideas with a continuing focus on identifying effects on individual organisms. Population modelling has the potential to contribute to improving the realism and ecological relevance of ecotoxicology (Chapman, 2002), but also develop a better understanding of important ecological processes that mediate the effects of pollution and how these vary between species, functional groups and environmental conditions.

Here, I briefly describe three possible reasons why population-level assessments are not common in ecotoxicology and why I believe they represent outdated viewpoints rather than fundamental reasons why this knowledge gap exists. The purpose of highlighting this continuing knowledge gap is to; (1) create awareness and facilitate the progress of routine population-level assessments into ecotoxicology and; (2) more broadly, contribute to the integration of ecology and ecotoxicology research disciplines (Gessner and Tlili, 2016).

## 2. Perceived barriers

### 2.1. Expensive and labor intensive

There is the perception that population-level assessments are expensive and labor intensive and therefore they are not useful (Simpson et al., 2013). Measuring a population over long periods of time in the field or laboratory to capture multiple generations is likely to be very costly. However, individual acute and chronic endpoints measured over shorter time periods can be used to predict the effects of toxicants on populations (Forbes and Calow, 1999; Stark and Banks, 2016). Life

table response experiments are one such example of this where individual mortality and reproduction are measured at each life history stage and used to model the effects on population growth (Stark and Banks, 2003). Although full life-cycle table response experiments have been criticized as too expensive (Sibly, 1999), even cheaper alternatives such as partial life-cycle toxicity tests that consider limited number of generations or only the early stages of development can also be considered (Stark and Banks, 2016).

Decision-making frameworks used to assess the environmental risk of sediment and water contamination require multiple toxicity tests for different test organisms (e.g. Simpson et al., 2005) and sometimes in-situ community-level assessments (Chapman and Anderson, 2005). This can also be an expensive and labor intensive process but in many cases the cost can be justified if the outcomes are targeted to answering management decisions (Kellar et al., 2014). Population models may be comparatively quite cost-effective as they can account for environmental stochasticity and underlying ecological processes, such as density dependence, thereby reducing uncertainty and likelihood of making costly decision errors (Hanson and Stark, 2011). Multiple toxicant combinations could also be built into model scenarios to make environmental realism easier to test (e.g. Galic et al., 2017). Furthermore, additional data collection for population models may be minimal as data on age-specific mortality and reproduction could easily be collected using existing resources and laboratory cultures used for standard toxicity testing (Gale et al., 2006).

### 2.2. Complexity of population models

The perceived complexity of population models may be an explanation as to why they are rarely used in ecotoxicology studies but there are, in fact, a vast range of models that can be used from simple to complex (Forbes et al., 2008). Simple demographic models using differential equations can be structured with size and age classes, and include spatial and/or demographic stochasticity. More complex individual-based matrix models allow parameterization of individual vital rates (i.e. fecundity, survivorship), and although they generally take more time to build and valid, provide predictions of future population size and potentially more direct relevance to management applications.

Population models could provide another line of evidence in the risk assessment process and do not necessarily need to introduce additional complexity for managers and decision makers. Predicting the size of a population with exponential growth is one of the simplest population models to use as it only requires information on initial and final numbers of individuals over time (Gotelli, 2001). Variations can be added to the model to improve the predictions, such as carrying capacities, density dependence and environmental stochasticity (McCallum, 2000). This approach may be particularly useful when there is limited information on the life history characteristics of the study organism and it is not possible to construct a full life-cycle response experiment

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