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Contribution to a Discussion Meeting Issue on: "Population growth rate: Determining factors and role in population regulation"

Population growth rate as a basis for ecological risk assessment of toxic chemicals

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Abstract

Assessing the ecological risks of toxic chemicals is most often based on individual-level responses such as survival, reproduction or growth. Such an approach raises the following questions with regard to translating these measured effects into likely impacts on natural populations. (i) To what extent do individual-level variables underestimate or overestimate population-level responses? (ii) How do toxicant-caused changes in individual-level variables translate into changes in population dynamics for species with different life cycles? (iii) To what extent are these relationships complicated by population-density effects? These issues go to the heart of the ecological relevance of ecotoxicology and we have addressed them using the population growth rate as an integrating concept. Our analysis indicates that although the most sensitive individual-level variables are likely to be equally or more sensitive to increasing concentrations of toxic chemicals than population growth rate, they are difficult to identify *a priori* and, even if they could be identified, integrating impacts on key life-cycle variables via population growth rate analysis is nevertheless a more robust approach for assessing the ecological risks of chemicals. Populations living under density-dependent control may respond differently to toxic chemicals than exponentially growing populations, and greater care needs to be given to incorporating realistic density conditions (either experimentally or by simulation) into ecotoxicological test designs. It is impractical to expect full life-table studies, which record changes in survival, fecundity and development at defined intervals through the life cycle of organisms under specified conditions, for all relevant species, so we argue that population growth rate analysis should be used to provide guidance for a more pragmatic and ecologically sound approach to ecological risk assessment.

Keywords: *Capitella*, density dependence, ecotoxicology, extrapolation, life cycles, pollution

1. Introduction

Ecological risk assessment tries to predict the likely impacts of human activities on ecological systems (USEPA 1992). In the case of toxic chemicals, the raw materials for ecological risk assessment involve exposure assessment based on predictions or measurements of environmental concentrations of toxic chemicals and an assessment of hazards, i.e. the potential of those chemicals to cause ecological harm. Hazard assessment is generally based upon observations on survival, growth or reproduction in a few individuals in a few species. We shall refer to these responses as individual-level variables. Variability in responses among species is expressed only in terms of differences in these traits as measured under standard laboratory conditions and hence only reflects physiological variability in sensitivity to chemicals. It is presumed that these kinds of observations are relevant for protecting populations and ecosystems. However, this raises at least three different questions, as follows.

- (i) To what extent do individual-level variables underestimate or overestimate population-level responses?
- (ii) How do toxicant-caused changes in individual-level

variables translate into changes in population dynamics for species with different life cycles?

- (iii) To what extent are these relationships complicated by population-density effects?

We have addressed these questions, which go to the heart of the ecological relevance of ecotoxicology, using the population growth rate as an integrating concept. We have limited our attention to modeling the links between development, fecundity and survival to population growth rate. Other models go beyond this to relate physiological processes to growth, fecundity and survival (e.g. Gurney *et al.* 1990; Kooijman 1993), but these are demanding in their requirement of detailed data.

For each question, we shall present a short review of the work done to date followed by one or two examples based on experimental data to illustrate how population growth rate can be used in a practical way to provide a more sound basis for ecological risk assessment. Our aim in the first instance is to develop an approach that can be applied to generalized ecological risk assessments of toxic chemicals, as is often required in the regulatory arena. This means that we are using population growth rate analyses to compare the effects of toxic chemicals on the same species among expo-

sure concentrations (i.e. to derive concentration–response relationships), on the same species among toxic chemicals (i.e. to rank chemicals in terms of their relative ecological hazard) and on the same chemical among species (i.e. so that the effects of chemicals on test species can be extrapolated to the effects on other, untested, species). Our analyses, therefore, have to be limited in detail and cannot take account of immigration–emigration effects or interactions with other species, the importance of which will vary from one habitat to another. In principle, when it comes to considering detailed impacts of specific chemical(s) on specific populations in particular habitats, it is possible to develop more detailed models. However, such models have had very limited applications in the regulatory arena to date.

2. Within a Life-Cycle Type, to What Extent Are Responses in Individual Variables More or Less Sensitive to Increasing Toxicant Concentration Than Population Growth Rate?

(a) Review

In practice, ecotoxicological tests focus on individual-level variables (i.e. survival in response to high chemical concentrations over short periods of time; survival, growth or fecundity in response to low chemical concentrations over long periods of time). However, most often, the targets of ecological risk assessments are not individuals but entire populations as, within limits, individuals can be removed from populations without any adverse effects on population persistence. Thus, for such individual-level responses to be useful endpoints, it is necessary that they adequately and consistently reflect the impacts of chemicals on populations. This means that there are two main issues of concern. The first is whether individual responses measured in terms of survival, fecundity or growth–development are more or less sensitive to chemical impacts than effects measured in terms of population growth rate. The second is whether there is consistency in the relationship between changes in the individual traits and changes in population growth rate, such that it is possible to identify those traits that are the best predictors of effects on population growth rate.

There is a number of reasons why differences in sensitivity between individual-level and population-level responses to chemicals may occur. These may arise from the nonlinear relationship between population growth rate and the demographic variables contributing to it; from the relative size of the demographic variables with respect to each other (i.e. life-cycle type); from species- and chemical-specific differences in the relative sensitivity of the demographic variables to chemical exposure; and from the demographic state from which the population starts (i.e. growing, stable, declining; see Forbes & Calow 1999). Clearly, whether population growth rate is expressed as the intrinsic rate of increase (r) or population multiplication rate (λ , where $\lambda = e^r$) is also important, particularly if proportional changes are used as a measure of relative sensitivity.

In a review of 41 studies, which included a total of 28 species and 44 toxicants, Forbes & Calow (1999) found that, out of the 99 cases considered, there were only five where chemical effects on population growth rate (most of which

were expressed as r) were detected at lower exposure concentrations than those resulting in statistically detectable effects on any of the individual demographic variables. In 81.5% of the cases considered (out of a total of 81), the percentage change in population growth rate (expressed as r) was less than the percentage change in the most sensitive of the individual demographic traits: 2.5% where the percentage change in population growth rate was equal to that of the most sensitive trait and 16% where the percentage change in population growth rate was greater than the percentage change in the most sensitive demographic trait. Despite the fact that any proportional changes in population growth rate were significantly correlated with the proportional changes in fecundity and with time to first reproduction, these correlations were rather weak and trend analysis indicated that these relationships were nonlinear. Surprisingly, the correlation between the proportional reduction in survival (i.e. the most frequently measured trait in ecotoxicological studies) and the proportional reduction in population growth rate was not statistically significant. Overall, there was no consistency in which of the measured individual-level traits was the most sensitive to toxicant exposure, and none of them, considered individually, could be said to be very precise predictors of toxicant effects on population growth rate.

Another way of approaching this question is to consider the sensitivity of population growth rate to changes in the life-history traits contributing to it. This can be formalized by assessing the percentage change in λ that arises from small percentage changes in individual-level variables. This quantity is referred to as the elasticity of λ with respect to the individual-level variables (De Kroon *et al.* 2000; Caswell 2000). It should be noted that these elasticities do not strictly represent contributions to λ and therefore do not necessarily sum to 1 (i.e. in this context there is no reason to expect that λ is a homogenous function of the individual variables (Caswell 2000, p. 232). Using a simplified two-stage life-cycle model, Forbes *et al.* (2001a) were able to show that when λ is close to 1 and the generation time is greater than or equal to 1, the elasticities with respect to the individual life-cycle variables were less than or equal to 1. In other words, in the neighborhood of the population steady-state, a small percentage change in the individual life-history variables, for example brought about by a toxicant, would result in at most the same percentage change in λ . However, if λ is allowed to increase above 1, the situation becomes more complex and in certain circumstances it is possible that a given proportional change in some of the individual-level traits leads to a proportionally greater change in λ .

(b) Example 1

The conclusion from the review is that, in general, the most sensitive individual life-cycle traits will be at least as sensitive as population growth rate to any increases in toxicant concentration. However, this assumes that the most sensitive trait(s) will always be measured in ecotoxicological assays, but this need not be the case. The following example, based on a life-table response experiment using the polychaete *Capitella* species I exposed to nonylphenol (Hansen *et al.* 1999a) illustrates this point. Table 1 shows the percentage reductions in life-cycle traits and population

Table 1. Percentage changes in individual demographic variables and λ for the polychaete *Capitella* species I exposed to 174 μg nonylphenol g^{-1} dry wt sediment. (The value of λ in the control was about 2.5 week^{-1} . Data are from Hansen *et al.* (1999a).)

Trait	Change relative to control (%)
Juvenile survival	0
Adult survival	0
Time to first reproduction	+ 17
Time between broods	+ 25
Total number of broods per individual	- 44
Total number of offspring per individual	- 78
Population growth rate (λ)	- 24

growth rate at the nonylphenol concentration at which the most sensitive trait was significantly impaired relative to the control. At this concentration there were wide differences in percentage effects on individual traits and population growth rate, with the reproduction effects being the most severe. There were no effects on either juvenile or adult survival over the entire concentration range used. So if the analysis had initially focused on just survival, a risk assessment based on this individual-level variable would have concluded that no effects of this chemical occurred at a concentration at which a 24% reduction in λ was calculated. Fecundity was the most sensitive trait to nonylphenol, with brood number and total offspring being reduced by 44 and 78%, respectively. Although the time to first reproduction was delayed by only 17%, a decomposition analysis of these data (Hansen *et al.* 1999a) showed that this trait contributed more to the effect on λ than did fecundity.

This example clearly demonstrates why life-table studies should take all effects into account and the value of population growth rate as an integrating variable. In addition, it highlights the necessity of considering both the toxicological sensitivity of individual-level variables and the demographic sensitivity of λ to changes in these variables for understanding the mechanisms of toxicant effects on population dynamics.

3. How Do Toxicant-Caused Changes in Individual-Level Variables Translate into Changes in Population Dynamics for Species with Different Life Cycles?

(a) Review

In ecotoxicology, concentration-response relationships are obtained for individual-level variables across a number of standard test species. It has been implicitly assumed that these responses have the same meaning, independent of the species involved; for example, 50% mortality is presumed to have the same effect, in terms of population dynamics, for all species. Alternatively, variability in individual-level traits across species has been used to construct species-sensitivity distributions that are now used as a basis for ecological risk assessment (Van Leeuwen & Hermens 1995). The implicit assumption here is that the variability in individual-level variables is di-

rectly related to variability in population responses. However, this ignores the possibility of complications from life-cycle differences across species. The analysis of demographic models very clearly shows that, indeed, different life-cycle types respond differently to changes in their corresponding demographic input variables (e.g. Stearns 1992; Caswell 2000).

Calow *et al.* (1997) considered a series of simplified but plausible scenarios to illustrate how information from ecotoxicological tests can be used to explore the effects on population dynamics for different life-cycle types. A number of general conclusions arose out of this analysis. (i) As expected, the effect on population growth rate of a toxicant that reduces juvenile survivorship or fecundity will be greater for semelparous species (i.e. species that reproduce once) as compared with iteroparous species (i.e. species that reproduce more than once), and the reverse will be the case for the effects of toxicants on adult survival. (ii) Iteroparous species with life cycles in which the time to first reproduction is shorter than the time between broods will be more susceptible to toxicant impacts on survival or fecundity than will species in which time to first reproduction is longer than the time between broods. (iii) Anything that shortens time to first reproduction relative to the time between broods (e.g. increased temperature, increased food availability) is expected to increase the population-level impact of toxicant-caused impairments in survival or fecundity. (iv) Lengthening of the time to first reproduction should lessen the population impact of toxicant-caused impairments in survival or fecundity. An additional important outcome from this analysis was a clear demonstration of the importance of a population's demographic starting point for relating toxicant-caused impairments on demographic traits to consequences at the population level. This is particularly so for time to first reproduction, t_j . Whereas, in growing populations, toxicant-caused delays in t_j have a negative effect on population growth rate, in shrinking populations such delays can have an ameliorating effect in that they slow the rate at which the population approaches extinction.

Extrapolating the effects of toxic chemicals from individual-level responses from a few species to the effects on entire communities is increasingly performed by fitting available ecotoxicological test data to a statistical distribution and using this to estimate the chemical concentration that is unlikely to impair most of the species in the distribution. However, the distributions of sensitivities based on individual-level variables are likely to differ from distributions based on population growth rate as discussed in the previous paragraph. Moreover, the species used to provide the input data for these distributions rarely, if ever, reflect the actual distribution of the life-cycle types in natural communities. Many of the species used routinely in ecotoxicological tests are chosen because they have life-cycle features that are amenable to laboratory work, and therefore cannot be considered to represent a random sample from nature. Forbes *et al.* (2001a) took an initial step towards exploring the importance of these considerations in ecological risk assessment by comparing sensitivity distributions that were based on the response of juvenile survival to a chemical with sensitivity distributions based on λ for

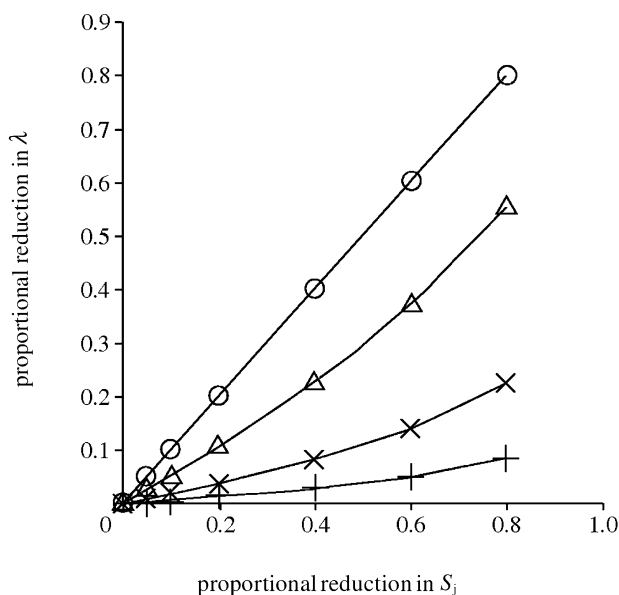


Figure 1. The proportional reduction in λ resulting from a given proportional reduction in juvenile survival (S_j) for different life-cycle types. Circles, benthic macroinvertebrate life cycle; crosses, fish life cycle; pluses, daphnid life cycle; triangles, algal life cycle (after Forbes *et al.* 2001a).

communities of varying life-cycle distributions. Nine scenarios were simulated in which the sensitivity of the life-cycle types and their proportions in the community were varied. For all of these cases, the sensitivity distributions based on juvenile survival gave lower effect concentrations (i.e. the concentration at which a defined percentage of the species was negatively affected) than the distributions based on λ and would therefore tend to provide an added measure of protection if used for risk assessment. However, the proportions of life-cycle types in a community must vary across communities and may have an important influence on sensitivity distributions. They therefore need to be given further consideration in ecological risk assessment.

(b) Example 2

Here, we provide two examples as follows: Example 2a illustrates how the same response of an individual demographic trait to a toxicant can have markedly different consequences on population growth rate as a result of life-cycle differences; Example 2b illustrates how very different responses of individual demographic traits to a toxicant can result in similar consequences for population growth rate as a result of life-cycle differences.

(i) Example 2a

Forbes *et al.* (2001a) considered the life cycles of the most widely used ecotoxicological test species (i.e. a green algal species, an iteroparous fish, a daphnid and a semelparous benthic invertebrate) and, using a simple two-stage demographic model, estimated the proportional decline in population growth rate (expressed as λ) resulting from a proportional decline in juvenile survival. Figure 1 summarizes the results of these analyses and shows that, for stable populations, the same toxicant-caused reduction in survival

would have very different effects on population growth rate, dependent on the life cycle. Daphnid population dynamics would be the least sensitive to impairments in juvenile survival, followed by the iteroparous fish and the alga, with the semelparous benthic invertebrate dynamics being the most sensitive to any impairments in juvenile survival. For all life cycles it could be shown that, for starting values of λ close to 1, toxicant-caused impairments in survival, fecundity or timing would result in equivalent (benthic invertebrate) or smaller (the fish, daphnid and algal life cycles) impacts on the population growth rate. Figure 1 shows that a 10% reduction in juvenile survival (hereafter referred to as LC_{10}) would result in a 10% reduction in λ for a semelparous benthic invertebrate life cycle, a 5% reduction in λ for a green alga life cycle, a 2% reduction in λ for an iteroparous fish life cycle and only a 0.6% reduction in λ for a daphnid life cycle. Clearly, a chemical having similar effects on juvenile mortality would be expected to have vastly different population-level consequences for these life cycles. In a similar manner, although the benthic invertebrate might have a higher LC_{10} value than the daphnid for a given chemical, its population dynamics could nevertheless be more sensitive. Our analysis indicated that a 5% reduction in juvenile survival of the benthic invertebrate life cycle would have the same effect on λ as an 80% reduction in juvenile survival of the daphnid life cycle.

The degree to which the responses of the individual demographic traits overestimated the impacts on population growth rate varied widely among the life cycles, and this may have important practical implications for risk assessment. An extension of the analysis showed that for starting values of λ that were much greater than 1, the effects on the individual demographic traits could, for at least some of the life-cycle types, result in proportionally greater effects on population growth rate.

(ii) Example 2b

Linke-Gamenick *et al.* (2000) examined the effects of the polycyclic aromatic hydrocarbon, fluoranthene, on survival, reproduction and the development time of three *Capitella capitata* sibling species (I, M, and S) and employed a two-stage demographic model to assess the consequences of the measured effects on population growth rate. In the absence of fluoranthene, the three species differed markedly in life-cycle traits and in λ (Table 2).

The percentage changes in the individual-level variables and λ following exposure to $50 \mu\text{g g}^{-1}$ of fluoranthene during a period of 25 weeks are summarized in Table 3. Without going into too much detail, it is clear that for all species some of the percentage changes in individual-level variables were greater than the percentage changes in λ . By comparing among the species studied, Table 3 shows that there were appreciable differences between the responses in the individual-level variables that were not matched by changes in λ . This can be explained in terms of (i) within species, some changes in individual-level variables acted to increase λ despite impairments in other variables (e.g. in Species I, the time between broods was shortened in the exposed populations; in Species M, the adult survival rate was increased in the exposed populations), or (ii) λ was relatively insensitive to changes in those variables that were impacted by toxicant exposure.

Table 2. Life-history traits of three sibling species of *C. capitata* grown under the same conditions in the laboratory. (Conditions: < 63 μm sediment with 6.6% organic matter, 30‰ seawater, 18 °C, constant darkness. For details on experimental design and sibling species, see Linke-Gamenick *et al.* (2000).)

Trait	Species S	Species M	Species I
Juvenile survival (proportion)	0.20	0.77	0.79
Adult survival (proportion)	0	0.61	0.79
Age at first reproduction (days)	115 \pm 20	58 \pm 5	76 \pm 10
Time between broods (days)	—	11 \pm 4	19 \pm 9
Number of offspring per brood per reproductive individual	10.3	15.6	13.4
Type of larval development	direct	lecithotrophic	lecithotrophic
Population growth rate (λ , d^{-1})	1.05	1.42	1.30

Table 3. Percentage changes in individual-level traits and λ between the control populations of three *Capitella* sibling species and populations exposed to 50 $\mu\text{g g}^{-1}$ of fluoranthene. (The values of λ under control conditions are given in Table 2. Data are from Linke-Gamenick *et al.* (2000).)

Trait	Species I	Species M	Species S
Juvenile survival	- 15.2	- 29.9	50.0
Adult survival	- 20.3	23.0	0
Time to first reproduction	15.7	15.4	0
Time between broods	- 47.2	45.9	0
Number of offspring per brood per reproductive individual	- 9.7	- 32.1	34.0
λ	- 4.6	- 10.5	4.3

It should be noted that we have chosen the concentration range for fluoranthene to illustrate the point that changes in individual-level variables do not necessarily translate into changes in population growth rate. However, at concentrations beyond this range, juvenile survival was markedly reduced and reproduction was completely inhibited in Species S, so that its population growth rate was reduced to zero. By contrast, there was little further effect either on individual-level variables or on population growth rate in the other two species up to a concentration of 95 μg fluoranthene g^{-1} dry weight of the sediment.

As a result of life-cycle differences among species (Table 2), λ is not equally sensitive to changes in each of the individual-level traits. Elasticity analysis can be used to identify particularly sensitive life-cycle types, i.e. life cycles whose population dynamics are very responsive to small changes in the individual survival, reproduction or timing, and to identify, for different life-cycle types, those demographic variables that have the greatest influence on the population dynamics. By combining traditional ecotoxicological measures of chemical effects on survival, reproduction and growth with demographic elasticity analysis, it is possible to tease apart the relative contributions of physiology and life cycle in determining the susceptibility of different species to toxicant exposure. This can be illustrated by considering the elasticities of the three sibling species of *Capitella* that have been estimated from the slopes of $\ln \lambda$ plotted against \ln trait (Caswell 2000, p. 226) and summarized in Table 4. From Table 2, it is clear that Species S has the lowest juvenile survival and fecundity of the three species under unexposed conditions. The analysis summarized in Table 4 indicates further that the life history of this species is such that its population growth rate is approximately twice as sensitive to changes in these traits compared with the other two species. In addition, Species S

was physiologically more sensitive than the other species in that its fecundity was reduced to zero at the highest exposure concentration. The greater sensitivity of Species S to toxicants compared with the other two species results from a combination of physiological and life-cycle differences. In addition, it is known that Species I is more widely distributed, particularly in heavily polluted sediments (Linke-Gamenick *et al.* 2000), than Species S, and this can be explained in the same way.

4. To What Extent Does Density Complicate Any Conclusions Drawn from Observations on the Response of Individuals to Toxicants under Nonlimiting Densities?

(a) Review

When, as is rarely the case, population growth rate is measured in ecotoxicological tests, it is generally done under conditions in which food and space are unlikely to limit population growth. There is concern, therefore, that conclusions drawn from such tests may have little relevance for field situations when populations are regulated by density dependence. Whether the combined effects of density and chemical exposure on individual survival, growth and reproduction interact to produce additive, more-than-additive or less-than-additive effects on population growth rate depends on (i) how, in combination, density and chemical exposure affect individual performance; (ii) the type of density-dependence operating (i.e. scramble or contest); and (iii) the life-cycle type of the species in question. Although the interactions of chemical exposure and density on population growth rate are theoretically predictable, the number of factors influencing the outcome is large and therefore simple, general, *a priori* predictions are not feasible (Forbes *et al.* 2001b). A few simulation stud-

Table 4. Elasticities of three sibling species of *Capitella* estimated from the slopes of relationships between each individual trait and λ (both on a ln scale), while holding all other traits constant (Caswell 2000, p. 226) and using a simple two-stage life-cycle model (Calow *et al.* 1997). (The values were rescaled so that the elasticities for each species summed to 1.)

Elasticities	Species I	Species M	Species S
Juvenile survival	0.199	0.210	0.391
Adult survival	0.048	0.040	n.a.
Time to first reproduction	-0.465	-0.477	-0.218
Time between broods	-0.089	-0.065	n.a.
Number of offspring per brood per reproductive individual	0.199	0.208	0.391

n.a. = not applicable.

ies have been performed, and these indicate that density is likely to ameliorate the effects of chemicals on population growth rate (Grant 1998; Hansen *et al.* 1999b). Experimental studies have produced mixed results, with some showing additive interactions between density and chemical effects on population growth rate (Winner *et al.* 1977; Klütgen & Ratte 1994), whereas others have found less-than-additive effects (Marshall 1978) or more-than-additive effects (Chandini 1988). There is even some indication that the form of the interaction may vary across a chemical concentration gradient, with effects shifting from less-than-additive at low-toxicant concentrations to more-than-additive at higher toxicant concentrations (Linke-Gamenick *et al.* 1999). As recent work on the effects of pulsed pesticide exposures on freshwater trichopteran populations has demonstrated, compensatory interactions between population density and toxicants on population dynamics can persist throughout a cohort's life cycle even if the period of toxicant exposure is very brief (Liess 2002). In addition, it appears that the kind of interaction that may be observed is, to an important extent, constrained by the kind of experimental design employed (Forbes *et al.* 2001b). The design most likely to approximate natural conditions is the so-called "bucket test" (Sibly 1999) in which populations are initiated with different combinations of food and chemical exposure and assayed over time. We illustrate in the example below how this might be put into practice.

(b) Example 3

Forbes *et al.* (2002) used a bucket-test design to explore the interaction between population density and toxicant effects in the polychaete, *Capitella* species I. Populations of worms were initiated with a stable age distribution and with different combinations of food availability and exposure to fluoranthene (0, 50, and 150 $\mu\text{g g}^{-1}$ dry weight sediment). The experiment was conducted over a period of 28 weeks. Further details of the experimental design can be found in Forbes *et al.* (2002). Figure 2 plots population growth rate against log density for control populations and those exposed to 50 $\mu\text{g g}^{-1}$ fluoranthene (populations exposed to 150 $\mu\text{g g}^{-1}$ fluoranthene became extinct by the eighth week of exposure). The results indicate that increasing population density ameliorates the effect of fluoranthene on population growth rate, and similar though weaker amelioration by high-population density of the effects of a toxicant were obtained for *Tisbe battagliai* exposed to pentachlorophenol (Sibly *et al.* 2000). Thus, in these particular cases, tests carried out under non-density-limiting

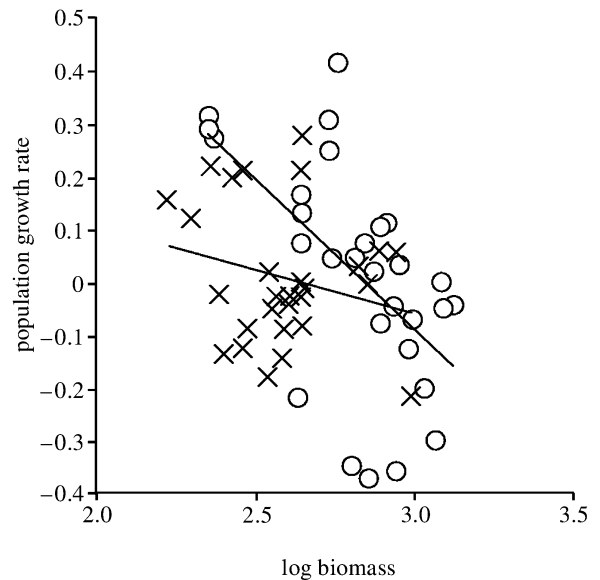


Figure 2. Population growth rate (r , week $^{-1}$) as a function of (Log transformed) population biomass in control populations (\circ) and populations exposed to 50 μg fluoranthene g^{-1} dry wt sediment (\times). The lines are linear regressions through the data. Control (\circ): population growth rate = $1.63 - 0.57$ (Log biomass), $n = 30$, $r = 0.57$, $p = 0.001$. 50 μg fluoranthene g^{-1} dry wt sediment (\times): population growth rate = $0.44 - 0.17$ (Log biomass), $n = 30$, $r = 0.24$, $p = 0.20$ (after Forbes *et al.* 2002).

situations would tend to overestimate the effects of the toxicants at high density, which might be closer to natural conditions. However, as the review indicated, this conclusion should not be taken to be a general one, and it underlines the point that density effects need to be taken more seriously in the design of ecotoxicological tests.

5. Conclusions

We have shown that individual-level variables are equally or more sensitive to increasing concentrations of toxic chemicals than is population growth rate. Hence, the concern that small effects on individual survival, growth or reproduction are magnified into large effects on populations is not supported by the available data. This is an important message for environmental protection given the large number of chemicals that have to be considered. However, the validity of relying on individual-level endpoints depends on the most sensitive variables always be-

ing measured. Due to the fact that these vary across species and chemicals, it is not feasible to identify which variables will be generally the most sensitive to toxicants, or the best general predictors of population growth rate *a priori*. Moreover, differences in life cycles across species mean that similar effects of chemicals on individual-level variables in different species can have vastly different consequences for population growth rate. Alternatively, very different effects on individual-level variables can sometimes translate into similar or no differences in effects on λ among species. Finally, the effects of toxicants in situations involving density limitations can differ from the effects recorded under low-density circumstances (as is usually the case in ecotoxicological tests) and it is not straightforward to predict actual outcomes *a priori*. More attention, therefore, needs to be given to the inclusion of appropriate and realistic density conditions in test scenarios.

Clearly, if we want to know more about the effects of toxic chemicals on population dynamics then we need to carry out more work on the relationships between individual-level responses and population growth rate under increasing toxicant concentrations. Moreover, if for particular chemicals we want to develop more ecologically relevant risk assessments then this should be done in terms of population growth rate rather than just observations on individual-level responses. Population growth rate analysis can, in principle, also indicate which species within communities will be the most or least susceptible to chemical pollution and which, after a pollution event, will be the most or least likely to recover and at what relative rates. For example, work with the polychaete *Capitella* would indicate that there are large differences among sibling species in their ability to persist in and recolonize polluted habitats and that these differences are at least partly due to life-cycle differences among species (Linke-Gamenick *et al.* 2000). For the species that are not amenable to laboratory testing, modeling the effects of toxicants using population growth rate analyses could be used to characterize their relative susceptibility to different toxicants.

These kinds of analyses have more general implications for understanding the ways that individual-level variables contribute to population dynamics. For example, in pest control, understanding how population growth rate responds to manipulations of different parts of the life cycle can enable the development of more effective control programs (e.g. McEvoy & Coombs 1999). A similar case can be made for the development of effective conservation strategies (see Sutherland & Norris 2002).

In conclusion, with appropriate time and resources, population growth rate analysis should form the basis of ecological risk assessment. It is desirable to incorporate more detail into models with regard to individual-level variables and the ecological context in which the species exist and pollution occurs. However, it will rarely be possible to conduct population growth rate analysis for all species and chemicals to a sufficient level of detail. One possible solution to this is to use population growth rate analysis to identify the most vulnerable species and to focus our assessments on them. Not only could this approach be useful for carrying out ecological risk assessments, but it could also contribute to the development of conservation strategies.

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