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TOXICANT IMPACTS ON DENSITY-LIMITED POPULATIONS: A CRITICAL REVIEW OF THEORY, PRACTICE, AND RESULTS

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Abstract. Most natural populations experience some density dependence, and long-term average rates of population growth are likely to be close to zero (i.e., steady state). An essential question, therefore, is how and to what extent do density-dependent effects influence the responses of populations to toxicant impacts? Here we consider three general types of interaction between density dependence and toxicant effects: additive, less than additive, and more than additive. If we know enough about the life-history dynamics of an organism and how its life-history traits are affected by density and toxicant exposure, we should be able to use life-history models to predict responses of populations living under density-dependent control to chemical exposure. However, because the number of factors influencing the outcome is large, we demonstrate that simple, general, a priori predictions are not feasible. A review of the literature confirms that a variety of interactions has been observed in experimental systems. It is essential that experiments are designed so that the interactions of interest can be determined. We review these critically. Experimental designs that are appropriate for exploring density–toxicant interactions on individual physiological performance may be unable to detect potential compensatory interactions at the population level. Designs most likely to simulate the dynamics of field populations will be unable to determine the mechanistic bases underlying population-level impacts. To facilitate an ecologically correct interpretation of density–toxicant interactions, it is therefore essential that the limitations of the chosen experimental designs be recognized and made explicit.

Key words: compensation; density dependence; ecotoxicology; experimental design; food limitation; population dynamics.

INTRODUCTION

A major criticism of standard ecotoxicological tests is that endpoints are measured in individuals, and there are uncertainties as to how effects at this level will translate into effects on populations. One concern is that small, statistically undetectable effects on one or more individual life-history traits (e.g., survival, developmental rate, reproduction) will be magnified into large impacts on population dynamics (Halbach et al. 1983). Alternatively, it has been suggested that exposing a density-limited population to a toxicant could, perhaps by removing a fraction of individuals, reduce the intensity of density dependence and therefore have less of an impact on population dynamics than if the population had been in exponential growth. This could occur if the toxicant removed a random selection of individuals, but could be particularly so if the toxicant acted nonrandomly and removed the weakest or least fit individuals. Indeed, such a buffering effect was hypothesized by Calow et al. (1997).

Forbes and Calow (1999) reviewed the literature in which toxicant effects on individual life-history traits and population growth rate were examined simultaneously. The review included a total of 28 species and 44 toxicants. There was no evidence that effects of toxicants on individual life-history traits were magnified at the population level. On the contrary, population growth rate (PGR, expressed either as r, the per capita rate of increase, or λ, which is e^r) was equally or less sensitive to toxicant exposure than the individual life-history traits contributing to it. However, there was no consistent relationship between which traits were most or least sensitive, and therefore no generally predictable relationship between effects on single life-history traits and PGR. In addition, a potentially important limitation in most of these studies is that the life-table-response measurements were performed, with few exceptions, under conditions in which population density did not influence population growth rate.

Population dynamics theory predicts that at low density populations grow exponentially at the per capita rate of increase r, but as density increases and competition between individuals intensifies, growth rate will decline until the carrying capacity for the population is reached. The growth rate will then maintain an average of zero, but with varying degrees of fluctuations around this value, especially initially. Most natural populations experience some density depen-

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dence, and long-term average rates of population growth are likely to be close to zero (i.e., steady state). Here we consider how and to what extent density-dependent effects influence the responses of populations to toxicant impacts.

In the first section we review the theory relating to individual- and population-level interactions. Then we consider in turn results from simulation and experimental studies. A main conclusion is that a wide range of interaction scenarios is possible and that experimental work is important in establishing what outcomes are likely in particular circumstances. In the final section, therefore, we pay attention to experimental designs and recommend which should be used to address specific questions.

Individual- vs. Population-Level Interactions: Theory

We presume for the following discussion that resources are shared equally among individuals in populations such that all individuals suffer as density increases, i.e., scramble competition (Akçakaya et al. 1999). A common example of this type of competition is competition for food, and much of the following discussion will focus on food limitation as the main density-dependent effect. However, a future step would be to take into account contest competition (i.e., unequal sharing of resources) in which winners are not affected and losers lose all.

In considering the possible ways in which density and toxicants might interact to influence the population dynamics of organisms we consider three general types of interaction: additive, less than additive, and more than additive. In the additive model, the shapes of the PGR vs. toxicant concentration curves would be parallel at high and low densities because the effects of each factor are independent (Fig. 1). In the less-than-additive model the high- and low-density curves would converge with increasing toxicant exposure because the density reductions caused by increasing toxicant concentrations would be compensated by an amelioration of density-dependent effects. In the more-than-additive model the high- and low-density curves would diverge with increasing toxicant exposure because stress caused by exposure to the toxicant exacerbates the density-dependent effects. Note that although in Fig. 1 we have drawn the curves as straight lines, they need not be. But likewise for nonlinear curves the extent to which they are parallel/converge/diverge indicates additivity/less than additivity/more than additivity. In addition, it is important to remember that whether or not the curves are additive is dependent on the choice of PGR measure. For instance, effects that are additive when PGR is measured as λ are more than additive if PGR is measured as *r*, and conversely, effects that are additive on an *r* scale are less than additive on a λ scale.

The mechanisms giving rise to effects on population

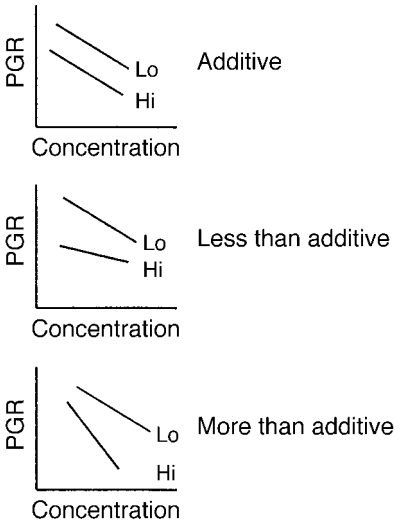

FIG. 1. Possible interactions between density-dependent effects and toxicant exposure on population growth rate, PGR. ''Lo'' and ''Hi'' refer to low density and high density, respectively. The slope of the low-density curves has been held constant for the three cases; as there is no density effect, the slope represents the effect of toxicant exposure alone.

dynamics are partly the result of interactions of density and toxicant exposure occurring at the individual level (which can be additive, less than additive, or more than additive) and partly the result of the nonlinear form of the Euler-Lotka equation relating individual performance to population growth rate. For example, food limitation and toxicant stress may have additive effects on time to first reproduction (individual level), but because time to first reproduction relates to PGR as an exponent in the Euler-Lotka equation, the combined effect of food limitation and toxicant exposure on PGR may be more than additive. Density and toxicant stress may alter more than one aspect of individual performance (juvenile- or adult survival (S_j and S_a, respectively), reproductive output (n), time to first reproduction (t_j), or time between broods (t_a)) simultaneously. Although effects on each might be additive, when the effects are combined to calculate PGR a different pattern may emerge at the population level. Assuming that there are three kinds of interaction possible at each of the individual and population levels, there is a total of 3^2, or nine, possible combinations.

At the individual level food limitation and toxicant exposure both can be expected to have negative effects on organism performance. If they act independently, then the effects on an organism exposed to both types of stress would be the sum of effects on an organism exposed to either stress alone (effects would be additive). Alternatively, food limitation might physiologically impair individuals (e.g., prevent them from synthesizing adequate amounts of detoxification enzymes) such that they become increasingly susceptible to toxicant exposure. Effects on an organism exposed to tox-

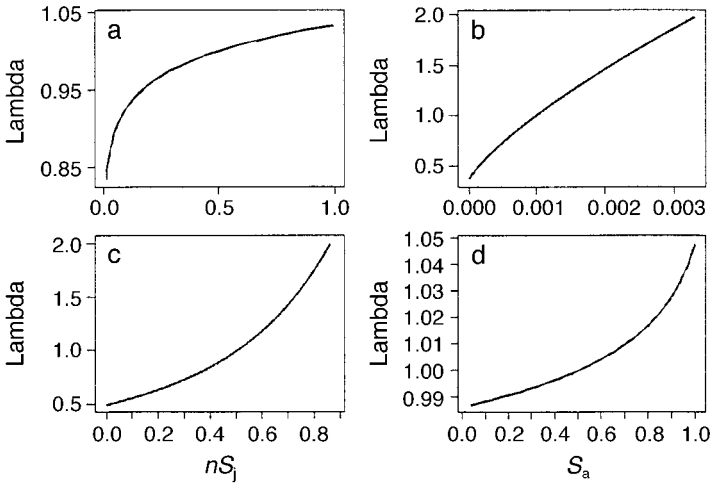

FIG. 2. Lambda plots may be concave or convex depending on the life history. In (a) and (c) λ is plotted against the number of surviving female offspring, nS_j. In both cases adult survivorship, $S_a = 0.5$, but in (a) age at first breeding $t_j = 0.2$, and in (c) $t_j = 20$; n represents the number of female offspring, and S_j represents juvenile survivorship. In (b) $nS_j = 0.999$ and $t_j = 0.001$; in (d) $nS_j = 0.5$, $t_j = 50$. Life-history parameters were chosen to give clearly contrasted curves. Curves were plotted using the two-stage Euler-Lotka equation, in the form $1 = nS_j\lambda^{-t_j} + S_a\lambda^{-1}$.

icants in a food-limited condition, following this scenario, would be greater than the sum of effects on an organism exposed to either food limitation or toxicant exposure alone (effects would be more than additive). A third scenario could occur if toxicant stress and density stress operate on the same life-history trait but in opposite directions as this could potentially have an ameliorating effect on individuals exposed to both (effects would be less than additive). For example, food limitation might cause organisms to slow their metabolism and hence to reduce their uptake and metabolic transformation of toxicants. In this case food-limited individuals could appear less sensitive to toxicant exposure than their well-fed counterparts.

Sibly and Calow (1989) used an energy-budget approach to calculate optimal stress responses for individuals. For the case in which the trade-off curve relating mortality to growth rate does not change shape when stress is applied, then the optimal strategy is to spend less on personal defense when stressed. If such an individual is food limited, it will defend itself less, so the chemical will damage it more than when it has plenty of food. The combined effects of chemical exposure and food limitation will then be more than additive. If, however, the trade-off curve relating mortality to growth changes shape in response to toxicant exposure, additive or less-than-additive effects could occur.

Kooijman and Metz (1984) developed a model for age-dependent growth and reproduction of individuals as a function of food supply and used this to predict effects of toxic chemicals on population growth rate. Their model assumed a Holling-type functional response with von Bertalanffy growth, a fixed ratio between energy used for reproduction and respiration, and a juvenile stage that lasts until the individual reaches a threshold body mass. The model predicted that chemical impairment of individual feeding, digestion, basal metabolism, and survival would have more serious impacts on the growth rate of food-limited populations. However, the population-level consequences of chemical effects on individual growth and reproduction were largely independent of food level (i.e., additive).

Whether the combined effects of density and toxicant stress on individual performance are translated into additive, less-than-additive, or more-than-additive effects at the population level, depends not only on the form of the interaction at the individual level but also on the life-history type of the study organism as well as on which life-history traits are affected. Although the relationship between PGR and the individual life-history traits contributing to it is predictable, e.g., by the Euler-Lotka equation, the sensitivity of PGR to changes in the different traits is not obvious. It appears that the sensitivity of PGR to changes in the traits contributing to it depends on (1) the starting value of PGR, (2) the absolute values of the traits (i.e., the life-history type of the organism), and (3) the relative responsiveness of the different traits to toxicant exposure (Calow et al. 1997, Hansen et al. 1999; Forbes et al. 2001).

To demonstrate how such factors might influence PGR, we consider some simple examples. If food limitation and chemical stress at the individual level act additively on juvenile survival, S_j, the effects on λ depend on the curvature of λ plotted against S_j. The curve may be concave or convex depending on the life history, as in Fig. 2. If the curve is concave seen from below, then additive decrements in survival have a more-than-additive effect on λ (Fig. 2a, b). Hence concavity seen from below corresponds to more-than-additive effects. Conversely, convexity produces a less-than-additive effect (Fig. 2c, d). Only if the λ plot is a straight line is the result λ additivity. These examples show that no general inferences can be made as to whether effects on λ are additive, more than additive, or less than additive from the nature of effects on individual life-history traits.

The argument can be formalized by calculating the second derivatives of λ with respect to individual life-history traits. Consider for instance how changes in

adult survivorship, S_a, influence λ. Convexity of the λ plot is equivalent to less-than-additive behavior of λ, and this corresponds to $\partial^2\lambda/\partial S_a^2 > 0$. Similarly concavity/more-than-additivity corresponds to $\partial^2\lambda/\partial S_a^2 < 0$, and linearity/additivity to $\partial^2\lambda/\partial S_a^2 = 0$. Which λ-outcome occurs depends on the sign of $\partial^2\lambda/\partial S_a^2$, and this can be calculated from the Euler-Lotka equation. In general the sign of $\partial^2\lambda/\partial S_a^2$ depends on the life history, as in the examples in Fig. 2.

What these simple examples show is that if we know enough about the life-history features of an organism and how its life-history traits are affected by food limitation and toxicant exposure, we should be able to predict responses of populations living under density-dependent control to chemical exposure. However, because the number of factors influencing the outcome is large, it is unlikely that simple, general, a priori predictions will be feasible. At least for the near future it is likely that we shall have to approach this problem experimentally, and it is essential that our experiments are designed so that the interactions of interest can be determined.

To date, the literature dealing with toxicant–density interactions on population dynamics is limited. In the next section we examine this literature and consider the results of both simulation studies and experimentally measured responses. Following this analysis, we evaluate the different general types of experimental protocols that have been used for exploring density–toxicant interactions and make recommendations as to how experiments should be designed in order to test these interactions more rigorously.

Results from Simulation Studies

The results of simulation studies have suggested that toxicants are likely to have less of an effect on density-limited populations than on exponentially growing populations. For example, Grant (1998) simulated two types of density dependence and explored the consequences of simulated effects on previously published life-table-response data for *Eurytemora affinis* exposed to dieldrin. Rather than examining effects on population growth rate, his analysis focused on equilibrium population size. His argument was that if a population at stable equilibrium is exposed to a toxicant so that individual performance is impaired, PGR would initially become negative, but that if exposure is continued the population would stabilize at a different equilibrium, and the value of PGR would return to zero. Therefore he argued that PGR is not a very helpful measure of population impacts, and that one should instead compare equilibrium population sizes. He considered first the case in which juvenile survivorship declines with increasing density and then a case in which fertility declines exponentially with population size. He found that the life-history traits that make the greatest contribution to dieldrin effects are altered by the form of the density dependence and that life-history traits later in life are more important than in the density-independent scenario. An important conclusion from this analysis is that substantial reductions in some life-history traits by toxicant exposure can have little impact on the population if they are compensated for by reductions in the intensity of density dependence. In other words, density dependence will act to buffer the effects of toxicant exposure on population dynamics, consistent with a less-than-additive model.

Hansen et al. (1999) simulated potential influences of competition and predation on population-level response of the polychaete *Capitella capitata* (Type I) to 4-*n*-nonylphenol. By considering scenarios in which fecundity was reduced, time to maturity was extended, and/or juvenile survival was reduced by realistic amounts, the authors explored how the sensitivity of population growth rate to toxicant-caused changes in individual life-history traits was influenced. They found pronounced changes in the relative sensitivity patterns among scenarios, which indicated that negative effects of nonylphenol on population growth rate were buffered when additional negative (i.e., density-dependent) influences operated on the individual life-history traits, again consistent with a less-than-additive model.

Results from Experimental Studies

Although a relatively large number of studies has explored the combined effects of food limitation and toxicant exposure on different aspects of individual performance, relatively few experiments have considered interactions of these effects on population dynamics. Here we present some examples of experiments performed with aquatic invertebrates since our aim is to see whether empirical evidence can be found for the different forms of relationship between density dependence and toxicant effects on populations, and not to perform a complete literature review on this topic.

Winner et al. (1977) examined the sensitivity of *Daphnia magna* to chronic copper stress under two food regimes, namely vitamin-enriched algae and trout granules. In the absence of copper the two diets yielded equivalent PGR values (measured as *r*); longevity and the number of broods were greater on the algal diet, brood size was greater on the trout diet, and time to first reproduction was equivalent on the two diet types. On the algal diet, exposure to copper increased time to first reproduction at concentrations >60 μg/L, reduced longevity significantly at concentrations >40 μg/L, increased brood size at concentrations of 20, 40, and 60 μg/L, and led to reduced PGR at concentrations >60 μg/L. On the trout diet, in contrast, exposure to copper increased time to first reproduction at concentrations >20 μg/L, reduced both longevity and brood size significantly at concentrations of ≥20 μg/L, and led to reduced PGR at concentrations >40 μg/L. Visual inspection of trends in these data (Fig. 3a–d) suggests that interactions between food quality and copper ex-

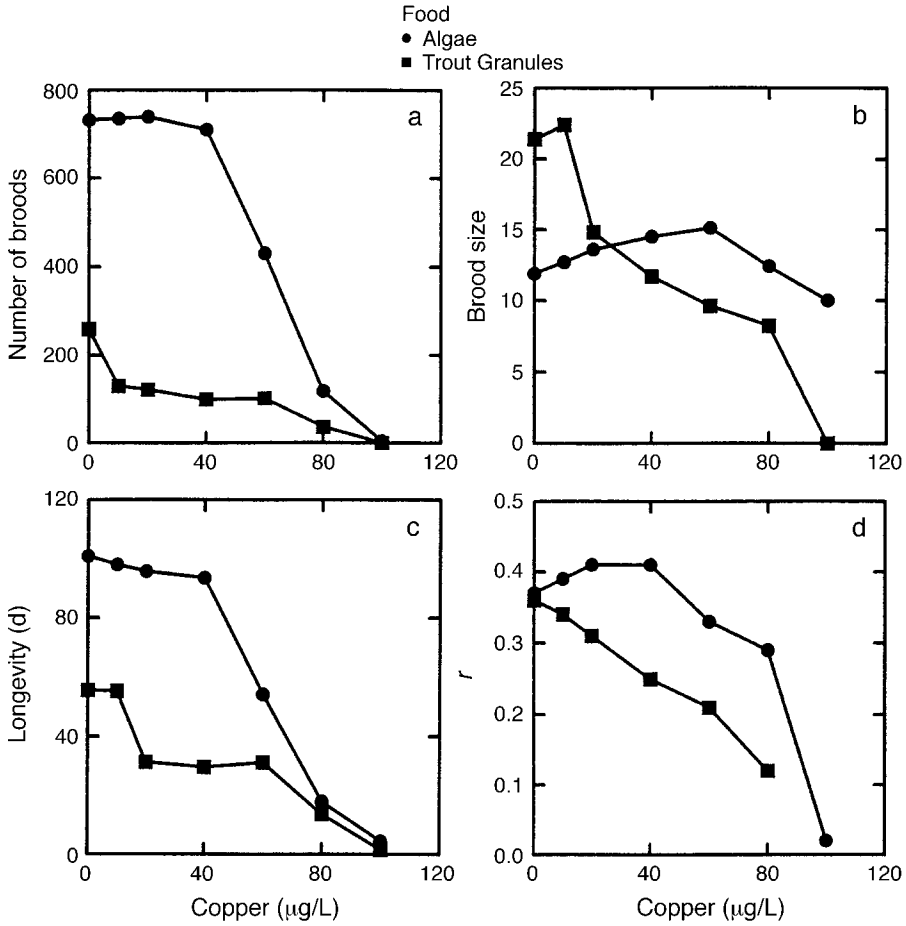

FIG. 3. The sensitivity of *Daphnia magna* to chronic copper stress under two food regimes, namely, vitamin-enriched algae and trout granules. Data are from Winner et al. (1977).

posure are less than additive for longevity and brood number, are more than additive for average brood size, and are near additive for PGR.

Marshall (1978) examined the effects of density dependence and toxicant exposure in *Daphnia galeata mendotae* populations exposed to cadmium. He found that toxicant-caused reductions in population density resulted in increased food supply per surviving individual. This was reflected in increased brood size per female and an increase in the proportion of egg-bearing females. Thus, because cadmium exposure reduced the strength of density-dependent effects, the interaction of density and cadmium with respect to PGR (measured as *r*) was less than additive.

Chandini (1988) studied the effect of different food levels on the chronic toxicity of cadmium to *Echinisca triserialis.* Low food levels and high Cd stress resulted in a delay in age at first reproduction, a decrease in age-specific fecundity, and a low frequency of reproduction, all of which contributed to a decrease in PGR (measured as *r*). He concluded that the high energetic costs of maintenance caused by Cd stress could reduce energy available for somatic growth and reproduction, particularly when food intake is low. Thus, the implication here was that food limitation would exacerbate the effects of toxicant stress at the population level. Visual inspection of the data (Fig. 4a–d) suggests that both additive (longevity) and more-than-additive (total fecundity, time to first reproduction) interactions describe the responses of different individual-level traits, whereas the interaction of food and Cd with respect to *r* is more than additive.

Klüttgen and Ratte (1994) examined the effects of Cd on *Daphnia magna* reared under four food levels. Visual inspection of their data (Fig. 5a–d) suggests more-than-additive effects on time to first reproduction (i.e., the delay in development was more pronounced at low food), less-than-additive effects on body length and brood size, and additive (or slightly more than additive if the lowest food level is included in the comparison) effects on *r*.

Linke-Gamenick et al. (1999) examined the effects of the polycyclic aromatic hydrocarbon fluoranthene on individual life-history traits and PGR (measured as λ) of *Capitella capitata* (type M) cultured at different densities. They found that, with respect to both indi-

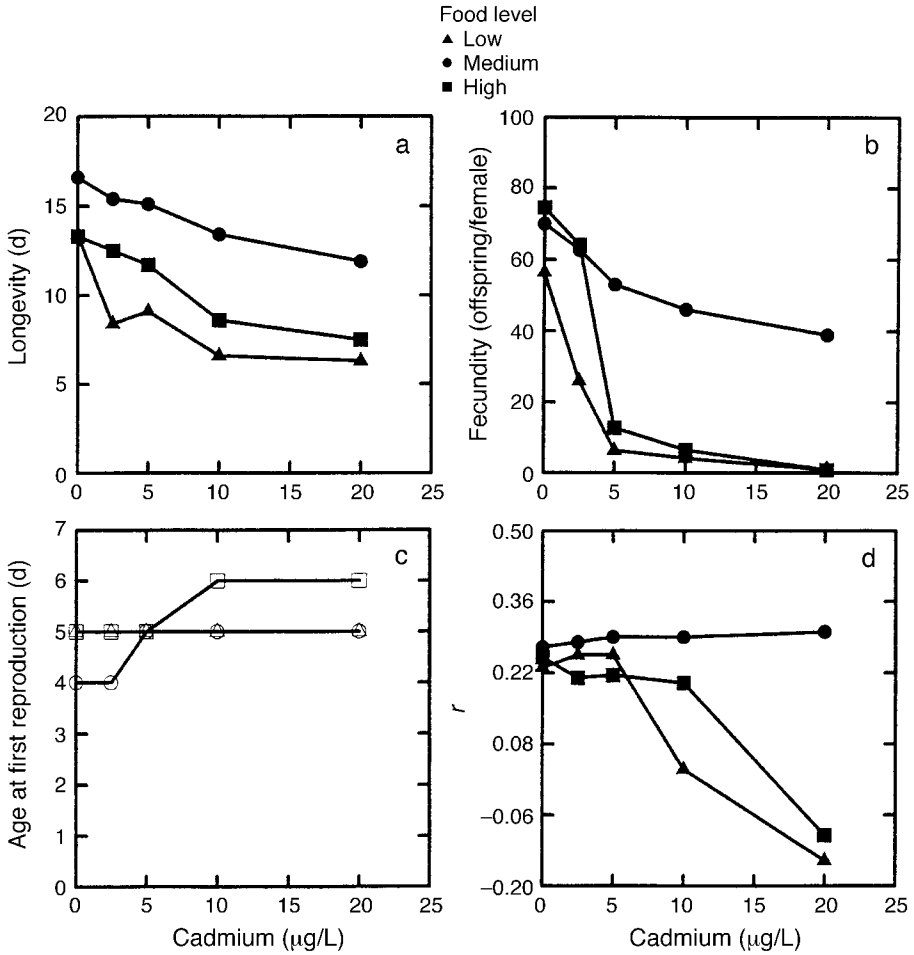

FIG. 4. The effect of different food levels on the chronic toxicity of cadmium to *Echinisca triserialis.* Data are from Chandini (1988).

vidual-level traits and λ, interactions between fluoranthene and population density appeared to be less than additive at low toxicant concentrations but more than additive at the highest fluoranthene concentration. Thus, although effects of low concentrations of fluoranthene on individual performance and population dynamics could only be detected for the low-density treatment, high-density populations were more likely to go extinct at lower fluoranthene concentrations than low-density populations. The results suggest that compensation at the population level (i.e., a less-than-additive interaction between density and toxicants) can only occur if the remaining individuals are in good enough physiological condition that they can continue to grow and reproduce. As toxicant concentration increases feeding rates may become impaired, and those individuals surviving toxicant exposure may be unable to benefit from an increasing food supply. Thus, we hypothesize that the form of the density–toxicant interaction may switch from less than additive to more than additive when exposure concentration reaches the level at which feeding rate is impaired.

Sibly et al. (2000) investigated effects of population density, food quality, food concentration and pentachlorophenol (PCP) on population growth rate and carrying capacity of populations of the marine copepod *Tisbe battagliai.* Most of their results showed additive effects, however there was a suggestion of less-than-additive effects of PCP on PGR. At higher food concentrations PCP reduced PGR at low population density but had little or no effect on PGR at population carrying capacity.

Cecchine and Snell (1999) examined the combined effects of food level and either PCP or mercury on PGR in the rotifer *Brachionus calyciflorus.* The slopes of the relationship between *r* and toxicant concentration were steeper in the low food than in the high food treatments for both toxicants, indicating more-than-additive effects. Rotifer populations exposed to either of the toxicants required several-fold higher food levels to maintain population size, compared to unexposed populations.

Clearly, even this limited selection from the experimental literature demonstrates a variety of interaction

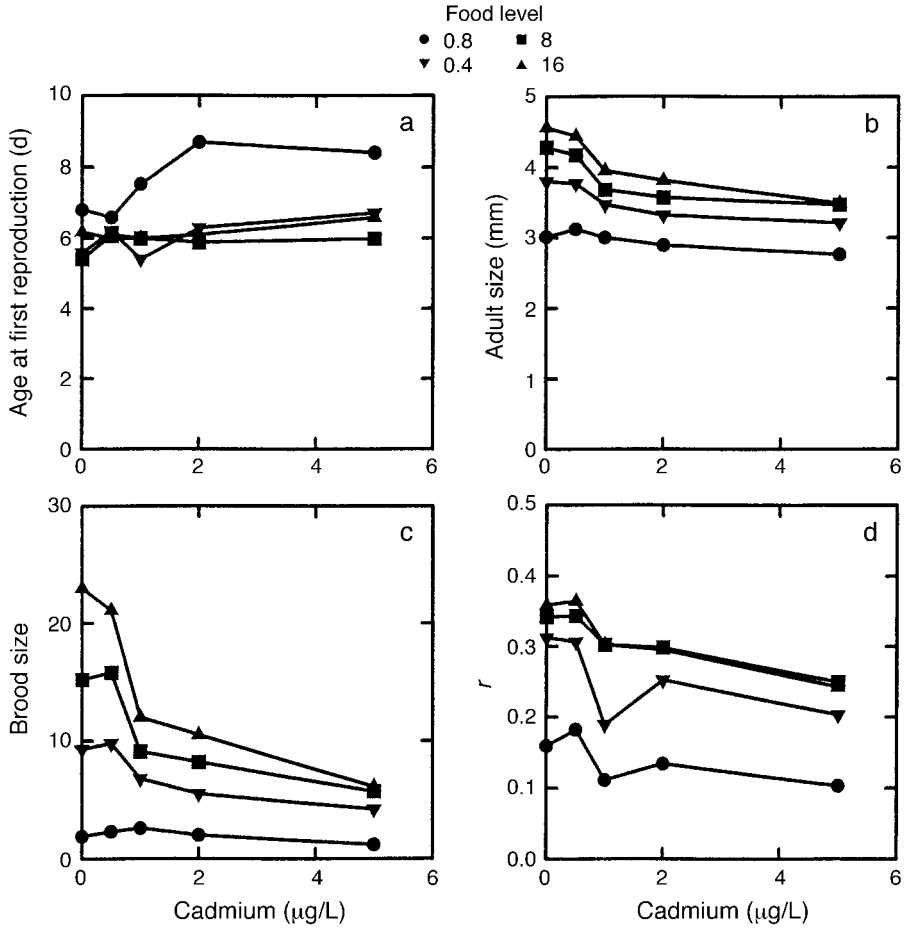

FIG. 5. The effects of cadmium on *Daphnia magna* reared under four food levels. Data are from Klüttgen and Ratte (1994).

possibilities at both the individual and population levels. In attempting to identify general rules for when the different patterns may apply it is important to consider the extent to which a given interaction is observed as a result of the biology of the system or as a result of constraints of the experimental design. We proceed to consider these issues in detail with an aim toward clarifying the advantages and limitations of different experimental approaches for studying density–toxicant interactions.

THE IMPORTANCE OF EXPERIMENTAL DESIGN

For exploring the effects of population density on toxicant responses several experimental designs have been employed in the literature. Which design is chosen limits the possibilities for interactions to occur and thus has a critical influence on the types of conclusions that may be drawn from the results.

Design 1.—Tests of individual performance (e.g., Klüttgen and Ratte 1994). In this design adults are replaced upon their death so that density remains at its starting value and offspring are removed as they are produced. Such a design can effectively be achieved either by exposing individuals to different levels of food and toxicant (the most common design in the literature) or by adding different numbers of individuals to a single fixed amount of food at different toxicant exposures. This design is most appropriate when the question of interest is ''How do the combined stresses of toxicants and population density (i.e., food limitation) influence the physiological performance of individuals?''. The weakness of this design for exploring density–toxicant interactions at the population level is that the questions that can be explored are limited to those not involving stress-induced changes in density. In practice this means avoiding cases in which stress affects survivorship. If stress lowers survivorship, then in this design dead animals are immediately replaced, so any effects of lowered density cannot be observed.

Design 2.—Life-table response experiments (LTREs) (e.g., Linke-Gamenick et al. 1999). In this design the effects of density–toxicant interactions on the dynamics of exponentially growing populations are tested. Groups of individuals are cultured at different food levels and toxicant exposures; there is no replacement of adults upon their death so that density is allowed to fluctuate in re-

sponse to toxicant exposure, but offspring are removed as they are produced. This design is useful for obtaining information on the mechanistic bases of density–toxicant interactions on population dynamics. Because density is allowed to vary from its starting condition in response to treatment effects, this design allows testing of feedback between the effects of toxicant-caused reductions in density on the performance of individuals and, by calculation, on the per capita rate of increase of the population. This design also allows some insight into links between individual performance and population dynamics. However, since offspring are removed from the system, the natural development of the population, with regard to achieving a stable age distribution and attaining an equilibrium population size, is disturbed. Estimates of PGR from LTREs therefore do not predict actual growth rates of populations, but are considered to project what the population would do if the environment were to remain constant at its present state (Caswell 1996).

Design 3.—''Bucket'' or mesocosm experiments (e.g., Sibly 1999, Sibly et al. 2000). In this design density–toxicant interactions on equilibrium population size are examined. Groups of individuals are cultured at different food levels and toxicant exposures; there is no replacement of adults upon their death (cf. Design 2), and juveniles are allowed to remain in the system. In this design population size is assayed over time, population growth rate can be measured as changes in number of individuals over a fixed period of time, and changes in equilibrium population size as a function of toxicant exposure estimated. Because this design does not involve artificial replacement or removal of individuals, it is probably the best design for simulating the likely effects of density and toxicants on field populations (given certain assumptions, e.g., about predators and migration) in a cost-effective manner (Sibly 1999). However, this design is not appropriate for determining the mechanistic bases of toxicant–density effects that link individual performance to population dynamics. Starting conditions, such as whether the population is initiated with juveniles, adults, or with a stable age distribution will also influence population development and need to be considered carefully (Stark and Banken 1999). Decisions about sampling frequency and protocol (i.e., harvesting entire replicates or subsampling replicates repeatedly over time) will also have to be made on a case-by-case basis.

Recommendations and Conclusions

Our analysis suggests that the observed interactive effects of population density and toxicant exposure on population dynamics are critically dependent on the details of study design. Designs that are appropriate for exploring the effects of interactions on individual physiological performance are unable to detect potential compensatory interactions at the population level. Designs most likely to simulate the dynamics of field populations will be unable to determine the mechanistic bases underlying population-level impacts.

Whereas theoretical calculations based on life-history models (see case for S_a given above) indicate that virtually all types of interactions between toxicants and density dependence are possible, intuitive arguments (Calow et al. 1997) and simulation studies (Grant 1998, Hansen et al. 1999) have suggested that compensatory interactions, and hence buffering of toxicant effects on the dynamics of density-limited populations are likely. Results of experimental studies are mixed: Winner et al.'s (1977) results and Klüttgen and Ratte's (1994) results suggest additive effects on r, Marshall's (1978) results suggest less-than-additive effects on r, and Chandini's (1988) results suggest more-than-additive effects on r. Linke-Gamenick et al.'s (1999) results suggest that the type of interaction that occurs may vary with increasing toxicant concentration, with effects on λ shifting from less than additive at low toxicant concentrations to more than additive at higher toxicant concentrations. This has important implications for predicting the likelihood of extinction of toxicant-exposed populations. Whereas impacts of initial or low levels of toxicant exposure may be less detectable in populations whose growth is already depressed as a result of density limitation, these populations may be more likely to go extinct as toxicant exposure increases.

An important consideration relevant to all of the above designs is the need to manipulate food availability independently from toxicant exposure. Many toxicants of environmental concern are particle reactive and will therefore tend to bind to surfaces, including food-particle surfaces. If toxicant uptake occurs primarily from the dissolved phase, increasing food level can result in a greater fraction of the added toxicant becoming surface-bound and therefore not bioavailable. For deposit feeders, whose uptake of contaminants often occurs primarily via ingested sediment (Selck et al. 1998), designs that manipulate food level by adding/removing organic material to/from sediment can also alter the degree and strength of contaminant binding to the sediment, and hence contaminant bioavailability.

We note here an added complication. It has been assumed throughout that the effects of toxicants and density on organism performance and thus population dynamics are generally negative. However, as Calow et al. (1997) pointed out, if the effects of either of these factors is on timing (e.g., lengthening time to first reproduction), then whether positive or negative effects on population dynamics occur may depend on whether the population is growing or shrinking (i.e., on the sign of the starting value of r).

Several important questions remain. Are similar patterns of interaction likely to occur for equilibrium population size as well as population growth rate? Does the type of interaction vary as a function of toxicant

concentration (as shown by Linke-Gamenick et al. 1999) in general, and if so, can the toxicant concentration at which the shift from less-than-additive to more-than-additive interactions occurs be predicted on theoretical grounds? To what extent is the effect likely to differ for different life-history types? Much more experimental work with a carefully selected range of life-history types and toxicants is needed before these questions can be adequately answered. But such answers are essential for improving our ability to predict the consequences of toxicant exposure on natural populations. In addressing them it is important to recognize that our choice of experimental design influences the types of interactions that can be detected. Further consideration (both theoretical and experimental) needs to be given to distinguishing the effects of scramble vs. contest competition. We conjecture that contest competition is more likely than scramble competition to buffer the effects of toxicants on populations (i.e., show less-than-additive interactions). The reason for this is that contest competition is more stabilizing such that when population size increases above carrying capacity, competition for the limited resource reduces population size back exactly to carrying capacity, and no further. Hence if there is contest competition and survivorship is reduced by a toxicant, there will still be stabilization as long as the toxicant does not reduce survivorship below carrying capacity. Below carrying capacity there would be no contest competition and therefore no interaction between toxicant and density effects. The net result is that below carrying capacity density has no effect, but effects of the toxicant may be seen, whereas above carrying capacity contest competition masks the toxicant's effects. It must be remembered, however, that scramble and contest competition are idealizations, and real cases are likely to be intermediate. Furthermore, there may be interactions between competitive ability and toxicant sensitivity that could complicate the argument, and these need further consideration.

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