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ON THE DYNAMICS OF CHEMICALLY STRESSED POPULATIONS: THE DEDUCTION OF POPULATION CONSEQUENCES FROM EFFECTS ON INDIVIDUALS

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On the Dynamics of Chemically Stressed Populations: The Deduction of Population Consequences from Effects on Individuals[1]

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A general, simple, and explicit model for the age-dependent growth and reproduction of individuals as a function of food supply is presented. The model assumes a Holling-type functional response coupled with a von Bertalanffy body growth law, a fixed ratio between the energy utilized for reproduction and respiration, and a juvenile stage that ends as soon as the animal attains a sufficient weight. This model is shown to fit the available data on the development of *Daphnia magna* quite well. The model is used as a basis for studying the effects of chemicals on population growth rate, given the effects on individuals. Effects on individual growth and reproduction are reflected in a concentration-dependent relative reduction of the population growth rate. Effects on feeding rate, digestion, basal metabolism, and survival work out much more dramatically at low natural population growth rates. This already follows from a much simpler model that assumes age-independent reproduction, as exemplified to a good approximation by the rotifer *Brachionus rubens*. The theoretical results are supplemented with experimental evidence, implying that the stress on a population at a certain concentration of a chemical may indeed be strongly dependent on the feeding state of that population.

INTRODUCTION

Probably owing to the pharmacological origin of the subject, the early literature on environmental toxicology largely concerns the physiological (in a wide sense) effects of toxic chemicals. However, in evaluating the toxic effects of chemicals in the environment, physiological effects should be reinterpreted and supplemented in an ecological sense. For that purpose, effects on artificial and seminatural ecosystems, indoors as well as out, have been ever more widely studied. Unfortunately, even large-scale experimental studies are also of limited applicability, owing to the inherent complexity of the processes involved, and the concomitant scatter of data.

In this paper we hope to help bridge the gap between the individual and the ecosystem-based approaches to environmental toxicology: we shall examine the ecological consequences of physiological effects of chemicals by considering properties of (monospecific) populations of individuals under chemical stress. Although our results today are far from sufficient as a basis for an impact study, they at least offer a guide to the kind of effects we should look for when studying ecosystems exposed to toxic chemicals.

Spin-offs of our theoretical exercises are that they provide a basis for modeling dose–response relations of physiological effects of chemicals, and for modeling natural

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populations on a physiological basis. We intend to explore these topics in greater detail in the near future.

Any understanding of the determinants of a population growth has to be based on a knowledge of individual survival probabilities and reproductive rates as functions of age. Since reproduction depends on weight and therefore on growth, which in turn depends on the available assimilation energy, we shall deal with these topics in the reverse order. The plausibility of our assumptions will be discussed on the basis of data for the water flea *Daphnia magna* and the wheel animal *Brachionus rubens*. These freshwater filter feeders reproduce parthenogenetically, so only females are involved. This of course simplifies the theory. However, if the sex ratio is constant, and if fertilization of females is almost certain, the theory immediately generalizes to sexual populations.

A list of symbols is included in the appendix. Where estimated values of parameters are given for the first time, they are followed in brackets by the 95% confidence limits if there are readily available.

INDIVIDUAL ENERGY BALANCE

Part of the food ingested by an animal is digested and can therefore be regarded as assimilation energy. A certain amount of this energy has to be spent on maintenance. The energy excess is either stored, spent on growth and reproduction, or both. When food is abundant, we can expect the storage capacities to be filled. The stored energy may be consumed again when there is less food. At such times, the animal can economize on growth and reproduction, but not on maintenance. In this paper we shall refrain from modeling these physiological alternatives. Instead we shall focus on situations of constant food supply. This enables us to assume steady-state rates of energy consumption, eliminating the need to deal with storage considerations. Although this restriction narrows the applicability of our model, the resulting relative simplicity reveals the salient implications much more clearly.

Food Intake

We shall assume that at any given food density, the food ingestion rate is proportional to the surface area of the animal (a mechanistic argument supporting this assumption will be presented below); we shall moreover assume that the animal is one that does not change very much in shape after birth, which is equivalent to its surface area being effectively proportional to the two-third power of its wet weight, or to the square of some appropriate linear measure (cf. von Bertalanffy, 1934, 1969). Our second assumption is that at any given size of an individual, its intake rate increases with food density as a (Holling, or Monod) rectangular hyperbola. Data in support of these assumptions are given in Fig. 1. Here the feeding rate at 20°C of the cladoceran *D. magna* on a suspension of the green alga *Chlorella* is given as a function of food density and size of the animal. Clearly the fit is excellent, corroborating our assumptions in this particular case.

In the ecological literature the intake rate as a function of food density, X, is known as the functional response. Many invertebrates have been shown to exhibit approximately hyperbolic functional responses. The first author to provide an explanation of this functional form was Holling (1959), who wrote it as $\rho X/(1 + \rho\theta X)$ and interpreted

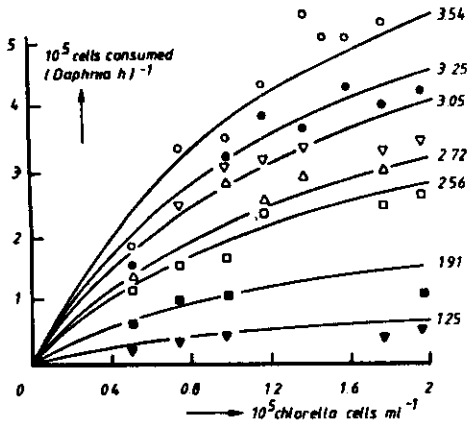

FIG. 1. Feeding rate Y at 20°C of *Daphnia magna* on *Chlorella* as a function of food density X for various body sizes L. The fitted curves are given by $Y = \alpha L^2 f(X)$, where $f(X) = \xi X/(1 + \xi X)$ and $\alpha = 0.74\ (0.60, 0.88) \times 10^3$ cells/(hr × mm^2), $\xi = 0.7(0.44, 0.96) \times 10^{-5}$ ml/cell. Data from McMacon and Rigler (1963), as reproduced by Wulff (1980).

ρ as the search or filtering rate (volume swept per unit of time) and θ as the average time the animal needs to cope with a single food particle. Alternatively, θ can be taken to be the inverse of the digestion rate. If Holling's explanation is correct, our assumptions imply that both the search rate and the digestion rate are proportional to the surface area of the animal. This does not seem too bad a first guess, because the search rate depends on the surface area of the food gathering apparatus, and the digestion rate on the surface area of the intestines. It should be noted, however, that neither the assumed weight dependence nor the specific functional form itself depends on the correctness of the Holling mechanism or on strict self-similarity during growth. There may well be other mechanisms leading to exactly the same results.

The functional response of filter feeders like *Daphnia* has long been assumed to be linear up to some threshold value of the food density, and constant thereafter (Frost, 1972). However, recent careful measurements give little support to this assumption. Fenchel (1980) found hyperbolic functional responses for filter feeding ciliates. Porter *et al.* (1982) report a close fit of a hyperbolic functional response for cladocerans. Our own reanalysis of the data of McMahon and Rigler (1963) shown in Fig. 1 led to the same conclusions. Finally, Fig. 2 shows the functional response of the rotifer *B. rubens*, again feeding on *Chlorella* at 20°C. The data points are redrawn from Pilarska (1977) on a linear instead of a logarithmic scale for the algal density. Here, too, a hyperbolic functional response is seen to fit the data reasonably well.

In the following discussion we shall assume that the energy gain per food particle is constant, so that the total energy intake can be written as

$$\nu W^{2/3} f(X) \tag{1}$$

where $f(X) = \xi X/(1 + \xi X)$, X is the food density, W the animal's wet weight, and ν and ξ are constants.

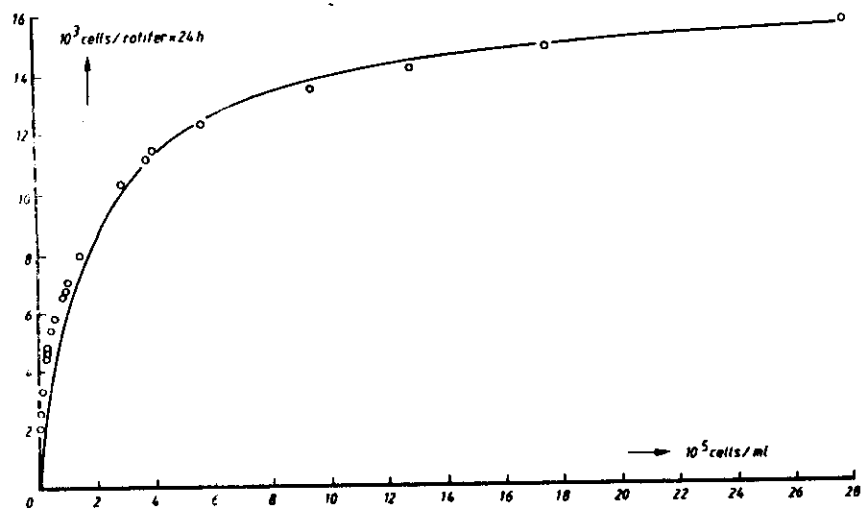

FIG. 2. Feeding rate Y at 20°C of *Brachionus rubens* on *Chlorella* as a function of food density X. The fitted curves are given by $Y = Y_m f(X)$, where $f(X) = \xi X/(1 + \xi X)$ and $Y_m = 16.6 \times 10^3$ cells/(24 hr × rotifer) and $\xi = 5.3 \times 10^{-5}$ ml/cell. Data from Pilarska (1977).

Growth

We shall assume the respiration energy to be a fixed fraction κ of the total, or assimilation, energy intake. This respiration energy can be divided into maintenance energy, which is proportional to wet weight, and energy spent on tissue growth, proportional to weight increase per unit of time. At constant food density, this results in an age-dependent weight increase of the type first discussed by von Bertalanffy (1934, 1969). In symbols we have

$$\kappa \nu f(X) W^{2/3} = \zeta W + \eta \frac{dW}{da}, \tag{2}$$

where ζ is the weight-specific maintenance energy (including the energy needed per unit of weight for molting, filtration, and the like), η is the conversion factor for energy to wet weight, and a is age. For a birth weight W_b, this results in

$$W(a) = f^3(X) W_m [1 - \{1 - f^{-1}(X)(W_b/W_m)^{1/3}\} \exp\{-\gamma a\}]^3, \tag{3}$$

where $\gamma = \zeta/(3\eta)$ the rate constant of growth, and $W_m = (\kappa\nu/\zeta)^3$ the wet weight of a very old individual reared at the highest possible food density. For length L, which is proportional to the third root of wet weight, this reduces to

$$L(a) = f(X) L_m - \{f(X) L_m - L_b\} \exp\{-\gamma a\}, \tag{4}$$

where $L_m = \kappa\nu/\zeta$. In Fig. 3 this family of curves has been fitted to the lengths of *D. magna* reared at various *Chlorella* densities. The fit is excellent, even though the number of parameters is but two more than the number of curves (L_b, γ, and the value of $f(X)L_m$ for each curve).

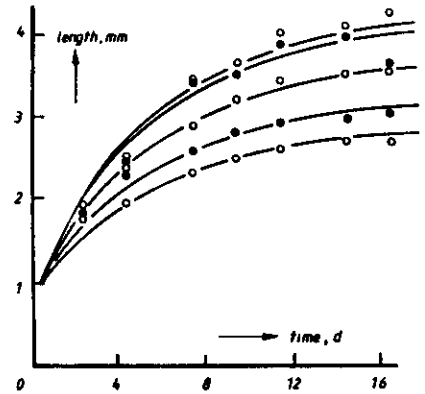

FIG. 3. Length L of *Daphnia magna* as a function of age a for various food densities X. The fitted curves have the form $L = f(X)L_m - \{f(X)L_m - L_b\}\exp\{-\gamma a\}$, where $f(X)L_m$ = 2.89, 3.24, 3.72, 4.17, 4.31 mm, L_b = 0.8 mm, γ = 0.17 (0.15, 0.20)/day.

Reproduction

Since the energy spent on reproduction is equal to the total energy input minus the respiration energy, the reproductive energy is found from (2) to be equal to $(1-\kappa)\psi f(X)W^{2/3}(a)$. If we divide this by the energy investment per young, ωW_b, we arrive at the number of young born per unit of time from a female of a certain age, or the age specific fertility. In symbols we have

$$R(a) = f^3(X)R_m[1 - \{1 - L_b/(L_m f(X))\}\exp\{-\gamma a\}]^2, \tag{5}$$

where $R_m = (1-\kappa)\psi L_m^2/(\omega W_b)$ is the maximum reproductive rate, i.e., the reproductive rate at a very high age of an animal reared on abundant food.

In cladocerans the young are released in batches just before molting. As pointed out by Tessier and Goulden (1982), the energy investment in reproduction should be considered a continuous process of energy storage in triglyceride droplets during the intermold periods. Toward the end of such a period, the stored energy is converted into young. Here we shall not try to incorporate such minor refinements in our model formulation.

In the ecological literature the production of young as a function of food density is known as the numerical response. When regarded as a function of X, $R(a)$ can therefore be called the age-specific numerical response. For fully grown individuals, it is plotted in Fig. 7.

Species for which ωW_b is small, so that the number of young released is large, are often classified in the ecological literature as r strategists, and animals for which the opposite is true as K strategists, referring to the supposed selective advantages of these two properties under conditions of, respectively, continued food abundance and steady-state energy limitation. In principle our type of model, based as it is on energy considerations, can be considered a necessary first step toward a thorough theoretical underpinning of such ideas. We intend to deal with this topic in greater detail in the future.

Juvenile Stage

Most animals have a juvenile stage, i.e., a stage during which they are not yet able to beget young. In our model we take account of this by assuming that during the first part of an animal's life the reproductive energy cannot yet be converted into young, but has to be spent on building the reproductive apparatus. Our assumption that there is a fixed ratio between the energy utilized for reproduction and that utilized for respiration implies that the animal's length and the energy invested in its gonads are related monotonically, at least until the end of its juvenile stage. If the gonads are fully grown at length L_j, the passing of this length marks the end of the juvenile period. So, if J denotes the length of the juvenile period, we get from (4)

$$J = \gamma^{-1}\ln\frac{f(X)L_m - L_b}{f(X)L_m - L_j}. \tag{6}$$

For $X < X_R(a)$, where

$$X_R(a) \overset{\text{def}}{=} \xi^{-1}\frac{L_j - L_b\exp\{-\gamma a\}}{L_m - L_j - (L_m - L_b)\exp\{-\gamma a\}}, \tag{7}$$

the animal will not start reproducing until age a. (To be precise, if young are born one at a time, the first will not be born before time P, where $\int_J^P R(a)da = 1$. Here, however, we shall neglect such minor refinements.)

To support our assumptions, we have plotted in Fig. 4 the length of *D. magna* as a function of age for two different food densities. Moreover, we have indicated when the first eggs were seen in the brood pouch. The figure shows that, in fact, this occurred at a constant size, and certainly not at a constant age.

For $X < X_R(\infty) = \xi^{-1}L_j/(L_m - L_j)$ the animal will never have any young. For *D. magna*, we have L_j = 2.5 mm (from Fig. 4), L_m = 6.6 mm (from Kooijman, 1983), $\xi = 0.7\times10^{-5}$ ml/cell, resulting in $X_R(\infty) = 0.87\times10^5$ cells/ml. The food consumption

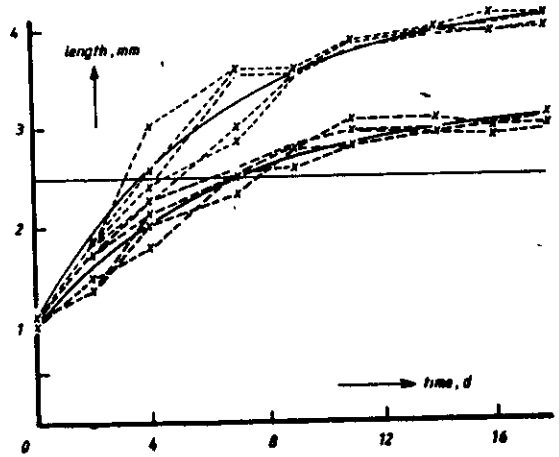

FIG. 4. Length L of a number of individual *Daphnia magna* as a function of age. Above the horizontal line eggs were visible for all the data points; below the line no eggs were visible.

rate at this density equals $Y_R = \alpha L_j^3 f(X_R) = \alpha L_j^3/L_m$, cf. Fig. 1. For *D. magna*, we have $\alpha = 0.74 \times 10^5$ cells/(hr $\times$ mm^2), resulting in $Y_R = 1.75 \times 10^5$ cells/hr.

Threshold Food Density

With some slight additional assumptions, our model formulation also enables us to calculate what is called the threshold food density, i.e., the minimum food density for survival (Lampert, 1977). The first assumption is that an animal dies when it cannot maintain its basal metabolism. The second is that an animal of a certain size that get less food is able to switch as much of its assimilation energy from reproduction to respiration as is necessary to cope with its bodily needs. These assumptions imply that to calculate the threshold food density X_s, we should set $\kappa = 1$ and $dW/da = 0$ in (2) to arrive at $f(X_s) = L\zeta/\nu$, or equivalently $X_s = \xi^{-1}L/(\nu/\zeta - L)$. The food consumption rate at this food density equals $Y_s = \alpha L^2 f(X_s) = L^3\alpha\zeta/\nu = L^3\alpha\kappa/L_m$ (cf. Fig. 1). The proportionality of threshold consumption rate to wet weight follows from the assumption that maintenance energy is proportional to wet weight. The expression for the threshold consumption rate can be used to estimate the value of κ, i.e., the proportion of assimilation energy spent on respiration when the supply of food is constant. For *D. magna*, Kooijman (1983) found $\kappa = 1/3$ from $L_m = 6.6$ mm.

POPULATION GROWTH RATE

Under constant environmental conditions the number of individuals N of a population is often observed to increase exponentially. This exponential growth law applies to animals as widely different as ciliates and reindeer (see, e.g., Scheffer, 1951). In symbols we have

$$N(t) = N(0)\exp\{rt\},$$

where t is time and r is a parameter known as the intrinsic rate of natural increase, or the Malthusian parameter, or simply as the population growth rate. This intrinsic rate of natural increase is a true population parameter. It is related to the individual survival and reproduction through the so-called characteristic equation

$$\int_0^\infty \exp\{-ra\}\mathcal{S}(a)R(a)da = 1, \tag{8}$$

where $\mathcal{S}(a)$ is the survival probability to an age of at least a. A derivation of (8) can be found, e.g., in Lotka (1956), Fisher (1958), Keyfitz (1968), Roughgarden (1979), Charlesworth (1980), or, in a stochastic context, in Jagers (1975).

For animals that die randomly, the survivor function has the form $\mathcal{S}(a) = \exp\{-a/T\}$, where T is the mean age at death. We expect such a survivor function, e.g., in a population under heavy predation pressure. At low predation pressure, the effect of aging will be much more pronounced, and the survivor function resembles the block survivor function $\mathcal{S}(a) = \chi(a \leq T)$, where χ denotes the indicator function, which takes the value one for true, and the value zero for false; i.e., the animal reaches age T, and dies immediately after. Most actual survivor functions are somewhere in between these two extremes.

To illustrate the relation between population growth rate, individual reproductive rate, and (mean) lifetime in a simple case, we shall put $R(a) = R$. This may be considered a limiting special case of our previous model for γ very large or L_b close to L_m. For rotifers, for instance, this may be considered an acceptable approximation. Under this simplifying assumption (8) reduces to

$$r = R - T^{-1} \tag{9}$$

for exponential survivor functions, and to

$$r = R(1 - \exp\{-rT\}) \tag{10}$$

for a block survivor function. The latter equation gives r only implicitly, and has to be solved numerically. In both cases dimensional considerations at once reveal that r/R, or alternatively, rT, depends only on the product RT. Figure 5, which is an evaluation of (9) and (10), shows three-dimensional plots of r as a function of R and T. For both (9) and (10), the population will be stable, i.e., $r = 0$, if the animals merely replace themselves, which happens for any survivor function when $RT = 1$. For small r, (10) reduces to $r \cong 2(R - T^{-1})$, which is more sensitive to the parameter T in the case of aging than in the case of random deaths. The difference becomes more dramatic for negative growth rates, i.e., when the population is dying off. With aging (10) may be seen to predict a steep decline for values of T where the random death Eq. (9) predicts only a slow decline. The biological interpretation of this effect is that in the latter case the population is saved from too rapid a decline by those few individuals that survive to a ripe old age. For large population growth rates of aging individuals, lifetime is not important so long as $RT > 3$. An increase in lifetime from $RT = 3.15$ to $RT = \infty$ raises the population growth rate by a mere 5%. In populations of nonaging individuals the comparable value for RT is 20. In such populations, therefore, the mean lifetime is important over a much wider range.

To illustrate the dependence of population growth rate on the duration of the juvenile stage (cf Cole, 1954), we shall consider a population of individuals that live forever and reproduce at a constant rate when mature. (This simplified model can be derived as a limiting case of our general energy-based model for $\gamma \to \infty$, and $L_j \to L_m$ in such a way that $L(J) = L_j$). For such a population the characteristic equation can be rewritten as $r = R\exp(-rJ)$. From dimensional considerations it follows that r/R depends only on the product RJ. For very small J we have $r \cong R(1 - RJ)$. Figure 6 shows the steep decline of r with increasing J. This explains why *B. rubens* and *D. magna* have a maximum population growth rate of 0.5 day^{-1} and 1 day^{-1}, respectively, whereas the first species produces one young every 2 days, and the second 60 young every 2 days: The juvenile period is negligible in *B. rubens*, and 2 days in *D. magna*.

For an age-dependent reproductive rate given by (5), a juvenile stage given by (6), and a survival up to a fixed age T, the population growth rate has to be found from

$$f^{-3}(X)\frac{1}{R_m} = \frac{1}{r}(e^{-rJ} - e^{-rT}) - \frac{2[1 - L_b/(L_m f(X))]}{r + \gamma}(e^{-(r+\gamma)J} - e^{-(r+\gamma)T}) + \frac{[1 - L_b/(L_m f(X))]^2}{r + 2\gamma}(e^{-(r+2\gamma)J} - e^{-(r+2\gamma)T}). \tag{11}$$

This equation has to be solved numerically. Figure 7 shows the result of such a calculation applied to *D. magna* reared at 20°C at various concentrations of *Chlorella*, using the parameters estimated in the previous sections. The figure shows that the

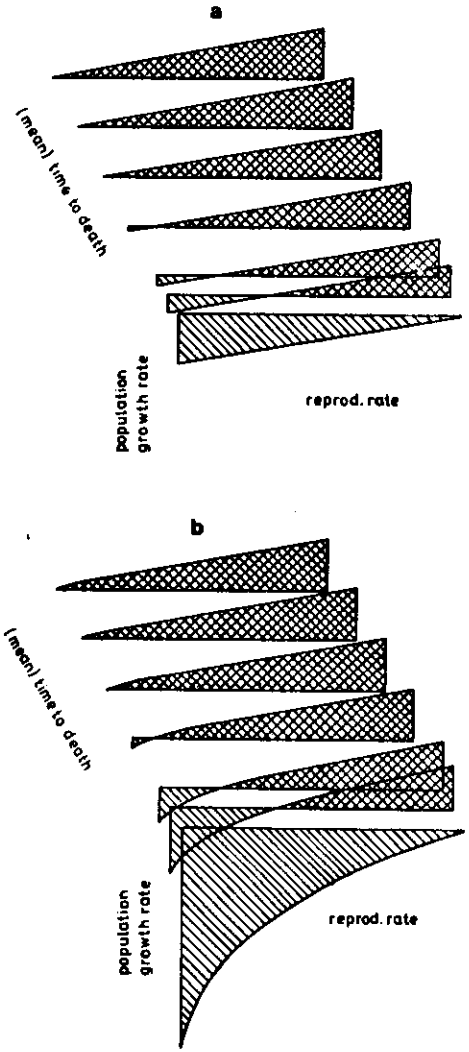

FIG. 5. Population growth rate r as a function of reproductive rate R and mean age at death T, for organisms which die randomly (a) or at a fixed age (b). The population is stable, i.e., $r = 0$, for $RT = 1$.

range of food densities within which the daphnid population changes from steeply declining to growing at almost the maximum rate is extremely narrow. Combined with the sampling problems encountered in the usually very clustered natural daphnid populations, this observation provides an explanation for the frequently erratic appearance of the population counts of daphnids recorded in the literature.

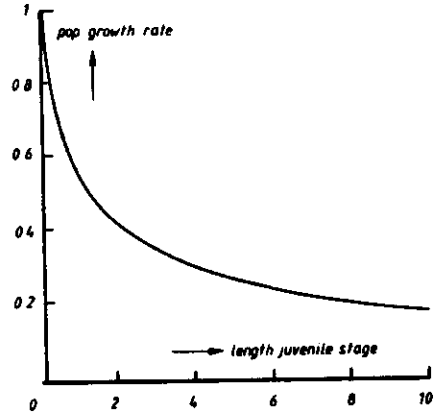

FIG. 6. Population growth rate r as a function of duration of juvenile stage for animals that grow very old. A time scaling is assumed such that the individual reproductive rate R equals 1.

TOXIC EFFECTS

In the literature on environmental toxicology the reproductive rate is usually considered to be a more "sensitive" parameter than survival. Our model framework allows one to distinguish between several possible ways in which the reproductive rate can be affected:

1. Test compounds whose toxic effects the animal is able to fight off, e.g., by metabolic deactivation or repair of the resulting damage, will cause an increase in maintenance energy per unit of body weight ζ. This will result in a decrease of growth rate and in a lower maximum weight, leading in turn to a lower reproductive output.
2. Chemicals which affect growth directly, for example, by impairing protein synthesis, will increase η, i.e., decrease γ (but not W_m), again suppressing reproduction.

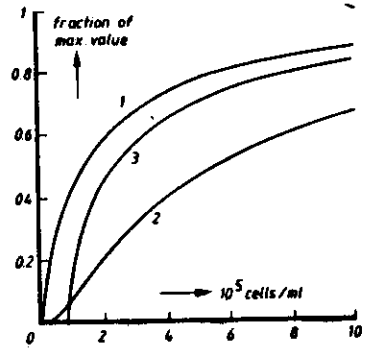

FIG. 7. Theoretical dependence on food density of the values of some individual and population characteristics of *Daphnia magna*, expressed as proportions of their maxima. (1) Feeding rate; (2) reproductive rate at old age; (3) population growth rate.

3. Compounds which affect reproduction will increase the energy required for the gestation of one young, ω, and thereby decrease R_m.

4. Chemicals which directly affect the available assimilation energy, e.g., by reducing the amount of digestible food or impairing the secretion of digestive juices, will decrease ν, leading to both slower growth and a lowered weight-specific reproductive rate.

5. Finally the last plausible type of effect to be distinguished within our modeling framework is impairment of the filtering rate, which manifests itself in a lowering of ξ. This effect has been described by von Pott (1980) and by Kersting and van der Honing (1981). Its frequency is not very well known, however, because it has no effect on growth and reproduction at the high food densities normally used in routine toxicity testing.

In routine chronic toxicity tests with daphnids the cumulative number of young up to and including 21 days is counted. From (5) and (6) the cumulative number of young up to time T equals

$$f^3(X)R_m\left[T - J - \frac{2}{\gamma}\{1 - L_b(f(X)L_m)^{-1}\}\{e^{-\gamma J} - e^{-\gamma T}\} + \frac{1}{2\gamma}\{1 - L_b(f(X)L_m)^{-1}\}^2\{e^{-2\gamma J} - e^{-2\gamma T}\}\right].$$

The concentration—EC_{50}—of test compound for which this number is half that in the blank is used as a measure of the toxicity of the compound. Figure 8 illustrates the dependence of the cumulative number of young on food density for the test compounds having various effects added in concentrations equal to the EC_{50} at a high food density. The parameters used were $\kappa = 1/3$, $R_m = 30$/day, $L_b = 0.8$ mm, $L_J = 2.5$ mm, $L_m = 6.6$ mm, $\gamma = 0.17$/day, and $\xi = 0.7 \times 10^{-5}$ ml/cell. It can be

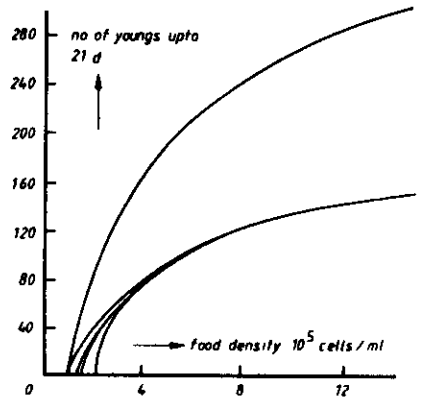

FIG. 8. The cumulative number of young of *Daphnia magna* per female up to 21 days as a function of the food density in the blank (upper curve) and under stress of compounds acting in different ways. The compounds are assumed to be present in concentrations equal to the EC_{50} at high food densities. For increasing abscissa intersections, the curves correspond to compounds affecting reproduction, digestion, growth, and basal metabolism, respectively.

seen that for food densities greater than 4×10^5 cells/ml, the total number of young cannot be used to distinguish between the various possible ways of action of a potentially toxic compound. The following will show, however, that the seemingly minor differences in impairment of reproductive rate at food densities below 4×10^5 cells/ml are greatly amplified in the effects on population growth rates.

Our general model predicts that any chemical that hampers growth will necessarily affect reproductive output. This seems to contradict the results presented by Winner (1981), who reports that low concentrations of zinc and copper affect the body size of *D. magna*, but not its reproductive rate. He concludes that there is no reason to believe that the chemicals can affect reproduction through an effect on body size. It should be noted, however, that recognizing such an effect is difficult because the scatter of reproductive data is usually much greater than that of growth data.

The general impression that the reproductive rate is a more sensitive parameter than survival should not prevent one from considering individual survival as a separate mechanism possibly affecting growth rate, because that impression may well be due to the short exposure times in routine toxicity tests. These usually last 48 hr, or at best 21 days. In the laboratory a water flea normally lives about 2.5 months at 20°C, and so even a considerable reduction in lifetime will never be noticed.

The time dependence of the survival probability is usually described very well by a model assuming that the animal dies upon accumulating a certain amount of the test compound by a simple one-compartment accumulation–elimination process (Kooijman, 1981). If chemical deaths occur only at higher ages, we may neglect the volume variation of the animal. If, moreover, the distribution of the threshold values is assumed to be of the usual log-logistic type, we get, in the absence of "natural" mortality,

$$\mathcal{F}(a) = [1 + (c/LC_{50} \cdot \infty)^{1/\beta}(1 - \exp\{-a/\tau\})^{1/\beta}]^{-1} \stackrel{\text{def}}{=} g_{\mathcal{L}}(a), \qquad (12)$$

where c is the concentration of the test compound in the medium, and $LC_{50} \cdot \infty$ is that concentration at which the eventual survival probability is 0.5, β is the shape parameter of the concentration-response curve, i.e., a measure of the variance of the $\ln\{LC_{50} \cdot \infty\}$ values among the individuals, and τ is the time constant of the elimination process.

CHEMICAL IMPAIRMENT OF POPULATION GROWTH RATE

In this section we shall evaluate the effect chemicals may have on population growth by comparing the growth rates, r_c and r_0, in situations with and without chemical stress for different food densities. For the sake of simplicity we shall assume that the stress, i.e., the concentration of the toxic compound and its effect on the individuals, does not vary with time.

In situations of stress, the population growth rate is by definition smaller, and so $r_c < r_0$. As well as biological intuition our model formulation tells us, moreover, that r_c and r_0 attain their highest values $r_{m,c}$ and $r_{m,0}$ when food is abundant. Therefore $r_{m,0}$ is the absolute maximum the population growth rate can reach in a given species. To study the impact of a chemical, we consider a stress which has a certain small effect p on the population growth rate under optimum feeding conditions, i.e., $r_{m,c} = pr_{m,0}$. We then calculate the stressed growth rates r_c for populations growing at lower blank rates r_0.

Age-Independent Reproductive Rates

Rotifers kept at 20°C attain their final size within a short time and live for about 12 days (Halbach, 1970). Moreover, their juvenile stage is extremely short, so we may safely make the approximating assumption that $R(a) = R$, a constant.

Suppose that a compound at low concentrations has no effect on survival, so that $\mathcal{S}_c = \mathcal{S}_0 = \mathcal{S}$, but does have an effect on reproduction: $R_c = (R_{m,c}/R_{m,0})R_0$. Then, for $\mathcal{S}(a) = \exp\{-a/T\}$, we get from (9) that $r_c = R_c - T^{-1}$ and $R_0 = r_0 + T^{-1}$, so that

$$r_c = (r_0 + T^{-1})(pr_{m,0} + T^{-1})/(r_{m,0} + T^{-1}) - T^{-1}. \tag{13}$$

This relation is depicted in Fig. 9 for $p = 0.9$. For $\mathcal{S}(a) = \chi(a \leqq T)$ we get from (10) that $r_c = R_c(1 - \exp\{-r_cT\})$ and $R_0 = r_0/(1 - \exp\{-r_0T\})$. Therefore,

$$r_c = pr_0 \frac{(1 - \exp\{-r_{m,0}T\})(1 - \exp\{-r_cT\})}{(1 - \exp\{-r_0T\})(1 - \exp\{-pr_{m,0}T\})}. \tag{14}$$

For this equation we numerically calculate the stressed population growth rate as a function of the blank population growth rate r_0.

The result (for $p = 0.9$) is shown in Fig. 9. For large mean lifetime T, both (13) and (14) reduce to $r_c = pr_0$, i.e., the growth rate of a population under stress is simply

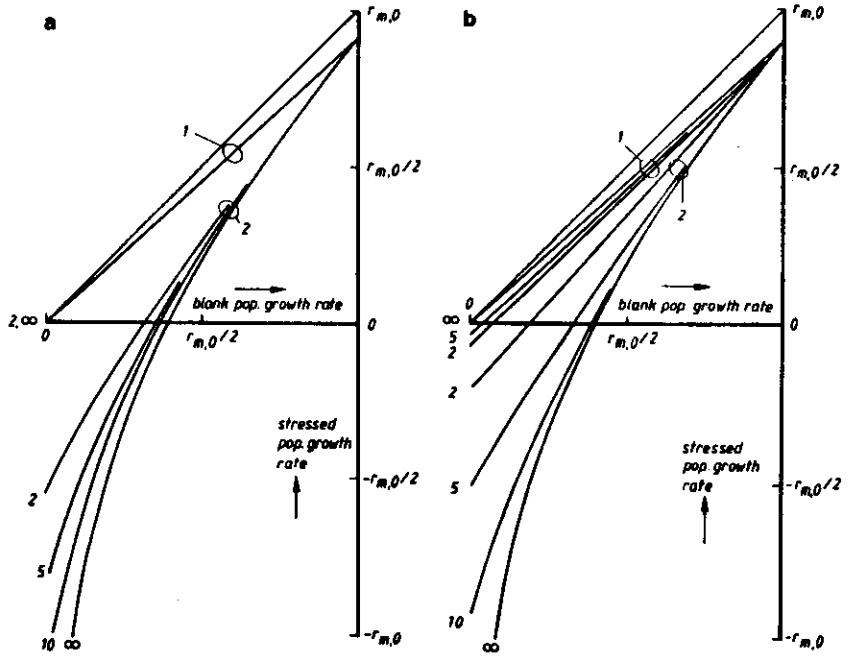

FIG. 9. Stressed population growth rates r_c as a function of the blank growth rates r_0 for compounds affecting reproduction only (1) and survival only (2) for individuals which in the blank either die randomly (a) or at a fixed age (b). The numbers along the curves correspond to the values of $Tr_{m,0}$, where T is the mean age at death and $r_{m,0}$ the maximum population growth rate in the blank. The maximum population growth rate in the stressed situation was set at $r_{m,c} = pr_{m,0}$, with $p = 0.9$.

a fixed fraction of the blank population growth rate. Figure 9 shows that, for smaller mean lifetimes, the interaction between chemical stress and poorer feeding conditions is still not a very dramatic, provided that only effects on reproduction are taken into account. The situation changes drastically, however, when the stress affects survival rather than reproduction.

From now we shall assume until further notice that $R_c = R_0 = R$. We begin our study of the effects of changing survival by making the simple assumption that the animals die from toxic effects at time $T_c \leqq T_0$. In our simple aging model the resulting survivor functions are $\mathcal{S}_c(a) = \chi(a \leqq T_c)$ and $\mathcal{S}_0(a) = \chi(a \leqq T_0)$. We then get from (10)

$$r_c = R(1 - \exp\{-r_cT_c\}) \qquad \text{and} \qquad r_{m,c} = pr_{m,0} = R(1 - \exp\{-pr_{m,0}T_c\}).$$

The latter equation can be solved for T_c, giving

$$T_c = -(pr_{m,0})^{-1} \ln\{1 - p(1 - \exp\{-r_{m,0}T_0\})\}.$$

Substituting this for T_c, and elimination R using (10), again with $r = r_0$, finally yields

$$r_c = r_0(1 - \exp\{-r_0T_0\})^{-1}(1 - [1 - p(1 - \exp\{-r_{m,0}T_0\})]^{r_c/(pr_{m,0})}). \tag{15}$$

Our simple assumption about chemical deaths changes the random death survival function $\mathcal{S}_0(a) = \exp\{-a/T_0\}$ to $\mathcal{S}_c(a) = \mathcal{S}_0(a)\chi(a \leqq T_c)$.

Proceeding as before, we get

$$\frac{r_c + T_0^{-1}}{r_0 + T_0^{-1}} = 1 - \left(\frac{(1 - p)r_{m,0}}{r_{m,0} + T_0^{-1}}\right)^{(r_c + T_0^{-1})/(pr_{m,0} + T_0^{-1})}. \tag{16}$$

For large T_0, both (15) and (16) reduce to

$$r_c = r_0[1 - (1 - p)^{r_c/(pr_{m,0})}]. \tag{17}$$

Figure 9 shows what the solutions to (15) and (16) look like in the general case. Clearly, the impact of an effect on survival for decreasing population growth rates rapidly builds up. This is in sharp contrast to the impact of effects on the reproductive rate. As is to be expected, this effect is less marked for populations suffering high predation, i.e., for $\mathcal{S}_0(a) = \exp\{-a/T_0\}$ and $T_0r_{m,0}$ small.

So far we have assumed that individuals die upon accumulating a certain amount of toxic chemical, all individuals having the same toxicity threshold. Under those conditions we may avoid the dynamic details of the accumulation–elimination kinetics. This is no longer the case when the threshold values vary within the population.

For a log-logistic threshold distribution and no natural mortality, the survivor function is given by (12). If chemical and natural mortality occur together, we get $\mathcal{S}_c(a) = \mathcal{G}_c(a)\mathcal{S}_0(a)$. For this survivor function we can no longer find an explicit expression for the integral in the characteristic Eq. (8), which then has to be tackled numerically. However, the basic ideas remain essentially the same. Some representative numerical results for $\mathcal{S}_0(a) = \chi(a \leqq T)$ are presented in Fig. 10, which shows the relation between the stressed and blank population growth rates r_c and r_0 for two types of compound, one having a small time constant for elimination, e.g., a detergent, and the other having a rather large elimination time constant, e.g., a heavy metal. These figures show that an increase of the variability β of the threshold levels widens the region of blank population growth rates in which the population does not succumb

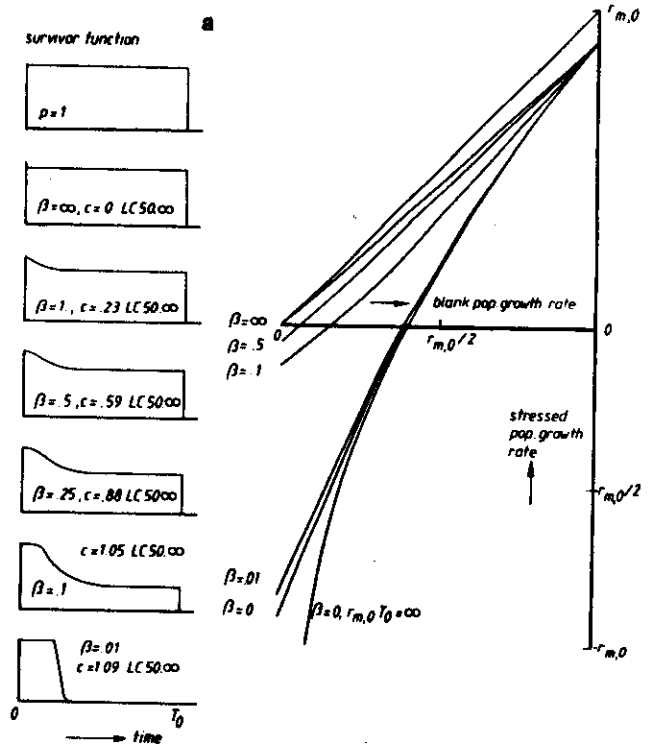

FIG. 10. Stressed population growth rates r_c as a function of the blank population growth rates r_0 for compounds with a small time constant of elimination, $\tau = 1$ (a), and with a large time constant of elimination, $\tau = 50$ (b), and an effect on survival. The numbers along the curves are the variances of the ln threshold concentrations among the individuals. The product of the maximum lifetime T_0 and the maximum reproductive rate in the blank situation $R_{m,0}$ was chosen as 10, as is reasonable for *Brachionus rubens*. The maximum population growth rate in the stressed situation was set at $r_{m,c} = pr_{m,0}$, with $p = 0.9$.

to chemical stress. Clearly, moreover, the effect is much more marked for the compound with the small time constant. This at once follows when a large value of β is put in (12): in that case animals either die from chemical causes very early on in their lives or experience no effect at all. When almost stillborn animals are regarded as having been unborn, the chemical effect on survival becomes one on reproduction. Biologically speaking, those individuals that escape early chemical death go on reproducing for their normal lifetimes, thereby effectively saving the population from declining rapidly. We have tested the theory set forth above in two sets of experiments. Kooijman (1983) subjected populations of *B. rubens* to a range of concentrations of sodium metavanadate. The observed population growth rates were in excellent agreement with the population growth rates calculated numerically from (8) on the assumption that $\mathcal{F}_c(a) = \mathcal{S}_c(a)\chi(a \leq 12 \text{ days})$ and $R(a) = R$.

The results of the second set of experiments are depicted in Fig. 11, which shows the growth rates at 20°C of populations of *B. rubens* for different *Chlorella* densities and different concentrations of the toxic compounds 3,4-dichloroaniline and potassium

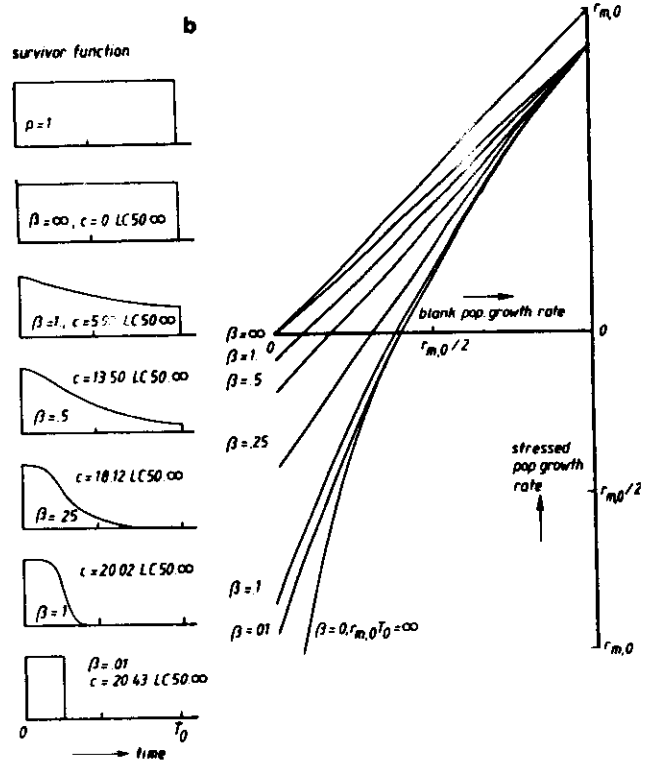

Fig. 10.—*Continued*

dichromate. The first compound is known mainly to affect reproduction in low concentrations, whereas the second compound mainly affects survival. Clearly, the results are in perfect correspondence with our predictions: the chemical impact of 3,4-dichloroaniline is almost independent of the blank population growth rate, whereas for dichromate there is a marked dependence.

Age-Dependent Reproductive Rates

As a final step we shall study the various possible effects of chemical stress on a population of individuals having a juvenile stage whose duration is given by (6), and whose age-dependent reproductive rates are given by (5). For this purpose we first solve (11) for the food density X for various blank population growth rates, using the parameter values of the blank situation. Next, we solve (11) for the value of that parameter that is supposed to be affected, assuming a very high food density and a given reduction p of the maximum blank population growth rate. Finally, we use (11) to calculate the stressed population growth rates for the food densities obtained in the first step and the value of the affected parameter obtained in the second step.

The result of the procedure is shown in Fig. 12. In calculating the curves, we used a block survivor function with $T_0 = 70$ days, and the parameter values for *D. magna*

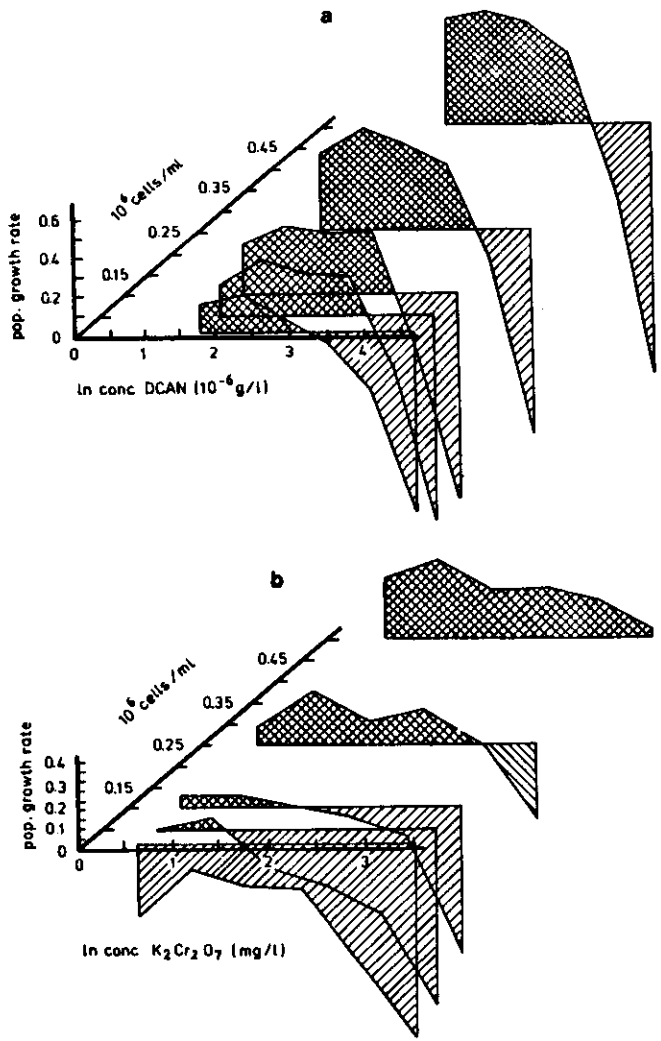

FIG. 11. Observed population growth rates of 20°C at *Brachionus rubens* (vertical in $days^{-1}$) for various concentrations of 3,4-dichloroaniline (a) and potassium dichromate (b) (to the right, in ln $\mu g/l$ and ln mg/l, respectively) feeding at various *Chlorella* densities (backward, in 10^6 cells/ml). The leftmost concentration is blank and should be positioned at minus infinity.

obtained previously. The values of those parameters were $\gamma = 0.17$ mm/day, $\xi = 0.7 \times 10^{-5}$ ml $cell^{-1}$, $R_m = 30$ day^{-1}, $L_b = 0.8$ mm, $L_J = 2.5$ mm. For ν and κ, we chose the values $1J$ day^{-1} mm^{-2} and 1/3, respectively. The value of ν is arbitrary, because it enters the calculations only as a scaling constant. The resulting values of the metabolic parameter are $\zeta = 0.05J$ day^{-1} mm^{-3}, $\eta = 0.1J$ mm^{-3}, and $\omega = 1.834J$ mm^{-3}. The

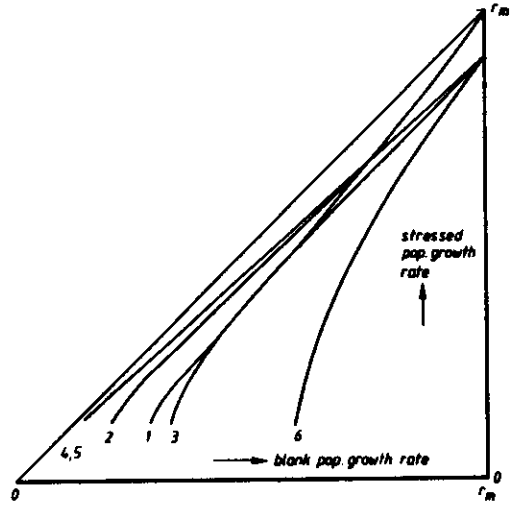

FIG. 12. Stressed population growth rates for compounds affecting (1) feeding rate, (2) digestion, (3) basal metabolism, (4) growth, (5) reproductive rate, and (6) survival. Except for the feeding rate, the concentration of toxic compound is chosen such that the maximum stressed population growth rate is 90% of the maximum blank population growth rate.

corresponding stressed parameter values for $p = 0.9$ were found to be $\nu = 0.924J$ day^{-1} mm^{-2}, $\zeta = 0.066J$ day^{-1} mm^{-3}, $\eta = 0.116J$ mm^{-3}, $\omega = 2.587J$ mm^{-3}, and $T = 4.07$ days. The stressed value of ξ was chosen arbitrarily as $0.5 \cdot 10^{-5}$ ml $cell^{-1}$. (Note that we here assume that only one parameter is affected at a time. This may be realistic for low concentrations which only affect that parameter which is most sensitive to the chemical in question. At higher concentrations, more parameters may be affected simultaneously.)

These stressed parameter values for ν, ζ, η, and ω correspond to a reduction factor for the cumulative number of young per female up to 21 days at high food densities of 0.79, 0.65, 0.92, and 0.71, respectively. So for compounds affecting growth, a reduction of the cumulative number of young per female up to 21 days at high food densities correspond to a similar reduction of the population growth rate, at least for *D. magna*.

The main conclusions that now emerge are that for *D. magna* (1) toxic effects on growth and reproduction result in a stressed population growth rate that is in effect proportionally reduced; (2) the impact of effects on survival, basal metabolism, and digestion increases with decreasing blank population growth rate; and (3) not unexpectedly, effects on filtering rates are only seen at low population growth rates.

DISCUSSION

If we are interested in the effect of toxic chemicals on the environment, our main concern should not be individuals but populations which, moreover, experience a variety of feeding conditions. Our models predict that the impact on population growth rate of chemical effects on filtration rate, digestion, basal metabolism, and

survival strongly depends on food availability. Unfortunately, this means that laboratory systems, in which food is normally abundant, cannot be but insensitive to such effects. However, our results can be put to two positive uses: (1) they can explain some of the difficulties encountered in the scientific study of chemically stressed experimental and natural ecosystems, and (2) they can warn us about the critical phases in the life of an ecosystem when chemical pollution will be most disrupting.

When an aquatic "ecosystem" is isolated for experimental purposes, it usually exhibits an algal bloom. Our theory predicts that a toxic compound will have its severest impact only after development of the bloom, when the food supply for phytoplankton as well as for zooplankton is falling short. Unfortunately such effects are difficult to study experimentally, because scaled-down ecosystems begin to diverge rapidly in behavior after a few weeks. For this reason it is nearly impossible to trace effects to their causes in any particular case. (Certain experimental results reported in the literature may not be as significant as they are held to be, because they may well be due to the organisms adapting themselves to their new experimental environment, as may be manifested, e.g., in an increase in the lag phase in populations of microorganisms; see Kooijman *et al.* (1983).) It is here that a modeling study from causes instead of effects should prove its worth.

Most animals in the relatively short-lasting spring and autumn blooms of freshwater zooplankton do not grow old. Therefore, it is those animals surviving between the blooms that will suffer especially from a low level of chemical stress. At such times the biomass per unit volume is necessarily low, and so difficult to sample. Early adverse effects may thus escape notice. This situation might best be helped by monitoring chemical influxes together with laboratory tests and fate studies in a modeling environment of the type discussed in this paper.

Finally we wish to emphasize once more that a shortage of food, even if temporary, is likely to greatly increase the chemical stress to which an ecosystem is subject.

APPENDIX: NOMENCLATURE

Symbol	Dimension	Interpretation
a	time	age
c	g · length^{-3}	concentration of toxic compound
f	—	functional response as a proportion its maximum
$\mathcal{F}$	—	survival probability
$\mathcal{F}_0$, $\mathcal{F}_c$	—	survival probabilities in the blank and stressed situations
$\mathcal{S}_c$	—	reduction of the survival probability due to chemical stress
J	time	duration of juvenile period
L	length	length of the animal
L_b, L_J, L_m	length	length at birth, at the end of the juvenile period, maximum length
$LC_{50} \cdot \infty$	g · length^{-3}	concentration of compound at which the survival probability upon continuous exposure is 0.5

APPENDIX—*Continued*

Symbol	Dimension	Interpretation
p	—	reduction of the maximum population growth due to chemical stress
$r_{(m)}$	time^{-1}	(maximum) population growth rate
$r_{(m),0}$, $r_{(m),c}$	time^{-1}	(maximum) blank and stressed population growth rate
$R_{(m)}$	time^{-1}	(maximum) reproductive rate
$R_{(m),0}$, $R_{(m),c}$	time^{-1}	(maximum) blank and stressed reproductive rates
T	time	mean lifetime
T_0, T_c	time	mean lifetime in the blank, upperlimit to the lifetime resulting from chemical stress
W	length^{-3}	volume of animal = wet weight
W_b, W_m	length^{-3}	wet weight at birth, maximum wet weight
X	cells · length^{-3}	food density
X_s, X_R	cells · length^{-3}	threshold food densities for survival, reproduction
Y	cells · time^{-1}	feeding rate
Y_s, Y_R	cells · time^{-1}	threshold feeding rate for survival, reproduction
α	cells · time^{-1} · length^{-2}	maximum feeding rate per unit of surface area
β	—	variance of the log threshold concentrations
γ	time^{-1}	rate constant of growth
ζ	energy · time^{-1} · length^{-3}	maintenance energy consumption rate per unit of weight
η	energy · length^{-3}	growth energy per unit of weight
κ	—	proportion of assimilation energy spent on respiration
υ	energy · time^{-1} · length^{-2}	maximum energy intake rate per unit of surface area
ξ	length^3 · cells^{-1}	filtering rate times the digestion time per cell
τ	time	time constant of elimination
χ	—	indicator function, giving 1 for a true argument and 0 for a false one
ω	energy · length^{-3}	invested energy per unit of birth weight

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