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On the relationships between bioassays and dynamics in chemically stressed, aquatic population models

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Abstract. *One purpose c f this article is to synthesize some recent results on the dynamics of mathematical models of chemically stressed aquatic populations and communities; in particular, we (1) illustrate some of the difficulties that might arise from extrapolation of bioassay results to dynamic, chemically stressed population and community models; and (2) indicate different ways in which chemicals can affect the dynamics of population models. Bioassays, an important component of ecological impact and risk assessment, can be. misleading if extrapolated to settings beyond experimental boundaries. Extrapolation of bioassays to the populations and community levels can not be direct because derived information is usually specific for a subset of individuals and obtained under experimental constraints on time and parameters. We present examples derived from a mathematical setting where consequences of bioassays, even when employed as the fundamental determinant of stress in the systeni, have no predictable relationship to the ultimate effect of the chemical on the system. The first illustration, at the population level, demonstrates that sublethal effects of a lipophilic chemical with a reversible mode of action on individuals attained at concentrations well below the LC_{50}, indeed even below the EC_{50} for growth, can drive the population to extinction so that the chemically stressed population is much more severely damaged than predicted by hioassays. The second illustration at the community level indicates that results of bioassays can also indicate outcomes that (ire worse than actually occurs for the community. Finally, we compare the outcome of a spectral analysis of time series of a sequence of chemically stressed populations, demonstrating that complex effects of lipophilic chemicals on population dynamics are not readily identifiahle from spectral signatures.*

Introduction

The theory needed to increase ecological assessment scope and capability to levels sufficient to meet current goals of determining the ecological health of the world's major ecosystems remains spurious. Construction of a predictive framework, one capable of integrating both regularities and differences in structure with causal mechanisms and processes, is arguably the central quest of science. For environmental systems, the cornerstone of such a predictive framework is a broadly based, dynamic approach to ecology postulating common linkages for all natural systems, such as energetic fundamentals and biogeochemical processes. All biologically important levels of scale contribute to the patterns of nature. At the same time, the level of the observer through experimental design acts to diminish the action of some mechanisms and processes while highlighting others. The observer problem is often present in bioassay extrapolation to the population level in that bioassays can be misleading unless the appropriate information for the system under investigation is also included in the bioassay formulations.

Bioassays, an important component of ecological impact and risk assessment, are often used to measure the effects of chemical stressors on a nurnber of usually, similar types of individuals. Examples of mathematical relationships derived from bioassays that provide useful information for ecological risk assessment are quantitative structure activity relationships (QSARs), bioassay data that delineate an effect of a concentration of a chemical on individuals. These encompass the toxicity syndrome from no observed effect concentration (NOEC) through the concentration that reduces the individual growth rate by a fixed percentage from the nominal unstressed environment, such as EC_{50}, the concentration that reduces the growth by 50%, to the lethal concentration that kills 50 % of the organisms, LC_{50}. These QSARs focus on

groups of individuals and are performed over a prescribed period of time (Hermens et al. 1984, 1985, de Wolf et al. 1988). While of utility as a screening device, extrapolation of bioassays to the population and community levels of organization has not been straight forward because this information is specific for a subset of individuals arid, because of experimental time constraints, needed interactions present in populations and communities are omitted.

A principle- and process-oriented approach that results in testable hypotheses has been and is the focus of our modeling approach. To accomplish a mechanistic model representation, the use of individual structure is necessary. We now indicate the forms of some of the population and community models utilized here.

Underlying our approach is the dynamics of an individual. Once a dynamic model of an individual is formulated (e.g. Hallam et al. 1990a), it can be incorporated into a population model via an extended McKendrick—von Foerster partial differential equation (Hallam et al. 1993); the details of incorporating the individual dynamics into a population are indicated in Hallam et al. (I 990b). This equation represents transport processes based upon a conservation law and is a hyperbolic partial differential equation of the form

$$\frac{\partial \rho}{\partial t}+\frac{\partial \rho}{\partial a}+\frac{\partial(\rho L)}{\partial m_L}+\frac{\partial(\rho S)}{\partial m_s}= -\mu\rho$$

where $\rho = \rho(t, a, m_L, m_S)$ is the density function (numbers; age^{-1}; mass^{-1}; mass^{-1}), μ is the mortality rate (d^{-1}), L is the growth rate of lipid in the individual (mass; d^{-1}), S is the growth rate of structure in the individual (mass; d^{-1}), t is time (d), a is age, m_L is the lipid mass of an individual (mass), and m_S is the structural mass of an individual (mass).

In order for the problem to make sense biologically and for the mathematical problem to be well posed, it is necessary to prescribe initial and boundary conditions for the population. An initial distribution $\rho(0, a, m_L, m_S)$ is assumed; it describes the population at time t= 0. A boundary condition, often called the renewal equation, indicates the birth process of the population. In structured models this can have numerous forms. For example, one form is

$$\rho(t,0,m_{L0},m_{S0})=\int_0^{\infty}\int_{m_{S0}}^{\infty}\int_{m_{L0}}^{\infty}\beta(t,a,m_{L0},m_{S0},m_L,m_S)\rho(t,a,m_L,m_S)dm_L dm_S da$$

where β is the fertility rate function that describes the number of eggs with the initial physiological dimensions m_{L0}, the lipid per egg, and m_{S0}, the structure per egg, produced by an organism of age a and physiological state m_L and m_S, at time t. The function ρ is determined by the individual model in that the allocation to eggs must be represented at the times of reproduction for each organism.

This modeling approach together with the technique for solving it (the method of characteristics) is advantageous because it allows for incorporation of individual dynamics, toxicarnt dynamics, and effects determination at the population level. The population, the combination of numerous ecotypes of individuals, can be of arbitrary dimension. We employ a model that consists of 27 extended McKendrick-von Foerster partial differential equations of type (1); one equation for each of 27 different ecotypes of individuals in the initial distribution.

For *Daphnia*, Hallam et al. (1990b) delineate characteristics of the ecotypes in the model employed here. The diversity of ecotypes in the population is determined by three distinct choices of the three parameters: resource level, quality of resource, mid filtering rate. The population composition reflects age, lipid and structure variation. The initial distribution contains a spectrum of individuals whose distributions of age, size mid lipid are realizable in known laboratory populations of *Daphnia magna* (Hallam et al. 1990b).

It is generally recognized that sublethal effects on individuals are important, but it is not generally known how these effects are manifested at the population level. To better understand implications of sublethal effects, a modeling approach was employed to study the effects of a class of chemicals (those with the nonpolar narcosis mode of action) on *Daphnia* populations. In Hallam et al. (1993), a quantitative

dose-response relationship (QDRR) was found by focusing on individual survival in toxicant stressed populations. A population is persistent if the population density function p is nonzero throughout the simulation nun time. A population goes to extinction if $\rho = 0$ at some time during the simulation run. This definition of survival is clearly a function of the length of the simulation; however, for sublethal exposures that produce growth effects, we find that extinction is independent of simulation nun time- if the run time is sufficiently long, generally exceeding loll days.

In our simulations, the exposure scenario consists of chronically exposing all individuals in a population to a toxic chemical at a constant concentration. The population extinction threshold, PET, is the ambient concentration of a chemical such that if the ambient chemical exposure concentration, C_w (mg/L), for the stressed population is less than PET then the population is persistent and if C_w is greater than or equal to PET, the. population goes to extinction. The QDRR represents the population extinction threshold as a function of the chemical property K_{OW}, the octanol-water partition coefficient. A point on the extinction threshold for the *Daphnia* population model is generated by selecting K_{OW} within the interval from 1 to 10^7, a range appropriate for narcotic chemicals, and performing numerous chemical stress simulation runs to calculate the minimum concentration where extinction of the population results. The process, repeated for numerous values of K_{OW}, generates the population extinction threshold QDRR (Figure 1). The three most important features of the population extinction threshold QDRR are that (i) it is linear in log K_{OW}; (ii) it has a slope of -l ; and, for the purposes of this article, it is important that (iii) it lies below the known QSARs for the 16 d EC_{50}, for growth and reproduction. The threshold is a function of many variables including the chemical mode of action and concentration, (lie structure of the population, and the mortality representations. The important conclusion is that the threshold will exist independent of most of these variables.

Hence, our first illustration where bioassays do not present a proper perspective is concerned with the determination of conditions for a population to be driven to extinction when subjected to chemical stressors at concentrations below that predicted by the bioassay, the 16 d LC_{50}. We emphasize that features different from mortality of individuals can drive a population to extinction. In this situation, the culprit is the inability of individuals to attain reproductive sizes over their lifetimes.

Spectral analysis of stressed population data

The dynamic behavior of a population subjected to chemical stress is difficult to classify although there are some indicators of stress such as age structure and size structure (Kersting 1975), a phenomenon we also observed in structured model analysis. Indeed, the presence of only young and old cohorts seems characteristic of populations tending towards extinction.

We next illustrate some difficulties that arise in the determination of the characteristic indicators obtained from dynamic behavior of stressed populations. Natural populations and structured population models frequently exhibit highly oscillatory dynamics. We utilize these oscillatory characteristics to attempt to differentiate various levels of stressors in the system.

In simulations including sublethal effects, oscillations are also prevalent and they change in appearance with the concentration of chemical in the water C_w (Hallam et al. 1993). This can be observed by comparing Figure 2 and Figure 3. We focus here on a fixed model population mid investigate the sensitivity of the dynantics to changing chemical stresses.

Both short term as well as long term oscillations are present in the dynamic behavior of the population. These oscillations are most prominent in individuals in the smallest size class where the short term fluctuations are associated with reproduction (Figure 4).

In the simulations we have assumed that the reproductive period of *Daphnia magna* is 4 days, so we expect some dominant frequencies close to one cycle every four days. The detenninants of the long term fluctuations are a complex function of physiological characteristics of the individuals mid ecotypes present in the population.

One methodology available to study oscillations is to fund the power spectrum of the time series; that is, to find “how much power” is contained in the frequency interval between f and f+df. We used the Fast Hartley Transform (FHT) method to compute the power spectrum (Bracewell 1984, Press et al. 1938). The FHT of a given function $h(\tau)$ for $\tau = 0, 1, \ldots, N-1$, is defined as follows:

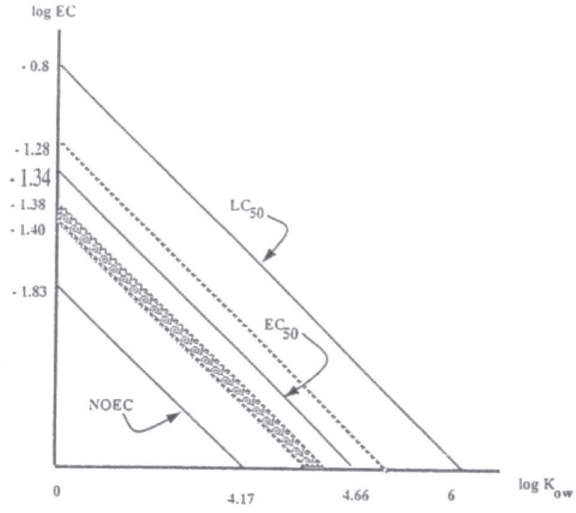

Figure 1. A comparison between quantitative structure activity relationships (QSARs) and quantitative dose-response relationships (QDRRs) for population extinction thresholds obtained from the simulations under different assumptions about mortality. The axes are the logarithm of the octanol-water partition coefficient and the logarithm of the effect concentration (mg/L). The solid lines are QSARs and the squiggled lines are computed.

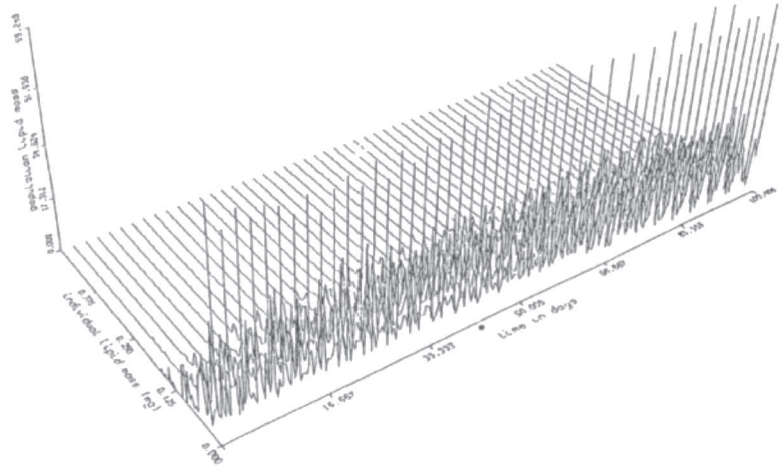

Figure 2. The figure illustrates the unstressed dynamic population as viewed through its total lipid density (#) on the interval 0 to 100 d. Output step 2 d.

$$H(f) = \frac{1}{N}\sum_{\tau=0}^{N-1} h(\tau)cas\{\frac{2\Pi\ f\tau}{N}\},\ f = 0,1,...N-1$$

here cas θ_{OW}= cos θ_{OW} + sin θ_{OW}. The squared magnitude spectrum is calculated from this (real) function by:

$$Z^2 = \frac{1}{2}\{[H(f)]^2 + [H(-f)]^2\}$$

In order to avoid aliasing effects, data was produced every day (sampling interval Δ = 1 day) for 2,048 days. We also applied a high-pass filter, to avoid noise at low frequencies. A high—pass filter passes all frequencies greater than a certain value Ω_C.

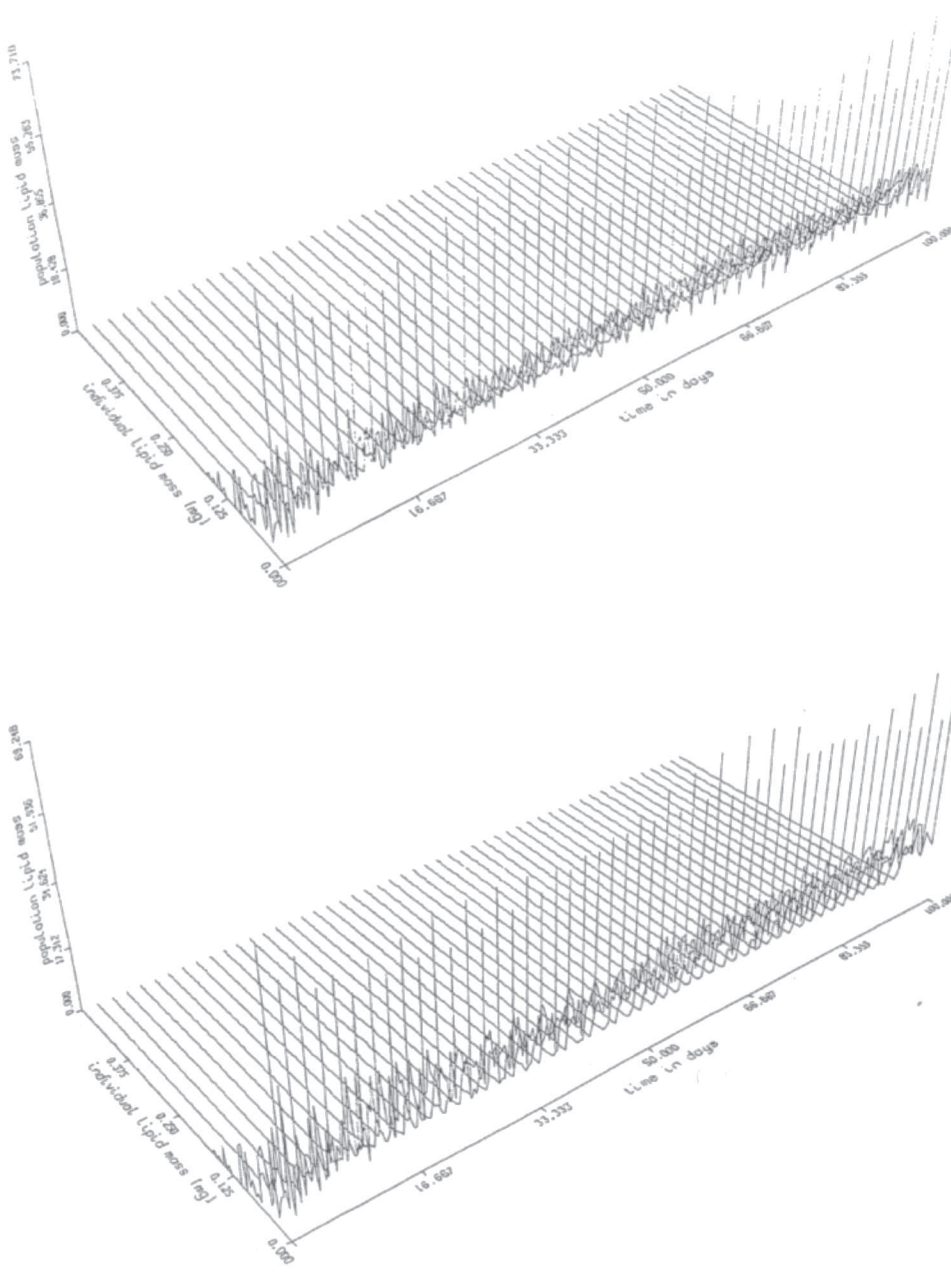

Figure 3 a and b. The figures illustrate the stressed population as viewed through its total lipid density (#) on the interval 0 to 100 d. K_{OW} =1000; (a) C_W= 0.30x10^{-4} (mg/1) , (b) C_W = 0.34x10^{-4} (mg/L).

In Figure 5 the magnitude is plotted against frequencies in such a way that high frequencies are on the right and the low frequencies are on the left. The reproductive period is represented in the middle spikes of the graphs (at $f \approx 0.25$). We used a high-pass filter with Ω_C,= 0.005, hence only frequencies greater than 0.005 are passed through the filter.

The power spectrum evolves as the concentration of toxic chemical in the water increased. Note that for concentration 0.32x10 $^{-4}$ mg/L the population is in resonance. As the concentration increases from C_W = 0.32x10^{-4} towards the threshold concentration for extinction ($C_W \approx 0.36x10^{-4}$) the behavior reverses in such

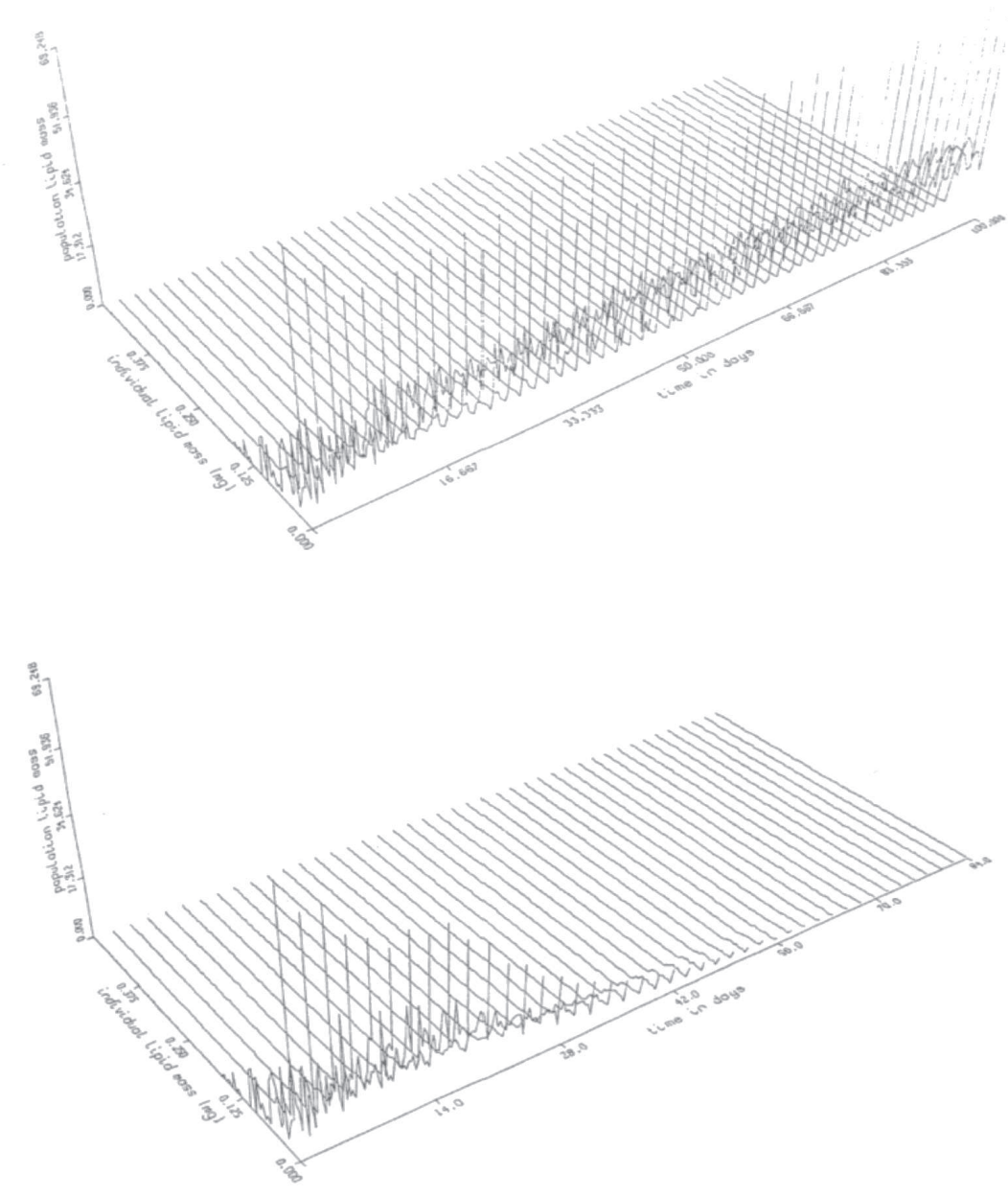

Figure 3 c and d. The figures illustrate the stressed population as viewed through its total lipid density (#) on the interval 0 to 100 d. K_{OW} =1000; (c) C_W= 0.36x10^{-4} (mg/L), (d) C = 0.37x10^{-4} (mg/L). Notice that this Ias concentration leads to extinction.

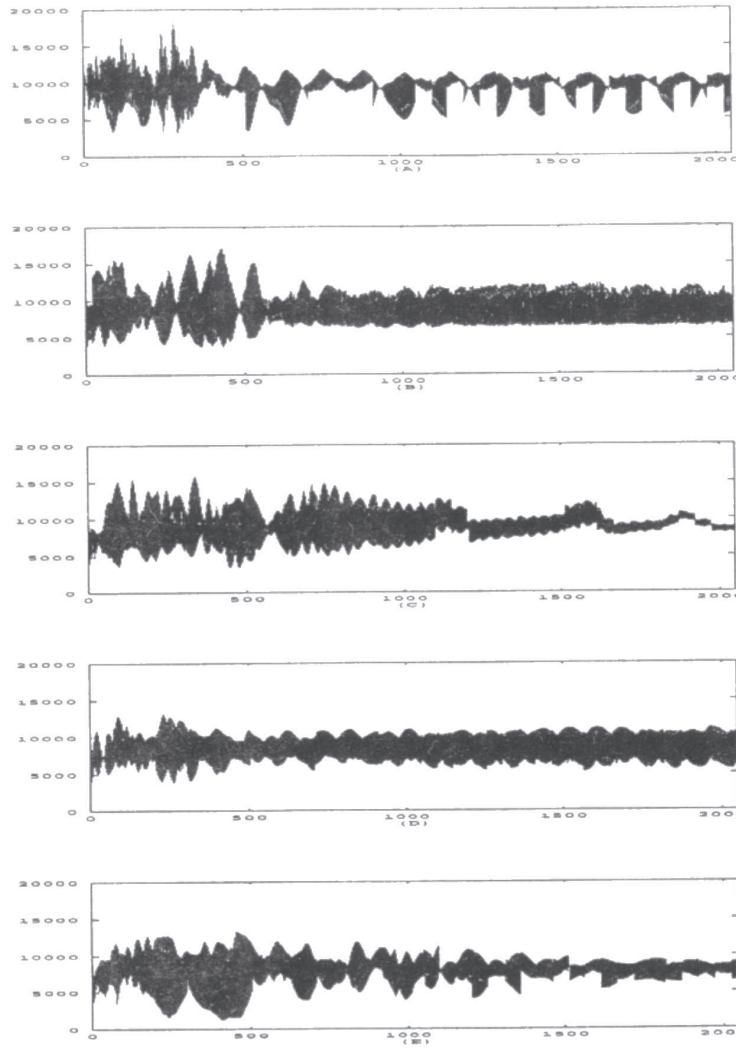

Figure 4. Dynamics of the newborns in the population, as viewed through lipid density, over 2,048d. (A) C_w= 0.0(mg.L), (B) C_w= $0.3x10^{-4}$(mg.L), (C) C_w= $0.32x10^{-4}$(mg.L), (D) C_w= $0.34x10^{-4}$(mg. L), (E) C_w= $0.36x10^{-4}$ (mg. L).

a way that at the threshold the dynamic is very much like the dynamic of the unstressed population. Unfortunately, our conjecture that the power spectrum is important in comparing effects of toxicants seems false as there appears to be no correspondence between the power spectrum and the severity of the chemical stressor, either in the ordering of the high, intermediate or low frequencies.

The second illustration relating bioassays and dynamics comes from some recent work by Jaworska et al. (1996; in press a; b; c) who consider a microbial food chain model where ciliates feed on bacteria indiscriminantly without respect to size selection and the ciliate predator has access to the entire prey population as a resource. The model is formulated for a batch system and because individual, population and community model details are available in the references, we present only the salient conclusions to demonstrate some feasible relationships between bioassays and dynamics. Model simulations are parameterized for *Escherichia coli* and *Tetrahymena pyriformis*. Because of the batch setting, *Tetrahymena*

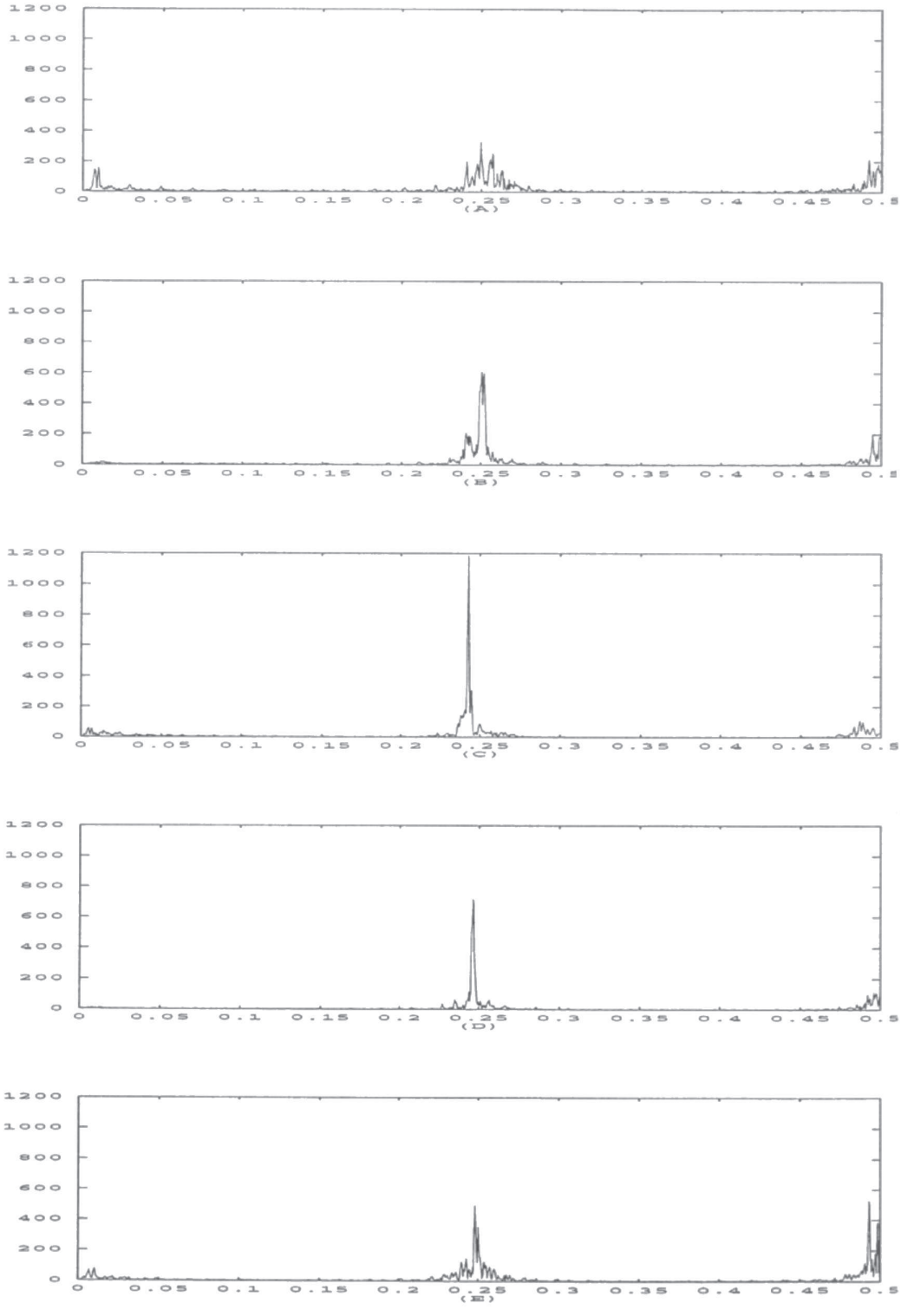

Figure 5. Power spectra of the time series shown in Figure 4. (A) C_w= 0.0(mg.L), (B) C_w= 0.3x10^{-4}(mg.L), (C) C_w= 0.32x10^{-4}(mg.L), (D) C_w= 0.34x10^{-4}(mg.L), (E) C_w= 0.36x10^{-4}(mg.L).

by-products of metabolism which are excellent nutrients for bacteria also need to be followed. The model assumes that the amount of these substances generated is proportional to the biomass of tetrahymenids with the carbohydrates to amino acids amount always being in a fixed ratio. This creates am interesting feedback in tile system.

Because of this model's setting, a batch culture, in which environmental conditions constantly change, it is important to follow the amount of organic material circulating in the environment such as dead organisms and feces. As time progresses additional toxicant is bound to this organic material, thus providing a buffer of protection for the living species. In an environment where stress is mild, it is possible that, at the end of a simulation, toxicant is present in insufficient quantity to cause any adverse effect even though initially there were effects. The dynamics of such a stressed community is generally only slightly perturbed, in that it takes longer for the community to organize compared to the unstressed behavior. With initial predation at low levels, the bacterial population is not depleted as fast as in tile unstressed experiment mid the massive death of ciliates also occurs later. A delay in development occurs because the growth rate of the tetrahymenids is reduced by the stressor.

Another important feature of this community is that ciliates and bacteria are not equally susceptible to the toxicant. According to QSARs results, bacteria are less sensitive than ciliates. Therefore, conditions can occur where only one population, the ciliates, is adversely affected. When both populations are affected by chemical toxicity, there are two types of outcomes. First, the stress has to be quite toxic to the ciliate population in order to have an impact on the bacterial population. Subsequently, ciliates die. quickly and bacteria are released from predation pressure but do not bloom because their growth is reduced by toxicant stress. The case when both populations go to extinction is less interesting with extinction of ciliates proceeding extinction of bacteria.

Simulations also indicate that the community can survive in environments where chemical concentrations are above the lethal concentrations predicted by bioassays for single populations of *E. coli* and *Tetrahymena.* Indeed, survival of the community was observed in the simulations at toxic levels of almost six times the LC_{50}, for *Tetrahymena.* At concentrations below the EC_{50} for growth of ciliates, negligible effects on both populations resulted because of the eventual low bioavailability of toxicant in the water.

In the initial phase of simulations covering the concentrations above LC_{50} but less than 5.9 x LC_{50} the chemical effects are a depression of ciliate density below that of the unstressed population. However, uptake of chemical results in a lower toxicant concentration in the water which allows deputation and growth of tile ciliates to levels that are almost equivalent to those of a healthy population.

At the beginning of the stress, the lower predation allows growth of the bacteria population. When die ciliates are recovering, increased predation pressure on the bacterial population results and the bacteria numbers decline.

Discussion

Bioassays, generally focusing on single populations, or oil specific subsets of populations for limited tune scales, can be misleading for studying the effects of chemicals on both tile population and community levels. We have illustrated models where perceived outcomes from bioassays may be changed in either direction; that is, the situation can be worse or better than indicated by the bioassay.

To anticipate the effects observed at the population or community level, a caricature of population dynamics requires knowledge of ecology (e.g. detailed individual life history mid physiological attributes as well as environmental factors affecting toxicant stressor levels) and toxicology (chemical mode of action, concentration).

We have presented only two illustrations to demonstrate that bioassay results might not have tile same consequences in population or community settings. It is our feeling that these phenomena are the rule rather than the exception because the mechanisms that determine response (reproduction attained at a fixed size; buffering characteristics) are robust. Consequently, (lie effects of chemical exposure in a system can be determined experimentally only in tile proper setting.

Not only does it appear impossible to extrapolate from bioassays, it is also difficult to relate dynamics

of chemically stressed populations to observed fluctuations in population numbers or biomass. We have considered model output from a structured population model that is subjected to chemical stresses of varying concentrations and analysed the resulting time series by employing a spectral analysis on the time series of reproductive output. We found no correlation or pattern between the level of concentration in the water and the dominant frequencies in the model solutions. Determination of effects of toxic chemicals on populations and communities appear difficult even in a model setting where almost every system control can be imposed.

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