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SIMULATION OF STRUCTURED POPULATIONS IN CHEMICALLY STRESSED ENVIRONMENTS

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DEDICATION

Professor MA Zhien, as a septigenarian, remains a significant international influence on the research and teaching of applied mathematics and mathematical ecology. We are delighted to join the authors of this special volume in dedicating this paper in honor of Zhien-an outstanding scientist, an exceptional mentor, a stimulating teacher, an impressive operatic talent even on the rare occasion when the English or Italian words evade him, a fine gentleman and an extremely good friend. It has been a pleasure to study and learn from Zhein many aspects of mathematical ecology and on this occasion to return to the subject of our first joint endeavors and the topic of this paper, ecotoxicology.

Abstract. A heterogenous environment usually impacts, and sometimes determines, the structure and function of organisms in a population. We simulate the effects of a chemical on a population in a spatially heterogeneous environment to determine perceived stressor and spatial effects on dynamic behavior of the population. The population is assumed to be physiologically structured and composed of individuals having both sessile and mobile life history stages, who utilize energetically-controlled, resource-directed, chemical-avoidance advective movements and are subjected to random or density dependent diffusion. From a modeling perspective, the presence of a chemical in the environment requires introduction of both an exposure model and an effects module. The spatial location of the chemical stressor determines the exposure levels and ultimately the effects on the population while the relative location of the resource and organism determines growth. We develop a mathematical model, the numerical analysis for this model, and the simulation techniques necessary to solve the problem of population dynamics in an environment where heterogeneity is generated by resource and chemical stressor. In the simulations, the chemical is assumed to be a nonpolar narcotic and the individuals respond to the chemical via both physiological response and by physical movement. In the absence of a chemical stressor, simulation experiments indicate that despite a propensity to move to regions of higher resource density, organisms need not concentrate in the vicinity of high levels of resource. We focus on the dynamical variations due to advection induced by the toxicant. It is demonstrated that the relationship between resource levels and toxicant concentrations is crucial in determining persistence or extinction of the population.

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1. **Introduction.** Populations are structured by organismal processes and intraspecific interactions in synchrony with the biotic and abiotic components of their environments. Previous results modeling the role of a toxic chemical in dynamically structuring the population have generally assumed a homogeneous environment [6, 10, 13, 16, 19, 20]. In this paper, we discuss mathematical model relationships between the processes specific to the individual organism (physiology and energetics), intraspecific interactions at the population level (mortality) and the heterogeneous abiotic environment (resource dynamics and chemical stress). This approach requires the integration of several representations including the following: (i) a model of the abiotic environment; (ii) a model of an individual organism that represents physiological processes fundamental to growth and is sufficient to measure effects of environmental stressors; (iii) a model of the exposure and effects of stressors at the individual level; and (iv) a model of the population that represents the growth dynamics, variation between individuals, and also tracks both birth and death events and their interspecific interactions. The novel aspects of this paper include the coupling of the chemical uptake and effects with the spatially heterogeneous environment model. For physiologically-based models, we present a general simulation approach, indicate numerical techniques, and give examples that illustrate the dynamics of a chemically stressed structured population in a spatially heterogenous environment. The numerical techniques of [4, 21] for age-structured models and the procedure indicated for physiologically structured models in [7] are generally applicable to this extended setting but require a technique that numerically destructures the physiological components of the individuals in the population. We liberally use the ideas, process representations, and fish parameter values obtained in our previous work [2, 4, 8, 9, 13, 16, 17]. An objective of this paper is to provide a synthesis that integrates biological and environmental concepts in stress ecology. To investigate the structured population in a heterogenous environment requires a new numerical scheme that is indicated here in some detail.

Spatial structuring of a population can occur when dispersion processes, such as advection or diffusion, force a change in the location of individuals. The numerical methods for models of classical advection (hyperbolic models) and diffusion (parabolic models) remain independent. For advective populations in a habitat characterized by a homogeneous resource, the method of characteristics where cohorts of individuals with the same age and physiological condition often forms the basis of the numerics [9, 17]. In heterogeneous environments where environmental characteristics associated with the location of the individuals and the movement behavior are similar, a uniform advective force makes it possible to regard members of a cohort as identical [9, 17]. However, when forces disperse cohorts of individuals, the situation is numerically complicated when similar individuals in the same age or size cohorts are physically transported to locations where they are exposed to different levels of toxicants or resources. Forces that disperse cohort individuals that were previously uniformly clustered require novel numerical treatments that are especially needed to address population models driven by individual control processes.

The dynamics of the stressed structured population models can be complex. Here we consider survival mechanisms for a population in a stressed environment and discuss aspects of persistence and extinction [11, 12] in a simulation model.

2. **The Mathematical Model.** Individual-based population models structured by physiological representations are a proven methodology for investigating the role of energetics in population dynamics [1, 6, 14, 15] in a homogeneous environment. The spatial environment has traditionally been suppressed in ecotoxicological modeling efforts that involve complexities such as individuals represented by physiological structure, reactions to chemical stresses, and movement in a heterogenous environment [3, 5, 10]. However, continuous spatio-temporal processes can be formulated within similar setting, an extended McKendrick–von Foerster framework; for example, advection can be directly included in the hyperbolic formulation, provided individual movement dynamics can be explicitly represented [9, 17] and diffusion incorporated in the traditional manner. We will investigate such a prototypic model, its numerical analysis, and its application to environmental stresses.

The mathematical model consists of an individual model describing the organism's life history in terms of physiological variables, a population model that integrates across all individuals in the population, an exposure model that determines the concentration of chemical in an organism, and an effects model to determine the consequences of a stressor concentration. Because of the intricacies of the ecotoxicological problem, we choose to employ minimalistic representations in our formulation, which helps to explain the relevance of each assumption. The life history of the organism is assumed to consist of two stages, one sessile and one mobile. The abiotic factors, the resource and the chemical, are assumed to be either homogeneous across the entire spatial domain or nonhomogeneous step functions along cells in the spatial domain. The chemical is assumed to be a nonpolar narcotic, which includes a large percentage of known chemicals (approximately 70%) and is a relatively simple mode of action that allows direct uptake and effects representations.

2.1. **The Population Model.** The population dynamics, as described by extended McKendrick–von Foerster-type partial differential equations, reflects changes in time and space through a density function: $\rho = \rho(t, a, m_1, ..., m_M, x)$ $(numbers/d \cdot m^M$, number per age per mass of physiological variable m_i, at time t and location $x)$. It is assumed that the organism's life history contains a stage where individual movement is not significant to the spatial modeling concerns such as can occur in the early life stages, the embryonic or the juvenile period. During this time of immobility, assumed to occur for $a \in (0, J]$, the population model is, for $t \in [t_0, T], a \in (0, J], x \in \Omega$,

$$\frac{\partial \rho}{\partial t} + \frac{\partial \rho}{\partial a} + \sum_{i=1}^{M} \frac{\partial(\rho\, g_i)}{\partial m_i} = -\mu\rho\,; \tag{1}$$

in the remaining mobile life stages (including the adult stages), for $t \in [t_0, T], a \in (J, A_m], x \in \Omega$,

$$\frac{\partial \rho}{\partial t} + \frac{\partial \rho}{\partial a} + \sum_{i=1}^{M} \frac{\partial(\rho\, g_i)}{\partial m_i} - \nabla \cdot (k\, \nabla\rho - \mathbf{q}\rho) = -\mu\rho\,; \tag{2}$$

and the initial-boundary condition is

$$
\begin{aligned}
&k\frac{\partial \rho}{\partial \nu} - \mathbf{q}\nu = 0 \quad or \quad \rho = 0, \quad \text{on} \quad \partial\Omega, \\
&\rho\,|_{t=t_0} = \rho_0 \\
&\rho\,|_{a=0} = \int_0^{A_m}\int_0^{\infty}\cdots\int_0^{\infty} \beta\rho da dm_1\cdots dm_M,
\end{aligned}
\tag{3}
$$

where A_m is the maximal attainable age, μ is the mortality function, k is the diffusivity coefficient, $\mathbf{q}$ is the advection rate function, g_i (briefly described in (4) below) represent the rates of change of the physiological variables, $\beta = \beta(t, a, x, P, m_1, \cdots, m_M)$ is the birth rate and $P(t, x)$ is the total population size at (t, x). This analysis considers diffusion [20, 25] that is a random walk plus density-dependent dispersal which is represented as $k = 1 + \kappa_p \hat{P}$, $\hat{P} = \int_J^{A_m} \rho\, da$, where κ_p is a constant and $\hat{P}$ stands for the number of mobile organisms. The function $\mathbf{q} = \mathbf{q}(a, m_1, m_2, ..., m_N, x, P, ...$ in (3) represents the advection movement velocity (m/d) and describes how an individual of age a with physiological condition represented by the variables m_i at location x alters its advection movement in response to environmental heterogeneity and energetic constraints. The ν is the unit outward normal direction vector of the boundary of the domain, $\partial\Omega$.

2.2. **The Individual Model.** On time intervals where there are no reproductive events, the dynamics of the physiological variables (such as lipids and proteins) are modeled as an M-coupled ordinary differential system:

$$
\begin{aligned}
&\frac{dm_1}{da} = g_1(t, a, m_1, m_2, \cdots, m_M, x) \\
&\cdots \\
&\frac{dm_M}{da} = g_M(t, a, m_1, m_2, \cdots, m_M, x),
\end{aligned}
\tag{4}
$$

where a represents age of the individual, t represents time, x represents the spatial location in the habitat $\Omega \subset R^Z$ $(Z = 1, 2, 3)$, and m_i $(i = 1, 2, \cdots, M)$ represents the masses of the M physiological variables governing the growth of the organism. The g_i include representation of the process effects of environmental variables such as temperature and toxic chemical concentrations [6, 9, 13, 17].

2.3. **The Exposure Model.** The exposure model is coupled with the individual model. The basic ideas employed to assess chemical exposure and effects on individuals are discussed in [18]. The uptake of chemicals from the environment and food represents the chemical exchange between the aqueous environment and the individual fish across the gill membranes and the chemical exchange between the fish and its food across the intestinal wall. The uptake model we use is a modification of the Food and Gill Exchange of Toxic Substances (FGETS) [18] to handle exposure of fish to nonpolar, hydrophobic, reversible chemicals.

The mathematical model that describes the processes of chemical uptake from the environment and food and includes dilution of chemicals due to organism growth is

$$
\frac{dC_T}{dt} = k_1 C_w + \frac{F}{W_T} C_F - k_2 C_T - \frac{E k_E}{W_T} C_A - \frac{1}{V}\frac{dV}{dt} C_T, \tag{5}
$$

where C_T, C_w, C_F, and C_A are respectively the concentrations of the chemical in the whole fish, in the environment, in the food, and in the aqueous portion of the organism. F is the weight of the food eaten per day, and E is the weight of

material defecated per day. k_E is the partition coefficient of chemical to excrement and is given by $k_E = \frac{C_E}{C_A}$, where C_E is the concentration of the chemical in the feces. k_1 and k_2 are the uptake and depuration rates of the environmental chemical, respectively, and are specified by

$$k_1 = S_g k_w V^{-1}, \quad k_2 = S_g k_w V^{-1}(P_A + P_L K_L + P_S K_S)^{-1}.$$

k_w measures the conductivity of the exposed surface area, S_g, and V is the volume of the organism calculated by using the total weight, W_T, and the density of the organism. P_A, P_L, and P_S are the aqueous, lipid, and structural fractions of the organism, respectively; K_L is the partition coefficient of the chemical between the organismal lipid and water; K_S is the partition coefficient of the chemical between the organismal structure and water. The last term in (5) represents the dilution of chemical due to organism growth. The relationships between this set of variables and those of the structured model are delineated in [6].

To calculate the concentration of the chemical in the food, we assume instantaneous chemical equilibration with the water and within the organism. The food, like the consumer, consists of the aqueous, lipid, and structural phases, and the chemical is distributed among them according to its affinity for these phases.

To model the effects of chemicals on individuals, the uptake model is coupled with models for the mode of action and models for concentration-response relations. Effects of chemicals on individuals focus on mortality but sublethal effects, such as reduction of growth rate, could be considered using a similar method. We do not include sublethal effects in our simulations. The assessment of mortality due to chemical action is implemented by utilizing formulations based on quantitative structure-activity relations (QSARs). We utilize results developed for baseline narcotic chemicals and relate a chemical property, the octanol/water partition coefficient, K_{ow}, to mortality of individuals. For a single individual, an effect occurs when the concentration of the chemical in the aqueous phase reaches a critical level, denoted by LC_{50}, and is calculated from the equation $\log LC_{50} = -0.8 - \log K_{ow}$ [16].

2.4. **The Dispersal Model.** Detailed information on the movement behavior of individuals is only recently becoming available, a result due to significant advances in technology. We simplify dispersal issues for illustrative purpose and assume that diffusion is a random walk dispersal and that advection is the sum of two forces, one of which is resource directed and the other is chemically directed. Individuals alter movement in response to changes in resource density and in response to concentration of contaminated media. Exposed organisms may avoid contaminated environments or may be attracted to them (for example, due to pesticide-debiliated prey) or may lose their ability to detect contamination due to toxicant effects. If the contamination is local and of short duration, avoidance can prevent the occurrence of effects on mobile organisms. However, avoidance of chronically contaminated media or food may result in resource deficiency, which could seriously reduce the population. There is no standardized procedure for determining avoidance and preference behaviors largely because they depend on the chemical and its concentration as well as the species.

Equations (1) and (2) incorporate the growth rates, g_i of the physiological variables m_i and the advection rate, $\mathbf{q}$, of an individual into the population. In particular, our simulations are based on the movement equation

$$\mathbf{q} = \mathbf{q_r} + \mathbf{q_c} \tag{6}$$

where $\mathbf{q_r}$ represents the advective movement velocity due to changes in food density and $\mathbf{q_c}$ represents the advective movement velocity due to changes in the concentration of contaminated media. The function $\mathbf{q_r}$ is

$$\mathbf{q_r} = \kappa_r v_s \frac{\partial r}{\partial x} , \tag{7}$$

where κ_r is a positive constant that measures the tendency of predators to pursue prey and represents the distance covered by the foraging predator per unit change in the prey density. The derivative $\frac{\partial r}{\partial x}$ represents the rate of change of the resource at x; v_s is an average swimming velocity (m/d) of an individual with length $L_f\,(m)$ and is given by $v_s = 8.64 \cdot 10^4 s L_f$, where s denotes the body lengths per second of the predator while performing sustained cruising. The function $\mathbf{q_c}$ is

$$\mathbf{q_c} = \kappa_c v_s \frac{\partial C_w}{\partial x} , \tag{8}$$

where κ_c is a constant that measures the tendency to move according to chemical concentration and represents the distance covered by the foraging fish per unit change in the contaminated media. If κ_c is positive, the fish is attracted to the contaminated location; if it is negative, then it tends to avoid the contamination.

The mortality function μ accounts for different types of mortalities. It includes mortality due to the physiological process of aging, mortality due to starvation, juvenile density-dependent mortality, and mortality due to exposure to lethal levels of a toxicant. Toxicant mortality is assessed at the individual level according to lipid content of the organism.

The toxicant-population model is formulated so that a toxicant may be released at different locations and at numerous times for an arbitrary exposure length, and as a result, we may obtain a spatially explicit variation in toxicant concentration. The spatial location of the chemical stressor determines the exposure, and the chemical concentration, which can be variable over the spatial domain. We do not simulate the chemical transport, but rather we mimic point sources and nonpoint sources with functional representations in the model.

Our numerical procedure follows cohorts of individuals that move continuously in the heterogeneous habitat, which results from both resource and toxicant distributions, and allows effects of spatially explicit toxicant exposure to be assessed at the individual level. A characteristic behavior of the unstressed model is that the fish and resource biomass as well as the age, lipid, and structural distributions are dynamically related to a period of one year, the same period as the reproductive events, for parameter values that result in the coexistence of the population. As will be indicated graphically below, the reproductive peaks in the population dynamics are dominant features that are present even in populations that are going to extinction.

3. **The Computational Model.** The computational model is based upon a localization technique, physiological destructurization (to decompose the structure related to the physiological model), and a linearization approach. To localize the problem, we introduce a small positive parameter for step size that leads to a sequence of local problems. Physiological destructurization is used at the local problem level where the individual growth model and the partial differential equations

for the population dynamics are considered sequentially. The initial value problem of the ordinary differential system describing the individual is approximated and this approximate solution is inserted into the partial differential problem. The partial differential equation solution is then approximated over the small time interval.

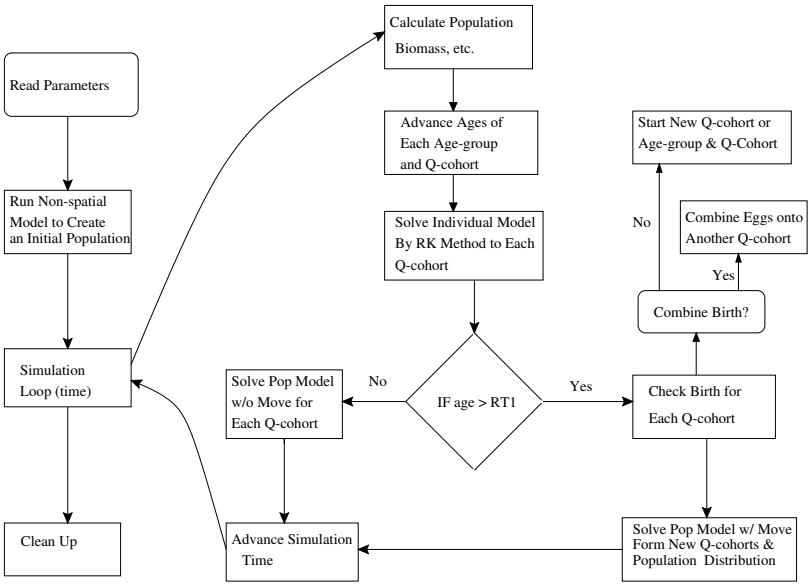

FIGURE 1. The complete flow chart for the numerical simulations.

The numerical solution of the local structured population model differs from classical approaches because the model form is a nonlocal initial-boundary value problem for a nonlinear partial differential equation with discontinuous coefficients. Then, this problem is linearized using the technique of a positive delay to handle and overcome the nonlinearity. The flow chart of the numerical simulation is provided in Figure 1.

Because of stiffness, the numerical methods use the implicit Runge-Kutta method for the individual model. For the partial differential equation problem, we use a characteristic finite difference discretization in the age-time domain and a finite element method with numerical integration and upwind modification of advective terms in the spatial domain. Our analyses for this and related age-structured models show that the numerical schemes not only yield optimal error estimates from the perspective of numerical analysis, but they also produce biologically reasonable approximate solutions [2, 4].

Let τ be the finite difference mesh size for age a as well as for time t,

$$\begin{aligned} 0 &= t_0 < t_1 < t_2 < \cdots < t_{N_t} = T, \\ 0 &= a_0 < a_1 < a_2 < \cdots < a_{N_a} = A_m, \end{aligned} \tag{9}$$

where $t_n = t_0 + n \cdot \tau$, $a_j = a_0 + j \cdot \tau$ and there is a N_j such that $a_{N_j} = J$. We apply the 1-stage implicit Runge-Kutta method for the individual model (4); see [2].

Our development of a computational scheme for individually-based population models respects the heterogeneity of the environment by employing the technique of tracking age-groups and the construction or reconstruction of special cohorts. An *age-group* is a collection of individuals with the same age. Because of the

heterogeneity, growth characteristics of individuals in the population may differ and, at times, be considerably distinct. We introduce the *growth pattern* (*q-cohort*) into the *age group* to account for these individual growth differences. The *q-cohort* consists of individuals of the same age whose physiological properties are *close* in that these individuals will have *similar* responses to the same environment over a time computational step and over a local spatial region. In a single age-time step of the computation, the *q-cohort* functions as a characteristic cohort in a spatially homogeneous model. Essentially, the final values of this step are calculated by numerical schemes of the individual model and the individual-based population model. For the transition from the current step to the next computation, the algorithm reconstructs the *q-cohorts* and initial values for the next step because the previous *q-cohorts* may have already changed in the spatial environment. Figure 2 presents a flow chart for one time-age step of a *q-cohort* of the numerical procedure. We use an array structure for a *q-cohort*, each of whose array elements stores a

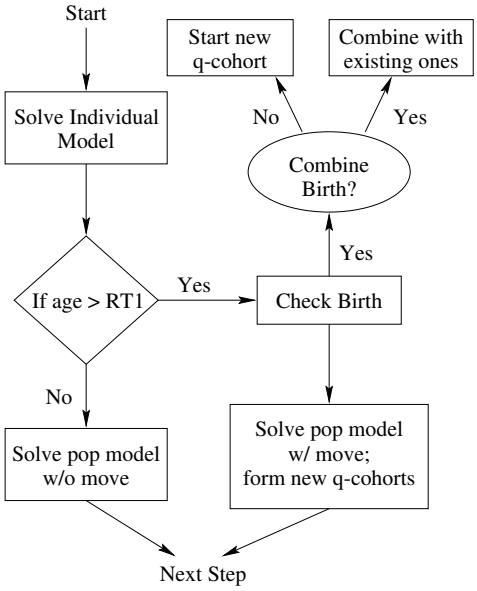

FIGURE 2. Flow chart for one time-age step of a *q-cohort*.

physiological characteristic value or age. A double-linked list of *q-cohorts* is used to describe the differences in individuals of the same *age-group* in a small spatial area (represented by a nodal point) at a special time. For a *q-cohort* with a known value, $(m_1^*, \cdots, m_M^*)$, as its representative physiological properties, obtained from the individual model (4) at a time step $t = t_0 \geq 0$, the population equation for this *q-cohort* at time interval $[t_0, t_0 + \tau]$ can be rewritten, for $t \in [t_0, t_0 + \tau]$, $a \in [0, A_m], and\ x \in \Omega$,

$$\frac{\partial \rho}{\partial t} + \frac{\partial \rho}{\partial a} + \sum_{i=1}^{M} g_i \frac{\partial \rho}{\partial m_i} - \nabla \cdot (\tilde{k}\, \nabla \rho - \tilde{\mathbf{q}} \rho) = \ -\tilde{\mu} \rho \,, \tag{10}$$

where $\tilde{k}$ and $\tilde{\mathbf{q}}$ are zero-extended to $a \in [0, J)$ and have the values k and $\mathbf{q}$ at $((m_1^*, \cdots, m_M^*, t_0, \cdots)$, respectively; $\tilde{\mu}$ stands for $\mu + \sum_{i=1}^{M} \frac{\partial g_i}{\partial m_i}$ evaluated at $(m_1^*, \cdots, m_M^*, t_0, \cdots)$.

Let $u_0(a, x) \in L^2([0, A_m],\ V)$ be nonnegative. Then, under sufficient smoothness conditions, there exists a unique nonnegative weak solution of the following age-structured population problem, That is, there exists a unique nonnegative $\rho \in L^2([t_0, t_0 + \tau] \times [0, A_m],\ V)$ such that, for $t \in [t_0, t_0 + \tau],\ a \in [0, A_m]$,

$$\begin{aligned}
&(\partial_\tau\ \rho,\ w) + \alpha(\tilde{k},\ \tilde{\mathbf{q}}\rho,\ w) = -(\tilde{\mu}\rho,\ w), \quad \forall\ w \in V,\\
&\rho(t_0, a, x) = \rho_0(a,\ x), \quad \forall\ x \in \Omega,\\
&\rho(t, 0, x) = \int_0^{A_m} \beta\rho(t, a, x)da, \quad \forall\ x \in \Omega,
\end{aligned}$$

where $V = H^1(\Omega)$ or $H_0^1(\Omega)$, $(\cdot,\ \cdot)$ is the inner product of $L^2(\Omega)$; and

$$\begin{aligned}
\partial_\tau\rho &\equiv \frac{\partial\rho}{\partial t} + \frac{\partial\rho}{\partial a} + \sum_{i=1}^{M} g_i \frac{\partial\rho}{\partial m_i},\\
\alpha(\tilde{k}, v, w) &= \int_\Omega (\tilde{k}\nabla u - \tilde{\mathbf{q}}u)\nabla w dx\ .
\end{aligned}$$

Since the above partial differential problem is linear, the verification is straightforward; see [2, 8, 9, 11] for the details.

Therefore, we can develop a meaningful numerical scheme for the population model in the time interval $[t_0, t_0 + \tau] = [t_{n-1},\ t_n]$. We introduce a weakly acute simplex triangulation $\mathcal{T}_h$ with mesh size h for Ω [2, 4]. In particular, if $\Omega = [0,\ L]$, $\mathcal{T}_h$ is:

$$0 = \ x_0 < x_1 < x_2 < \cdots < x_{N_h+1} = L, \tag{11}$$

where $x_i = x_0 + ih$ and $h = L/(N_h + 1)$. Let V_h be the linear finite element space over $\mathcal{T}_h$ to approximate V and $\mathcal{N}_h$ be the set of all true unknown nodal points of V_h. Thus, the numerical scheme on $[t_{n-1},\ t_n]$ is

$$\tilde{\partial}_\tau \rho_h^{n,j}(i) = -\tilde{\mu}_h^{n-1,j}(i)\ \rho_h^{n,j}(i),\ i \in \mathcal{N}_h, 1 \le j \le N_j,$$

$$\begin{aligned}
&(\tilde{\partial}_\tau \rho_h^{n,j}, w)_h + \alpha_h(\tilde{k}_h^{n-1}, \tilde{\mathbf{q}}_h^{n-1}, \rho_h^{n,j}, w) =\\
&\ -(\tilde{\mu}_h^{n-1,j}\rho_h^{n,j}, w)_h,\ \forall w \in V_h, N_j < j \le N_a,\\
&\rho_h^{n,0} = \frac{1}{2}\tilde{\beta}_h^{n-1,0}\rho_h^{n,0}\tau + \sum_{j=1}^{Na-1} \tilde{\beta}_h^{n-1,j}\rho_h^{n,j}\tau\\
&P_h^n = \frac{1}{2}\rho_h^{n,0}\tau + \sum_{j=1}^{Na-1} \rho_h^{n,j}\tau,\ 0 \le n \le N_t,
\end{aligned} \tag{12}$$

where $\tilde{\partial}_\tau \rho_h^{n,j} = \frac{1}{\tau}[\rho_h^{n,j} - \rho_h^{n-1,j-1}]$; $(\cdot,\ \cdot)_h$ and $\alpha_h(k, v, w)$, $(\cdot,\ \cdot)$ and $\alpha(k, v, w)$ are calculated by a numerical integration,

To solve the above scheme, a parallel algorithm has been developed in [2]. This is especially relevant for 2- or 3-dimensional settings where the computational effort can be voluminous. The following proposition is important to the numerics (for the age structure analogue, see [4]).

Let $\mathcal{T}_h$ be a weakly acute simplex triangulation. Then under appropriate smoothness conditions and if $\tau > 0$ is sufficiently small, the numerical scheme (12) has a unique nonnegative solution.

4. **The Simulation Model.** For the convenience of graphical presentations, the habitat is assumed to be linear, $\Omega = (0, L)$. L is taken to be 1 Km and is divided into 10 equal length cells. Each of the resource density and chemical concentration is assumed to be homogeneous within a cell but can differ between cells.

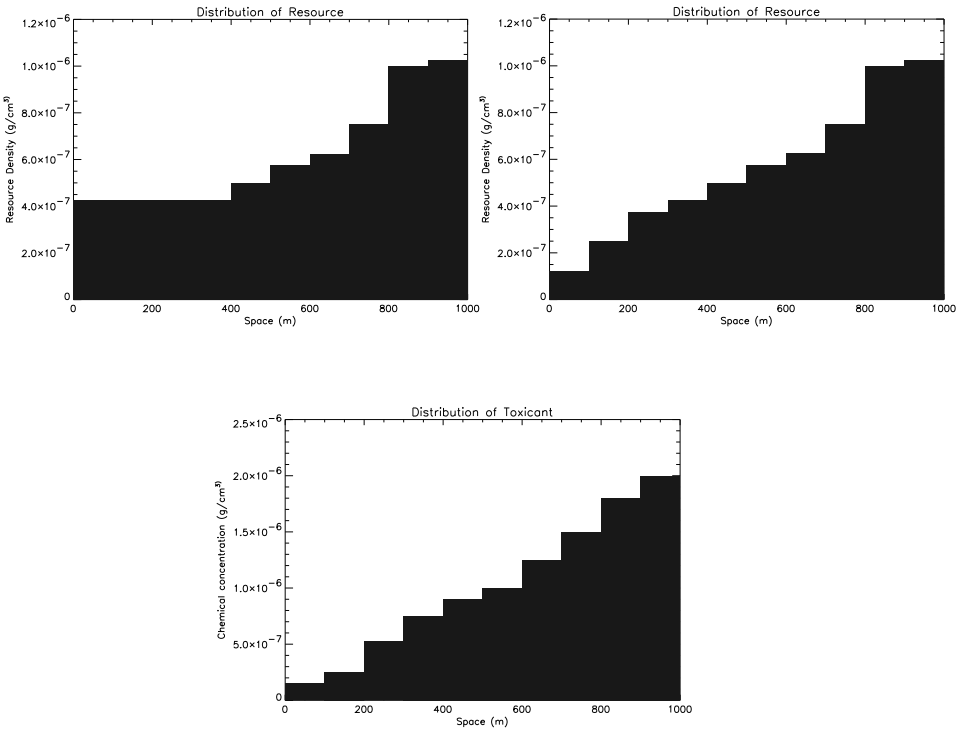

FIGURE 3. a: (Upper left) and b: (upper right) Resource Distribution Patterns; and c: (lower) Point Source Chemical Toxicant Distribution.

We choose the same step size for age and time as in (5) with $\tau = 1/20$ *days* and the triangulation $\mathcal{T}_h$ as in (7) with $h = 0.0025Km$. We focus on investigating the dynamics of an *Oncorhynchus mykiss* (rainbow trout) population under stress. Numbers and size as measured by total fish nonlabile structure are among the variables we will track in the simulations. For *O. mykiss*, an energetics-based individual model has been described in the form (4) with $M = 2$, and the state variables are lipids and proteins. We refer to [2, 8, 13] for the explicit formulations of g_L and g_S.

In the simulations, the mortality function μ is assumed to have the form $\mu = \mu_a + \mu_w + \mu_y + \mu_d$ where μ_a, μ_w, μ_y, and μ_d represent the mortality related to age, size, the younger age classes and density, respectively (cf. [6, 8]). The population birth process is determined at the individual level by accumulating the totality of births resulting from the individual model outputs. The resource-directed advection

velocity, $\mathbf{q_r}$, of an individual generally will depend on the size of the individual, the individual energy available, and the gradient of the resource at the position where the individual is located.

The advective movement rule requires that individuals tend to move towards neighboring areas with higher food densities. $\mathbf{q} = \kappa v_s \dfrac{\partial r}{\partial x}$. We refer to [6] for the details and their parameter values.

5. **Methodology.** Simulation results for individual-based populations with both diffusion and advection are few, although there are numerical and theoretical studies for some special cases [4]. We have tested some of our models without toxicant stress and reported the results elsewhere [4, 7]. Here we present simulations carried out for situations that include differences in the resource distributions (homogeneous or heterogeneous step functions), a movement type (random diffusion-advection), and special boundary conditions (Dirichlet). We can consider chemical effects that range from sublethal to lethal although here we report on the results for lethality.

To demonstrate the importance of environmental heterogenity, a chemical stressor is introduced into the spatial environment, and we investigate the effects of varying the spatial distribution of the environmental chemical concentration.

In the numerical simulations, we take the time-age step size $\tau = 1/20$ days, the spatial mesh size $h = 0.0025$ km and the simulation end time $T = 5,400$ days. For simplicity, we assume that the initial population occupies only a 1% interval located at the middle of the habitat domain Ω.

The heterogeneous environment consists of a step function chemical distribution and either a uniform resource or a step function resource distribution (Fig. 3). We then illustrate the dynamic behavior of the population when a particular heterogeneity is present.

6. **Results.** Lassiter and Hallam [16] developed an approach to evaluate the effects of the lipid distribution on population dynamics in response to an acute exposure of a lipophilic narcotic chemical in a homogeneous environment. According to this theory, in an assessment of mortality, an individual with smaller lipid fraction content will die before another individual with a larger lipid fraction, given equal exposure. This theory, the survival of the fattest, considers homogeneous toxic exposures to a static population that are necessarily acute. The theory is not valid when these assumptions are violated [6].

For dynamic populations, survival of the population after chronic exposure is determined not only by the lipid distribution, but also by the growth rate of the individuals in the population [6]. The prediction of survivors of toxic stress in dynamic populations is difficult even in homogeneous environments. Some scenarios of exposure that can lead to slowly growing organisms as the dominant survivors, intermediate growing organisms as the dominant survivors, and fast growing organisms as the dominants in the population. This can depend on seemingly innocuous characteristics such as sex and reproductive state [6].

In addition to the physiological aspects of the population, the response of a fish population to a toxicant exposure depends on the spatial pattern of the toxicant and resource as related to the distribution of individuals in time and space, the duration of the exposure, and the concentration of the toxicant, as is demonstrated below. The spatio-temporal pattern of release of chemicals from a source(s) is a major aspect of uncertainty in an ecological risk assessment, but this aspect has

been addressed in some useful software (Spatial Analysis Decision Assistance; see www.tiem.utk.edu/SADA).

We consider two simple scenarios for the release of a chemical: 1) point-source release (taken as an inhomogeneous spatial distribution of the chemical) and 2) non–point source release (taken as a homogeneous spatial distribution of the chemical). We incorporate release of the chemical in our model by dividing the habitat into cells that are assumed chemically homogeneous. We have analogous test distributions for the resource, as indicated in Figure 3. For the following simulations, the habitat in which the populations live is taken to be 1 km long and is divided into 10 cells. The parameter values used in the simulations may found in [17].

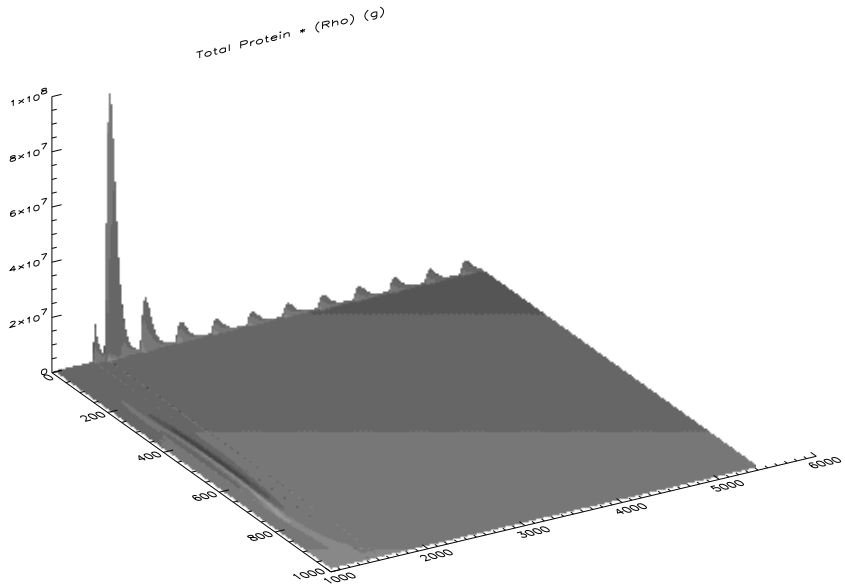

FIGURE 4. Simulation Results for the Illustration 1. The graph represents the protein dynamics in the population as a function of space and time. This is an example of persistent dynamics when the fish can avoid the chemical.

To demonstrate the effects of chemicals and the importance of chemical heterogeneity, we impose a chemical stress on the theoretical population employing the same toxic chemical throughout the examples and investigating the outcomes by varying the resource distribution, the spatial distribution of the environmental chemical concentration and the initial time of exposure. The dynamic structure of the population is compared before and after the exposure and the spatio-temporal evolution of the stressed population is compared to that of the nominal, unstressed population.

Illustration 1. This illustration compares two situations with different movement laws. The chemical distribution is the step function (Figure 3c), and the distributions of resource in each illustration are assumed to be uniform with a resource density of $5.0 \times 10^{-7} g/cm^3$. A chronic chemical exposure begins at day 1,400 in the simulation and continues to the end of the simulation, which is day 5,400.

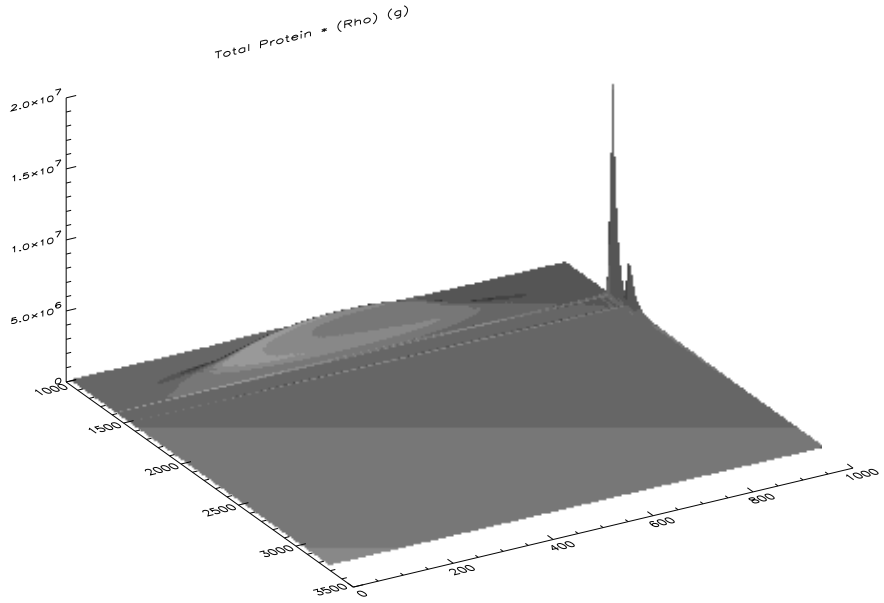

FIGURE 5. Simulation results for Illustration 1. The protein distribution of the population as a function of space and time. The example is of a population that has gone to extinction at day 3,500 due to toxic exposure. The time and distance axes have been interchanged from Figure 4 to allow viewing the extinction result more clearly.

When fish have a tendency to avoid the chemical (so $\mathbf{q_c} < \mathbf{0}$), the dynamic behavior is a plume-like structure. The main reason for the dynamic spatial structuring is that chemically induced advection dominates the diffusion process forming the plume. The population persists until the end of the simulation (see Figure 5). The graphics in Figure 5 represent the total protein components of the population in the simulation. Protein is one component of the total size of an individual, and the total protein has a close correlation with the total numbers in the population. The peaks represent the annual birth events in the population.

Under the same environmental conditions, but with the advection term $\mathbf{q_c} > \mathbf{0}$, so that the movement into toxic areas is now feasible, the advective force remains dominant but the population now goes to extinction at day 3,500. These outcomes are presented in Figure 6.

Illustration 2. In the next two examples, both the resource and the chemical are assumed to be distributed according to the step functions in Figures 3a and 3c respectively. The chemical exposure begins at day 1,400 and remains chronic throughout the simulation period of an additional 4,000 days. When fish avoid the chemical, ($\mathbf{q_c} < \mathbf{0}$), the dynamic results indicate that the population persists until the end of the simulation, taken as day 5,400. Here, chemically-induced advection dominates both the diffusion and the resource-directed advection.

The slight reduction in resource distribution as indicated in Figure 3b and the same chemical exposure scenario produces extinction at day 4,000 (Figure 6). When $\mathbf{q_c} > \mathbf{0}$ and the original resource distribution (Figure 3b) applies, the interesting case where the population still persists results despite higher exposure to the toxicant. This scenario might occur when a chemical is present in the food and the resource directed advection is operational.

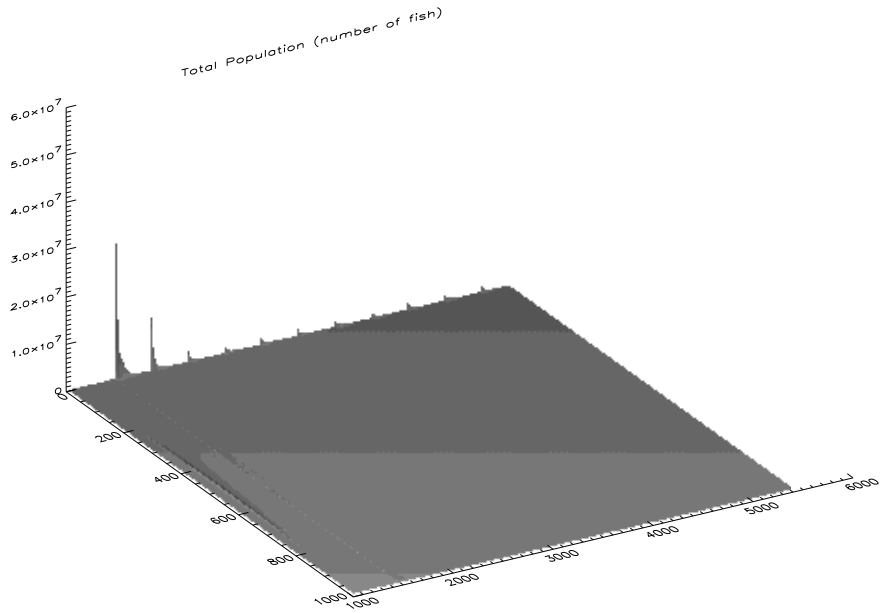

FIGURE 6. Simulation result for Illustration 2. The figure represents the protein dynamics in the population as a function of space and time when the fish can move through the chemical. The population persists despite higher toxic exposures because of an adequate supply of resource.

7. **Discussion.** These illustrations indicate that the dynamics of our physiologically structured model is sensitive to several temporal and spatial assumptions. These include (i) behavioral mechanisms that relate chemical concentrations to movements and (ii) physiologically directed mechanisms that relate resouce abundance to movement. We have shown previously that fish may not congregate in areas of highest resource level because of resource-directed movement. Here we find that exposure via movement can determine persistence or extinction of the population, and there is a trade-off between the concentrations of toxic chemical and the levels of resource available.

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