A New Approach to Ecological Risk Assessment: Simulating Effects of Global Warming on Complex Ecological Networks

Yun Zhou¹, Ulrich Brose², William Kastenberg¹ and Neo D. Martinez³

> ¹University of California at Berkeley Berkeley, California, USA 94720-1730

²Technical University of Darmstadt Schnittspahnstr. 3, 64287 Darmstadt, Germany

³Pacific Ecoinformatics and Computational Ecology Lab Rocky Mountain Biological Laboratory, P.O.Box 519, Gothic, CO 81224 USA

1. Introduction

1. 1 Ecological Risk Assessment

The field of Ecological Risk Assessment (ERA) has been under development since the 1970s. Early ERA borrowed basic concepts from human health risk assessment (HRA) methodology [NAS 1983]. However, because of the nature of an ecosystem, there is a fundamental difference between HRA and ERA. In an HRA, the only receptor is a single human being and the concerned endpoints are always associated with human health issues, such as the risk of cancer. In ERA, however, entire populations, communities and ecosystems are at risk, and ERA must rigorously assess these more complex and larger scaled concerns. Many investigators have attempted to develop a new paradigm for ERA that can deal with this intrinsic distinction. Currently, a six-step framework is now widely used by the U.S. EPA and its contractors. This new paradigm is characterized by: (1) receptor identification, (2) hazard identification, (3) endpoint identification, (4) exposure assessment, (5) doseresponse assessment and (6) risk characterization [Lipton et al. 1993, Suter 1993]. The six-step framework identifies receptors at risk, possible hazards related to certain receptors, and chooses appropriate assessment and measurement endpoints [Suter 1990]. While the additional receptor and endpoint identifications improve on the

traditional framework, single-species laboratory toxicity tests typically estimate ecological responses simply by predicting an environmental concentration associated with a certain stressor divided by the no-observed effect concentration (NOEC) for that stressor. This "Hazard Quotient" (HQ) approach ignores interactions between species that are critical to the functioning of communities and ecosystems.

1.2 Shortcomings and Challenges of Current Ecological Risk Assessment

As noted above, the major shortcoming of any ERA is that it ignores most, if not much, of ecology, Ecology focuses on relationships among species and interactions among species and the environments they live in. A well-developed ERA should focus on the structure (e.g., food-web structure) and function (e.g. biomass production or decomposition) of communities and ecosystems [Burger and Gochfeld 1992]. However, due to the ecosystem complexity and limited information, ERA in practice typically focuses on toxicity at smaller scales of biological organization, such as physiological mechanisms of toxicity and the responses of discrete endpoints in a single species to toxicant exposure [Preston 2002]. This approach to ERA employs a linear, reductionist and determinist paradigm that considers risks to each species to be independent of one another and determined by "causal" relationships inferred between input and output, such as dose and response [Kastenberg 2002]. Another major shortcoming is the absence of predictive tests [Holdway 1997] stemming from simplistic methods employed in the ERA. One prevalent method relies on singlespecies acute and chronic toxicity test data obtained in the laboratory to predict population impacts of environmental stressors in the field. However, such predictions are not field tested. Also, little if any effort is expended on understanding how estimates of toxicity vary among different environmental contexts. Prediction of longterm population-level effects in the field by applying data from short-term laboratory experiments may be particularly problematic due to the large discrepancy between the spatiotemporal scales of the experiments and the predictions they generate [Martinez and Dunne 1998]. Ecotoxicologists have been facing the challenge and working on developing a more holistic approach to ERA. Both the excedence profile (EP) [Solomon and Takacs, 2001] and the potentially affected fraction (PAF) methods illustrate a relationship between the proportion of species affected and the likelihood that their response concentrations to certain toxicant will be exceeded. The strength of the PAF method is that it considers multiple-species, instead of single-species in an ecosystem. However, species interactions, especially those that depend on species exposed to higher than NOEC levels, are not addressed well. Some mesocosm studies have estimated effects of toxicants on small-scale ecosystems. These mesocosm studies can observe both direct and indirect effects on multiple species and inform ERAs beyond information derived from laboratory single-special toxicity data. However, the mesocosm studies are almost always conducted after toxicant releases and are applied to specific species and areas, such as lakes. This limits more general understanding of ecological risks to wide varieties of ecosystems and species.

We propose a novel and more holistic *in silico* complement to ERAs that simulates the structure and dynamics of complex ecological networks to generally estimate and predict the risk under environmental stressors, such as global warming to entire communities including species' extinction risks. So far, such holistic effects of global warming that account for species' interactions have been poorly studied. In this paper, our main objective is to examine the persistence of community structure and species under a warmed climate and to provide a more synthetic, integrated, and holistic model to help decision makers and the public learn more about ecological risks associated with environmental stressors.

2. Approach

2.1 Food-web and Trophic Dynamics Models

Our *in silico* approach combines models of food-web structure [Williams and Martinez 2000] and trophic predator-prey dynamics [Yodzis and Innes 1992] with competitive consumption of multiple limiting abiotic resources by producer species [Tilman 1982, Huisman and Weissing 1999]. A detailed description of the synthetic model is given in Brose et al. (in press). Here, we briefly describe the model's basic components including the "niche model" of network structure and the bioenergetic model of feeding and consumer growth dynamics.

The network structure of the food webs are constructed by the stochastic "niche model" that uses species richness (*S*) and directed connectance (C, number of directed feeding links divided by S^2) as input parameters. The niche model hypothesizes that food-web structure is a result of a particular arrangement of a one-dimensional community niche space where all species and their diets are located as described in Figure 1.

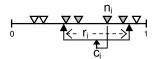


Figure 1. Niche model diagram. *S* (trophic species richness, here S = 7, shown by inverted triangles) and *C* (connectance) are set at the observed values for the empirical web being modeled. The niche model assigns each of S species a uniformly random "niche value" $0 \ge n_i \ge 1$ that establishes each species' location in the community niche. Each species is then assigned a beta distributed feeding range $0 \ge r_i \ge 1$ with a mean equal to connectance ($C=L/S^2$). Each *i*th species consumes all species within its r_i which is placed on the niche by choosing a uniformly random center (c_i) of the range between $r_i/2$ and n_i . The species with the lowest n_i is assigned $r_i = 0$ so that each "niche web" has at least one basal species. All other species that happen to eat no other species are also basal species.

Following previous works [McCann and Yodzis 1994, McCann and Hastings 1997, McCann *et al.* 1998, Brose *et al.* 2003], we use a bioenergetic consumerresource model for the species' consumptive interactions that has been recently extended to *n* species [Williams and Martinez 2001]. The rate of change in the biomass, M_i of species *i* changes with time *t* is modeled as:

$$\frac{dM_i(t)}{dt} = G_i(R) - x_i M_i(t) + \sum_{i}^{n} (x_i y_{ij} \alpha_{ij} F_{ij}(M) M_i(t) - x_j y_{ji} F_{ji}(M) M_j(t) / e_{ji})$$
(1)

where $G_i(R)$ describes the growth of producer species; x_i is the mass-specific metabolic rate; y_{ij} is species *i*'s maximum ingestion rate of resource *j* per unit metabolic rate of species *i*; a_{ij} is species *i*'s relative strength of consuming species *j*, which is equal to the fraction of resource *j* in the diet of consumer *i* when all *i*'s resources are equally abundant, normalized to one for consumers and zero for producers, and e_{ji} is the biomass conversion efficiency of species *j* consuming *i*. We used a type II functional response, $F_{ij}(M)$ that indicates the flow of biomass from resource *j* to consumer *i*.

2.1 Climate Change and Metabolic Rates

In this study, we consider a single environmental stressor, temperature change due to a warmed climate, by focusing on the effects increased temperature on species' metabolic. Earth's climate has warmed by approximately 0.5 °C over the past 100 years [Pollack *et al.* 1998], and global warming is an ongoing process. The Intergovernmental Panel of Climate Change (IPCC) (2001) has projected the mean global surface air temperature to increase by 1.4 °C to 5.8 °C from 1990 by 2100, with the magnitude of the increase varying both spatially and temporally. Coastal ocean temperature increases are expected to be slightly lower than the IPCC projected increases for land, but are still expected to rise measurably. This increase in temperature causes increases in species' metabolic rates [Gillooly *et al.* 2001]. Given that these metabolic rates define the species' expenditure of energy on maintaining themselves – their cost of life – we hypothesize that increased metabolic rates might severely impact their extinction risks.

Gillooly *et al.* (2001) proposed that species' metabolic rates depend on their body-sizes and temperatures in a highly predictable manner. The metabolic rates, *B*, scale with body mass M as $B \propto M^{3/4}$ so that their mass specific metabolic rate equals $B/M \propto M^{-1/4}$. Temperature governs metabolism through its effects on rates of biochemical reactions. Reaction kinetics vary with temperature according to the Boltzmann's factor $e^{-E/kT}$, where *T* is the absolute temperature (in degrees K), *E* is the overall activation energy, and *k* is Boltzmann's constant. When considering both species' body mass and temperature dependence of the whole organism, the metabolic rate can be derived by

$$B = B_{o} M^{3/4} e^{-E/kT}$$
 (2)

In Equation (2), B_0 varies according among taxonomic groups and metabolic-statedependent normalization constant, and M is the body mass of an individual [West *et al.* 1997, Gillooly *et al.* 2001]. T is the body temperature of organisms, at which different biochemical reactions occur. For ectotherm species – invertebrates and ectotherm vertebrates, T is nearly equal to the environmental temperature [Savage *et al.* 2004]. Equation (1) fits metabolic rates of microbes, ectotherms, endotherms, and plants in temperatures ranging from 0° to 40°C [Gillooly *et al.* 2001]. When the temperature Tchanges from T_1 to T_2 , metabolic rates ΔB , can be derived by

$$\Delta B = \frac{B_2(T_2)}{B_1(T_1)} = \frac{B_o M^{3/4} e^{-E/kT_2}}{B_o M^{3/4} e^{-E/kT_1}} = \frac{e^{-E/kT_2}}{e^{-E/kT_1}} = e^{\frac{E}{k} (\frac{T_2 - T_1}{T_2 T_1})}$$
(3)

We assume that body mass is the same before and after the temperature change. ΔB depends on $\Delta T = T_1 - T_2$, and the initial temperature T_1 . Therefore, if T_1 and ΔT are known, equation (3) can be used to predict metabolic rates.

2.2 An Example for Community Risk Assessment

We simulate the response of *in silico* species and complex ecological communities to global warming and the subsequent increase of the species' metabolic rates as an example in order to demonstrate the framework for community risk assessment we proposed in the previous section. Numerical integration of 30-species invertebrate food webs including eight producer species and 135 trophic interactions (connectance = 0.15) over 2000 time steps yields long-term predictions on community-level effects. The model parameterization assumes invertebrate species, predators that are on average ten times larger than their prey, and producer species with a relative metabolic rate of 0.2 in model cases without global warming effects. The sensitivity of the simulation results to these assumptions will be studied elsewhere. In particular, we address changes in persistence, i.e. extinction risk, for species in general and for producer species (species without prey), consumer species (species with prey) and omnivores (species feeding on multiple trophic levels). Furthermore, we study changes in food-web structure in terms of connectance (links/species²). To study the effects of global warming, we compare numerical integration results averaged over 50 replications with and without increases in metabolic rates that are subsequently indicated by the subscripts 'warm' and 'const', respectively. For instance, proportional change in species extinction risks, ΔR due to global warming are calculated as

$$\Delta R = 1 - \frac{R_{warm}}{R_{const}} \tag{4}$$

where *R* is species richness. R_{warm} is the species richness after a temperature increase and R_{const} is the species richness in the benchmark case in which the global temperature remains constant at it currently observed values. The proportional changes in the other dependent variables are calculated similarly.

To help understand our model framework, we examined the model's sensitivity to temperature change. Three climate change scenarios are studied: the anticipated "best-case scenario" minimally increases global temperature by 1.4 °C, the "average-case scenario" increases global temperature by 3.6 °C, and the "worst-case scenario" increases global temperature by 5.8 °C. Based on these three scenarios and equation (4) with E/K = 9.15 for multicellular invertebrates [Gillooly *et al.* 2001], proportional metabolic rate changes are evaluated in Table 1. Simulations concern a thirty-species invertebrate food web living in an area with a 0 °C average annual temperature.

Table 1. Proportional metabolic rate change for three climate change scenarios

| Temperature Increase | Proportional Metabolic Rate Change* | | |
|----------------------|-------------------------------------|--|--|
| 1.4 °C | 0.017% | | |
| 3.6 °C | 0.044% | | |
| 5.8 °C | 0.070% | | |

* We assume an initial temperature of 0 °C.

3. Quantitative Results and Discussion

The mean values of our dependent variables for the benchmark case ($\Delta T = 0$) and each climate change scenario are given and compared in Table 2 and Table 3. For example, with a 5.8 °C temperature increase, 0.2 species die out after 2000 time steps due to the temperature increase in the invertebrate food-web. Total species richness among all scenarios ranges from 24.3 to 23.5 and the proportional change in species extinction risks due to warming (ΔR) ranges from -2.36% to 1.1% under the projected global temperature increases of 1.4 °C to 5.8 °C, respectively. The number of links decreases , then increases from a baseline of 92.3 links to 94.9-88.9 links. The richness of top species ranges from 2.0 to 2.4 and the proportional changes in extinction risks for top species (Δ Top) ranges from 25.2% to 11.11%. The richness of basal species remains at the original number, 8 despite the temperature increases of 1.4 °C to 5.8 °C. In other words, all producer species typically survive the climate warming. Connectance varies from 0.161 to 0.162 as the temperature increases. With global temperature increases of 1.4 and 3.6 °C, species richness are greater than the benchmark species richness. In the worst temperature increase scenario, species richness drops down below 23.7, which indicates a proportional change in species extinction risk, 1.11%.

Table 2. Output parameters from the simulations for three climate change scenarios

| Temperature Increase | Species Richness | Connectance (Link/Species ²) | Links | Тор** | Basal** |
|-------------------------|---------------------|---------------------------------------------|-------|-------|---------|
| 0.0 °C* | 23.7 | 0.163 | 92.3 | 2.7 | 8 |
| 1.4 °C | 24.3 | 0.161 | 94.9 | 2.0 | 8 |
| 3.6 °C | 23.8 | 0.162 | 91.8 | 2.4 | 8 |
| 5.8 °C | 23.5 | 0.161 | 88.9 | 2.2 | 8 |

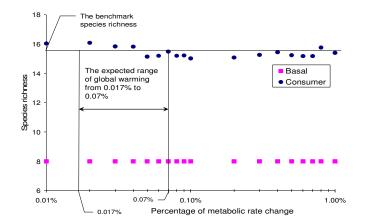
* The benchmark case, ** Top = top species richness and Basal = basal species richness.

| Table 5. Comparisons between three enhance enange secharlos and the benefiniark case | | | | | | | |
|--------------------------------------------------------------------------------------|-------|-------|---------|--------|--|--|--|
| Temperature | ∆R* | ∆Тор* | ∆Basal* | ∆Conn* | | | |
| Increase | (%) | (%) | (%) | (%) | | | |
| 1.4 °C | -2.36 | 25.2 | 0 | 1.85 | | | |
| 3.6 °C | -0.34 | 11.1 | 0 | 1.21 | | | |
| 5.8 °C | 1.10 | 18.5 | 0 | 1.56 | | | |

Table 3. Comparisons between three climate change scenarios and the benchmark case

* ΔR = proportional change of species richness, ΔTop = proportional change of top species richness, $\Delta Basal$ = proportional change of basal species richness and $\Delta Conn$ = proportional change of connectance.

From the above data analysis, we hypothesize that small temperature increases may favor the species richness. However, beyond this range, temperature increases can decrease biodiversity and increase species' extinction risks. In order to understand



better, we simulated the responses to varying the possible change of metabolic rates by 0.01% to 1% based to the temperature range from 0° C to 40° C used in equation (2).

Figure 2. Effect of metabolic rate on species richness for basal (producer) and consumer species with a metabolic rate change range from 0.01% to 1% after 2000 time steps

Figure 2 gives the tendencies of species richness with varying the metabolic rate changes from 0.01% to 1%. The trend in species richness basically suggests that global warming presents very little if any extinction risks to consumer species due only to temperature effects on metabolic rates. Basal species such as plants appear unaffected by increases in metabolic rates while such temperature increases affect consumer's abilities to persist.

Here, we propose a generic framework for estimating and predicting community risks that may be extended to address many other risks such as those posed by toxics in the environment combined with global warming. We have examined how a warmed climate in terms of metabolism change, affects biodiversity and species' extinction risks. Surprisingly, a warmed climate in terms of metabolism change makes minor effects on biodiversity and species' extinction risks, which is not as what we expected. We will explore how different mechanisms, such as assimilation efficiency will affect different ecosystems due to climate change and other stressors. In this paper, we have more simply explored how an invertebrate ecosystem may respond to a warmed climate. However, this is more a description and exploration of our methods rather than a specific prediction of future events. In order to make such predictions, further research will examine more specific food webs, such as invertebrate, ectotherm vertebrate and endothermic vertebrate food webs parameterized for more specific habitats including terrestrial and marine systems in tropical, temperate, and polar climates. Results from these future studies may help scientists to understand, and society to decide, which geographic areas and which habitats and species are most at risk and in need of the most urgent attention and action. Temporal predictions of ecosystem response to external perturbations should be very valuable for predicting and understanding ecological risks associated with climate change and other anthropogenic effects on ecosystems.

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