PROJECTED POPULATION-LEVEL EFFECTS OF THIOBENCARB EXPOSURE ON THE MYSID, AMERICAMYSIS BAHIA, AND EXTINCTION PROBABILITY IN A CONCENTRATION-DECAY EXPOSURE SYSTEM

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(Received 8 April 2004; Accepted 4 August 2004)

Abstract—Population-level effects of the mysid, Americamysis bahia, exposed to varying thiobencarb concentrations were estimated using stage-structured matrix models. A deterministic density-independent matrix model estimated the decrease in population growth rate (λ) with increasing thiobencarb concentration. An elasticity analysis determined that survival of middle stages provided the largest contribution to λ. Decomposing the effects of λ in terms of changes in the matrix components determined that reduced reproduction had a large influence on population dynamics at lower thiobencarb concentrations, whereas reduced survivorship had the largest impact on populations at higher concentrations. A simulation model of a concentration-decay system was developed to demonstrate the importance of integrating chemical half-life and management practices in determining population viability. In this model, mysids were originally exposed to a high thiobencarb concentration (300 µg/L) that decayed an order of magnitude in the number of mysid generations corresponding to thiobencarb half-life values under three different exposure regimes. Environmental stochasticity was added to the model to estimate the cumulative extinction probability of mysids exposed to fluctuating concentrations of thiobencarb in random environments. The cumulative extinction probability increased with thiobencarb half-life, stochasticity, and concentration present at the time of a new exposure. The model demonstrated the expansion of population projection models in determining the ecological impact of a population exposed to pesticides.

Keywords—Americamysis bahia  Thiobencarb  Matrix models  Cumulative extinction probability

INTRODUCTION

The mysid shrimp, Americamysis bahia, is considered a primary candidate for monitoring the health of estuarine habitats [1] and has been adopted as a standard marine and estuarine test organism by the U.S. Environmental Protection Agency (U.S. EPA) [2]. Americamysis bahia is more sensitive to toxic substances than many other marine species [3,4] and has been used to demonstrate the toxicity of heavy metals [5–7] and pesticides [8–10] on the physiological and individual levels. Although the cellular-level mechanism of and respective individual-level response to a toxicant provide valuable information on toxicant mode of action and potential species impacts, a hierarchical approach must be taken to understand ecological impacts. Hierarchical toxicology bridges the gap between levels of biological organization to obtain a comprehensive view of the potential ecological impacts of a pollutant [11]. As such, the projection of population-level effects from the response of individual mysids exposed to a pollutant is necessary to assess potential long-term ecological impacts to both the population and estuarine ecosystems, yet is limited to just a few studies [12–15].

Because the long-term field experiments that are required to monitor populations exposed to pollutants are confounded with time and financial constraints, modeling approaches are commonly employed to project population-level consequences from the biological responses of individual organisms [14,16–18]. Although modeling tools are well-developed and readily available [19], their employment to project population-level effects of toxic substances is not yet widespread. Furthermore, other tools, such as sensitivity and elasticity analyses, that are frequently applied in conservation management [20–22], are seldom applied in toxicological studies. Caswell [11,19] outlined methods for a decomposition analysis that further breaks down the observed changes in population growth rate in terms of individual-level responses. This method helps to identify the individual-level responses that result in the observed population-level effect and links the two levels of organization as they pertain to pollutant toxicity. As with sensitivity and elasticity analyses, however, this method is infrequently applied in toxicological studies.

To address the concern of pesticide exposure in estuarine ecosystems and to apply the above outlined tools of hierarchical toxicology, this study projects A. bahia population-level effects following exposure to thiobencarb (S-[4-chlorophenyl] methyl diethylcarbamothioate), a thiocarbamate herbicide primarily used to suppress annual grasses and sedges in rice cultivation. The application of thiobencarb to inundated rice fields places it in direct contact with aquatic organisms, where it poses a high risk to freshwater and estuarine invertebrates [23]. Thiobencarb has caused adverse physiological effects on a number of nontarget organisms [23–26] and has decreased the survival, growth, and young production of A. bahia in laboratory experiments [9].

Laboratory data collected from exposing A. bahia to a range of lethal and sublethal thiobencarb concentrations throughout a complete life cycle were used to project the population-level response of A. bahia to thiobencarb using stage-structured matrix models [19]. The model quantified population-level effects as the changes in population growth rate (λ) with changing thiobencarb concentration. An elasticity analysis was then used to determine which individual-level response (survival, reproduction) provides the largest contribution to λ. In another analysis, the observed changes in λ were decomposed in terms...
Population growth rate is a valuable endpoint that is indicative of biomass production and can be used to estimate population viability under varying environmental conditions [27]. Changes in population growth rate or reduced population viability can then be used to infer broader community-level consequences. Studies that derive such an estimate of a population persistence assume a constant exposure concentration over long periods of time [28, 29]. In such cases, the time to extinction can be extrapolated by the rate of population decline (λ) and environmental stochasticity [30]. However, in pesticide application, organisms are exposed at scheduled times of the season or intermittently throughout the year. Additionally, if the half-life of the chemical is short relative to the rate at which the population declines, estimating population viability from a constant exposure concentration does not accurately reflect the population dynamics as a result of pesticide exposure.

Using the observed effects of thiobencarb on A. bahia population dynamics, a simulation model was developed of a concentration-decay system that demonstrates the importance of chemical half-life, the concentration of chemical present at the time of a new exposure (management practices), and environmental stochasticity when interpreting population-level effects. The model assumes that thiobencarb is applied at regular intervals, thiobencarb half-life is constant through time, and mysid generations are nonoverlapping. Using the matrix models described above, the model determined cumulative extinction probability of mysid populations exposed to periodic, pulse thiobencarb exposures in random environments.

**METHODS AND MATERIALS**

**Model input**

Mysids were exposed to seven environmentally realistic [31] treatment concentrations of thiobencarb (0, 18.8, 37.5, 75, 150, 300, 600 μg/L) throughout the complete life cycle [9]. Newly released juvenile A. bahia were obtained from ovigerous females maintained in static, recirculating cultures. Fifteen juveniles less than 24 h old were placed into each of three replicates of each treatment concentration. Every 3 min, thiobencarb was added to the treatments using a proportional diluter as described by Schoor and McKenny [32]. Daily survival observations were made at each exposure concentration throughout the 29-d life cycle. Ovigerous females were isolated and paired with mature males in brood cups made from glass Petri dishes and nylon net (mesh size 210 μm) and daily observations were made of the number of young released by each female. For each treatment concentration, sex ratio within the replicates was approximately 50:50, and the replicates contained an average seven breeding pairs. Stage-specific survival and reproduction data were used for model input.

**Density-independent, deterministic model**

To develop a stage-structured model, the mysid life cycle was first divided into seven stages containing a variable number of time steps with a projection interval equal to 1 d (Fig. 1a): Early juveniles, less than 24 h old; juveniles, 1 to 4 day old; advanced juveniles, 5 to 10 d old; young adult, 11 to 16 d old; early breeders, 17 to 20 d old; intermediate breeders, 21 to 24 d old; and late breeders, 25 to 29 d old. Juvenile stages were selected based on ages that were representative of various mysid life stages [9]. Reproductive adults were separated into three stages (early breeders to late breeders) because preliminarily analysis of the data indicated that thiobencarb may delay reproduction in mysids. The distinction of the three adult stages was used to identify potential effects of delayed brood release on population dynamics through the elasticity analysis and decomposing the effects of λ in terms of the matrix components.

The initial model was a density-independent, stage-structured matrix model of the general form:

\[ n_{t+1} = A n_t \] (1)

where \( n_t \) was a vector of the numbers in each stage in the population at time \( t \) and \( A \) was a population projection matrix [19]. The population growth rate, \( λ \), was the dominant eigenvalue of \( A \) and was related to the continuous-time rate of increase as \( r = \log λ \). The components of \( A \) were the probability of surviving and remaining in the same stage, \( P_i \); the probability of transitioning into the next stage, \( G_i \); and the average reproductive output per female, \( F_i \), of the adult stages and are collectively referred to herein as the population vital rates, \( a_i \) (Fig. 1b).

For each treatment level, \( x \), a projection matrix \( A(x) \) was constructed using the vital rates, \( P_{λm}, G_{λm}, \) and \( F_{λm} \) such that

\[ P_{λm} = σ_{λm}[1 - γ_{λm}] \] (2)

\[ G_{λm} = σ_{λm}γ_{λm} \] (3)

\[ F_{λm} = σ_{λm}^{1/2}[(1 + P)m + Gm_{λm}]/2 \] (4)

where \( σ_{λm} \) was the stage-specific survival probability, \( γ_{λm} \) was the transition probability, and \( m \) was the average number of offspring per female in stage \( i \). The lower order vital rates, \( σ_{λm} \) and \( γ_{λm} \), were calculated according to Caswell [19].

**Fig. 1.** (a) Stage-structured life cycle of *Americanamysis bahia* showing probability of survival, \( P_i \), probability of transitioning from one age class to the next, \( G_i \), and fertility, \( F_i \), for each stage \( i \). Numbers in circles represents the maximum age, in days, of the stage. (b) The population projection matrix, \( A \), of the corresponding life cycle, comprised of stage-specific \( P_i, G_i, \) and \( F_i \).
In Equation 6, \( T \) was the duration of the stage interval and \( \gamma_{\ell_0} \) was the population growth rate normally calculated as the dominant eigenvalue of the matrix \( \Lambda_{\ell_0} \). Because the estimate of \( \gamma_{\ell_0} \) required the eigenvector of the matrix that it was projecting, an iterative approach was used to calculate values of \( \gamma_{\ell_0} \) by setting an initial value of \( \lambda_{\ell_0} \) to 1.1. The initial value of \( \lambda \) can be set as 1.0 for the first iteration \([19,33]\); however, 1.1 was selected as the initial value here to avoid mathematical complications where the denominator of Equation 6 was reduced to zero. The resulting values of \( \gamma_{\ell_0} \) were used to estimate the entries of a second matrix, which was then used to produce a second value of \( \lambda_{\ell_0} \). Parameters were recalculated until the resulting values of \( \lambda_{\ell_0} \) were stabilized \([19]\). Parameter estimates for each treatment matrix are shown in Table 1. The final values of \( \lambda_{\ell_0} \) were used to determine the change in \( \lambda \) with increasing thiobencarb concentration, \( \Delta \lambda / \Delta c \).

**Decomposing the effects of \( \lambda \)**

To determine the contribution of each vital rate, \( a_q \) to \( \lambda \) for each concentration, \( x \), the sensitivity \( (s_q) \) of \( \lambda \) to changes in \( a_q \) was first determined for each concentration as
where \( w \), the stable age distribution, was the right eigenvector of the matrix and \( v \) was the corresponding left eigenvector representing the reproductive value of each stage, and \( \langle vw \rangle \) was the scalar product. A sensitivity matrix derived from Equation 7 determined how much would change if a change occurred to \( \lambda_p \). An elasticity matrix for each treatment concentration determined the relative contribution of each vital rate to \( \lambda \) and was related to sensitivity as

\[
e_{ij} = \frac{a_{ij} \lambda}{\lambda} \frac{\partial \lambda}{\partial a_{ij}}
\]

Elasticities, which sum to one within a matrix, are presented for each treatment concentration in Table 1.

We used a regression design of the decomposition method outlined by Caswell [11] to determine how each vital rate changed with thiobencarb concentration and the relative contribution of these impacts on changes in \( \lambda \). The decomposition analysis was based on the sensitivity of \( \lambda \) to the vital rates, \( \partial \lambda/\partial a_p \), and the change of each vital rate with thiobencarb concentration, \( \partial a_{ij}/\partial x \). The latter expression was obtained as the slope of a nonparametric regression using a loess smoother [11,34]. The smoothing parameter was selected using cross-validation, in which the curve is fit closest to the data points, to minimize the prediction error [11]. The decomposition of the change in \( \lambda \) with thiobencarb concentration was modeled as

\[
\frac{\Delta \lambda}{\Delta x} = \sum \frac{\partial \lambda}{\partial a_{ij}} \frac{\partial a_{ij}}{\partial x}
\]

and is a function of the sensitivity value of each vital rate and magnitude of its change.

Stochastic concentration-decay model

A simulation model was developed to demonstrate how management practices (exposure regime), chemical half-life, and environmental stochasticity affect the extinction risk of mysid populations where a pesticide is applied. Simulated mysid populations were first exposed to a lethal thiobencarb concentration of 300 \( \mu g/L \), which decayed one order of magnitude. The variable \( t_{1/2} \) (ranging in value from 1 to 7) represented thiobencarb half-life and was the number of mysid generations exposed to each concentration before it decayed one order of magnitude. For example, if thiobencarb had a half-life of \( t_{1/2} = 2 \) and the number of concentrations is six (300, 150, 75, 39, 18, 0 \( \mu g/L \)), thiobencarb decayed one order of magnitude in two mysid generations and the time between exposures is 12 mysid generations. The half-life of thiobencarb in aquatic systems has been estimated between 3 and 190 d and is dependent on various physical parameters of the system [23,31,35,36]. The values of \( t_{1/2} \) were selected to include the range of observed half-lives, assuming a 29-d mysid life cycle.

While exposed to each concentration level, \( x \), mysid dynamics were determined by the corresponding matrix \( A_{iij} \). Density dependence occurs in both survival and reproduction of mysids [37] and was added to each matrix \( A_{iij} \) by modifying the vital rates by a function of population density (Ricker-type density dependence) such that

\[
S_{ij} = \frac{\partial \lambda}{\partial a_{ij}} = v_{Wj} \langle vw \rangle
\]

where \( N \) is the total population size and \( b \) is the strength of the density dependence [38]. When \( b = 0 \), the model reduces to the density-independent model and, when \( b = 1 \), the population experiences strong density dependence. There is very limited information characterizing the density dependence of mysids and \( b \) functions only for theoretical purposes as a product of \( N \). It was therefore set at 1 for all vital rates in all simulations.

Three time-varying scenarios were developed to simulate three different exposure regimes. In exposure regime 1, a new exposure of thiobencarb occurred following total recovery (zero concentration) of the environment. Exposure regimes 2 and 3 simulated the addition of a new dose before total recovery of the environment at 18 and 39 \( \mu g/L \), respectively. These two regimes do not account for any accumulation of concentration that may occur by adding a new dose to a system that contained an existing concentration, and the effects may be underestimated. The highest exposure concentration in the simulation was 300 \( \mu g/L \) because mysids experienced 100% mortality in less than 5 d when exposed to 600 \( \mu g/L \).

Environmental stochasticity was added to the concentration-decay model to determine the probability of mysid extinction with exposure to thiobencarb of varying half lives and different exposure regimes. Mysids have a simplified life history and all life stages are found in the same habitat. Therefore, it was assumed that environmental stochasticity affected the stage-specific survivorship (\( P_i \)) and transition (\( G_i \)) probabilities of all stages equally. Stochasticity was linked to the vital rates by varying \( P_i \) and \( G_i \) under the assumption of independent and identically distributed environments such that, for each time step,

\[
P_{sij} = P_{mij} \pm \sigma \xi_i
\]

\[
G_{sij} = G_{mij} \pm \sigma \xi_i
\]

where \( P_{sij} \) and \( G_{sij} \) are the stochastic survival and transition rates, respectively, with a mean of \( P_{mij} \) and \( G_{mij} \), and a standard deviation of \( \sigma \xi \). The variable \( \sigma \) represents the intensity of the environmental fluctuation and \( \xi \) is normally distributed white noise with a mean 0 and a standard deviation of 1. The shared \( \sigma \xi \) term among survival and growth rates assumes direct correlation between the environment and both vital rates, which are equally affected by the environment. The vital rates will either increase or decrease as a result of environmental variance; however, to ensure that \( 0 \leq P_{sij}, G_{sij} \leq 1 \) for all time steps, \( t \), values were truncated at either zero or one when variance resulted in \( P_{sij} \) or \( G_{sij} \) that were less than zero or greater than one, respectively [39].

Three levels of environmental intensity, \( \sigma \), were used to represent a range of environmental stochasticity. In natural environments, stochasticity is a difficult parameter to quantify but can be estimated by temporal variance of natural populations [30]. Very limited information on natural A. bahia populations is available to measure the temporal variance; however, previously published data on the population dynamics of other mysid species produced coefficients of variation (CV) between 50 and 250 [40,41]. The three values of \( \sigma \) (0.155, 0.165, 0.175) were selected with preliminary CVs calculated...
within the range of CVs for the previously reported mysid populations. These values were large enough to have a random effect on the population and small enough to prevent the population dynamics from fluctuating chaotically.

Simulations for each exposure regime (1–3) were conducted over the seven values of $t_{1/2}$ and three values of $s$. Each simulation was run over 500 generations and 10,000 iterations to determine the probability of quasi-extinction for mysid populations under the various conditions. For the first 10 generations of each simulation, populations were maintained at the stable age distribution determined from the deterministic model at the control concentration (0 µg/L). An exposure of 300 µg/L was added at generation 11 and the number of iterations that fell below a quasi-extinction threshold (1% of the control equilibrium density) determined the cumulative extinction probability (CEP). For each level of environmental intensity, a CEP of control populations (0 µg/L, no exposure added) in stochastic environments was also determined from 10,000 iterations run over 500 generations.

**RESULTS**

**Density-independent, deterministic model**

Based on the deterministic stage-structured model, $\lambda$ decreased with increasing thiobencarb concentration (Fig. 2). For all treatment concentrations less than 300 µg thiobencarb/L, $\lambda$ was greater than one, whereas $\lambda$ was less than one for mysids exposed to concentrations of 300 and 600 µg thiobencarb/L. The decrease in $\lambda$ with exposure from 0 to 600 µg thiobencarb/L was a significant, 96%, decline ($r^2 = 0.88, p < 0.002$). From 0 to 300 µg thiobencarb/L, $\lambda$ decreased 15% ($r^2 = 0.94, p < 0.002$) and, from 0 to 150 µg thiobencarb/L, $\lambda$ decreased approximately 5% ($r^2 = 0.85, p < 0.05$).

Based on the elasticity analysis, $F_i$ and $G_i$ had small relative contributions to $\lambda$ compared with $P_i$ of intermediate stages for all treatment concentrations (Table 1). The nonparametric regression of the effects of treatment on the vital rates showed $P_i$ and $G_i$ were affected across all concentrations (Fig. 3a and b). From 0 to 18 µg thiobencarb/L, $P_i$ decreased across all stages. Although there was variation in the response of $P_i$ at lower concentrations, the greatest decrease occurred to $P_i$ at higher concentrations and was most dramatic for the later stages (Fig. 3a). In general, $G_i$ was not impacted as severely as $P_i$ and was most dramatic for juveniles between 1 and 4 d old at lower concentrations and younger stages at higher concentrations (Fig. 3b). Overall, the most dramatic effect occurred in $F_i$ of late breeders at lower concentrations (Fig. 3c). Primarily, a large reduction in $F_i$ occurred between 0 and 18 µg thiobencarb/L for later reproductive stages. Although early and intermediate breeders did not exhibit an effect between 0 and 39 µg thiobencarb/L, reproduction was delayed at concentrations greater than 39 µg thiobencarb/L. This delay is depicted in Figure 3c as the negative changes in $F$ that occurred in young adult (age 16 d) and early breeder stages (age 20 d) from 39 to 75 µg thiobencarb/L and then in early and inter-
mediate breeders (age 24 d) from 75 to 150 μg thiobencarb/L. Between concentrations of 39 and 75 μg thiobencarb/L, young production was reduced to zero in young adults and decreased 90% in early breeders; as concentration increased from 75 to 150 μg thiobencarb/L, young production was reduced to zero in early breeders and reduced by 80% in intermediate breeders (Table 1). At concentrations greater than 150 μg thiobencarb/L, young production was reduced to zero.

Decomposing the effects of λ in terms of changes to the vital rates showed no contribution of early-stage $P_i$ and only a small contribution of late-stage $P_i$ at lower concentrations. However, changes in λ at high concentrations was attributed entirely to the changes in $P_i$ that occurred in the later stages (Fig. 4a). The contribution of $G_i$ to changes in λ was restricted to effects observed in early stage at lower concentrations (Fig. 4b). Decreased $F_i$ contributed to the changes in λ at lower concentrations only (Fig. 4c).

Stochastic concentration-decay model

In the absence of environmental stochasticity, mysid populations in the time-varying model deterministically fluctuated between two points, dependent on $t_{1/2}$ and the exposure regime. Throughout the range of $t_{1/2}$ and exposure regimes included in the model, thiobencarb decayed to sublethal concentrations ($\leq$150 μg/L) and populations increased before they were driven to extinction from the lethal thiobencarb concentration.

When environmental stochasticity was added to the model, extinction occurred with some probability for all exposure regime and half-life combinations. For all values of $\sigma$, control populations had very low CEPs (0.008–0.047). In general, CEP increased as half-life, environmental stochasticity, and thiobencarb concentration present at the time of a new exposure increased (Fig. 5). Cumulative extinction probability was low (<0.3) for all values of $\sigma$ when half-life was less than or equal to two mysid generations (~56 d) and the environment was allowed to completely recover from the thiobencarb application (exposure regime 1). Conversely, CEP approached 0.6 in highly variable environments ($\sigma = 0.175$) where thiobencarb had a longer half-life and a new thiobencarb dose was added under exposure regime 3.

To relate the values of environmental intensity used in the simulation model to variation that may be observed in natural populations, the average CV was calculated for the control population of each value of $\sigma$. Values were 60.85, 64.82, and 68.65 for values of $\sigma$ equal to 0.155, 0.165, and 0.175, respectively.
DISCUSSION

The population growth rate of mysids decreased with increasing thiobencarb concentrations throughout the range of concentrations applied. When exposed to concentrations of 300 μg thiobencarb/L and greater, λ was less than one and would result in population decline at these values. The values of λ measured for concentrations less than 300 μg thiobencarb/L demonstrated a significant decrease with increasing thiobencarb concentration (r² = 0.85, p < 0.05), indicating a significant population-level effect even at sublethal concentrations. Although results obtained from these data are reflective of laboratory conditions and cannot directly infer what may happen in field populations, demographically structured population models have been shown to be good predictors of A. bahia populations in controlled environments [15]. Indeed, the significant decline in growth rate that occurred with increasing thiobencarb concentration would result in reduced mysid biomass production in impacted estuarine ecosystems, the effects of which may have greater implications on the community level.

The elasticity analysis determined that Pᵢ of the intermediate stages (specifically of ages 5–16 d old) contributed the most to the value of λ for each treatment concentration. This is in contrast to a previous study [14] that found, through a different statistical procedure, that survival of earlier juvenile stages (ages 24–96 h old) and reproduction had more of an impact on population-level effects than the subadult stages (7–14 d old) and female survivorship, respectively. The elasticity analysis used in our study is applied extensively in perturbation and conservation studies [42,43, and references cited therein] and is a mathematically derived method for determining the contribution of each vital rate to population growth rate [19]. The elasticity values were consistent across all treatment concentrations but only reflect the relative role of each vital rate in determining the value of λ. They do not reflect how the changes in each vital rate altered λ, which was addressed by decomposing the effects of λ with changes in the vital rates.

The decomposition analysis determined that the vital rate responsible for the majority of the population-level effect was different for sublethal (<300 μg/L) and lethal (≥300 μg/L) concentrations. At sublethal concentrations, altered Fᵢ had a large effect on the reduction in λ, whereas decreased Pᵢ was the overriding influence on populations at concentrations ≥300 μg/L. At concentrations less than 300 μg/L, all vital rates were affected by thiobencarb; however, the changes to Fᵢ were orders of magnitude greater than those observed for Pᵢ and Gᵢ, and provided a large contribution to the reduction of λ, despite its inferior elasticity. The reduction of Fᵢ to zero in early and intermediate breeders (delayed reproduction) was also determined in the decomposition analysis to contribute to changes in λ, further emphasizing the importance of altered reproductive capacity. At higher concentrations, juvenile stages did not survive long enough to reproduce; hence, survival was the overriding factor affecting the value of λ.

The observed alterations of Fᵢ at sublethal concentrations support recent suggestions that thiobencarb may be acting as an endocrine-disrupting chemical [23], which disrupts normal hormone functions and may result in reproductive impairment [44]. The mechanisms of action of endocrine-disrupting chemicals occur on the cellular level and their consequences are expressed at the individual level. Population models reflect the effects on individuals and define population and ecological impacts from chemicals acting as endocrine disruptors. Considering the complexity of environmental toxicology and chemical-organism relationships, the potential endocrine-disrupting effect of any chemical warrants research on all hierarchical levels.

The lethal effect observed at concentrations above 300 μg/L is consistent with other studies measuring the lethal dose of thiobencarb on mysids. The median lethal concentration (LC50) of thiobencarb has been reported as 0.33 ppm (330 μg/L) and 0.29 ppm (290 μg/L) for mysids less than 1 d old and between 6 and 8 d old, respectively [23,45]. Although the LC50 (or median lethal dose) values of thiobencarb have been determined for mysids in several studies [24,45,46], this is the first study to identify the response of mysid populations exposed to these lethal concentrations of thiobencarb. Our results demonstrate that, at concentrations near or above the LC50, decreased survival was the vital rate that caused decreased population growth rate, λ. This result is supported by Kuhn et al. [14], who determined a strong correlation between LC50 and toxicant concentration that predicted a population growth rate equal to one for several chemicals.

Models that center around changes in λ do not account for density dependence and may fail to document population behavior where density dependence is known to occur [38]. Although density dependence has been documented for A. bahia [37], information characterizing the density dependence is lacking. Earlier research using the same data applied in this study determined that selecting an arbitrary strength of density dependence (b in Eqn. 10) yields results that are highly correlated with the density-independent model (S. Raimondo, U.S. Environmental Protection Agency, Gulf Breeze, FL, unpublished data). Additionally, Caswell [19] noted that elasticities of density-dependent vital rates were highly correlated with those of corresponding density-independent vital rates. Without more detailed information characterizing the density dependence of A. bahia, incorporating it into age-structured models such as the one used here are not likely to improve the predictability of the model.

Population growth rate, λ, remains a useful population-level endpoint and has been used to determine critical concentration values of chemicals that would result in population decline [12–14]. In general, the probability of extinction is one where λ is reduced to less than one by a pollutant and, the lower the value of λ, the less time it would take for a population to go extinct in stochastic environments [47]. Under the first of these two lemmas, mysid populations in the deterministic model will decline to zero when exposed to thiobencarb concentrations of ≥300 μg/L. Under the second lemma, populations exposed to sublethal thiobencarb concentrations are at a risk of extinction that increases as population growth rate is reduced by thiobencarb concentration.

Seldom are deterministic, time-invariant models accurate depictions of the processes occurring in real environments. As such, they have limited power for determining extinction risk of a population exposed to sublethal pollutant concentrations. Extinction risk is better determined from stochastic models and is generally a function of environmental variation, carrying capacity, and population growth rate [30,47,48]. In addition to these factors, the concentration-decay model presented here demonstrates the importance of including chemical half-life and management practices when determining population viability of species exposed to pulse exposures of a pesticide. Thiobencarb is generally applied to rice paddies within a few
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Effects Research Laboratory. It has been reviewed by the U.S. EPA's National Health and Environmental Effects Research Laboratory (Gulf Breeze, FL, USA). The information in this document does not necessarily reflect the views and policies of the U.S. EPA.

REFERENCES


days of planting and will provide residual control of weeds for up to one month following application [49]. Depending on the pest management strategy, thiobencarb may not be applied again until the following season, creating pulse-exposures of the pesticide into the environment. Considering the importance of half-life and exposure frequency on population responses to a chemical, ignoring these parameters would lead to erroneous population projections.

The concentration-decay model showed that half-life and pest management practices (exposure regime) influence population viability and are important factors in projecting population-level consequences. Over all values of ς and t½, CEP was low provided the environment completely recovered from thiobencarb before the next treatment exposure (exposure regime 1). Cumulative extinction probability was also low when thiobencarb half-life was only as long as one or two mysid generations. Half-life is an easily measured variable and, together with the management practices, may provide a useful estimate of the ecological impacts of pesticides on mysid populations.

Cumulative extinction probability also increased with environmental intensity. Although little information exists on the fluctuations of natural mysid populations, estuaries are characterized by marked environmental fluctuations that may lead to more stochastic abiotic conditions. We provided CVs for our simulated populations to relate our values of environmental intensity with population fluctuations. The range of CVs calculated from the model was small (60.85–68.65); however, our simulation demonstrated increased extinction risk with increased environmental randomness. Our data, along with other studies concerning population extinction [30,47,48], conclude that species subjected to more erratic environmental fluctuations will be at a greater risk of extinction.

Deterministic matrix models and life-table response experiments are commonly used to estimate the population-level response of an organism exposed to a pollutant [14,16–18]. At best, these analyses can determine the change of λ with varying pollutant concentration or environmental state [19]. Reduced values of λ with increasing chemical concentration, as noted here, can yield larger ecological consequences, such as local extinction of the population or reduced biomass production. For organisms such as mysids, which play a critical role in the trophodynamics of the community [50], these population-level effects may find larger consequences on the community level. For toxicologists to understand the ecological consequences of a cellular mechanism, a hierarchical approach, such as the one applied here, must be taken. Bridging the gap between a population and the ecosystem requires the application of population-level effects in realistic environmental scenarios. The concentration-decay model in this study conceptualized how the population-level endpoint, λ, can determine population viability in a time-varying scenario, which in turn helps attain a clearer view of ecological consequences. Although the trends observed in our concentration-decay model may be intuitive, the model allowed us to demonstrate the expansion of population models to integrate other factors that are important to population viability.

Acknowledgement—The data used in this analysis were generated by the staff of the U.S. EPA, Gulf Ecology Division. We wish to thank Doug Middaugh, Kenneth Rose, and the anonymous reviewers for their suggestions that improved this manuscript. This manuscript is contribution 1202 of the Gulf Ecology Division of the U.S. EPA Office of Research and Development’s National Health and Environmental

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