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PREDICTION OF POPULATION-LEVEL RESPONSE FROM MYSID TOXICITY TEST DATA USING POPULATION MODELING TECHNIQUES

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Abstract—Acute and chronic bioassay statistics are used to evaluate the toxicity and risks of chemical stressors to the mysid shrimp *Americamysis bahia* (formerly *Mysidopsis bahia*). These include LC50 values from acute tests, chronic values (the geometric mean of the no-observed-effect concentration and the lowest-observed-effect concentration from 7-d and life-cycle tests), and U.S. Environmental Protection Agency water quality criterion continuous concentration (CCC). Because these statistics are generated from responses of individual organisms, the relationships of these statistics to significant effects at higher levels of ecological organization are unknown. This study was conducted to evaluate the quantitative relationships between toxicity test statistics and a concentration-based statistic derived from exposure–response models relating projected population growth rate to stressor concentration. This statistic, C*, describes the concentration above which mysid populations are projected to decline in abundance as determined using population modeling techniques. An analysis of responses of *A. bahia* to 10 metals, nine organic compounds, and ammonia surprisingly indicated the acute LC50 to be the best predictor of C*, followed by the chronic value from life-cycle tests, which predicted population-level response almost equally as well. The chronic value for the 7-d test was less predictive of population-level effects. The CCC was lower than C* for 94% of the compounds evaluated, indicating the criterion value to be protective of population-level effects for *A. bahia,* as intended.

Keywords—*Americamysis* *Mysidopsis* Population model Exposure–response models Toxicity tests

INTRODUCTION

The acute and chronic responses of the mysid shrimp *Americamysis bahia* (formerly *Mysidopsis bahia*) are used by the U.S. Environmental Protection Agency (U.S. EPA) in a number of regulatory and research programs addressing marine water quality [1–8]. Bioassays established with this species include a 96-h acute mortality test [9]; a 7-d survival, growth, and fecundity test; and a 28-d life-cycle test with survival, growth, and reproduction endpoints. The 7-d test was developed as a rapid chronic test by the U.S. EPA to evaluate the toxicity of complex effluents and associated marine receiving waters for National Pollutant Discharge Elimination System permits [10]. The 28-d life-cycle test was designed to evaluate chronic toxicity through the first brood release of the life cycle of *A. bahia* [11].

The standard concentration-based endpoint statistics calculated from data generated in these assays include concentrations lethal for 50% of the test organisms (LC50s) from the acute test and chronic values from both the 7-d and life-cycle tests [9–11] (Table 1). A chronic value is defined as the geometric mean of the no-observed-effect concentration (NOEC) and the lowest-observed-effect concentration (LOEC). These statistics are designed to indicate the potential of chemical stressors to elicit biological effects in natural systems. The acute and chronic data are also used to calculate criterion continuous concentrations (CCCs) in development of U.S. EPA water quality criteria [12]. The water quality CCC is an estimate of the threshold concentration producing an unacceptable biological effect and is intended to provide protection to aquatic species from long-term chronic exposure to chemical stress.

A long-standing interest has existed in the relation between laboratory toxicity tests results and risks to natural ecological systems [13–15] and in the statistical relations among endpoints [16,17]. The central question is whether test endpoints that focus upon the responses of individuals can be extrapolated to evaluate the long-term risks of contamination at higher levels of ecological organization (populations, communities, and ecosystems). Several studies have been conducted to assess population-level effects of environmental contaminants on aquatic animals [18–38]. Unlike statistical analyses of individual response endpoints from a typical bioassay, these studies provide evidence of contaminant effects on the demographic responses of populations.

The primary objective of this study was to evaluate the ecological relevance of mysid bioassays by comparing statistics from standard toxicity tests with a statistic calculated from population modeling. This latter statistic, C*, is derived from exposure–response models relating population growth rate to contaminant concentration, and describes the concentration predicted to result in zero population growth. This calculation of a projected ''threshold'' concentration for mysid populations is based on an assumption that a model-derived population-level statistic is more ecologically relevant than a statistic based upon responses of individual mysids. The comparison of C* with the CCC for a given contaminant also permits evaluation of the degree of protection afforded by the CCC. Another objective was to identify the 28-d chronic test

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Table 1. Summary of mysid toxicity test statistics and relative exposure periods

Test type	Exposure period (age in days)	Measured endpoints	Test statistic	Methods reference
96-h Acute	0–4	Survival	LC50	[9]
7-d Subchronic	7–14	Survival, growth, and fecundity[a]	Subchronic value[b]	[10]
Life-cycle chronic	0–28 (35)[c]	Survival, growth, and reproduction[d]	Chronic value[b]	[11]

[a] Fecundity represents the presence of eggs in the oviducts or brood sac.
[b] Subchronic and chronic values are the geometric mean of the no-observed-effect concentration and lowest-observed-effect concentration.
[c] Chronic life cycles performed between 1979 and 1981 were performed at 22 ± 2°C, and usually ran for 35 d to allow for development and reproduction.
[d] Reproduction represents the number of live young produced.

endpoint (reproduction or survival) that best predicts population-level effects. The analysis of which endpoint best predicts population-level effects may provide important information that will enable the development of efficient methods that maximize predictability of long-term and higher level effects.

MATERIALS AND METHODS

Toxicity tests

All of the response data used in this study were generated at the U.S. EPA Atlantic Ecology Division in Narragansett, Rhode Island, over a time span of about 14 years (1979–1993). During this period, the toxicities of 10 metals, three organic contaminants, and ammonia were evaluated in single-contaminant exposures using all three toxicity tests (96-h acute, 7-d subchronic, and life-cycle). An additional six organic contaminants were evaluated using only the 96-h and life-cycle tests. The results of many of these tests have been reported elsewhere [25,26,28]. Chemical concentrations were measured for all tests and their methods were described [25,26,28]. Toxicity tests were conducted according to the conditions briefly described below.

The 96-h acute tests were performed under flow-through conditions following American Society for Testing and Materials standard methods [9]. These were initially conducted as range-finder tests and then repeated as definitive tests with a narrower range of concentrations. Resulting data were subjected to probit analysis [39] or Spearman–Karber methods [40] to calculate LC50s on the basis of the average measured contaminant concentration.

The 7-d survival, growth, and fecundity assays were performed from 1987 through 1988 under daily static-renewal (90% renewal) conditions according to U.S. EPA guidance [10]. These tests required a pre-exposure holding period of 7 d, during which test animals were acclimated to the salinity and temperature (26–27°C) conditions of the test. The exposure period for this test encompasses the age period of 7 to 14 d old, a subadult phase that involves rapid growth and gonadal development (hence the term fecundity instead of reproduction). The most sensitive measure of the three test endpoints was used to determine the NOEC and the LOEC for the 7-d test [10]. The geometric mean of those concentration limits was used to determine the subchronic value or 7-d test statistic. The NOECs and LOECs were obtained using Dunnett's procedure, which identifies toxicant concentration means that are statistically different from the control mean at the chosen 5% level of significance.

Life-cycle tests were performed from 1979 through 1993 under flow-through conditions following American Society for Testing and Materials guidance [11]. Earlier tests (1979–1981) were performed at 22 ± 2°C, and usually ran for 35 to 40 d to allow for development and release of the first brood. After 1981, tests were performed at 25 ± 1°C, shortening the duration of tests to 28 d. Tests were checked daily through day 14, when the sex of individuals was determined [11]. Individual female and male pairs were observed daily for survival and reproduction through the remainder of the test. As with the 7-d test, the test statistic for the life-cycle test is the chronic value, determined by the geometric mean of the NOEC and LOEC for the most sensitive of the three endpoints [11]. Arcsine-transformed data for all percentage values from life-cycle tests were statistically analyzed by analysis of variance and contingency table analyses [41], followed by a hypothesis test approach such as Dunnett's procedure [42,43] or Tukey's no statistical significance of trend test [44].

Contaminant-specific values for CCCs were obtained from the national water quality criteria documents for 15 of the 20 compounds [45–59]. The CCCs were calculated for deriving water quality criteria following U.S. EPA national guidelines [12]. In essence, this calculation takes into account both acute and chronic toxicity information from a representative assemblage of aquatic species to derive a threshold concentration below which unacceptable biological effects are not expected to occur. The CCCs for carbaryl, diazinon, and propoxur were obtained from draft water quality criteria documents (G.Thursby, personal communication). The CCCs for pyrene and carbofuran are not published; we used methods in [12] to calculate preliminary values using the minimum database for saltwater species.

Values of the test statistics described above are given in Table 2. Growth was never the most sensitive endpoint, so the chronic and subchronic values were determined by either survival, reproduction, or in some cases both. The statistics in Table 2 are all based on direct chemical measurement of contaminant concentrations during tests.

Population model

The life history of *A. bahia* is very amenable to demographic modeling because of its rapid growth (almost daily molts), sexual differentiation at 14 d of age, reproduction commencing around 17 d of age, and numerous broods produced (average of five to seven per female) over the average full life span of 90 d. Data collected daily during life-cycle tests were used to model the dynamics of mysid populations exposed to chemical contaminants using an age-classified population projection matrix model [60–62]. The model permits projection of population abundance through time as a function of age-specific schedules of survivorship and fecundity. A daily time step was used so that each day of the life-cycle assay was treated as an age class in the model. Daily reproduction (start-

Table 2. Toxicity statistics (μg/L) derived from *Americamysis bahia* bioassays for 20 contaminants. Criterion continuous concentrations (CCCs) were taken from U.S. Environmental Protection Agency water quality criteria documents. These statistics are all based on direct chemical measurement of contaminant concentrations during test performance. C* estimates (λ = 1) represent concentration thresholds (μg/L) above which populations of *A. bahia* would be expected to decrease in abundance

Contaminant	96-h Acute LC50	7-d Subchronic value[a]	Life-cycle chronic value[a]	Life-cycle endpoint affected[b]	Water quality CCC	C*
Metals						
Arsenic	1,740	849	895	S, R	36	—[c]
Cadmium	110	12	7	S	9.3	15.3
Chromium	2,030	34	132	R	49.9	—[c]
Copper	164	71	54	R	3.1	49
Lead	3,130	41	25	R	5.6	—[c]
Mercury	3.5	1.3	1.1	S, R	1.1	2.0
Nickel	508	217	93	S, R	8.3	136.8
Silver	249	190	19	R	1	26.9
Thallium	4,830	2,076	272	R	162	155.3
Zinc	499	47	166	S, R	58	223.4
Organics						
Acenapthene	460	NC[d]	63.9	R	31.1	75
Aniline	1,930	1,120	771	R	37	1,932
Carbaryl	8.998	3.5	9.92	S	0.968[e]	2.5
Carbofuran	3.19	NC	0.842	S	0.093[e]	3.6
DDT	0.24	NC	0.056	S	0.001	—[c]
Diazinon	4.82	NC	3.04	R	0.395[e]	3.2
Phenol	8,500	844	4,071	S, R	474.7	3,155.7
Propoxur	36.02	NC	6.805	S	1.5[e]	17.4
Pyrene	28.28	NC	4.53	R	1.66[e]	5.0
Other						
Ammonia[f]	1,700	224	232	R	35	561

[a] Subchronic and chronic values are the geometric mean of the no-observed-effects concentration and lowest-observed-effects concentration.
[b] S = survival; R = reproduction.
[c] Concentrations for these compounds not high enough to produce a C* estimate.
[d] NC = test not conducted.
[e] Unpublished values from draft water quality criteria document.
[f] Concentrations reported as unionized NH_3.

ing about 17 d into the life-cycle assay) as well as survival was incorporated in this model. Age-specific schedules of female survival and fecundity were calculated for controls and exposure concentrations of each chemical evaluated in life-cycle tests. Female survivorship was calculated by comparing the number of females alive each day with the number alive on the previous day. Age-specific fecundity was calculated as the number of female offspring produced per female per day, assuming a 1:1 sex ratio for the offspring. These age-specific survival and fecundity data were then used as parameters for the mysid population model constructed using RAMAS/stage software [63].

Under stable demographic conditions (an underlying assumption of analyses for matrix models) the model yields an estimate of the finite rate of population increase, λ (the dominant eigenvalue of the projection matrix). The value of λ provides information regarding the status of the population: $\lambda > 1$ indicates a growing population, $\lambda < 1$ indicates a declining population (ultimately leading to extinction), whereas $\lambda = 1$ indicates no change in population size through time (i.e., zero population growth).

The relationship between estimates of λ and a measured exposure concentration was modeled using a nonlinear regression estimation procedure described in Bruce and Versteeg [64]. The concentration predicted to produce $\lambda = 1$ (i.e., C*) was estimated for each chemical using the following equation:

$$R = R_{ctrl} \cdot \Phi[(\log(EC_x) - \log(C))/s + Z_x]$$

where R is the predicted population growth rate at concentration C, R_{ctrl} represents the estimated control response for population growth rate, Φ is the cumulative area under the standard Gaussian distribution, EC_x is the xth percentile effects concentration, Z_x is the normal deviate above which x percent of the Gaussian distribution lies, and s is the standard deviation of the Gaussian distribution. We used the NLIN procedure [65] to fit a nonlinear regression model using least squares method to estimate model parameters [64]. The C* was estimated from the resulting model.

The life history characteristics of *A. bahia* are sensitive to temperature [66]. The potential effects of differing temperature regimes used among life-cycle tests were evaluated before estimation of C* by comparing population growth rates calculated for control treatments.

Endpoint comparison

Rank order parametric correlation analyses were conducted comparing C*, 96-h acute LC50, 7-d subchronic values, life-cycle chronic values, and the CCC, treating chemical stressors as replicate observations. These analyses were performed on all chemicals combined, as well as on the metals and organics (including ammonia) subgroups. Significant correlations ($p \leq 0.05$) were used to identify statistical relationships among test statistics. The C* was then regressed against each positively correlated test statistic to quantify predictive relationships and the uncertainties associated with extrapolation to population-level effects. Because values of the individual toxicity test statistics were not fixed a priori, least squares linear regression was used in this analysis [67]. For these regression anlayses, log values for all contaminants and C* estimates were used

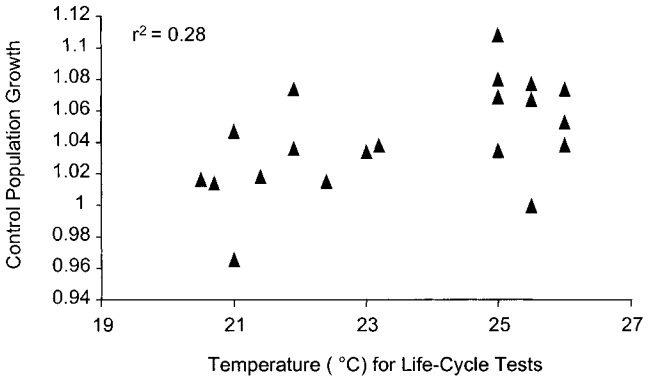

Fig. 1. Relationship between estimated *Americamysis bahia* population growth rate (λ) and life-cycle test temperature.

because of the wide range of concentrations among contaminants.

Additional analyses were performed to determine which individual measure of response from the life-cycle chronic test (survival or reproduction) correlated most closely with C*. We selected compounds for which survival was the most sensitive and only endpoint affected in the life-cycle test (four contaminants) combined with compounds for which both survival and reproduction were affected (four contaminants) and correlated the ranks of the respective chronic values with C* (total n = 8) . This analysis was repeated for contaminants where reproduction was the most sensitive and only endpoint effected (eight contaminants) combined with compounds for which both survival and reproduction were affected (same four contaminants as above) and correlated the ranks of the respective chronic values with C* (total n = 12). A simple numerical comparison between C* and the CCC for each chemical was performed to determine if the water quality criteria CCC was indeed protective of estimated *A. bahia* population-level effects (e.g., CCC < C*).

RESULTS

The effects of differing test temperature regimes used for the life-cycle tests were evaluated by regression analysis to identify influences of temperature on population growth rates. An estimate of population growth for each control treatment was calculated and plotted against its respective test temperature for the 20 chemicals tested. No relationship was observed between temperature and λ within the range of temperatures used in these tests (Fig. 1). Therefore, we considered temperature to be unimportant in the rest of our analyses.

Population-level exposure–response relationships

For all chemicals except arsenic, chromium, lead, and DDT population growth rates for *A. bahia* were concentration-dependent (Fig. 2). The typical response consisted of population growth rates slightly greater than 1.0 for controls and low-concentration treatments, followed by a fairly rapid decrease to extremely low population growth rates (λ < 1) at higher concentrations. The population-level responses to arsenic,

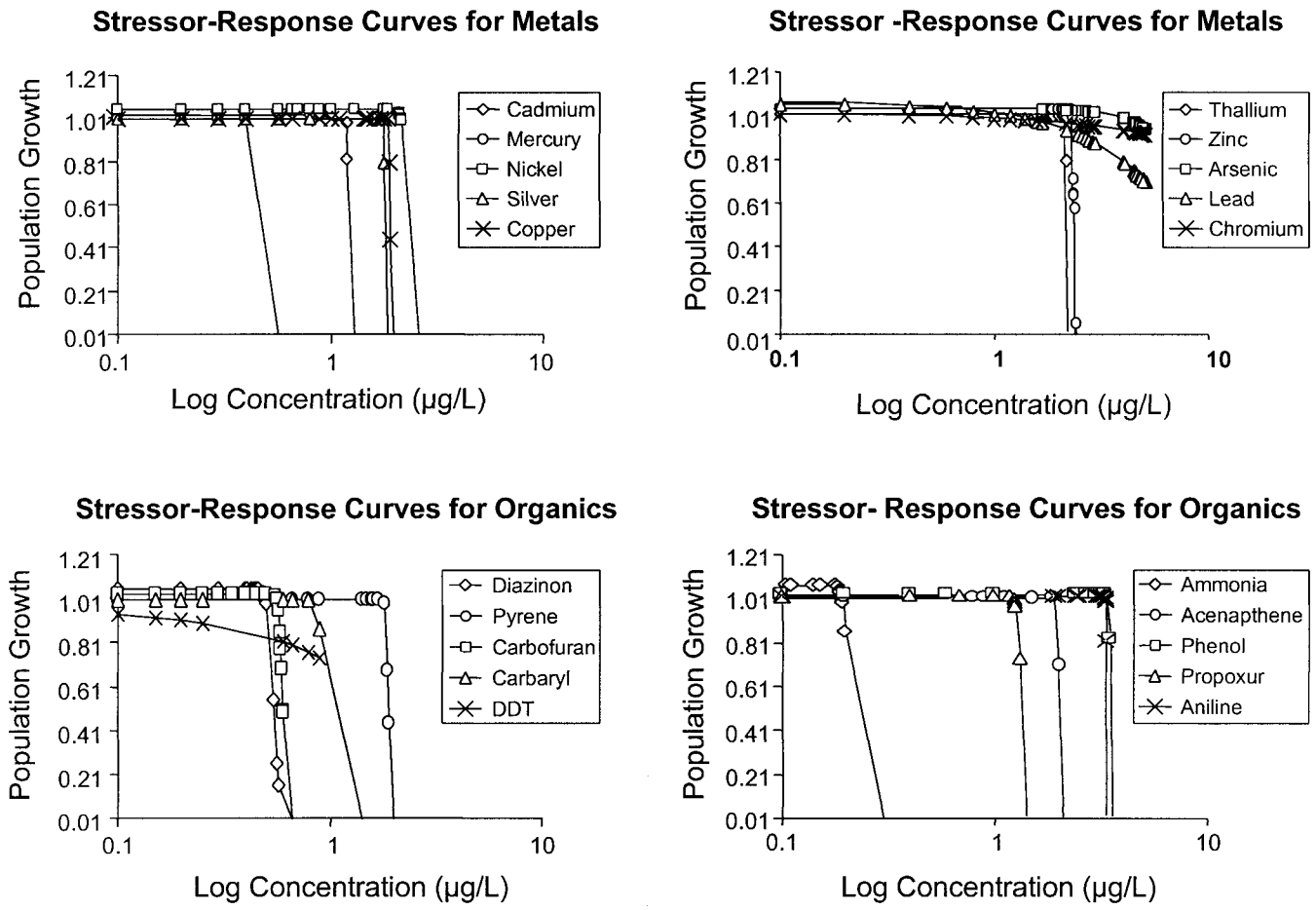

Fig. 2. Stressor–response curves (population growth rate vs log contaminant concentration) for metals and organics.

Table 3. Rank order parametric correlations among *Americamysis bahia* test statistics. Italicized entries denote statistical significance ($p \leq 0.05$)

Data set[a]	*n*	Test statistic	Test statistic: Acute LC50	Test statistic: 7-d Sub-chronic	Test statistic: Life-cycle chronic	Test statistic: C*
Full (metals and organics)	16	7-d Subchronic	0.67			
		Life-cycle chronic	*0.89*	*0.78*		
		C*	*0.96*	*0.81*	*0.93*	
		CCC	*0.86*	0.58	*0.90*	*0.87*
Metals subgroup	7	7-d Subchronic	*0.89*			
		Life-cycle chronic	*0.93*	*0.75*		
		C*	*0.86*	0.61	*0.96*	
		CCC	0.57	0.29	*0.71*	0.68
Organics subgroup[b]	9	7-d Subchronic	*0.80*			
		Life-cycle chronic	*0.83*	*0.80*		
		C*	*0.97*	*0.80*	*0.82*	
		CCC	*0.87*	*0.80*	*0.93*	*0.92*
Survival chronic endpoint[c]	8	Life-cycle chronic				*0.79*
Reproduction chronic endpoint[d]	12	Life-cycle chronic				*0.98*

[a] Including only chemicals for which the model-derived population statistic C* ($\lambda = 1$) could be calculated.
[b] Including ammonia.
[c] Includes compounds with survival alone as most sensitive endpoint ($n = 4$) plus compounds with survival and reproduction both being most sensitive endpoints ($n = 4$), for a total $n = 8$.
[d] Includes compounds with reproduction alone as most sensitive endpoint ($n = 8$) plus compounds with survival and reproduction both being most sensitive endpoints ($n = 4$), for a total $n = 12$.

chromium, DDT, and lead exposure were uniformly high across all concentrations ($\lambda > 1$), which suggests that the ranges of concentrations used in these life-cycle tests were insufficient to cause a decrease in population growth rate for *A. bahia*. Thus, C* for these four contaminants could not be estimated.

Endpoint comparison

The results from the rank order parametric correlation analyses identified the acute LC50 value as being strongly correlated with C* (correlation coefficient $r = 0.96$) (Table 3). The life-cycle chronic also correlated highly with C* ($r = 0.93$) (Table 3), followed by the 7-d mysid test, demonstrating a relatively weaker correlation with C* ($r = 0.81$) (Table 3). As a subgroup, organic contaminants (including ammonia) generally demonstrated a closer correlation with C* for all three test statistics (the acute, 7-d, and life-cycle) than did the metal subgroup (Table 3). Within the metal contaminant subgroup, only the life-cycle chronic value and the acute LC50 value were significantly correlated with C* (Table 3). These three test statistics (log concentration values) were then individually regressed against C* to quantify their predictive relationship in extrapolation to population-level effects (r^2 values, Fig. 3a to c). These results demonstrate that the life-cycle chronic value has a clear linear relationship with C* ($r^2 = 0.92$), followed by the acute LC50 with $r^2 = 0.88$ and the 7-d chronic value with $r^2 = 0.73$. Analyses of the two life-cycle chronic value response measures indicated that reproduction correlates more closely to C* ($r = 0.98$) than does the survival endpoint ($r = 0.79$) (Table 3 and Fig. 3a).

DISCUSSION

The results of the rank order parametric correlation analyses comparing established bioassay statistics with C* indicate the mysid 96-h acute test to be the best predictor of demographic response for mysid populations, followed closely by the life-cycle assay. The relatively weaker correlation of the 7-d mysid test with C* was surprising considering the longer exposure of the 7-d test versus the 96-h acute test because sublethal effects (growth and fecundity) were measured in the 7-d test, whereas only mortality was measured for the acute test. A plausible explanation for this is the relative sensitivities of the various life stages exposed in the two tests. The 96-h assay exposes 24- to 48-h-old individuals, which is generally considered a very sensitive life stage for this species. The 7-d test exposes mysids at a later stage of development (7–14 d old). Analysis of these data suggests that survival of the earlier juvenile life stage (24–96 h old) has more of an impact on population-level effects than the subadult life stage (7–14 d old) for chronic toxicant exposure.

The results from linear regression analysis of positively correlated test statistics with estimates of C* demonstrate that the life-cycle chronic value would be the most powerful tool for predicting population-level effects (Fig. 3a). The life-cycle chronic values and the estimates of C* for each contaminant evaluated averaged within a factor of 1.6 (ranging from 0.25 to 2.5) of each other.

The correlation analyses of the life-cycle effect measures suggest that changes in female reproduction rather than female survival cause a greater effect on population growth rate. However, the survival endpoint also demonstrates a statistically significant correlation with C* and also seems to be an important indicator of population growth rate.

The comparison of the water quality criteria CCC to C* for each contaminant illustrates the protective nature of the CCC for this species, because the CCC was set at a concentration lower than the C* estimate for 15 out of the 16 compounds (94%) in this study. For thallium, the one contaminant that had a C* estimate lower than the CCC, the two values were very close: 155 and 161 μg/L, respectively. The national water quality criterion for a contaminant consists of two concentrations: the criterion maximum concentration and the CCC. These two concentrations are used in combination to describe threshold concentrations and to set limits for a given contaminant that provide an appropriate degree of protection (95% of representative species from an appropriate variety of taxonomic and functional groups). In general, concentrations

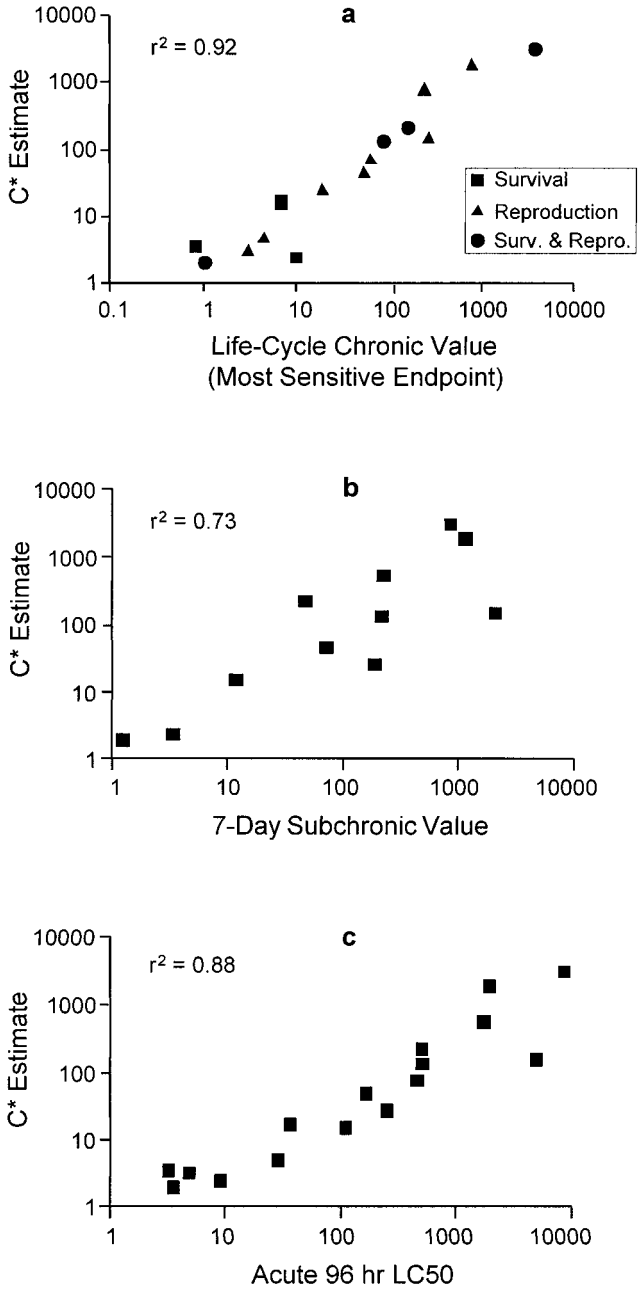

Fig. 3. Linear regression analysis for chronic (**a**), subchronic (**b**), and acute (**c**) statistics with estimated populatin growth rates.

above the CCC for a particular contaminant are expected to cause an unacceptable effect. The magnitude and duration of pollutant excursions above the CCC are factors that can govern the extent of the unacceptable effect. For chronic criteria the U.S. EPA uses the CCC along with a measurement of the pollutant over a consecutive 4-d averaging period for regulatory compliance. This average contaminant concentration should not exceed the CCC more than once every three years on average. In addition to complying with the CCC to satisfy the criterion, a 1-h average concentration of the pollutant of concern must not exceed the criterion maximum concentration more than once every three years on the average. The four most sensitive species tested for a particular contaminant are used to calculate the national criteria final acute value, which is then used in calculating the criterion maximum concentration, which is equal to one half of the final acute value. The CCC is equal to the lowest of the final chronic value, the final plant value, and the final residue value [12]. This derivation of the criterion maximum concentration from the four most sensitive species tested is important to understand because it exemplifies the importance of *A. bahia,* which was one of the four most sensitive species tested for 14 of the 20 (70%) contaminants evaluated in this study. For 18 out of 20 (90%) of the contaminants *A. bahia* was one of the eight most sensitive species tested.

In this study, population model estimates have been applied to laboratory-derived toxicity data for a single species, *A. bahia,* which is often one of the most sensitive tested species used for deriving saltwater national water quality criteria. The age-classified population matrix model links the effects of contaminants on individual mysids with their population-level effects. In an editorial essay Luoma and Carter [14] discussed the inherent uncertainties that exist when making predictions of toxicity by extrapolating laboratory results to ecosystem levels. They noted that these uncertainties can be lessened by using long-term studies of contaminated ecosystems and processes at many levels of biological organization. Our mysid population model is just one of the steps in identifying the biological processes that control toxicity at a population level. This model is an attempt to reduce one of the many uncertainties and simultaneously enhance the ecological realism of a risk-based approach to environmental regulation. The population model we used in this study is basic in that it does not consider density dependence, migration, predation, competition, and other factors that affect population growth. Our conclusions regarding population-level effects for this species are constrained to predictions of population dynamics under controlled laboratory conditions favoring exponential growth and therefore should not imply general patterns for natural environmental conditions.

The next phase of this research is to validate this population model with a multigenerational laboratory study. An evaluation of the efficacy of this population model for predicting effects on *A. bahia* populations will allow us to define the uncertainty associated with using laboratory-derived data to extrapolate effects to the population level.

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