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ENVIRONMENTAL SCIENCES DIVISION

USER'S MANUAL FOR ECOLOGICAL RISK ASSESSMENT

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ABSTRACT

BARNTHOUSE, L. W., and G. W. SUTER II. 1986. Users' manual for ecological risk assessment. ORNL-6251. Oak Ridge National Laboratory, Oak Ridge, Tennessee. 220 pp.

This report presents the results of a four-year project on environmental risk analysis of synfuels technologies, funded by the Office of Research and Development (ORD), U.S. Environmental Protection Agency. The overall objective of the project was to support the ORD's synfuels research program by developing a risk assessment methodology capable of (1) ranking the waste streams in a process by risk to the environment, (2) estimating the change in environmental risk that would be achieved using alternative control technology options, (3) estimating the sensitivity of risk estimates to site-dependent variables, and (4) identifying research problems contributing the greatest uncertainty to risk estimates.

At the time the project was initiated, the kinds of environmental risk analyses desired by ORD had never been performed, and proven quantitative methods analagous to the methods used to perform human health risk assessments or engineering safety assessments did not exist. Consequently, methods for quantifying ecological risks had to be developed de novo and/or borrowed from other fields. An initial suite of five potentially useful techniques was applied in a preliminary risk analysis of indirect coal liquefaction technologies. As a result of this application, it was determined that two of the original five techniques were unsuitable for synfuels risk assessments. The remaining three were developed further and applied in a unit-release

risk assessment, a revised indirect liquefaction risk assessment, a direct liquefaction risk assessment, and an oil shale risk assessment.

The methodology used in the synfuels environmental risk assessments has many potential applications, in addition to the specific purpose for which it was developed. This users' manual is intended to faciiitate wider use of ecological risk analysis techniques by (1) presenting the rationale for the approach developed in this project, (2) describing the derivation and mechanics of the three techniques used in the synfuels risk assessments, and (3) discussing the limitations and other potential applications of ecological risk assessment methods.

1. INTRODUCTION

L. W. Barnthouse and G. W. Suter II

This report presents the methodological results of a 4-year project on an environmental risk assessment of synfuels technologies, funded by the Office of Research and Development (ORD), U.S. Environmental Protection Agency. The overall objective of the project was to support the ORD's synfuels research program by developing a risk assessment methodology capable of (1) ranking waste stream components in a process by risk to the environment, (2) estimating the change in environmental risk that would be achieved by alternative control technology options. (3) estimating the sensitivity of risk estimates to site-dependent variables, and (4) identifying areas of research most likely to reduce uncertainty in the risk estimates. The methodology would be required to address both atmospheric and aqueous releases of chemical contaminants, but would not be required to address nonchemical effects such as thermal pollution or habitat disturbance. In addition, the methodology would be required to produce best estimates of environmental risk rather than worst-case estimates, and to explicitly quantify uncertainties concerning magnitudes of risk. The methodology would be demonstrated by using it to perform risk assessments for three classes of synthetic liquid fuels technologies: direct coal liquefaction, indirect coal liquefaction, and surface oil shale retorting.

At the time the project was initiated, environmental risk assessments of the type desired by ORD had never been performed, and proven quantitative methods analogous to the methods used to perform

human health risk assessments or engineering safety assessments did not exist. Consequently, methods for quantifying ecological risks had to be developed de novo or borrowed from other fields. An initial suite of five potentially useful techniques were described by Barnthouse et al. (1982). These five were applied in a preliminary risk assessment for indirect coal liquefaction technologies. As a result of this application, it was determined that two of the original five techniques, specifically fault tree analysis and the analytic hierarchy process, were unsuitable for synfuels risk assessments. The remaining three were further developed and applied in a unit-release risk assessment (Barnthouse et al. 1985a), a revised indirect coal liquefaction risk assessment (Barnthouse et al. 1985b), a direct coal liquefaction risk assessment (Suter et al. 1984), and an oil shale risk assessment (Suter et al. 1986).

The methodology used in synfuels environmental risk assessments has many potential applications in addition to the specific purpose for which it was developed. This users' manual is intended to facilitate wider use of ecological risk assessment techniques by (1) presenting the rationale for the approach developed in this project, (2) describing the derivation and mechanics of the three techniques used in synfuels risk assessments, and (3) discussing the limitations and other potential applications of ecological risk assessment methods.

1.1 CONCEPTS AND DEFINITIONS

The approach described here is based on the concepts of risk assessment and risk management, as defined by Ruckelshaus (1983) and

Moghissi (1984). The stimulus for adopting risk assessment as a fundamental component of environmental regulation is the recognition that (1) the cost of eliminating all environmental effects of technology is prohibitively high, and (2) regulatory decisions must usually be made on the basis of incomplete scientific information. The objective of risk-based environmental regulation is to balance the degree of risk permitted against the cost of risk reduction, against competing risks, or against risks that are generally accepted by the public. Scientific risk assessment has two roles in this process. First, it provides the quantitative bases for balancing and comparing risks. Second, it provides a systematic means of improving the understanding of risks by comparing the relative magnitudes of uncertainties concerning different steps in the causal chain between initial event (e.g., release of a toxic chemical) and ultimate consequence (cancer in humans or extinction of a bird population).

Risk assessment may be defined as the process of assigning magnitudes and probabilities to adverse effects of human activities (or natural catastrophes). This process involves identifying the adverse effects to be addressed in the assessment and using mathematical or statistical models to quantify the relationship between initiating events and ultimate effects. Ideally, although not always in practice, the results of a risk assessment reflect both the inherent uncertainty of events (e.g., probabilities of pipe ruptures or frequencies of rainstorms) and the scientific uncertainty resulting from an inadequate understanding of cause/effect relationships.

A risk-based approach to ecological effects assessment and management differs fundamentally from conventional impact or hazard assessment. In ecological risk assessment, uncertainties concerning potential effects must be explicitly recognized and, if possible, quantified. It is necessary to consider not only uncertainty regarding the biological effects of environmental stressors, but also the inherent variability of natural populations and ecosystems. Moreover, ecological risk assessments used in decision making should be based, to the greatest extent possible, on objective estimates of ecological damage (e.g., probabilities of population extinction or reductions in abundance of plants and animals). Such assessments require more information about the environments and organisms potentially affected than is used in current hazard assessment schemes for effluent discharges or toxic chemical releases.

1.2 ELEMENTS AND RATIONALE FOR RISK ASSESSMENT METHODOLOGY

The ecological risk assessment scheme adopted for this project consists of the components outlined in Fig. 1.1. First, the specific adverse effects to be evaluated, known as "end points," are selected. Second, the environment within which the technology being assessed is located (the "reference environment") is described. Third, a technical description of the facility that is the source of potential impacts is developed, and estimates of effluent magnitudes and compositions, or "source terms," are developed. Fourth, appropriate environmental transport models are used to perform an "exposure assessment," i.e., to estimate patterns of contaminant distribution in time and space.

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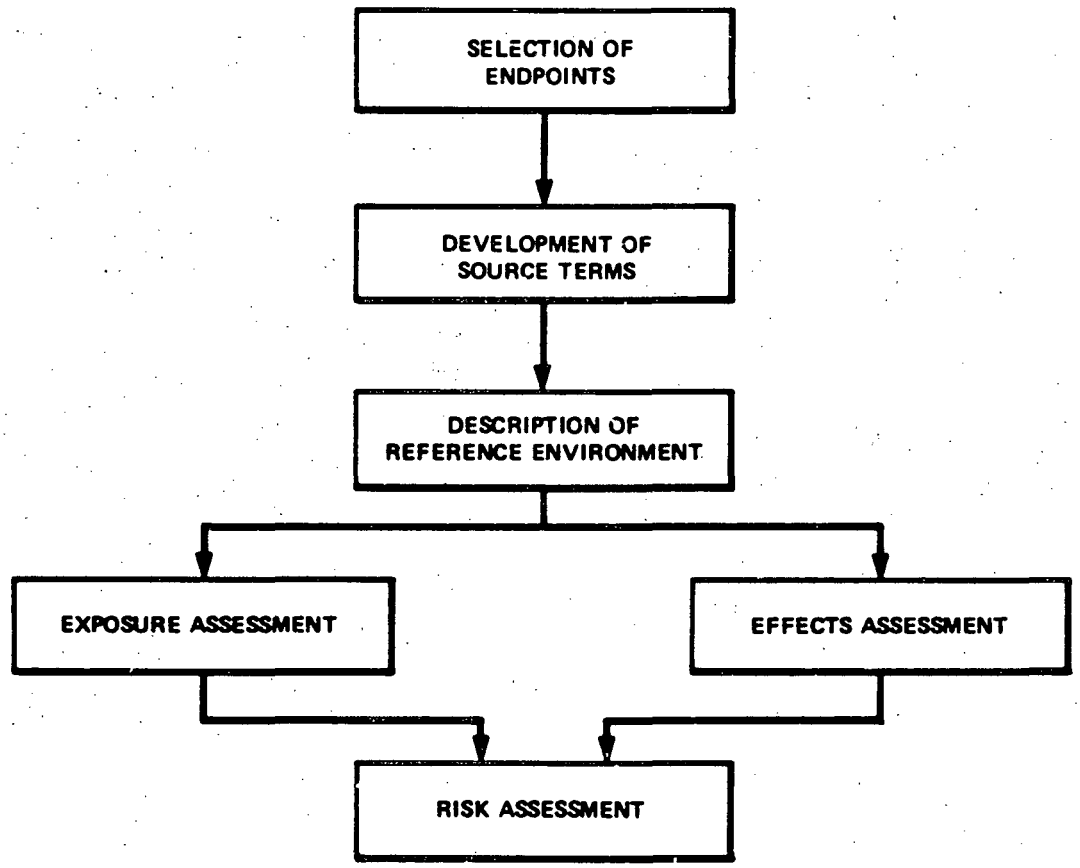

Fig. 1.1. Flow chart for ecological risk assessments of toxic chemicals.

Fifth, in the "effects assessment," available toxicological data are analyzed to determine the effects of the released contaminants on the organisms exposed. Finally, all of the previous steps are combined to produce the final risk assessment, which expresses the ultimate effects of the source terms on the end points in the reference environment.

The above scheme closely parallels risk assessment schemes used in human health risk assessments. The components that are unique to ecological risk assessment, and for which no previous guidance was available, include the selection of (1) end points and (2) methods for effects assessment. Rationales for the decisions made regarding these two components are presented here.

1.2.1 End Points for Environmental Risk Assessment

There are no obvious ecological equivalents of cancer or core meltdown, hence, there can be no standardized list of universally applicable ecological end points for risk assessment. To be useful in risk assessment, however, any end point should (1) have biological relevance, (2) be of importance to society, (3) have an unambiguous operational definition, and (4) be accessible to prediction and measurement. For synfuels risk assessments, it was concluded that the most appropriate end points were impacts on biological populations of importance to society. Societal importance was emphasized because assessments of risks to insects, zooplankton, or other organisms not perceived by society as being valuable are not likely to influence decision making unless they can be clearly shown to indicate risks to fish, wildlife, crops, or forest trees. Biological populations were

emphasized because (1) the death of an individual organism is usually biologically meaningless, and (2) current scientific understanding of higher levels of organization (communities and ecosystems) is insufficient to support the use of higher-level end points.

Specific descriptions and rationales for the five classes of end points used in synfuels risk assessments are presented here. They were chosen on the basis of their perceived importance and the availability of methods for quantifying population-level effects, without regard to any known or hypothesized vulnerability to synfuels-derived environmental contaminants. The existence and quantity of toxicity data relating to the end point biota were not considered.

1.2.1.1 Reductions in abundance and production of commercial or game fish populations. Impacts on fish species harvested by man are among the most socially important impacts on aquatic ecosystems. These species are also important indicators of the ecological health of aquatic ecosystems. Many harvested fish, especially game fish, are predators at the top of aquatic food chains; these top predators are frequently among the first species to disappear as a result of disturbances.

1.2.1.2 Development of algal populations that detract from water use. Undesirable blooms of algae commonly occur as consequences of nutrient additions to lakes or reservoirs. These blooms are a nuisance to shoreline residents and recreational lake users; they can affect fish populations and cause taste and odor problems in drinking water. Although changes in the abundance and relative concentrations of inorganic nutrients are responsible for most such blooms, they can also

be caused by reductions in grazing pressure from zooplankton that are sensitive to toxic chemicals, and they could, at least in theory, be caused by species-specific differences in sensitivity to toxic chemicals.

1.2.1.3 Reductions in timber yield and undesirable changes in forest composition. Forests have direct economic, aesthetic, and recreational values as well as indirect values. Direct economic values are the easiest to quantify. Aesthetic and recreational values of forests can be related to primary production because of the general preferences for mature forests with large trees, however, pollution-induced chlorosis and necrosis of tree leaves is also an important aesthetic impact, even when reductions in yield cannot be detected. The indirect values of forests are possibly the most important, but they are difficult to analyze. These values include erosion and flood control, removal and detoxification of pollutants, and climate moderation. Although production has been used as an index of indirect values, community structure and composition are also clearly important.

1.2.1.4 Reductions in agricultural production. The value of agriculture is self-evident. For the purpose of synfuels risk assessment, agriculture is assumed to refer only to crop production. Livestock and poultry are considered with wildlife, because assessments of risks to all vertebrate animals are based on the same toxicological data base.

1.2.1.5 Reductions in wildlife populations. Wildlife is valued as game and as an object of various forms of nondestructive

appreciation. Hunting, bird watching, and other wildlife-oriented forms of outdoor recreation are economically and psychologically important. Effects of pollutants on wildlife may result from direct toxicity, habitat modification, or food-chain dynamics.

1.2.2 Methods for Ecological Effects Assessment

Direct information on risks to populations in nature, comparable to human epidemiological data, is rarely available and often unobtainable even in principle. For the case of ecological effects of toxic chemicals, it is inevitably necessary to extrapolate risk estimates from laboratory toxicity test data or from limited field experiments. The quantity, quality, and applicability of available test data varies vastly among chemicals and end point biota. In addition, extrapolations from even the best laboratory data are compromised by incomplete characterization of the species compositions of affected environments, biotic interactions among the exposed populations, and interactions with other stresses (e.g., exploitation by man) that affect the exposed populations.

Given the diversity of end points and the variety of data types that must be accommodated, it is clear that no single method can be adequate for making all of the necessary extrapolations for all chemicals and end points of interest. Moreover, confidence in the conclusions from any risk assessment is increased if similar conclusions can be reached using several independent methods. Consequently, at the initiation of the project, it was determined that five distinctly different methods for assessing ecological effects of

toxic chemicals for risk assessment would be investigated. The following subsections briefly describe the major characteristics of the five methods and present the rationales for their choice. As previously noted, fault tree analysis and the analytic hierarchy process were abandoned following application in a preliminary risk assessment for indirect coal liquefaction. To illustrate the difficulty of applying methods borrowed from other fields to ecological assessment problems, the reasons for failure of our applications of these two methods are discussed.

1.2.2.1 Fault tree analysis. Fault tree analysis is a standard method used in engineering safety assessments to identify events and system states that can lead to disastrous failures of complex systems such as nuclear power plants and space shuttles. A fault tree is a model that graphically and logically represents these events and states. When the probabilities of each of the possible initiating events are specified, the fault tree can be used to calculate the probability of failure of the whole system.

There is an appealing analogy between complex engineering systems and complex ecosystems, and it is even possible to define ecological "failures," such as population extinctions, that are analogous to boiler explosions or core meltdowns. Based on this analogy, fault trees were developed for (1) recruitment failure in a fish population and (2) local extinction of a bird population. These fault trees proved useful in illustrating the various possible direct and indirect pathways through which toxic chemicals can affect populations; however, it is clearly impossible to perform quantitative analyses of ecological

fault trees. One major problem is the difficulty of estimating probabilities for the various initial states that make populations vulnerable to additional stresses (e.g., habitat restrictions). More fundamentally, the continuous responses and cumulative effects that characterize responses of biological systems to stress cannot be represented using the binary logic of fault trees. However, even without quantification, construction of ecological fault trees can serve important heuristic functions.

1.2.2.2 Analytic hierarchy process. The analytic hierarchy process (Saaty 1980) is a decision-making technique developed for use in economic planning. Its two basic components are (1) the ordering of the elements of a decision into a hierarchy and (2) the use of expert opinion to rank the elements of each level in the hierarchy. This approach was intended to be used in situations where qualitatively different attributes must be compared, quantitative measurement scales are unavailable, and/or subjective judgments are necessary. Because all of these characteristics are typical attributes of environmental assessment problems, it seemed possible that the analytic hierarchy process could be fruitfully used as an alternative to quantitative assessment models. For example, the decision about the relative hazard of 17 components of a complex effluent mixture can be hierarchically ordered into comparisons of the relative importance of different fish populations that may be exposed, the relative importance of direct and indirect effects of chemicals on each fish population, and so forth down to the effects of each effluent component on the exposed organisms.

When this approach was applied using expert ecologists and toxicologists, interesting results were, in fact, obtained. Taking into account information and opinions that could not be objectified with any of the strictly quantitative methods used in the preliminary risk assessment for indirect coal liquefaction (e.g., microbial degradation of contaminants in soils), both aquatic and terrestrial experts rated organic contaminants as substantially less hazardous than would be predicted based on toxicity alone. However, the analytic hierarchy process proved to be prohibitively cumbersome when applied to the synfuels risk assessment problem because of the necessity for large numbers of pair-wise comparisons among classes of chemicals. For example, applying the method to 17 contaminant classes requires 136 pair-wise comparisons of relative toxicity for each type of organism exposed. Although the method appears promising, adapting its use with synfuels risk assessment was judged to be beyond the scope of this project.

1.2.2.3 Quotient method. The quotient method entails a direct comparison of the estimated concentration of a chemical in the ambient environment with a measured toxicological benchmark concentration (e.g., an LC_{50}) for that chemical. No attempt is made to quantify uncertainties or to extrapolate to population-level effects. As such, the quotient method is not a quantitative risk assessment technique according to the definition used in this project. However, this method is nonetheless an important component of any risk assessment scheme for toxic chemicals. There are two major reasons for this. First, the quotient method is a valuable screening technique because environmental

concentrations of chemicals that are several orders of magnitude below concentrations that affect laboratory test organisms are unlikely to have serious ecological consequences. Second, direct comparisons between environmental concentrations and laboratory test data are the basis for all existing chemical hazard assessment protocols. Thus, the quotient method provides a means of comparing results obtained using more sophisticated, quantitative risk assessment techniques with results obtained using conventional procedures.

Not all toxicological benchmarks are equally useful in applying the quotient method; moreover, substantial care must be used in comparing toxicity test data obtained under differing experimental conditions. These issues, as well as (1) criteria for interpreting values of quotients and (2) procedures for evaluating complex effluents using the toxic units approach, are discussed in detail in Section 3 of this report.

1.2.2.4 Analysis of extrapolation error. The classical approach to assessing potential ecological effects of toxic chemicals is based on laboratory testing using one or a few standard species and life stages. Variability among species, life stages, and exposure durations is accounted for by using correction factors, supposedly sensitive test species, and subjective judgment. The usual objective of this approach is to estimate a "safe" level, below which no effects will occur. It is not possible, using this approach, to estimate the consequences of exceeding the safe level; moreover, it is still possible, because of the sources of variability previously mentioned, that effects will occur even if the safe level is not exceeded.

Section 4 of this report presents a method for explicitly quantifying uncertainty resulting from (1) interspecies differences in sensitivity and (2) the variable relationship between acute and chronic effects of chemicals. The method, known as analysis of extrapolation error, is based on statistical analysis of acute and chronic toxicity test data sets collected using uniform experimental protocols. At the time technology risk assessments for this project were performed, adequate data sets were available only for fish.

Given a chemical and species of interest, regression equations derived from the data base can be used to estimate a chronic effects threshold for the species of interest from a 96-h LC_{50} for either (1) the species itself or (2) any other species that has been tested. Residual errors from the regressions are used to estimate the prediction error of the estimated effects threshold and, consequently, the risk that a given environmental concentration of the chemical being assessed exceeds the chronic effects threshold of the species of interest.

Section 5 presents an extension of analysis of extrapolation error that enables extrapolation of individual-level effects of toxic chemicals to effects on populations. This extrapolation involves estimating concentration-response functions, with confidence bands, and linking these functions to a life-cycle model of the species of interest. The objective of this extension of the original methodology is to enable extrapolation to the level of ultimate end-points, that is, reductions in valued populations. Development of the population-level assessment model was not completed in time for use in the four synfuels technology assessments.

1.2.2.5 Ecosystem uncertainty analysis: As heretofore noted, effects of environmental stresses on real populations depend on complex biotic and abiotic processes that cannot be reproduced in the laboratory. Although many stresses can be usefully studied in field experiments, such experiments are impossible for some risk assessment problems. Mathematical models of the biological systems of interest provide an alternative means of incorporating environmental complexity in risk assessments. In particular, ecological models can incorporate biological phenomena, such as competition and predation, that can magnify or offset the direct effects of contaminants on organisms. For the synfuels risk assessment project, recent developments in systems ecology were exploited to develop an assessment method known as ecosystem uncertainty analysis.

In ecosystem uncertainty analysis, effects of stress on individual organisms are extrapolated to net effects on populations and trophic levels using an ecosystem simulation model. Estimates of uncertainties associated with individual-level effects are translated into estimates of risks of significant adverse changes in the model populations. An existing ecosystem model, the Standard Water Column Model (SWACOM), was used for the synfuels risk assessment, however, it was necessary to develop a procedure for translating laboratory test results, such as LC_{50}s, into changes in model parameters, such as photosynthesis and respiration rates.

In Section 6 of this report, the basic concepts used in ecosystem uncertainty analysis are described, and several applications of the method are presented and discussed. The fundamental components of the

method include (1) the linking of toxicity data to changes in ecological rate processes and (2) the use of efficient uncertainty analysis techniques to extrapolate from parameter uncertainties to ultimate risks. The specific ecological model used in an assessment can be selected to meet the needs of the problem at hand. It is expected that in many future applications SWACOM will be replaced by a more appropriate model.

1.3 ORGANIZATION OF USERS' MANUAL

The remaining sections of this report describe the steps in an ecological risk assessment for a synfuels facility, any other facility producing chemical effluents, or an individual chemical. It is assumed that source terms, in units of mass per unit time, have been provided to the risk assessor.

Section 2 describes the process of modeling the transport and transformation of contaminants in air, surface water, and groundwater. Because of the large number of existing models available for use in exposure assessments, the emphasis in this section is on criteria for selecting models that are properly matched to the available information concerning (1) the environmental chemistry of the contaminant(s) being modeled, (2) the spatiotemporal resolution of data on the characteristics of the reference environment, and (3) the requirements of the effects assessment methods being used.

Sections 3 through 6 document the effects assessment methods used in the synfuels risk assessments. Throughout these sections, the emphasis is on explanation and documentation of biological assumptions,

statistical/mathematical methods, and data sources. No attempt was made to document the computer codes used by the project staff in implementing the methods. It is expected that, because of differing computing configurations and assessment needs, the code modifications required by most users of the risk assessment methodology would render any such documentation effectively useless.

Section 7 discusses the integration of exposure and effects assessments to produce overall ecological risk assessments for toxic chemicals. In addition, Section 7 discusses the application of the methods documented in this report to problems other than technology risk assessment and also outlines the project staff's views on the research needed to increase current utility and scientific credibility of ecological risk assessment.

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2. EXPOSURE ASSESSMENT

L. W. Barnthouse

For the purpose of risk assessments for toxic chemicals, exposure assessment may be defined as the "determination of the concentration of toxic materials in space and time at the interface with target populations" (Travis et al. 1983). Before an exposure assessment can be performed, it is necessary to develop (1) source terms for the technology (or other contaminant source) being assessed and (2) a description of the environment into which contaminants will be released. The source terms are simply estimates of the quantity and composition of contaminant releases. They may be either time dependent, as in accidental spills or upset events, or time independent, as in continuous routine emissions. Reference environmental descriptions are those of (1) the biota that may be exposed to contaminant releases and (2) the hydrological, topographical, geological, and meteorological characteristics of the environment that affect the transport and transformation of contaminants. Environmental characteristics may vary in time and space. Given source terms and a reference environment, the key step in exposure assessment is the use of a model of contaminant transport and transformation to quantify the movement of contaminants from the source, through the environment, to the target populations.

Many atmospheric, surface water, groundwater, and multimedia models have been developed for quantifying the environmental fate of radionuclides and toxic contaminants. Rather than developing entirely

new models for the synfuels risk assessments, existing models that appeared appropriate were selected and, where necessary, modified. Only general descriptions of the models are presented here; detailed documentation is provided elsewhere (Travis et al. 1983). Only the atmospheric and surface water pathways are discussed in this section, because these are the primary routes of exposure for aquatic and terrestrial biota. The particular models chosen for the synfuels risk assessments were selected based on the following considerations:

1. Risk assessments were to be performed for technologies and processes rather than specific plants and sites. Only engineering judgments of routine emission compositions were available.

2. Exposure assessments were needed for a large number of complex effluent components, both organic and inorganic. The environmental chemistry of most of the organic chemicals to be assessed was poorly understood.

3. Both acute and chronic ecological effects were to be considered.

4. For ecological effects at the screening level, near-field exposure assessments should be sufficient. The concentrations of toxic contaminants would be expected to decline with decreasing distance from the source; therefore, if risks are minimal in the near field, they should also be minimal in the far field.

5. Both the inherent variability of environmental processes and scientific uncertainty concerning the fate of synfuels-derived contaminants should be explicitly modeled.

6. Models used in synfuels risk assessment should rely, to the extent appropriate, on models that have proved useful in other types of environmental assessments.

The above considerations suggested that relatively simple but flexible environmental transport models would be best suited for synfuels risk assessments. Because of the lack of specificity of the

source terms and the generic nature of the assessment, it was determined that generalized site descriptions characteristic of broad regions in which synfuels facilities might be sited, rather than detailed descriptions of particular sites, would be used. Given the use of generalized site descriptions, high spatiotemporal resolution in the models would be irrelevant. Moreover, because of the large number of chemicals involved and the poor understanding of the environmental chemistry of most of them, it seemed prudent to limit the modeling of chemical transformations and mass transfers to simple, first-order rates based on direct measurements or structure-activity relationships. Whatever information exists should be incorporated to avoid undue conservatism (e.g., by assuming complete solubility and no degradation of organic chemicals); however, consideration of higher-order processes and multistep transformations could be deferred to subsequent assessments focused on those contaminants identified in initial assessments to be potentially hazardous.

Because of the need to consider both acute effects of short-duration, high-level exposures and chronic effects of long-term, low-level exposures, the models would have to operate on time scales ranging from hours to months and years. Uncertainty and variability are important aspects of risk analysis; therefore, it was desirable for the models to be amenable to error analysis (Gardner et al. 1981), both to quantify scientific uncertainty regarding transport processes and to model hydrological and meteorological variability that affects the transport and fate of chemicals.

Because of the many similarities between the transport of radionuclides from power plants and the transport of chemical contaminants from industrial facilities, the models used in radiological impact assessments performed for the U.S. Nuclear Regulatory Commission and the U.S. Environmental Protection Agency were taken as the starting points for choosing environmental transport models for synfuels risk assessments.

2.1 SURFACE WATER TRANSPORT AND TRANSFORMATION

The surface water transport model used in the synfuels environmental risk assessment project is a steady-state model similar in concept to the EXAMS model (Baughman and Lassiter 1978) but simpler in terms of process chemistry and environmental detail. This model is also similar to the radionuclide transport model described by Niemczyk, Adams, and Murfin (1980). It is intended as a flexible descriptor of the transport and fate of contaminants in streams and rivers. Rivers, rather than lakes, were chosen as model environments because the most common proposed sites for synfuels plants are on rivers. As in EXAMS, a river is represented as a connected series of completely mixed reaches. Within each reach, steady-state contaminant concentrations are estimated based on dilution and on physical/chemical removal from the water column. The steady-state contaminant concentration ($C_{w,1}$) in the first reach downstream from a continuous effluent discharge is given by

$$C_{w,1} = (I/V_1)/[(Q_1/V_1) + k_{t,1}] \quad , \qquad (1)$$

where

I = contaminant input rate (kg/s),

V_1 = volume of first reach (m^3),

Q_1 = stream discharge of first reach (m^3/s), and

$k_{t,1}$ = first-order contaminant removal rate for the first reach.

The steady-state concentration for the nth reach downstream from the first is given by

$$C_{w,n} = [(C_{w,n-1}/Q_{n-1})/V_n]/[(Q_n/V_n) + k_{t,n}] \qquad (2.2)$$

The first-order removal rate ($k_{t,n}$) is equal to the sum of first-order rates due to volatilization, settling, direct photolysis, and biological/chemical degradation. With the exception of biological/chemical degradation, all of the above rates are modeled as functions of environmental parameters and physical/chemical properties of the contaminants. Procedures for estimating rate constants for volatilization, settling, adsorption, and photolysis are presented in Section 2.3.2 of Travis et al. (1983).

For the purpose of ecological risk assessment, only a 1-km stream reach immediately downstream from the assumed contaminant release point was modeled. In effect, the released contaminants were assumed to be completely diluted within a "box" 1 km in length. This reach size was selected on the basis of biological/social significance. It is unlikely that adverse ecological consequences would ensue from the killing of one fish at the end of a discharge pipe. However, the

biological degradation of a 1-km river segment could significantly reduce biological production or disrupt local fish populations (either through direct mortality or through indirect effects such as interference with migration). An impact on this scale would also likely be considered unacceptable by local residents.

The requirement to assess both short-term and long-term effects was met by modeling the effects of stochastically varying hydrologic parameters such as stream discharge, temperature, and sediment load. Realistic distributions for these parameters were obtained from U.S. Geological Survey water resources monitoring data for streams typical of those on which synfuels plants might be sited (Travis et al. 1983, Sect. 3). Frequency distributions for contaminant concentrations were computed as functions of the distributions of hydrologic parameters, according to the procedure of Gardner et al. (1981). For assessing chronic effects, the median daily concentration was chosen as the best estimator of the long-term average concentration to which organisms would be exposed. For assessing acute effects, the concentration chosen was the upper 95th percentile concentration, that is, the concentration expected to be met or exceeded on only 5% of days.

In practice, it was found that an even simpler model would have been sufficient for the purpose of ecological risk assessment. Estimated water-column half-lives for contaminants of interest in synfuels risk assessment were on the order of 10^2 to 10^4 h (Barnthouse et al. 1985a). Processes operating at these rates have negligible effects on water-column concentrations in the near field.

Near-field concentrations suitable for ecological risk assessment can be obtained by modeling only (1) dilution, as determined by stochastically varying stream discharges; and (2) essentially instantaneous chemical processes such as ionization and complexation.

2.2 ATMOSPHERIC TRANSPORT, TRANSFORMATION, AND DEPOSITION

Many computer codes exist for calculating the transport, transformation, and deposition of radionuclides and toxic contaminants within 50 km of a pollutant source. Most are variants of a single underlying model, the Gaussian plume. In its simplest form, the Gaussian plume predicts the diffusion and dispersion of a conservative, gaseous substance from a continuous point source elevated above the ground, under constant wind speed and homogeneous atmospheric conditions, and over uniformly flat terrain. The basic model can be modified to account for such phenomena as plume buoyancy, atmospheric stratification, contaminant degradation or decay, and wet and dry deposition of particles and aerosols.

Because of the relative ease of application of Gaussian plume models and the large accumulated experience with these models, a Gaussian plume model was used to calculate atmospheric exposures for synfuels risk assessment. The specific code chosen was AIRDOS-EPA (Moore et al. 1979). This model was chosen over five alternatives because it (1) incorporates first-order degradation rates for pollutants, (2) can estimate surface deposition rates, and (3) provides output in a form suitable for calculating exposures to human populations. The equations for estimating plume dispersion,

contaminant degradation, dry deposition, and wet deposition in AIRDOS-EPA are presented in Section 2.2.2 of Travis et al. (1983). The AIRDOS-EPA code calculates average ground-level atmospheric concentrations and surface deposition rates for sixteen 22.5° sectors surrounding the plume source.

Adverse meteorological conditions (such as inversions) can lead to high ground-level concentrations that cause acute toxicity to exposed plants and animals. Such conditions occur on time scales of from 8 h to a few days. Unfortunately, Gaussian plume models are relatively poor predictors of short-term plume behavior (Hoffman et al. 1978). These models are much better predictors of annual average concentrations. As a substitute for short-term exposure estimates, annual average concentrations were calculated at 500 m intervals over the 16 sectors modeled in AIRDOS-EPA, and the highest of these averages was used in the synfuels risk assessments (Barnthouse et al. 1985b, Sect. 2.3).

Deposited contaminants, when dissolved in soil water, can cause toxic effects on exposed plant roots. To provide root exposure estimates for ecological risk assessment, the deposition rates from AIRDOS-EPA were used to estimate accumulation of contaminants in soil over an assumed 35-year operational lifetime of a synfuels plant. As with ground-level atmospheric concentrations, accumulation was estimated at the point of greatest annual deposition. The soil solution exposure estimates incorporate both degradation of contaminants in soil and partitioning of contaminants between soil particles and solution (Barnthouse et al. 1985b, Sect. 2.3).

The atmospheric exposure assessments performed using AIRDOS-EPA did not meet all of the requirements for ecological risk assessments described in the introduction to this section. Specifically, short-term exposures were not addressed, only worst-case exposures were estimated, and no error analyses were performed. These deficiencies result in part from the use of a computer code designed for estimating long-term exposures to human populations, however, any Gaussian plume model would have been of uncertain utility for estimating short-term exposures. Although other classes of models are more suitable for this purpose, such models require far more site-specific meteorological data than are appropriate for technology-level risk assessments. Given necessary code modifications, error analyses of AIRDOS-EPA or any other similar code could be performed. It was not deemed necessary to perform such analyses for the synfuels risk assessment project, because preliminary screening using worst-case exposure estimates suggested that the majority of synfuels-related chemicals present negligible risks to terrestrial plants and animals (Suter et al. 1984, Barnthouse et al. 1985b). Future ecological risk assessments could, however, benefit from the development of atmospheric exposure assessment models designed specifically for ecological risk assessment, with capabilities for modeling short-duration events and incorporating error analyses.

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3. TOXICITY QUOTIENTS

G. W. Suter II

3.1 DEFINITION

The quotient method is simply the direct arithmetic comparison of a benchmark concentration (BC) from a toxicity test with an expected environmental concentration (EEC). It is typically calculated as the quotient of the ratio EEC/BC. It is the basis for nearly all assessments of the environmental hazards of chemicals. In this basic form, the method amounts to an assumption that the test benchmark is a good model of the assessment end point (i.e., the level of toxic effect that is not to be exceeded in the ambient ecosystem). This assumption is most likely to hold when the toxicity tests have been performed for the particular assessment, using the anticipated temporal pattern of exposure and dilution water and organisms from the site. When it is recognized that this assumption may not hold, multiplicative factors are often applied to the quotients.

3.2 FACTORS

The most common method of allowing for imperfect correspondence between the benchmark concentration and the end point is to multiply the quotient or either of its components by factors. These are variously referred to as safety factors, uncertainty factors, or correction factors, depending on whether the goal is to ensure safety, account for a recognized source of uncertainty, or correct for proportional differences between types of data. Traditionally, a

single number was used that incorporated all of the assessor's knowledge and beliefs about the relationship between the test result and the anticipated effect in the field (Mount 1977). More recently, it has become common to use multiplicative strings of factors, each of which accounts for a different correction or source of uncertainty (e.g., EPA 1985). These multiplicative chains imply an assumption that everything will go wrong at once. For example, the most sensitive life stage of the most sensitive species will be exposed to the most concentrated effluent at low-flow conditions while debilitated by stress, and the actual response is at the limit of our range of uncertainty. If carried out consistently, this approach would be extremely conservative. In actual applications, only a fraction of the possible uncertainties and corrections are included, so that the product of the factors will not be unacceptably large. To avoid the problems of subjectivity and conservatism, we have used unadorned quotients in our assessments and left the consideration of uncertainty and data extrapolation to methods that use more appropriate statistical models.

3.3 IMPLEMENTATION

The critical decisions in implementing the quotient method are (1) selection of expressions of the expected environmental concentration that reflect the pattern of exposure in the field, (2) selection of toxicological benchmarks that correspond to the effect of concern in the field, and (3) matching the benchmarks and environmental concentrations

so that they logically correspond. The selection and derivation of estimates of the expected environmental concentration is discussed in Sect. 2. The other two decisions are discussed here.

3.3.1 Matching Exposure and Effects

If the quotient is to be consistent, the toxicological benchmark must bear a logical relationship to the expected environmental concentration. The first major problem is ensuring that the medium and mode of exposure are consistent. For example, the environmental concentration that should be estimated for benthic infauna is the pore water concentration rather than the free water concentration, and per cutaneous toxicities should be compared with concentrations in films on traversed surfaces rather than with bulk concentrations.

The second major problem is ensuring that the response of the organism to the toxicant does not change the exposure. The most conspicuous example is avoidance of polluted food or media. However, toxicants may also reduce feeding, thereby reducing the oral dose, or may cause aquatic organisms to lose contact with the substrate and drift out of the area. Since behavioral data are lacking for most chemicals, this problem is relatively seldom addressed, but it should be kept in mind.

The third major problem is duration, which is a major source of confusion, largely because of ambiguities concerning the terms acute and chronic. The ambiguity arises from the use of these terms to describe severity as well as duration. Acute exposures and

toxicities are assumed to be both of shorter duration and more severe than chronic exposures and toxicities. The implicit model behind this assumption is that chronic effects are sublethal responses that occur because of the accumulation of the toxicant or of toxicant-induced injuries over long exposures. Conversely, it has become clear that the most sensitive responses in chronic toxicity tests for aquatic organisms are typically effects on sensitive life stages or processes that occur fairly quickly, do not require long prior exposures, and may be quite severe (McKim 1985). As a result, duration is now often defined both in temporal terms and in terms of the life cycle of an organism (i.e., a chronic exposure is one that potentially involves all life stages).

The resulting confusion is illustrated by the standard toxicological benchmarks for fish. The standard acute benchmark is the 96-hour median lethal concentration (LC_{50}) for adult or juvenile fish (EPA 1982, ASTM 1984, OECD 1981). The duration of this test was selected because most mortality in most such tests occurs in the first four days; in fact, this acute benchmark is considered a good estimate of the time-independent or incipient LC_{50} (Ruesink and Smith 1975). The standard chronic benchmark is the maximum acceptable toxicant concentration (MATC), which is the threshold for significant effects on survival, growth, or reproduction (EPA 1982, ASTM 1984). Since this benchmark is based on only the most sensitive response, life stages that are generally less sensitive have been dropped from chronic tests so that those tests have been reduced from life cycle (12 to 30 months)

to early life stages (28 to 60 days) (McKim 1985). Tests that expose larvae only for 11 (Birge et al.1981) or 7 days (Norberg and Mount, 1985) have now been proposed as equivalent to the longer chronic tests. As a result, the chronic benchmark for fish is now tied to events of short duration (the presence and response of sensitive larvae), whereas the acute benchmark is applicable to exposures of indefinite duration and life stages that are continuously present. Even the severity distinction is not clear. Although the LC_{50} clearly indicates a severe effect, the fact that the MATC is tied to a statistical threshold rather than a specified magnitude of effect means that it too can correspond to severe effects (e.g., failure of more than half of the females to spawn at the MATC for chlordane in Cardwell et al. 1977).

The solution for the assessor is to disaggregate the concept of duration from severity when categorizing exposures. In the simplest case the temporal pattern of exposure falls into distinct categories, based on characteristics of the source and its interactions with the environment. If the aqueous dilution volume is relatively constant, exposures may be divided into those that result from spills and other short-term upsets and those that result from routine releases. Exposures to an atmospheric release might be divided into plume strikes (an hour or less), stagnation events (a week or less), and the growing season average exposure. In these cases the durations are determined by the exposure, and the toxicological benchmarks must be selected to match.

In other cases it may not be possible to identify distinct and relatively constant categories of exposure; there may simply be a continuous spectrum of fluctuations in exposure concentrations. In such cases the biology of the toxicological responses must be used to select durations, and the exposure must be selected to match. For example, if the most sensitive response to a chemical is mortality of larval fish, which begins within a day of the beginning of exposure, then the appropriate exposure concentration could be based on dilution of the effluent in the 24-h low flow that recurs at an average interval of 10 years during the months in which larval fish are present at the site. In any case, the matching of exposure with a toxicological benchmark should be based on an analysis of the situation being assessed rather than on preconceptions about acute and chronic toxicity.

3.3.2 Benchmark Selection

In many cases the selection of toxicological benchmarks for an assessment is largely constrained by the availability of published data, by differences in the quality of available data, or by the need to match the benchmark to the mode and duration of exposure. However, when data are abundant or when testing can be prescribed by the assessor, toxicological benchmarks should be selected on the basis of their statistical form and their expression of the important responses of the organism of interest.

3.3.2.1 Statistical form. There are two statistical types of toxicological benchmarks: (1) those that are based on a concentration-response function and prescribe a level of effect and

(2) those that are based on hypothesis testing. The first type is obtained by fitting a function to sets of points relating the level of response (proportion dying, mean weight, etc.) to an exposure concentration (dose, concentration in water, concentration in food, etc.). The concentration causing a particular level of effect is then obtained by inverse regression. Examples of this type of benchmark include the LC_{50}, median lethal dose (LD_{50}), median effective concentration (EC_{50}), and lethal threshold concentration (LC_1).

The other statistical category of benchmarks consists of those that are derived by hypothesis testing techniques. Responses at the exposure concentrations are compared with control (unexposed) responses to test the null hypothesis that they are the same as the control responses. Benchmarks of this type include the no observed effect level (NOEL), the lowest observed effect level (LOEL) and the MATC, which is assumed to lie between the LOEL and the NOEL.

The disadvantages of benchmarks based on hypothesis testing relative to those based on curve fitting have been discussed by Stephan and Rogers (in press). They include (1) the use of conventional hypothesis testing procedures (with $\alpha = 0.05$ and β unconstrained) implies that it is very important to avoid declaring that a concentration is toxic when it is not, but it is not so important to declare that a concentration is not toxic when it is; (2) the threshold for statistical significance does not correspond to a toxicological threshold or to any particular level of effect; (3) poor testing procedures increase the variance in response and therefore reduce the apparent toxicity of the chemical in a hypothesis test; and (4) the

results are relatively sensitive to the design of the test. The advantages of hypothesis testing benchmarks are that they can be calculated even when the test data are too poor or meager for curve fitting and they allow the assessor to avoid specific decisions about what constitutes a significant level of effect. We feel that hypothesis testing is generally an inappropriate way to calculate benchmarks; however, in many cases, the use of such benchmarks by the assessor is unavoidable.

3.3.2.2 Taxon-specific factors. We discuss here benchmarks currently used to express toxic effects on the four end point taxa in our risk analyses: fish, planktonic algae, terrestrial vascular plants, and vertebrate wildlife.

1. Fish

The most abundant toxicological benchmark for fish is the 96-h LC_{50} for adult or juvenile (post-larval) individuals; for most chemicals, it is the only type of data available. As previously described, it is acute in terms of severity but is often applicable to extended durations. Since it does not protect early life stages and implies mortality in all life stages, it can be thought of as a benchmark for conspicuous fish kills (large numbers of large dead fish). Although the median response was chosen for the benchmark because of its small variance relative to other levels of mortality, a correction factor must be applied if the assessor is interested in preventing low-level mortality (EPA 1985), a process that adds considerable variance.

Another problem with this benchmark is that in most cases only the response at 96 h is reported. Many assessments involve transient events, and the time to mortality is more important than the percent mortality. However, despite the suggestions of Sprague (1973), Alabaster and Lloyd (1982) and others, the time course of mortality is seldom reported. In defense of the 96 h LC_{50}, it might be argued that it is only meant to be used for comparative purposes and not for assessment of effects. However, assessments have been conducted and criteria have been set on the basis of this benchmark because it is available and better numbers are generally not.

The standard benchmark for chronic effects on fish is the MATC. As previously discussed, MATCs have all of the considerable faults of benchmarks that are derived from hypothesis tests. In this context, it is important to reiterate that assessments based on MATCs do not provide a consistent level of protection, and the industry that performs the poorest tests will, on average, be the least regulated.

The most generally useful benchmarks for assessing effects on fish by the quotient method would be a set of LC_1 values for each of the life stages that will be exposed at 1, 24, 48, and 96 h (or longer if mortality continues), plus EC_1 values for growth and fecundity in suitably long exposures. Individual thresholds could then be selected for each assessment, depending on the life stages that will be exposed and the duration of the exposure.

If all life stages will be exposed to a relatively constant concentration of the toxicant, then a global benchmark [one that integrates the individual measured effects (Javitz, 1982)] may be

preferred as an expression of chronic effects. The simplest such benchmark is the standing crop of fish at the end of the test. More commonly, the weight of young per initial female (or initial egg, in the case of early life stage tests) is calculated as

$$\sum S_1 S_2 \ldots S_n MW \quad ,$$

where S_x is the survivorship of life stage x, M is fecundity, and W is the weight of the final cohort (e.g., Eaton et al. 1978). A third global benchmark (which can only be used with life-cycle results) is the intrinsic rate of increase r which is calculated from:

$$\sum l_x m_x e^{-rx} = 1 \quad ,$$

where l is the proportion surviving to age x, and m is the number of female offspring produced by a female of age x during the next interval (e.g., Daniels and Allan 1981). The intrinsic rate of increase, r, is a more appropriate benchmark for invertebrates than fish, since life-cycle tests are still routinely performed with invertebrates, and effects on growth (which are not included in the formula for r) are reflected in fecundity in invertebrate chronic tests.

The main advantage of global benchmarks is that they combine a diversity of individual responses, some of which have little intuitive significance, into a parameter that has the form of a population-level response. Global responses may be more sensitive than individual responses when a number of small toxic effects are combined into one large global response; however, sensitivity can also be reduced if

toxic effects are combined with hormetic or pseudo-hormetic effects or (if hypothesis testing is used) with highly variable effects.

2. Algae

Benchmarks for effects on algae have been poorly standardized. Reported responses included mortality, growth, CO_2 fixation, cell numbers, chlorophyll content, and others. Durations were various, and a variety of statistical expressions derived from both hypothesis testing and curve fitting were used. There is now some agreement on the use of 96-h EC_{50} values for some measure of productivity. However, there is still no agreement on whether the appropriate measure is weight, number of cells, chlorophyll, or carbon assimilation, and whether the benchmark should be based on the final value, the time-integrated value, or the maximum rate of increase. The EPA calls for the use of final cell weight, cell number, or an equivalent indirect measurement, whereas OECD calls for the use of the maximum growth rate based on cell number (EPA 1982 and OECD 1981). If, as is often the case, planktonic algae are limited by nutrient availability, then equilibrium biomass or cell numbers may be more relevant. However, if algae are limited by herbivory, the ability of a population to replace losses (i.e., maximum growth rate) may be more relevant.

Since the life cycles of microalgae in a rapidly growing culture are much shorter than test durations or most effluent releases, these test results can be used in most assessments. However, it should be remembered that algal communities are generally nutrient limited, and, over the course of chronic exposures, resistant algal species will tend

to replace sensitive species. The implications of these changes in community composition depend on the effects of the algae on water quality and their palatability to herbivores (Sect. 6).

3. Terrestrial plants

Existing toxicity data for terrestrial plants are even more diverse and nonstandard than for aquatic algae. Although (as with algae) production is measured and statistically analyzed in a variety of ways, terrestrial plants also have long life cycles with distinct stages and organs, and they can be exposed through the stomates, leaf surfaces, or roots. We have confronted this chaotic situation by limiting the benchmarks used to those such as yield, growth, or numbers of particular organs that directly express productivity (visible injury and changes in gas exchange rates are commonly reported responses that do not correlate with production), and by trying to match the duration and route of exposure in the test to the exposure being assessed.

The most common general type of phytotoxicity test is the seedling growth test. This type of test can be conducted in soil or hydroponic systems and can be adapted to test chemicals in air, sprays, soil, or irrigation water. There is little agreement on durations or responses, but the EPA (1982) recommends the determination of EC_{10} and EC_{50} values for weight and height after 14 days. Tests for effects on seed germination and hypocotyl elongation have been used as quicker and less-expensive phytotoxicity tests, as well as indicators of effects on those particular life stages (EPA 1982); however, their relationship to other plant responses has not been established. A definitive test

would include the entire life cycle from seed germination to germination of daughter seeds, but such tests are rarely performed. A life-cycle test using Arabadopsis is being developed by the EPA.

4. Wildlife

The most common benchmark available for assessing effects on wildlife is the acute, oral, median lethal dose (LD_{50}) for laboratory rodents. Avian toxicologists have followed the mammalian example by relying largely on acute LD_{50}s for adults (e.g., Hudson et al. 1984), but subacute median lethal dietary toxicities for young birds (LC_{50}s) have become more common (e.g., Hill et al. 1975) and have been adopted by the EPA (1982) and ASTM (1984). These benchmarks are applicable to short-term exposures such as result from application of nonpersistent pesticides. In most such cases, the concentration in food is the primary expression of exposure; therefore, oral LC_{50}s are directly applicable, whereas intake must be estimated to calculate doses before LD_{50}s can be used (Kenega 1973). In a few cases, notably when the exposure results from consumption of granular pesticides or cleaning pelt or plumage, an oral LD_{50} is more directly applicable. Since the relative sensitivities of adults and young and the effects of exposure duration are less well known for birds than fish (Tucker and Leitzke 1979), the comparability and usability of these benchmarks are uncertain.

The other standard wildlife benchmark is the threshold for effects in the avian reproduction test (EPA 1982, ASTM 1985). This test resembles the MATC for chronic and subchronic effects on fish, in that the benchmark is usually derived by applying hypothesis testing statistics to an array of measured parameters. Like the MATC, it would

be more useful for assessment if curve fitting were used to establish a consistent level of effect, and if a global parameter (such as the weight of young per female) were calculated along with the individual measured responses. The duration of exposure in this test (6-10 weeks) can be considered to represent a chronic adult exposure for all but the most persistent and bioaccumulated chemicals; however, since the young are not exposed, this cannot be considered a full chronic (i.e., life-cycle) test.

There are very few data available for assessing the toxic effects of nonpesticide chemicals and effluents on wildlife. It is generally necessary to resort to the use of the health literature for such assessments. We have used rodent LD_{50} values as a relatively consistent benchmark for comparative purposes and the lowest-reported toxic effect as a benchmark for suggesting where hazards may exist.

3.4 DISCUSSION

The chief advantages of the quotient method are that it is quick, easy, generally accepted, and can be applied to any data. Because the effects benchmark is directly compared with the expected environmental concentration, the burden of ensuring realism in the description of the effects and their relationship to exposure falls largely on the toxicologist rather than the assessor. As previously discussed, the use of multiplicative factors to modify quotients amounts to treating uncertainty in a deterministic manner, and this logical inconsistency has resulted in incomplete and inconsistent treatments of corrections and uncertainties. However, without the factors, the assumptions

concerning the appropriateness of the toxicological benchmark and the estimated environmental concentration are not incorporated in the analysis. Therefore, this method is useful when (1) a large number of chemicals must be screened to find potent l hazards, (2) the toxicity data are unconventional, or (3) the data ... believed to be completely appropriate to the assessment, or at least cannot be improved by available analytical techniques.

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4. ANALYSIS OF EXTRAPOLATION ERROR

G. W. Suter II, A. E. Rosen, and E. Linder

4.1 DEFINITION

Analysis of extrapolation error (AEE) is a method of calculating the probability of exceeding assessment end points to be used in those cases where the end points can be expressed as standard toxicological benchmarks. The method has two components: (1) the extrapolation component that, like the factors used with the quotient method (Sect. 3.2), is used to estimate the value of the assessment end point from the available test data and to account for the uncertainty in the estimate; and (2) the risk component that calculates the probability of exceeding the assessment end point using the results of the extrapolations. Since the extrapolation component treats extrapolation and uncertainty in a more rigorous and conceptually appropriate manner than does the use of chains of multiplicative factors, it can be used in place of such factors in hazard assessment. However, it is the calculation of the probability that an expected environmental concentration will exceed the end point (rather than simply comparing them arithmetically as in the quotient method) that makes AEE a true risk assessment method.

In the following sections we will explain the assumptions and statistical procedures for AEE and provide numerical examples; however, the method can be best introduced by presenting an example graphically. Assume that we wish to estimate the probability that the expected environmental concentration of a chemical will exceed the

threshold for life-cycle effects on survival, growth, or reproduction of brook trout (Salvelinus fontinalis) and that we only have an LC_{50} for rainbow trout (Salmo gairdneri). In that case we must extrapolate between the genera Salmo and Salvelinus, and we must extrapolate between the LC_{50} and the chronic threshold. The relationship between the two genera is illustrated in Fig. 4.1. Each of the points represents an individual chemical for which a member of both genera has been tested using a common protocol and with the results expressed as 96-h LC_{50}s. The relationship between LC_{50}s and life-cycle effects thresholds (expressed as MATCs) is shown in Fig. 4.2. The points here represent different species-chemical combinations for which both an LC_{50} and a life-cycle or partial life-cyle MATC have been determined in the same laboratory. If we use the rainbow trout LC_{50} as the x value in the Fig. 4.1 relationship, we can estimate a brook trout LC_{50} and an associated variance that can be used in the Fig. 4.2 relationship to estimate a brook trout MATC and associated variance. The estimated MATC and its total variance can be represented as a probability density function, as in Fig. 4.3. The risk that the MATC will in fact be exceeded is the probability that a realization of the MATC, chosen at random from that probability density function, will be less than a similarly chosen value from the probability density function for the expected environmental concentration.

AEE differs from previous approaches to extrapolating environmental toxicology data in its emphasis on the uncertainty associated with the extrapolations and the contribution of that uncertainty to the risk. The traditional approach is to ask whether

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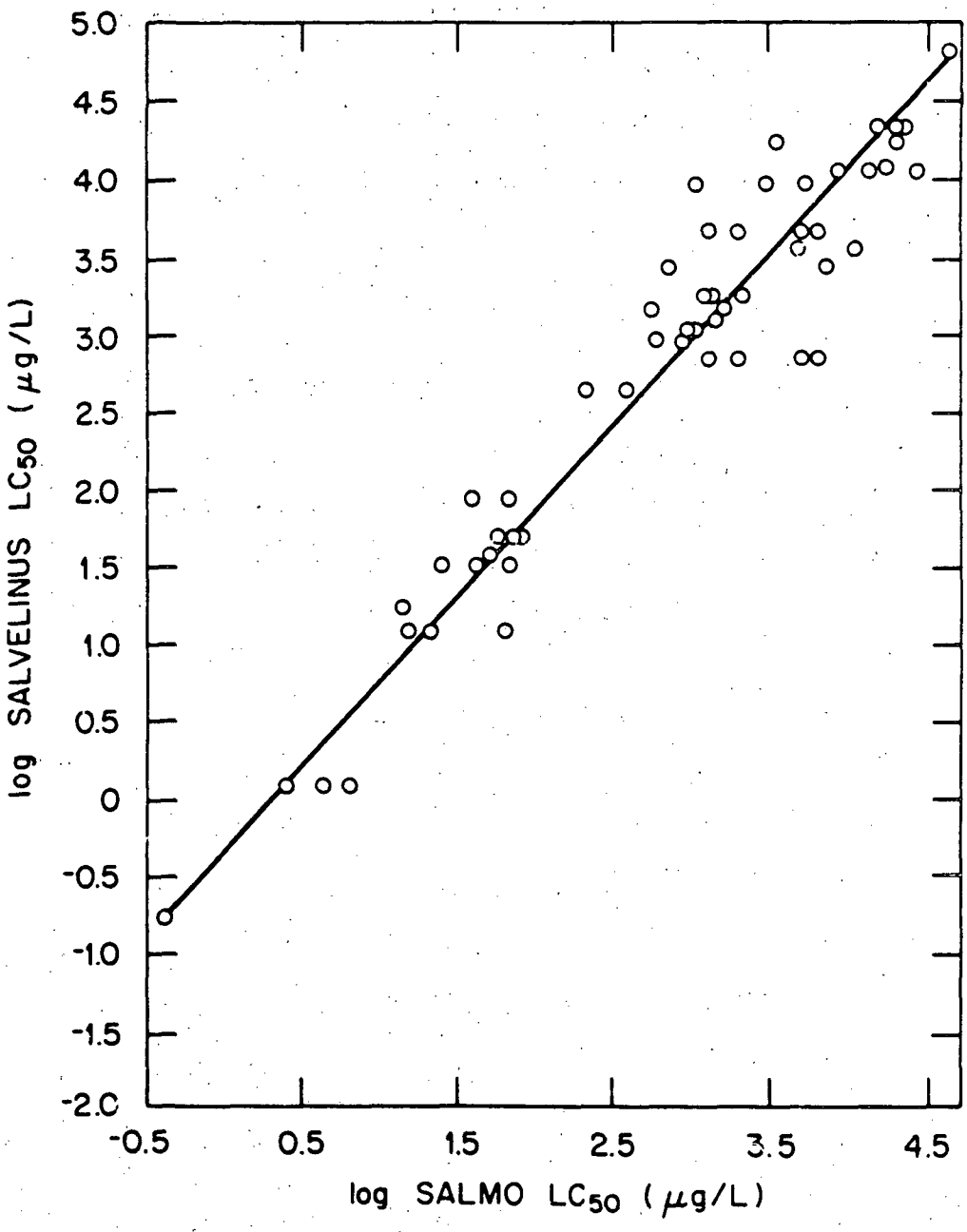

Fig. 4.1. Logarithms of LC_{50} values for Salvelinus plotted against Salmo. The line is determined by an errors-in-variables regression; the parameters are presented in Table 4.1.

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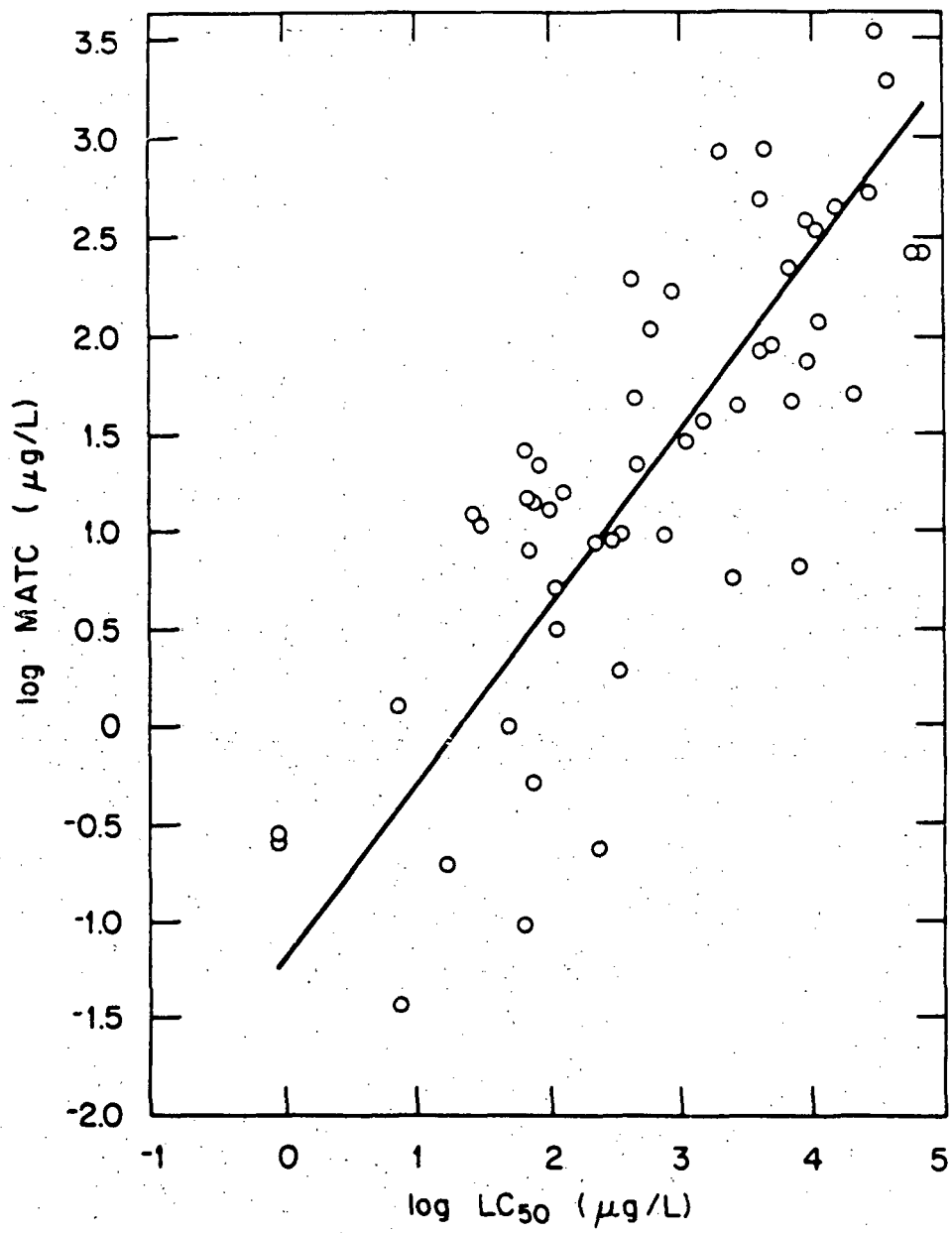

Fig. 4.2. Logarithms of MATC values from life-cycle or partial life-cycle tests plotted against logarithms of 96-h LC_{50} values determined for the same species and chemical in the same laboratory. The line is derived by an errors-in-variables regression; the parameters are presented in line 4 of Table 4.3.

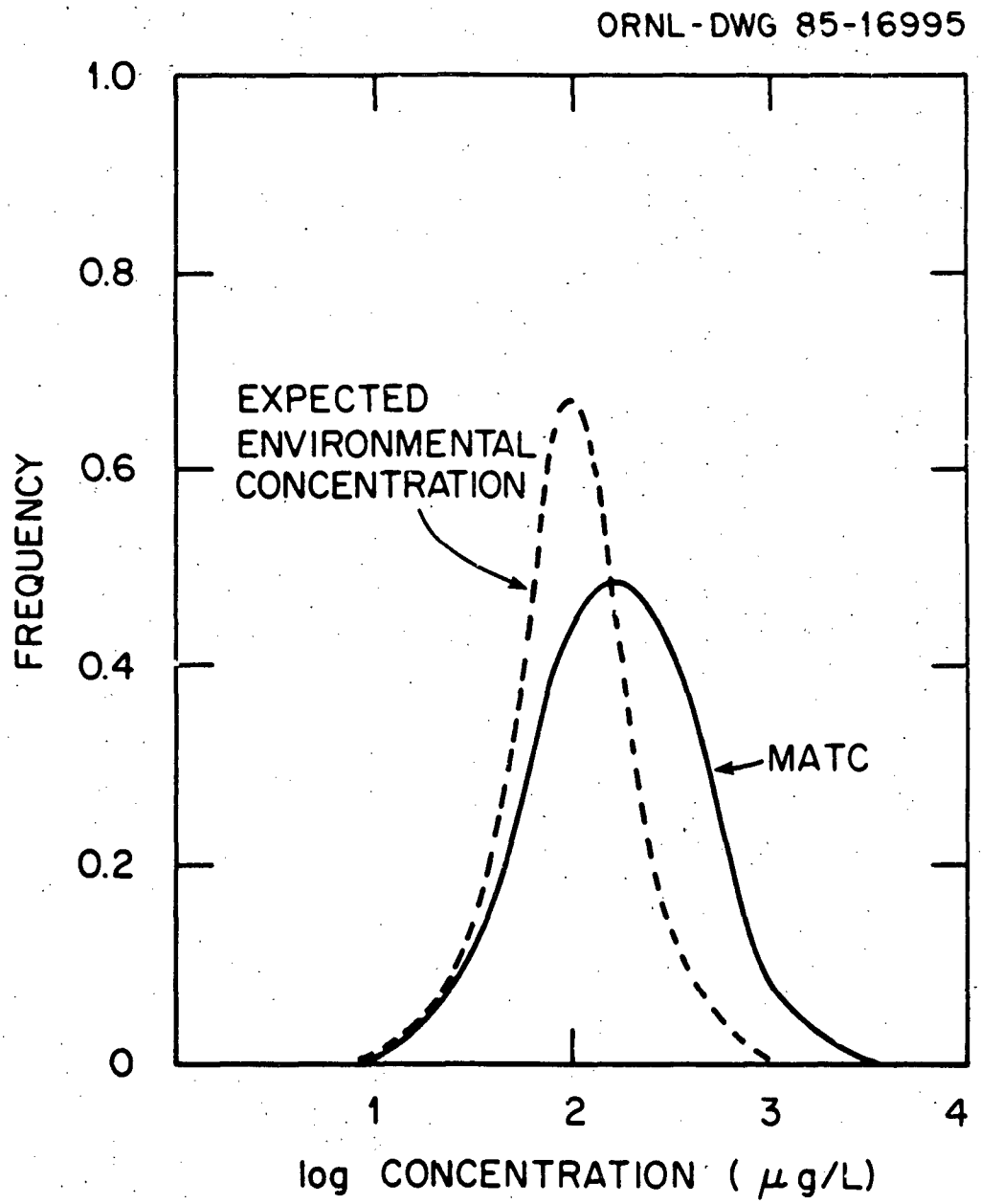

Fig. 4.3. Probability density functions for a predicted Salvelinus MATC (solid line) and an expected environmental concentration (dashed line).

one particular species, life stage, or test duration is an acceptable surrogate for another. When this question is asked, it is invariably discovered that no two tests give identical results, and that the results are not consistently proportional across test chemicals. This discovery can lead to the pessimistic conclusion that toxicity data should not be extrapolated (Tucker and Heagele 1971), which implies that only tested species can be protected. However, since no test is perfectly precise or accurate, even test results have associated uncertainty that can prevent fine discrimination between effective and ineffective exposures. Thus, the relevant question is: Does a particular benchmark, whether derived by testing alone or by testing and extrapolation, provide sufficient accuracy so that an acceptable level of risk can be determined?

4.2 IMPLEMENTATION

AEE consists of five steps: (1) define the end point of the risk assessment (e.g., the probability of causing reductions in brook trout productivity) in terms of a toxicological benchmark (e.g., the probability of exceeding the brook trout MATC); (2) identify the existing datum for the chemical of interest that is most closely related to the end point (e.g., a rainbow trout 96 h at LC_{50}); (3) break the relationship between the datum and the end point into logical steps (e.g., rainbow trout to brook trout and LC_{50} to MATC); (4) calculate the distribution parameters of the end point extrapolated from the datum; and (5) calculate the risk that the expected environmental concentration (EEC) will exceed the end point concentration. Step 1

is dependent on the assessment situation and on the assessor's and decision-maker's conceptualization of environmental values; however, steps 1, 2, and 3 are severely constrained by the state of the science of environmental toxicology as reflected in the available benchmarks and data for the organisms in question (Sect. 3.3).

4.2.1 Risk Calculation

In this method, risk is defined as

$$\text{Risk} = \text{Prob}(\text{EEC} > \text{BC}) \quad , \tag{4.1}$$

where BC is the benchmark concentration that is used as the estimator of the assessment end point. If we assume that the EEC and BC are independent and log-normally distributed, then

$$\text{Risk} = \text{Prob}(\log \text{BC} - \log \text{EEC} < 0) \tag{4.2}$$

$$= \text{Prob}[Z < [0 - (\mu_b - \mu_e)] / (\sigma_b^2 + \sigma_e^2)^{1/2}] \tag{4.3}$$

$$= \Phi_z[(\mu_e - \mu_b) / (\sigma_b^2 + \sigma_e^2)^{1/2}] \quad , \tag{4.4}$$

where (μ_b, σ_b^2) and (μ_e, σ_e^2) are the mean and variance of the log BC and log EEC, respectively and

$$Z = [(\log \text{BC} - \log \text{EEC}) - (\mu_b - \mu_e)] / (\sigma_b^2 + \sigma_e^2)^{1/2} \quad , \tag{4.5}$$

a standard normal random variable with Φ_z as its cumulative distribution function. If it is assumed that the EEC is constant and certain, then the risk calculation reduces to

$$\text{Risk} = \text{Prob}\{Z<[(\log \text{EEC} - \mu_b) / \sigma_b]\} \tag{4.6}$$

$$= \Phi_z[(\log \text{EEC} - \mu_b) / \sigma_b] \quad . \tag{4.7}$$

Given this definition, risk depends on the definitions of the EEC and BC and their associated uncertainties (i.e., on μ_e, μ_b, σ_e^2, and σ_b^2). For the BC, the mean and variance can be estimated by statistical extrapolation of the toxicity data.

4.2.2 Extrapolation

The choice of extrapolation model for this method was based on the following characteristics of toxicity data:

1. the observed values X and Y are subject to error of measurement and to inherent variability,
2. X is not a controlled variable (like settings on a thermostat),
3. values assumed by X and Y are open-ended and non-normally distributed.

These characteristics suggest that an ordinary least-squares model would be inappropriate and an errors-in-variables model should be used. Since we can estimate the value of λ, the ratio of the point variances of Y to X, a functional model provides maximum likelihood estimators of the regression parameters.

The estimators of the slope (β) and intercept (α) are

$$b = \{\Sigma y^2 - \lambda\Sigma x^2 + [(\Sigma y^2 - \lambda\Sigma x^2)^2 + 4\lambda(\Sigma xy)^2]^{1/2}\}/2\Sigma xy \text{ and} \quad (4.8)$$

$$a = \bar{y} - b\bar{x} \quad , \quad (4.9)$$

where $x = X_i - \bar{X}$ and $y = Y_i - \bar{Y}$ for $i = 1 \ldots n$. The variance of a single predicted Y-value for a given X-value ($X = X_o$) is given in Mandel (1983) as

$$\mathrm{var}(Y|X_o) = s_e^2\{1 + 1/n + [1 + (b^2/\lambda)]^2[(X_o - \bar{X})^2/\Sigma u^2]\}, \text{ where} \tag{4.10}$$

$$s_e^2 = (b^2\Sigma x^2 - 2b\Sigma xy + \Sigma y^2)/(n - 2), \text{ and}$$

$$\Sigma u^2 = \Sigma x^2 + 2b/\lambda\Sigma xy + (b/\lambda)^2\Sigma y^2.$$

This variance is the appropriate value to use in calculating confidence intervals and risk estimates because the interest in this case is the certainty concerning an individual future observation of Y, such as a toxic threshold, for an untested species-chemical combination. This variance is larger (by a factor of s_e^2) than the variance of the mean of a $Y|X_o$, which is in turn larger than the variance of the regression coefficient--the number provided by most programmable calculators. Confidence intervals calculated from this variance are larger than those that are conventionally reported and are referred to as prediction intervals.

For ease in using this method we reduce the variance formula to

$$\mathrm{var}(Y|X_o) = F_1 + F_2(X_o - \bar{X})^2 \tag{4.11}$$

and provide values for F_1 and F_2 in the tables.

All of the data used in our extrapolations are log transformed, and the reported variances and prediction intervals are for the transformed values. The log transformation was used to increase the homogeneity of the variances and the linearity of the relationships.

4.2.3 Double Extrapolation

In some cases it is necessary to make multiple extrapolations; the most common example is the combination of acute/chronic and taxonomic extrapolations. In those cases the Y from the first extrapolation becomes the "independent" variable in the second extrapolation, and the parameters of the second regression ($z = c + dy$) are determined as for the first, that is substituting y for x and z for y. The total variance for the two extrapolations is

$$\mathrm{Var}(Z|X_0) = \mathrm{var}(Z|Y_0) + d^2\mathrm{var}(Y|X_0) \quad . \qquad (4.12)$$

4.3 AN EXAMPLE: AQUATIC INVERTEBRATES AND FISH

4.3.1 Data Sets

The data set for the taxonomic extrapolations of LC_{50}s is based on an expansion of the Columbia National Fisheries Research Laboratory data set in Johnson and Finley (1980); the expansion was prepared by Mayer and Ellersieck (in press). This is the largest and most taxonomically diverse set of publicly available aquatic toxicity data that is reasonably uniform with respect to test procedures. We have created a more uniform subset of the data by limiting it to tests performed in soft water (except for those organisms such as *Daphnia* that are not tested in soft water), with post-larval fish weighing between 0.4 and 2.0 g, or with invertebrates belonging to the most often-tested life stage. Tests with aged test solutions, results expressed as > or < values, nonstandard temperatures or pHs, or

forms of a chemical other than the most often-tested form were not used. If, after these criteria were applied, there were still replicate LC_{50}s for a combination of species and chemical, one of the replicates was chosen at random. This subset contains 61 species and 327 chemicals.

The data sets for the extrapolations involving chronic effects on fish are presented in Appendices A and B. The chronic fish data are a compilation of published results of life cycle, partial life cycle, and early life-stage tests of freshwater fish. The concentration-response data for hatch of normal larvae, larval survival, early juvenile weight, eggs produced per female, and adult survival (Appendix B) were extracted from the tests listed in Appendix A. In Appendix B replicate results were averaged, and relationships were not used if there was not at least a 25% reduction in performance at the highest concentration, if there was greater than 30% mortality in the controls, or if there was not a significant positive slope to a fitted logit function. Since these studies were designed for calculating MATCs rather than for curve fitting, most of the responses did not pass these lenient criteria. However, they are the only chronic data available for fish and they serve to illustrate the use of benchmarks based on chronic effects levels and population models (Sect. 5).

The invertebrate chronic data are limited to life-cycle tests with Daphnia spp., since there are few good chronic data for any other freshwater invertebrate. Those data are from the 1980 and 1984 EPA ambient water quality criteria support documents and are not reproduced here.

4.3.2 Extrapolation Results

The taxonomic extrapolations of acute data are presented in Table 4.1. The extrapolations were performed between taxa having the next higher taxonomic level in common rather than simply matching all possible species combinations. For example, the extrapolation between the fathead minnow (*Pimephales promelas*) and largemouth bass (*Micropterus salmoides*) constitutes an extrapolation between the Cypriniformes and Perciformes. This system allows extrapolation to species that have rarely or never been tested by assuming that they are represented by tested species that are members of some common higher taxonomic level. The taxonomic hierarchy is based on the concept that greater evolutionary distance implies greater morphological and physiological dissimilarity, which implies greater dissimilarity in response to toxicants. It is the basis for preferring mammals over nonmammals and primates over nonprimate mammals in testing for effects on humans. It will not hold if the traits that determine sensitivity are extremely evolutionarily labile or conservative. The concept has been shown to hold on average for aquatic organisms (Suter et al. 1983, Suter and Vaughan 1984, and LeBlanc 1984).

As shown in Table 4.2, most extrapolations between taxa within the same family (i.e., between congeneric species and between confamilial genera) can be made with fair certainty, but extrapolations between orders of arthropods, classes of chordates or arthropods, and between the phyla Chordata and Arthropoda are highly uncertain. We use the prediction interval rather than the correlation coefficient (r),

Table 4.1. Taxonomic extrapolations [units are log(μg/L)].

Level[a]	Taxon X[b]	Taxon Y[c]	N[d]	Icept[e]	Slope[f]	Xbar[g]	F1[h]	F2[h]	Ybar[i]	G1[j]	G2[j]	P1[k]
SPECIES												
	CUTTHROAT TROUT	RAINBOW TROUT	18	0.04	0.98	2.47	0.24	0.01	2.45	0.25	0.01	0.96
	CUTTHROAT TROUT	ATLANTIC SALMON	6	-0.25	1.00	2.99	0.16	0.01	2.74	0.16	0.01	0.78
	CUTTHROAT TROUT	BROWN TROUT	8	-0.20	1.02	2.42	0.14	0.01	2.26	0.14	0.01	0.74
	RAINBOW TROUT	ATLANTIC SALMON	10	-0.51	1.20	2.61	0.20	0.01	2.62	0.14	0.01	0.87
	RAINBOW TROUT	BROWN TROUT	15	-0.21	1.09	2.16	0.08	0.00	2.15	0.07	0.00	0.56
	ATLANTIC SALMON	BROWN TROUT	7	0.09	1.01	2.53	0.13	0.01	2.65	0.13	0.01	0.70
	BLACK BULLHEAD	CHANNEL CATFISH	12	-0.11	1.00	2.23	0.11	0.00	2.13	0.11	0.00	0.66
	GREEN SUNFISH	BLUEGILL	14	-0.62	1.09	2.39	0.17	0.01	1.99	0.14	0.00	0.80
	D. MAGNA	D. PULEX	9	0.26	0.81	0.68	0.59	0.07	0.81	0.90	0.16	1.51
	G. FASCIATUS	G. LACUSTRIS	11	-0.06	0.84	1.32	0.15	0.01	1.05	0.21	0.03	0.76
GENUS												
	ONCORHYNCHUS	SALMO	56	-0.13	1.02	2.63	0.11	0.00	2.56	0.10	0.00	0.65
	ONCORHYNCHUS	SALVELINUS	13	-0.47	1.09	2.40	0.08	0.00	2.15	0.07	0.00	0.57
	SALMO	SALVELINUS	56	-0.33	1.10	2.86	0.14	0.00	2.82	0.11	0.00	0.73
	CARASSIUS	CYPRINUS	8	-0.47	1.05	3.04	0.09	0.01	2.73	0.08	0.01	0.58
	CARASSIUS	PIMEPHALES	19	-0.27	1.03	2.79	0.17	0.00	2.61	0.16	0.00	0.82
	CYPRINUS	PIMEPHALES	10	0.24	0.93	2.90	0.17	0.01	2.95	0.20	0.01	0.82
	LEPOMIS	MICROPTERUS	30	-0.20	1.05	2.33	0.22	0.00	2.24	0.20	0.00	0.92
	LEPOMIS	POMOXIS	8	-0.01	0.82	1.28	0.23	0.01	1.04	0.34	0.02	0.94
	DAPHNIA	SIMOCEPHALUS	51	0.35	0.92	1.48	0.16	0.00	1.71	0.19	0.00	0.78
	PTERONARCELLA	PTERONARCYS	8	-0.05	1.03	1.34	0.15	0.01	1.33	0.14	0.01	0.75
FAMILY												
	BUFONIDAE	HYLIDAE	6	1.26	0.56	2.34	0.34	0.14	2.58	1.06	1.37	1.14
	CENTRARCHIDAE	PERCIDAE	47	-0.02	0.95	1.96	0.27	0.00	1.85	0.29	0.00	1.01
	CENTRARCHIDAE	CICHLIDAE	6	0.93	0.40	0.90	0.08	0.04	1.29	0.51	1.67	0.56
	PERLIDAE	PTERONARCYIDAE	11	0.21	1.11	0.17	0.40	0.19	0.39	0.32	0.12	1.24
	PERLODIDAE	PTERONARCYIDAE	9	0.54	0.75	1.12	0.22	0.01	1.39	0.39	0.05	0.92
	SALMONIDAE	ESOCIDAE	11	-0.49	1.40	1.05	0.23	0.13	0.99	0.12	0.03	0.94
	PERCIDAE	CICHLIDAE	5	0.15	1.43	1.42	0.33	0.13	2.19	0.16	0.03	1.12
	ASTACIDAE	PALAEMONIDAE	6	0.27	0.54	1.89	1.37	0.05	1.29	4.67	0.55	2.30

Table 4.1. (Continued)

Level[a]	Taxon X[b]	Taxon Y[c]	N[d]	Icept[e]	Slope[f]	Xbar[g]	F1[h]	F2[h]	Ybar[i]	G1[j]	G2[j]	PI[k]
ORDER												
	SALMONIFORMES	CYPRINIFORMES	225	0.90	0.87	2.32	0.45	0.00	2.92	0.59	0.00	1.31
	SALMONIFORMES	SILURIFORMES	203	0.87	0.85	2.35	0.66	0.00	2.86	0.91	0.00	1.59
	SALMONIFORMES	PERCIFORMES	443	0.33	0.94	2.34	0.31	0.00	2.53	0.35	0.00	1.09
	CYPRINIFORMES	SILURIFORMES	111	0.23	0.93	2.59	0.28	0.00	2.63	0.33	0.00	1.04
	CYPRINIFORMES	PERCIFORMES	219	-0.39	0.99	2.66	0.59	0.00	2.24	0.61	0.00	1.51
	SILURIFORMES	PERCIFORMES	190	-0.74	1.08	2.67	0.82	0.00	2.15	0.71	0.00	1.78
	CLADOCERA	OSTRACODA	22	0.79	0.62	1.05	0.96	0.04	1.44	2.53	0.28	1.92
	CLADOCERA	AMPHIPODA	105	0.27	0.91	1.14	0.63	0.00	1.31	0.76	0.00	1.56
	OSTRACODA	ISOPODA	7	-1.10	2.05	1.26	1.23	0.61	1.49	0.29	0.03	2.17
	OSTRACODA	AMPHIPODA	14	-2.74	2.30	1.62	2.07	0.33	0.99	0.39	0.01	2.82
	ISOPODA	AMPHIPODA	20	-0.22	0.45	1.92	0.92	0.04	0.66	4.45	0.87	1.88
	ISOPODA	DECAPODA	5	-2.31	1.85	2.00	4.42	2.09	1.39	1.29	0.18	4.12
	AMPHIPODA	DECAPODA	14	0.65	1.67	0.89	2.73	0.25	2.14	0.98	0.03	3.24
	PLECOPTERA	ODONATA	13	0.60	0.53	0.55	0.61	0.10	0.89	2.16	1.26	1.53
	PLECOPTERA	DIPTERA	18	0.77	2.46	0.18	3.15	1.68	1.22	0.52	0.05	3.48
	SALMONIFORMES	ATHERINIFORMES	6	0.37	0.66	0.17	0.10	0.00	0.48	0.24	0.02	0.63
	CYPRINIFORMES	ATHERINIFORMES	5	0.02	0.74	0.95	0.06	0.00	0.72	0.12	0.01	0.50
	SILURIFORMES	ATHERINIFORMES	5	-0.48	0.85	0.84	0.91	0.09	0.23	1.25	0.17	1.87
	ATHERINIFORMES	PERCIFORMES	10	-0.10	1.03	0.77	0.21	0.01	0.70	0.20	0.01	0.91
	OSTRACODA	DECAPODA	9	-1.05	1.37	1.86	1.34	0.13	1.51	0.71	0.04	2.27
CLASS												
	AMPHIBIA	OSTEICHTHYES	206	-6.97	3.34	2.57	3.84	0.16	1.63	0.34	0.00	3.84
	CRUSTACEA	INSECTA	373	0.01	0.83	1.19	1.33	0.00	0.99	1.94	0.01	2.26
PHYLUM												
	CHORDATA	ARTHROPODA	2103	-0.55	0.77	2.35	1.76	0.00	1.27	2.94	0.00	2.60
SPECIAL												
	FATHEAD MINNOW	CYPRINIFORMES	30	0.26	0.95	2.63	0.19	0.00	2.77	0.21	0.00	0.85
	BLUEGILL	PERCIFORMES	65	0.16	0.95	2.13	0.22	0.00	2.19	0.24	0.00	0.91
	RAINBOW TROUT	SALMONIFORMES	88	-0.11	1.04	2.59	0.17	0.00	2.59	0.16	0.00	0.81
	FATHEAD MINNOW	OSTEICHTHYES	354	-0.30	1.01	2.77	0.45	0.00	2.49	0.44	0.00	1.31
	BLUEGILL	OSTEICHTHYES	500	0.17	0.96	2.52	0.49	0.00	2.60	0.53	0.00	1.37
	RAINBOW TROUT	OSTEICHTHYES	480	0.29	0.99	2.42	0.38	0.00	2.67	0.39	0.00	1.20

[a]Taxonomic level at which the extrapolation is made.
[b]Taxon from which values of the independent variable are drawn.
[c]Taxon from which values of the dependent variable are drawn.
[d]Number of points in the regression.
[e]Estimated intercept (a).
[f]Estimated slope (b).
[g]Mean of X.
[h]Factors used in calculating the variance of an individual Y.
[i]Mean of Y.
[j]Factors used with the inverse regressions to calculate the variance of an individual X.
[k]The 95% prediction interval on the point XBAR is YBAR + PI.

Table 4.2. Summary of aquatic taxonomic extrapolations

Taxonomic level	n[a]	n Weighted mean 95% prediction interval
Species		
Fish	8	0.76
Arthropods	2	1.10
Genera		
Fish	8	0.74
Arthropods	2	0.78
Families		
Fish	4	0.97
Arthropods	3	1.37
Amphibians	1	1.14
Orders		
Fish	10	1.35
Arthropods	10	2.06
Classes		
Chordates	1	3.84
Arthropods	1	2.26
Phyla	1	2.60

[a]Number of pairs of taxa at that taxonomic level.

because we are interested in the precision of the estimate rather than the ability of the model to "explain" the data. In addition, the r values for this regression model are considerably higher than those for ordinary least squares; therefore they could not be used for comparison with other results.

Because these extrapolations are made between identical benchmarks (96-h LC_{50}s) determined at a single laboratory, λ was set to 1. This assumption was tested by pair-wise comparisons of the 95% confidence intervals reported by Johnson and Finley (1980). Average ratios of confidence interval widths on LC_{50}s for pairs of taxa at each taxonomic level were all found to be very close to 1.

Table 4.1 can be used to extrapolate between taxon X and taxon Y, as previously explained (Sect. 4.2.1). Since we are using an errors-in-variables model, the inverse regression (X from Y) can be calculated as $x = (y - a)/b$. Variance for this inverse regression (Mandel 1983) reduces to $\mathrm{var}\,(X|Y_0) = G_1 + G_2(Y_0 - \bar{Y})^2$, with G_1 and G_2 provided in the table.

Four special taxonomic extrapolations are presented at the end of Table 4.1. These are extrapolations between the three most common test species of fish [fathead minnow, bluegill (Lepomis macrochirus), and rainbow trout], and both the Order to which they belong and the entire Class Osteichthyes. The extrapolations are useful for assessments in which members of an entire higher taxon are to be protected or for which an appropriate lower-level extrapolation is not available. This type of extrapolation also serves to indicate how well these species serve as representatives for the taxa as a whole. The measure of

predictive power provided by the prediction intervals for these equations is a better guide to the selection of test species than relative sensitivity, importance of the species, or its similarity to currently used species (Suter and Vaughan 1984). By this criterion, the three fish species are about equally good representatives, but the rainbow trout is slightly better.

A variety of acute-chronic extrapolations are presented in Table 4.3 for different chronic benchmarks and subsets of the data. The values of λ for these extrapolations are estimated from the ratios of the mean variances of benchmarks from replicate tests in Appendix A. The choice of extrapolation depends on the input data and on the end point desired, that is, MATC vs effects levels, all chronics vs life-cycle, or specific categories vs all chemicals. Clearly the extrapolations presented are only a fraction of those that could be created from different subsets of data.

The first extrapolation in Table 4.3 relates fathead minnow MATCs to those of all other freshwater Osteichthyes. Although the predicted Y for this type of extrapolation is meaningless (there is no mean fish), this relationship can be used to estimate the risk that the MATC (for some species of fish) will be exceeded, given a fathead minnow MATC and an expected environmental concentration. The prediction interval for this extrapolation is similar to that for the analogous extrapolation in Table 4.1 between fathead minnow LC_{50}s and those for all other Osteichthyes; however, the interval is slightly smaller, possibly due to the smaller array of species that have been used in chronic tests. One might expect that there would be greater variance

Table 4.3. Acute-Chronic Extrapolations. Units are log(μg/L).

OBS[a]	X[b]	Y[c]	Condition[d]	Lamda[e]	N[f]	Icept[g]	Slope[h]	Xbar[i]	F1[j]	F2[j]	Ybar[k]	PI[l]
1	FM MATC	All Fish MATC	All	1.0	52	-0.04	0.79	1.80	0.33	0.01	1.37	1.13
2	FM MATC	Salmoniformes MATC	All	1.0	27	-0.10	0.80	1.87	0.39	0.02	1.38	1.22
3	FM MATC	Perciformes MATC	All	1.0	8	-0.26	0.93	1.97	0.45	0.11	1.56	1.31
4	LC_{50}	MATC	Type = LC	1.5	55	-1.16	0.90	2.75	0.51	0.01	1.31	1.40
5	LC_{50}	MATC	All	1.5	98	-1.51	1.07	3.13	0.59	0.00	1.85	1.50
6	LC_{50}	MATC	Class = N	1.5	23	-0.42	0.90	3.87	0.09	0.00	3.05	0.59
7	LC_{50}	MATC	Class = M	1.5	25	-0.70	0.73	3.25	0.37	0.02	1.68	1.19
8	LC_{50}	EC_{25} Mort1	Type = LC	1.0	15	-1.46	0.96	2.71	0.53	0.03	1.14	1.43
9	LC_{50}	EC_{25} Mort2	All	1.0	30	-1.69	1.21	2.98	1.10	0.03	1.91	2.06
10	LC_{50}	EC_{25} Mort2	Species = FM TYPE = ELS	1.0	16	-2.33	1.33	3.35	1.52	0.06	2.12	2.42
11	LC_{50}	EC_{25} Hatch	All	1.0	13	-2.24	1.34	3.40	1.46	0.06	2.33	2.37
12	LC_{50}	EC_{25} Eggs	Type = LC	1.0	26	-2.43	1.19	2.83	0.75	0.04	0.94	1.70
13	LC_{50}	EC_{25} Weight	All	1.0	37	-2.03	1.24	3.40	0.77	0.01	2.18	1.72
14	LC_{50}	EC_{25} Weight	Species = FM TYPE = ELS	1.0	24	-1.72	1.18	3.70	0.84	0.02	2.66	1.79
15	LC_{50}	EC_{25} Wt of Juveniles/Egg	All	1.0	14	-1.88	1.10	3.20	1.49	0.04	1.66	2.39
16	LC_{50}	EC_{25} Wt of Juveniles/Egg	Species = FM TYPE = ELS	1.0	11	-2.00	1.16	3.18	1.60	0.05	1.68	2.48
17	LC_{50}	Daphnia MATC	All	1.3	57	-1.30	1.11	2.73	0.48	0.01	1.72	1.35
18	LC_{50}	Daphnia MATC	Class = M	1.3	27	-1.08	0.96	2.44	0.63	0.02	1.26	1.56

[a]OBS = Observation number.
[b]Independent variable. FM MATC = MATC values for fathead minnows. LC_{50} = LC_{50} values for the species and chemical corresponding to those of the dependent variable.
[c]Dependent variable. All Fish MATC = values for all freshwater fish other than fathead minnows. Salmoniformes MATC = values for members of the order Salmoniformes. Perciformes MATC = values for members of the order Perciformes. MATC = Values for fish. EC_{25} Mort1 = a concentration estimated to cause a 25% increase in mortality of parental fish. EC_{25} Mort2 = a concentration estimated to cause a 25% increase in mortality of larval fish. EC_{25} Hatch = a concentration estimated to cause a 25% decrease in normal hatches of fish eggs. EC_{25} Eggs = a concentration estimated to cause a 25% decrease in the number of eggs produced per female fish. EC25 Weight = a concentration estimated to cause a 25% decrease in the weight of fish at the end of the larval stage. Daphnia MATC = values for members of the genus Daphnia.
[d]Subset of the data used in the extrapolation. All = all pairs of X and Y points are used. Type = types of tests included: LC = life cycle or partial life cycle, ELS = early life stage. Species = Species of test organism: FM = fathead minnow. Class = Chemical class: M = metal, N = narcotic.
[e]Ratio of the variances of the Y and X variables.
[f]Number of points in the regression.
[g]Estimated intercept (a).
[h]Estimated slope (b).
[i]Mean of X.
[j]Factors used in calculating the variance of an individual Y.
[k]Mean of Y.
[l]The 95% prediction interval at the point XBAR is YBAR ± PI.

among species in chronic toxicity than in acute toxicity because of the greater variety of responses potentially involved, particularly in life-cycle tests. However, this analysis does not support that idea, and the substitution of larval mortality or growth for life-cycle responses in chronic tests suggests that acute and threshold chronic responses may be equally simple; therefore the true variances may be equal. Extrapolations 2 and 3 are analogous but extrapolate to specific orders. There is no gain in precision by this increased specificity. All extrapolations have negative intercepts and slopes less than 1, indicating that fathead minnows are a little less sensitive than most other fish in chronic tests.

The next four extrapolations in Table 4.3 predict MATCs from LC_{50}s for the same species. Extrapolations 4 and 5 include all species and chemical types, but 4 includes only life-cycle tests (which are somewhat more reliable than early life-stage tests), whereas 5 includes all MATCs for which there is a corresponding LC_{50}. Extrapolations 6 and 7 include all species and test types but are limited to narcotics and metals, respectively. The chemicals identified as narcotics belong to the classes of chemicals identified as such by Veith et al. (1983) and Call et al. (1985). The particularly narrow prediction interval for this extrapolation reflects the precision of the quantitative structure-activity relationships (QSARs) for narcotics presented in those reports, thus reinforcing the idea that the action of these chemicals is highly predictable. In fact, the fathead minnow LC_{50}s and MATCs generated by the QSARs in these reports, or by any other QSAR with precision as good as that of replicate tests, could be used in the

extrapolations between fathead minnow benchmarks and those for other taxa, if there is reasonable certainty that the chemical in question belongs to the correct category. QSARs can be more precise than individual tests because they summarize large amounts of information, and because chemical measurements are generally much more precise than biological tests (Craig and Enslein 1981).

The next nine extrapolations (8-16) constitute an examination of the predictability of particular levels of chronic effects (LC_{25}s and EC_{25}s) from acute LC_{50}s for the same species. Mort1 is mortality of parental fish; Mort2 is mortality from hatching to the early juvenile stage; Hatch is the proportion of eggs failing to successfully hatch; Eggs is the reduction in the number of eggs produced per female relative to controls; Weight is the proportional reduction in the average weight of early juveniles relative to controls; and Wt of Juveniles/Egg is the proportional reduction in the weight of early juveniles per initial egg. We used a 25% reduction in performance in this exercise largely as a matter of convenience in dealing with this data set rather than as a proposed assessment end point, but 25% could be defended as a level of effect that would be barely detectable in the field. These extrapolations are more imprecise than those from acute LC_{50}s to MATCs. This result is surprising since we expected that an acute median lethal concentration would be a better predictor of a chronic quartile lethal concentration than of a hypothesis-testing-derived benchmark that is not indicative of any particular type or level of effect. Limitation of the data set to only early life-stage tests with fathead minnows does not reduce the uncertainty. The most obvious

explanation is that the chronic LC_{25}s and EC_{25}s contain much extraneous variance because of the poor data from which they were derived. Nearly all of the chronic concentration-response data would fail to pass conventional requirements for calculating acute LC_{50}s and EC_{50}s because of the lack of partial kills, lack of effects levels of 50% or greater, or high control mortality. In addition, many of the chronic results show apparent hormesis at low concentrations, which complicates curve fitting.

The last two extrapolations in Table 4.3 are for predicting life-cycle MATCs for Daphnia from 48-h LC_{50}s, first for all chemicals and then for metals only. These extrapolations have about the same uncertainty as the corresponding LC_{50} to MATC extrapolations for fish (Nos. 4 and 7 in Table 4.3). These LC_{50} to MATC extrapolations for fish and Daphnia have about the same average level of uncertainty as the extrapolations of LC_{50}s between families of arthropods or orders of fish (Table 4.2).

One potential source of bias in these extrapolations is the fact that investigators will sometimes report results as being greater than or less than some value because the highest or lowest concentration tested was not high or low enough to allow the benchmark to be determined. Since the true value of the benchmark is unknown, these results cannot be used in the extrapolations. However, since these are likely to be chemicals with extreme application factors (MATC/LC_{50} values), they would presumably increase the variance in the extrapolations if their true values were known and included. In addition, there may be a bias in the centroids because there are more

< than > values for MATCs in the data set (17 vs. 6, - App. A). However, this does not appear to be a significant problem since all but one of the > or < estimates of the MATC fall within the 95% PI for extrapolation 5, Table 4.3. In addition, an examination of these studies indicates that the failure to show a statistically significant effect at the highest concentration tested is due primarily to high variance in the test data rather than extremely low chronic toxicities. These observations suggest that the true application factors for these chemicals may not be extremely high or low.

4.3.3 A Demonstration

As an example of the use of these extrapolations, consider the estimation of the risk of exceeding the threshold for chronic effects on brook trout beginning with a rainbow trout LC_{50} of 5300 µg/L for the chemical of concern. Substituting the log of that LC_{50} into the *Salmo*-*Salvelinus* extrapolation (Table 4.1) gives a log brook trout LC_{50} of 3.77; using Eq. (4.11), the variance is 0.14 (the second term of the variance equation, $F2(X_o - \bar{X})^2$, is trivial in this case). Substituting 3.77 into extrapolation 4, (Table 4.3), gives an estimate of 2.22 for the log brook trout life-cycle MATC, with a variance for this extrapolation of 0.53. Using Eq. (4-12), the total variance for the double extrapolation is 0.14 + (0.81 x 0.53) = 0.57.

If the log of the expected environmental concentration (EEC) is 2.0 with a variance of 0.5, then the probability that a realization of the brook trout MATC is less than a realization of the EEC is determined from Eq. (4.4), by calculating

$$(2.0 - 2.22) / (0.57 + 0.5)^{1/2} = -0.21 \quad .$$

The cumulative probability for this Z value (obtained from a Z table) is 0.42. Thus, the risk that the threshold for chronic effects on brook trout would be exceeded is 0.42, or we are 58% certain that chronic effects would not occur.

4.4 RISK WITHOUT REGRESSION

In a few cases the assessor will have in hand the benchmark that corresponds to his assessment end point; for example, he is interested in chronic effects on rainbow trout and he has a rainbow trout MATC for the chemical of concern. In that case uncertainty (as a result of the variance between replicate tests) must be accounted for, because the assessor will be uncertain as to the representativeness of the sample of fish used in the test and the biases introduced by variation in procedures and conditions. This variance is not accounted for separately when regressions are used for extrapolation, because it contributes to the total uncertainty in the regression estimates.

Pooled variances for particular test types and taxa are presented in Table 4.4. These are averages of the variances of replicate benchmark values, weighted by the degrees of freedom for each set of replicate tests. The sets are drawn from Appendix A and the EPA ambient water quality criteria support documents. Since we have determined the variances to be homogeneous, this pooled variance can be applied to unreplicated data. If we assume that an individually measured toxicological benchmark is the best estimate of the mean of such benchmarks, then that benchmark and the appropriate pooled variance can be used to estimate the risk that the benchmark will be exceeded by a particular distribution of environmental concentrations (Sect. 4.2).

Table 4.4. Pooled variances of log LC_{50}, EC_{50}, and MATC values from replicate tests

Taxon	Benchmark	n[a]	Pooled variance[b]
Osteichthyes	LC_{50}	37/333	0.018
	MATC	15/66	0.22
Daphnia	EC_{50}	11/81	0.15
	MATC	10/33	0.17

[a]Number of species-chemical combinations/total number of tests.

[b]Mean variance of log values weighted by the degrees of freedom.

If in our example the rainbow trout MATC for the chemical of interest is 20 µg/L, then the mean and variance of the log MATC are 1.3 (log 20) and 0.22, respectively. If the environmental concentration is known with certainty to be 10 µg/L, then the cumulative Z value calculated from Eq. (4.7) is -0.64; the probability (risk) that this concentration is higher than the MATC is 0.26. In other words, we are 74% certain that the environmental concentration will not exceed the rainbow trout MATC.

We have limited ourselves to empirically derived estimates of variance in this section, thereby implicitly assuming that the variance in response between the laboratory and the field is no greater than the variance between one laboratory and the next. The assessor who does not believe that the toxicological benchmark adequately represents his assessment end point may readily incorporate that subjective uncertainty by adding an increment of variance before calculating the risk. It is important to clearly document such judgments, including who made them and on what basis, and to separate the judgment from the calculation of end point values and risks so as to avoid the temptation to fiddle with the conclusion.

4.5 COMPARISON OF METHODS

We examine here the efficacy of AEE by comparing its ability to predict the MATC for particular fish species from a fathead minnow LC_{50}, with the ability of an untransformed fathead minnow MATC, a fathead minnow MATC with an application factor, and LC_{50}s with acute/chronic correction factors to predict the MATC for that species.

Although the double extrapolation used as an example of AEE is not intended to be used if a measured MATC is available (one would use extrapolations from the fathead minnow MATC to MATCs for the taxa of interest), it does provide an instructive comparison of the predictive power of AEE using a double extrapolation to that of the quotient method and the quotient method with factors.

The results of this comparison are presented in Table 4.5. All of the numbers in the table are derived from data in Appendix A. The measured fathead minnow MATC is in error by at least a factor of 2 in 71% of the cases and by a factor of 10 in 10% of the cases. The application factor MATC [(true LC_{50}/FM LC_{50}) x FM MATC] is in error by a factor of 2 in 57% of the cases and by a factor of 10 in 19% of the cases. The extrapolation MATC is in error by a factor of 2 in 71% of the cases and by a factor of 10 in 19% of the cases. In pair-wise comparisons of the methods, the extrapolated MATC was closer to the true MATC than the fathead minnow MATC in 44% of the cases. The extrapolation MATC was closer than the application factor MATC in 43% of the cases. Thus, the use of AEE with acute fathead minnow data is approximately as accurate in predicting the chronic toxicity to a particular species (other than the fathead minnow) as is fathead minnow chronic data, with or without an application factor.

The use of LC_{50}s with the most common acute/chronic correction factors (1/20 and 1/100) gives somewhat worse results. When these correction factors are applied to the fathead minnow LC_{50}s, the 1/20 factor fails to predict the true MATC within a factor of 2 in 80% of the cases and within a factor of 10 in 39% of the cases; the 1/100

Table 4.5. Comparison of methods for estimating the MATC for a species other than fathead minnow (all values are µg/L)

Chemical	Species	FM LC_{50}[a]	True LC_{50}[b]	True MATC[c]	FM MATC[d]	AF MATC[e]	Extrapolated MATC[f]
Arsenic	Flagfish	14,200	14,400	2962	3026	3251	62.7[h]
Atrazine	Bluegill	15,000	6700	218	430[g]	192	306
	Brook trout	15,000	4900	88	430[g]	140	338[g]
Cadmium	Bluegill	6000	21100	50	46	162[g]	56
	Brook trout	6000		2.4	46[h]		54[h]
	Flagfish	6000	2500	5.3	46[g]	19[g]	23[g]
	Walleye	6000		15	46[g]		56[g]
	Channel catfish	6000		14	46[g]		112[g]
	White sucker	6000		7.1	46[g]		138[h]
	Small mouth bass	6000		7.4	46[g]		56[g]
	Northern pike	6000		7.4	46[g]		54[g]
	Lake trout	6000		7.4	46[g]		54[g]
	Coho salmon	6000		7.2	46[g]		54[g]
	Brown trout	6000		6.7	46[g]		54[g]
Chromium	Brook trout	36,900	59,000	265	1987[g]	3177[g]	255
	Rainbow trout	36,900	69,000	265	1987[g]	3715[h]	255
	Bluegill	36,900		765	1987[g]		214
	Channel catfish	36,900		214	1987[g]		389
	Lake trout	36,900		143	1987[h]		255
	Northern pike	36,900		720	1987[g]		255[g]
	White sucker	36,900		395	1987[g]		498
Copper	Bluegill	253	1100	29	25	109[g]	5.6[g]
	Bluntnose minnow	253	230	8.8	25[g]	23[g]	14.7
	Brook trout	253	100	13	25	10	3.64[g]
	Brown trout	253		32	25		3.64[g]
	Lake trout	253		31	25		3.64[g]
	Northern pike	253		60	25[g]		3.64[h]
	White sucker	253		21	25		14.7
	Channel catfish	253		15	25		12.7
	Walleye	253		17	25		5.6[g]
	Rainbow trout	253	80	20	25	7.9[g]	3.64[g]
Hexachloro-cyclohexane	Bluegill	69	30	10.7	14.6	6.3	1.02[h]
	Brook trout	69	26	12.1	14.6	5.5[g]	0.44[h]
Malathion	Bluegill	10,500	110	5.2	341[h]	3.6	210[h]
	Flagfish	10,500	349	9.7	341[h]	11.3	49[g]
Methyl mercury	Brook trout	65	75	0.52	0.09[g]	0.10[g]	0.41
	Flagfish	65	240	0.2	0.09[g]	0.33	0.87[g]
Toxaphene	Channel catfish	7.2	16.5	0.20	0.037[g]	0.085[g]	0.38
Zinc	Brook trout	2349	2000	852	88[g]	75[h]	24[h]
	Rainbow trout	2349	430	191	88[g]	16[h]	24[g]
	Flagfish	2349	1500	36	88[g]	56	14[g]

[a]Measured fathead minnow LC_{50}; only LC_{50}s from the same study as the FM MATC determination are used.
[b]Measured LC_{50}s for the listed species; only LC_{50}s from the same study as the MATC determination are used.
[c]The measured MATC for the listed species. Life-cycle MATCs are preferred over early life-stage MATCs, otherwise the geometric mean of replicate MATCs is used.
[d]A measured MATC for fathead minnows; replicates are treated as in note (c).
[e](True LC_{50}/FM LC_{50}) x FM MATC.
[f]MATC calculated from a fathead minnow LC_{50} using taxonomic and acute/chronic extrapolations.
[g]Estimates that differ from the true MATC by a factor of 2 or greater.
[h]Estimates that differ from the true MATC by a factor of 10 or greater.

factor fails to predict within a factor of 2 in 76% of cases and within a factor of 10 in 29% of cases. When applied to the true LC_{50}, the 1/20 factor fails to predict the true MATC within a factor of 2 in 81% of the cases and within a factor of 10 in 24% of the cases; the 1/100 factor fails to predict within a factor of 2 in 86% of cases and within a factor of 10 in 38% of cases. These factors and LC_{50}s are poorer predictors of MATCs than the methods previously discussed, and neither correction factor does significantly better than the other in this exercise.

AEE has the advantage over the other methods of indicating how inaccurate it is likely to be. In this exercise the 95% prediction intervals (PIs) for the extrapolated MATCs includes the true MATC in all but one of the 41 cases; therefore, using the lower 95% PIs as standards would have prevented exceeding the true MATC in 98% of the cases. This result suggests the reasonableness of the variance terms used in this version of the method.

While this exercise does not constitute a validation of AEE, it does indicate that it is a good predictive tool relative to methods that are currently used. It also demonstrates that all of the methods have large associated errors; therefore, it is important to explicitly account for uncertainty in predictions, as is done with AEE.

4.6 DISCUSSION

The chief advantage of the analysis of extrapolation error method is that it provides an objective, quantitative estimate of risk without departing from the generally accepted practice of defining assessment

end points in terms of toxicological benchmarks. Compared with the quotient method, the extrapolation error method has the advantages of making assumptions concerning the relationship of the data and the end point explicit, treating the relationship as a set of quantitative extrapolations, estimating the uncertainty in the relationship, and producing an estimate of risk based on estimates of the end point and of the associated uncertainty. If the data available for an assessment are not from the needed test type and species, the quotient method requires that one use the data available and pretend that they are appropriate, use correction factors without considering the associated uncertainty, or aggregate the uncertainty factors with the correction factors and treat the assessment deterministically. Compared with population and ecosystem models (Sects. 5 and 6), AEE has the advantage of using as its end point the toxicological benchmarks that constitute the end points for all existing regulatory assessment schemes and environmental quality criteria.

The limitations of AEE are that the method (1) is limited to end points that can correspond to standard toxicological benchmarks; consequently, unless subjective corrections and uncertainties are used, it cannot address effects on entities or processes that occur on spatial or temporal scales beyond the range of toxicity testing; (2) is computationally difficult relative to the quotient method and conceptually opaque to decision-makers who lack statistical training; and (3) assumes that existing data sets are representative of future toxicity data. The problem of the representativeness of existing data sets is characteristic of any method that attempts to extrapolate

beyond the existing data. However, it is important to pay close attention to the potential biases in available data sets and to be aware of which sources of variability (e.g., water chemistry, interlaboratory variability, or different strains of the test species) are represented in the data set and which are implicit in the assessment (e.g., should data from laboratories of unknown reliability be used, and should the results of the assessment apply to a variety of sites). In some cases, the extrapolations can be inappropriately precise as the result of using a highly standardized data set. For example, studies of the acute effects of narcotic chemicals in Lake Superior water on the Duluth population of fathead minnows (Veith et al. 1983) are used in QSARs that generate predicted LC_{50}s that are more precise than replicate tests in different laboratories using different waters and fish populations. More often, there will be sources of variance in the data sets that are extraneous to the assessment but cannot be avoided because a more appropriate data set is not available. In those cases the extraneous variance is simply part of the uncertainty associated with performing assessments with limited knowledge, which is similar to the uncertainty concerning future emission rates or dilution volumes.

While the AEE method was developed to provide estimates of risk, it has a variety of other potential uses. The regression and error propagation portions can be used to estimate toxic effects for population and ecosystem models and to generate the parameter distributions used in Monte Carlo simulations. This use is described in Sect. 5 and 6. Another potential use is in designing testing

programs. Decisions about the need for additional testing of a chemical could be made on the basis of the expected reduction in the total uncertainty concerning the true value of the end point, the expected reduction in risk, or the probability that the test will cause a change in a regulatory decision. In addition to making decisions for testing individual chemicals, AEE could be used to elucidate the implications of the decision rules in tiered testing schemes or to devise new decision rules.

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5. EXTRAPOLATION OF POPULATION RESPONSES

L. W. Barnthouse, G. W. Suter II, A. E. Rosen,
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As noted in Section 1 of this report, the end points of ultimate interest in ecological risk assessment are effects of long-term exposures on the persistence, abundance, and/or production of populations. In contrast, the data available for assessing ecological risks of toxic contaminants are nearly always restricted to effects of contaminants on individual organisms. If assessments of ecological effects of toxic contaminants are ever to reach the same level of sophistication as assessments of nontoxicological stresses, such as fishing and power plants, it will be necessary to develop analytical techniques for extrapolating from individual-level responses to population-level responses.

Many of the components necessary for this task already exist. Section 4.1 of this report showed that statistical relationships (1) among 96-h LC_{50}s for different fish taxa and (2) between 96-h LC_{50}s and maximum acceptable toxicant concentrations (MATCs) can be used to extrapolate chronic effects thresholds for untested fish species from acute LC_{50}s for tested species. The literature on fish population modeling contains a variety of techniques for estimating population-level responses to age-specific changes in mortality, fecundity, and growth.

In this section we describe a method of generating life-stage-specific concentration-response functions for either tested or untested fish species. We demonstrate the linking of the estimated

concentration-response functions, together with their associated uncertainties, to simple fish population models that have proved useful in other problems involving anthropogenic stresses on fish populations. Our objectives are, first, to quantify the uncertainty resulting from extrapolation from bioassay results to population responses, and second, to express effects of toxic contaminants in common units with effects of other anthropogenic stresses on fish populations.

5.1 FORMULATION OF CONCENTRATION-RESPONSE MODEL

The concentration-response function used in this study is the logistic model

$$P = (e^{\alpha+\beta X})/(1 + e^{\alpha+\beta X}) \quad , \tag{5.1}$$

where

P = fractional response of the exposed population,

X = exposure concentration, and

α,β = fitted parameters with no biological interpretation.

When fitted to concentration-response data, the logistic function has a sigmoid shape similar to the probit model. Because ecological risk assessment does not involve extrapolation to extremely low doses, it does not matter which model is used. The logistic model has convenient properties that can be seen by reformulating it as

$$X_p = [\ln[P/(1 - P)] - \alpha]/\beta \quad , \tag{5.2}$$

where

X_p = concentration producing a fractional response equal to P.

If α and β are specified, then X_p can be directly calculated from Eq. (5.2). Alternatively, if X_p and β are specified, then α can be calculated from

$$\alpha = \ln[P/(1 - P) - \beta X_p] \quad . \tag{5.3}$$

In other words, the complete concentration-response function can be obtained by specifying either α and β or β and the concentration associated with a single response level (e.g., the LC_{25}). The parameter β specifies the curvature of the logistic function and is independent of the position of the curve on the concentration axis. If two logistic functions have different LC_{25}s but the same curvature, their β parameters will be equal.

If a chronic concentration-response data set is available for a species and contaminant of interest, then a logistic concentration-response function and associated confidence bands can be obtained by fitting the logistic model to the data. If, however, directly applicable data are not available, a function and confidence bands can be obtained using extrapolated values of β and LC_{25}. The following subsections describe methods for calculating concentration-response functions and confidence bands directly from data and by extrapolation.

5.2 FITTING THE LOGISTIC MODEL TO CONCENTRATION-RESPONSE DATA

Concentration-response data sets can be fitted to Eq. (5.1) using nonlinear least squares regression. This section describes the procedure for fitting chronic concentration-response data sets from

whole life cycle experiments to the logistic model. Although a variety of test end points can be used (e.g., growth or fecundity), only the method used to model mortality is described here. The data required are (1) the number of replicates tested at each concentration (including the controls), (2) the number of organisms in each replicate, and (3) the number of organisms dying in each replicate (including the controls). As in the extrapolation models described in Section 4, test concentrations are entered as $\log_{10}$(concentration in μg/L) so that the units represent orders of magnitudes of concentrations. The fraction of organisms dying in each replicate is corrected for control mortality using Abbott's formula (Abbott 1925), as described in Section 4. We use the SAS procedure NLIN to produce estimates of α and β and a variance-covariance matrix for α and β.

Uncertainty concerning the shape and position of the concentration-response function, as reflected in the variances and covariances of α and β, can be represented graphically as a confidence band surrounding the fitted function, as illustrated in Fig. 5.1. Brand et al. (1973) described a procedure for calculating confidence band functions for the logistic model from the elements of the variance-covariance matrix. Alternatively, confidence bands can be calculated numerically by iterative random sampling (i.e., Monte Carlo simulation) from the bivariate normal distribution defined by the variance-covariance matrix. Published data from full life cycle tests for fish are commonly broken out by life stage (e.g., eggs, larvae, and juveniles). To perform a population-level assessment using these data,

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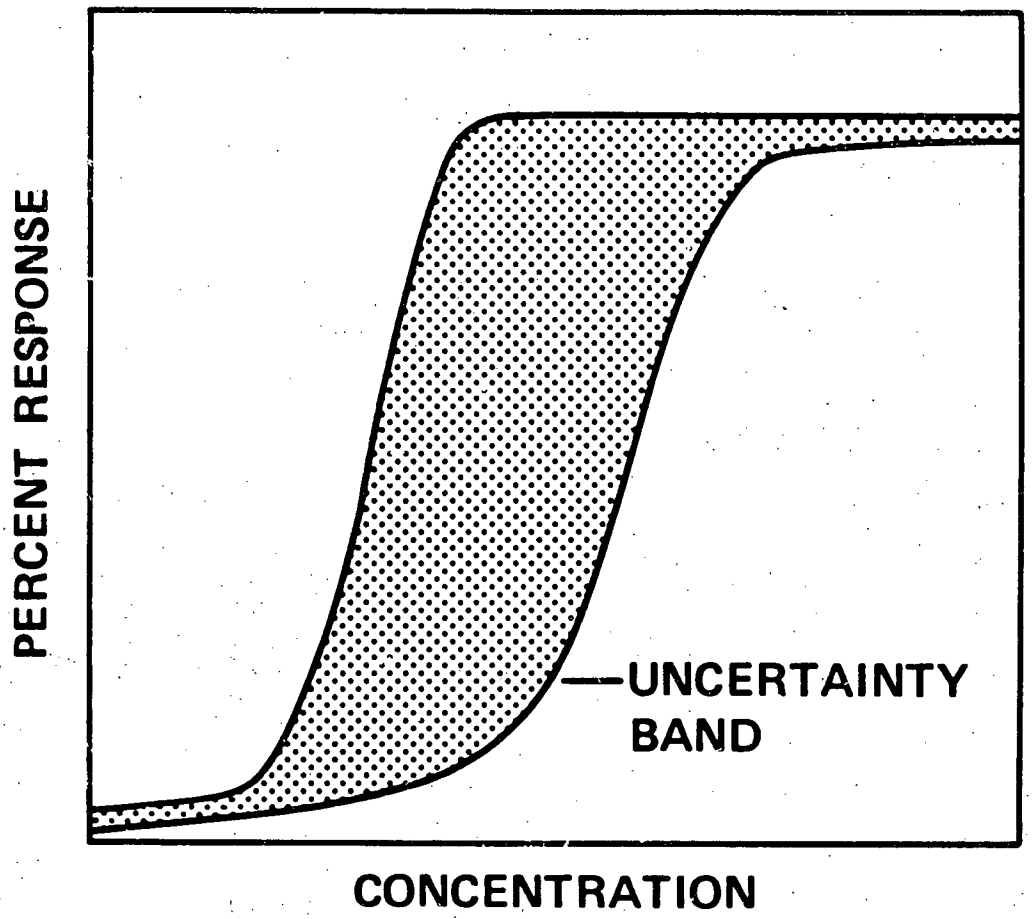

Fig. 5.1. Uncertainty band for the logistic model fitted to concentration-response data. For any contaminant concentration, there is a 90% probability that the fraction of organisms responding will lie within the shaded region.

concentration-response curves must be calculated separately for each life stage and then combined. We use Monte Carlo simulation for analysis of these data sets.

5.3 EXTRAPOLATION OF CONCENTRATION-RESPONSE FUNCTIONS AND CONFIDENCE BANDS FOR UNTESTED SPECIES

Because full life cycle concentration-response data are rarely available for species-contaminant combinations of interest in risk assessments, we developed a method for extrapolating logistic functions and confidence bands using data sets presented in Appendix B. We used data sets for mortality to three life stages (eggs, larvae, juveniles) that together encompass the fish life cycle from egg to first reproduction. The data were screened, and sets for which (1) mean control mortality was 30% or larger or (2) the range of test concentrations did not span the LC_{25} were deleted.

5.3.1 Extrapolation of β and LC_{25}

The chronic LC_{25}, rather than the LC_{50}, was chosen as a benchmark because, in the majority of available data sets, the range of concentrations used (usually 5-7 values per experiment, excluding controls) did not span the LC_{50}. The logistic model was fitted to the data sets that satisfied the exclusion criteria using the procedure described in Section 5.1. Data sets for which confidence intervals for the fitted β values included zero were excluded from further analysis. When the fitted β values for the remaining 77 data sets were examined, they were found to fit a lognormal distribution

with a median of 6.08, a 5th percentile of 1.87, and a 95th percentile of 16.43. No significant difference was found between the distributions of β's for the three life stages, and no correlation was found between the β's and the LC_{25}s.

Equations for estimating chronic LC_{25}s (with associated confidence intervals) from acute LC_{50}s were derived using the procedure described in Section 4. Separate equations were developed for each of the three life stages represented in the chronic concentration-response data sets.

5.3.2 Calculation and Verification of Synthetic Concentration-Response Functions

Given extrapolated estimates of β (β*) and LC_{25} (LC_{25}*), an extrapolated estimate of α (α*) can be obtained from

$$\alpha^* = \ln(1/3) - \beta^* LC_{25}^* \quad . \tag{5.4}$$

When substituted into Eq. (5.1), the extrapolated values of α* and β* permit the calculation of the expected response associated with any contaminant concentration. Uncertainty concerning the expected response is quantified, using Monte Carlo simulation, from (1) the observed distribution of fitted values of β and (2) the extrapolated error around the estimated LC_{25} (Sect. 4). Each distribution is sampled 1000 times, and the randomly chosen paired values of β* and LC_{25}* are used to calculate a statistical distribution for the response associated with a given contaminant concentration. When this procedure is repeated for a range of concentrations, the plotted values form a confidence band around the extrapolated concentration-response function (Fig. 5.1).

Of the 77 chronic concentration-response data sets used in this analysis, corresponding 96-h LC_{50}s (i.e., same species, contaminant, and experimental conditions) were available for 60. We used this subset of 60 data sets to verify the extrapolation method. First, one data set was arbitrarily deleted from the subset. A distribution of β's and a set of acute-chronic regression equations were then calculated using the remaining 59 sets. A synthetic concentration-response function and 90% confidence bands for the contaminant-species life-stage combination represented in the deleted data set were then extrapolated from the appropriate acute LC_{50}. Finally, the logistic model was fitted to the deleted data set and overlaid on the extrapolated uncertainty band. An example is presented in Fig. 5.2.

This process was repeated for each of the 60 data sets in the verification subset. The number of times the empirically estimated LC_{10}s, LC_{25}s, and LC_{50}s fell outside the extrapolated 90% confidence bands were counted. There were seven "misses" at each of the three response levels. These compare favorably with the expected number, six.

5.4 CALCULATING REDUCTION IN REPRODUCTIVE POTENTIAL

The population-level variable chosen as a response variable is the reproductive potential of a female recruit, defined here as a 1-year-old fish. The reproductive potential of a female recruit is defined as the expected contribution of that female to the next generation of recruits, taking into account her annual probability of survival at different ages; her expected fecundity at different ages, provided that

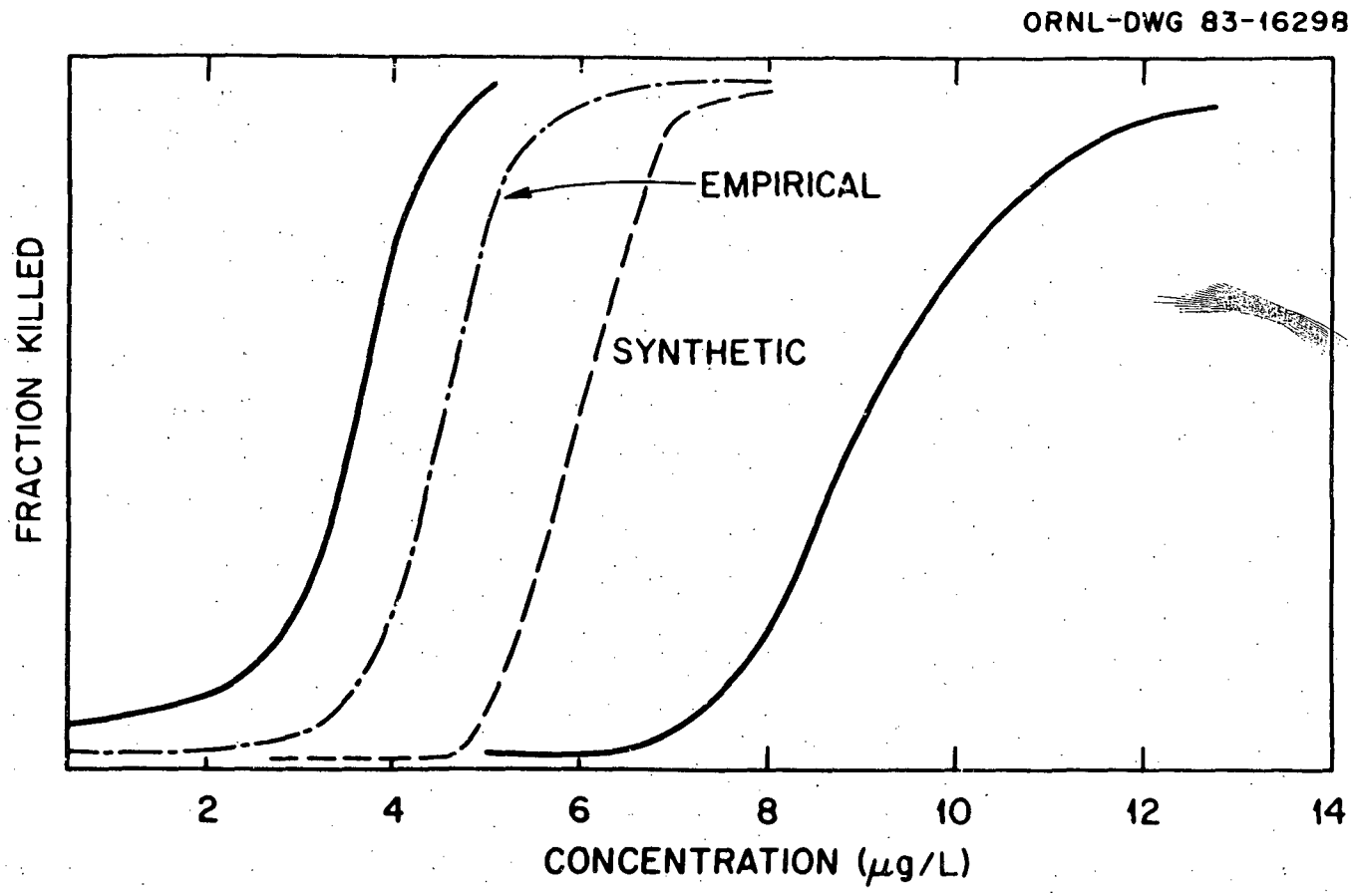

Fig. 5.2. Example of the procedure used to verify the synthetic concentration-response modeling technique. A logistic model fitted to an actual concentration-response data set is overlaid on the uncertainty band of a synthetic concentration-response model constructed for the same chemical, species, and life stage. When many such comparisons are made, 90% of the fitted functions should fall within the uncertainty bands of the synthetic functions.

she survives; the probability that a spawned egg will hatch; and the probability that a newly hatched fish will survive to age 1. The ability of a fish population to sustain exploitation (harvesting) by man and to persist in a variable environment is directly related to the reproductive potential of female fish.

Models based on reproductive potential have been used to assess the effects of fishing and of power plant cooling systems on the risk of catastrophic declines in fish populations (Goodyear 1977). Toxic contaminants, like fishing, reduce the reproductive potential of a female recruit. Mortality rates for fish exposed to toxic contaminants can be translated into changes in reproductive potential, thus allowing comparisons between the population-level consequences of fishing and toxic contaminants. The reproductive potential of a 1-year-old female recruit is given by:

$$P = S_0 \sum_{i=1}^{n} S_i E_i M_i \quad , \tag{5.5}$$

where

S_0 = probability of survival of eggs from spawning to age 1 year,

S_i = probability of survival of female fish from age 1 to age i,

E_i = average fecundity per mature female at age i,

M_i = fraction of age i females that are sexually mature,

n = number of age classes in the population.

Toxic contaminants may reduce the survival of fish at all ages. The reproductive potential of a female recruit exposed to a toxic contaminant throughout her life cycle is given by

$$P_s = S_0(1-m_0)\sum_{i=1}^{n} S_i(1-m_r)^{i-1} M_i E; \qquad (5.6)$$

where

m_0 = probability of contaminant-induced mortality during the first year of life, and

m_r = probability of contaminant-induced mortality for 1-year-old and older fish, assumed equal for all age classes.

The fractional reduction in reproductive potential because of toxic contaminants (R_s) is given by

$$R_s = (P - P_s)/P \quad . \qquad (5.7)$$

Note that natural young-of-the-year survival (S_0), for which reliable estimates are almost never available, cancels out of Eq. (5.7) and is not required for the assessment.

5.5 APPLICATION OF THE MODEL TO RAINBOW TROUT AND LARGEMOUTH BASS

The rainbow trout (Salmo gairdneri) and largemouth bass (Micropterus salmoides) were chosen as examples for illustrating the above extrapolation techniques. Tables 5.1 and 5.2 present life tables for representative populations of these species. The life-stage-specific mortality estimates obtained from the

Table 5.1. Life table for rainbow trout (Salmo gairdneri), modified from Boreman (1978).

Age	M^a	E^b	S_i [c]
1	0.151	207	1.0
2	0.234	850	0.31
3	0.995	1787	0.090
4	1.00	2734	0.013
5	1.00	4685	0.0020
6	1.00	5424	0.00030

[a]Proportion of mature females.

[b]Fecundity per mature female.

[c]Cumulative probability of survival from age 1 to age i.

Table 5.2. Life table for largemouth bass (*Micropterus salmoides*), modified from Coomer (1976).

Age	M^a	E^b	S_i [c]
1	0.0	0	1.0
2	0.17	5,243	0.52
3	1.00	10,830	0.19
4	1.00	16,190	0.085
5	1.00	24,500	0.039
6	1.00	29,973	0.018
7	1.00	36,287	0.0073
8	1.00	42,600	0.0029

[a]Proportion of mature females.

[b]Fecundity per mature female

[c]Cumulative probability of survival from age 1 to age i.

concentration-response model are translated into age-specific survival probabilities using the following equation:

$$(1 - m_0) = (1 - m_e)(1 - m_l)(1 - m_j) \qquad (5.8)$$

where

m_e = probability of mortality for the egg stage,

m_l = probability of mortality for the larval stage, and

m_j = probability of mortality for post-larval stages.

In the chronic toxicity tests, m_j applies roughly to the period from the end of the larval stage to the age of first reproduction. The total duration of the egg and larval life stages is only a few months, whereas juvenile females in both example populations do not reach sexual maturity until two years of age. In theory, therefore, some fraction of juvenile mortality should be allocated to older age classes. However, if mortality due to contaminants is restricted to prereproductive fish, then the allocation of a given fractional mortality $(1 - m_j)$ among prereproductive age classes does not affect the predicted population response. It is common practice in life-cycle toxicity tests to sacrifice the test fish after one spawning; thus, there is normally no information on the effects of toxic contaminants on adult age classes. It can be assumed either that (1) adults suffer the same mortality as juvenile fish; or (2) all susceptible fish are killed during the first reproductive cycle; therefore, fish surviving their first spawning will not suffer excess mortality for the remainder of their lives (i.e., $m_r = 0$). Assumption (2) is adopted here.

We note that Eqs. (5.6) and (5.7) are highly sensitive to errors in estimates of adult mortality because of the cumulative effect of applying $(1 - m_r)$ to each successive age class.

5.5.1 Comparison of Fitted and Extrapolated Concentration-Response Functions and Uncertainty Bands

Full life cycle toxicity data are not available for either the rainbow trout or the largemouth bass for any chemical. However, full life cycle toxicity data exist for brook trout (*Salvelinus fontinalis*) exposed to methylmercuric chloride (Appendix B). Figure 5.3 shows a concentration-response function and confidence bands constructed by using the brook trout as a surrogate for rainbow trout. The logistic model was fitted to egg, larval, and juvenile test data for brook trout. The reproductive potential index was then calculated using the life-table data for rainbow trout (Table 5.1). The brook trout MATC for methylmercuric chloride, as calculated from the same data set used to construct the concentration-response functions, is plotted on the concentration axis. The median value of the EC_{10} is 0.07 µg/L, and the prediction interval (i.e., the 90% confidence interval around the median) is approximately 0.03 to 0.1 µg/L. The brook trout MATC for methylmercury, 0.53 µg/L, corresponds to a 60 to 78% (median 68%) reduction in reproductive potential.

A methylmercuric chloride acute LC_{50} is available for rainbow trout. Figure 5.4 shows a concentration-response function constructed from a single-step extrapolation, from rainbow trout acute LC_{50} to chronic LC_{25}, using the method described in Section 5.3. The median

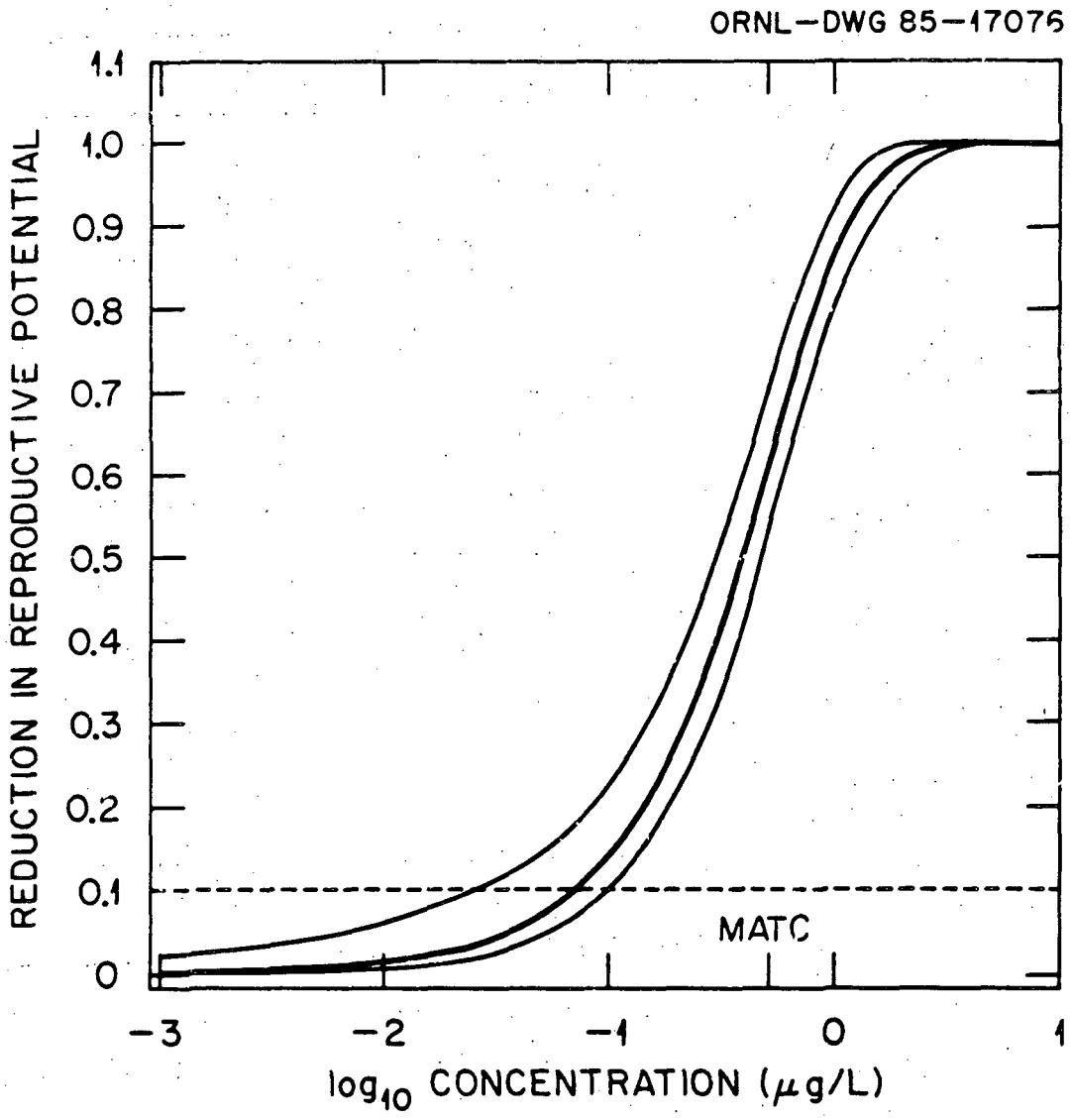

Fig. 5.3. Fitted concentration-response function and uncertainty band for the reduction in female reproductive potential of brook trout (Salvelinus fontinalis) exposed to methylmercuric chloride. The dashed line denotes the 10% effects level (EC_{10}).

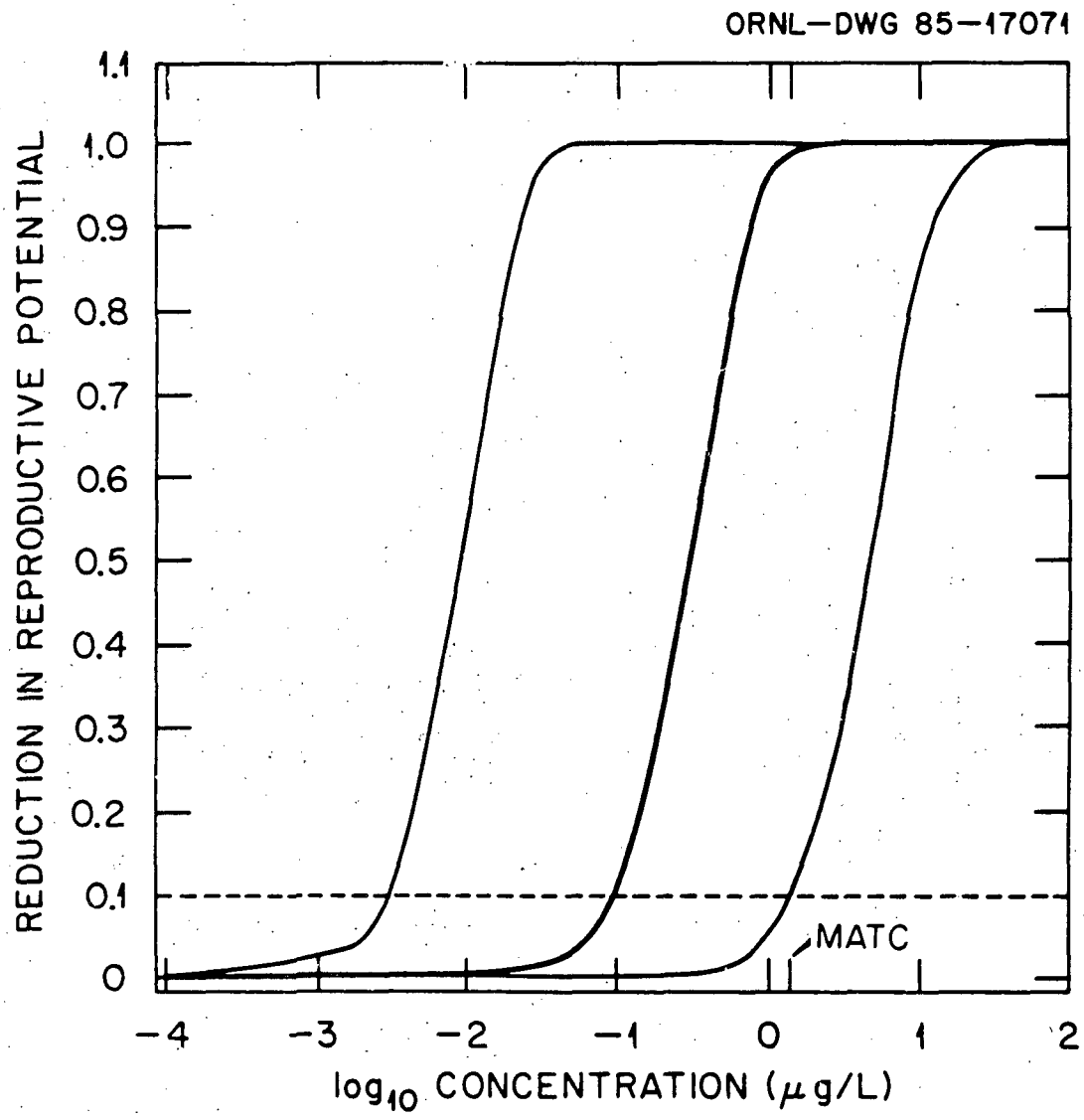

Fig. 5.4. Synthetic concentration-response function and uncertainty band for the reduction in female reproductive potential of rainbow trout (Salmo gairdneri) exposed to methylmercuric chloride. Chronic LC_{25}s for the three life stages were obtained by single-step extrapolation from an acute LC_{50} for rainbow trout.

responses from the extrapolated model (Fig. 5.4) are very close to the median responses (Fig. 5.3) from the fitted model (median EC_{10} = 0.09 µg/L for the fitted model and 0.10 µg/L for the extraplated model). The prediction intervals, however, are much wider. The prediction interval for the EC_{10} in Fig. 5.4, for example, ranges from 0.003 to 1.2 µg/L. The rainbow trout MATC for methylmercuric chloride (1.2 µg/L, extrapolated from brook trout using the method described in Section 4), corresponds to a 10-100% reduction in reproductive potential.

If no acute LC_{50} had been available for rainbow trout, it would have been necessary to extrapolate a value from an acute LC_{50} for another species. Figure 5.5 shows a concentration-response function constructed from a two-step extrapolation (Section 4), from fathead minnow (Pimephales promelas) to rainbow trout acute LC_{50} to chronic LC_{25}. The prediction interval for the EC_{10} obtained from the two-step extrapolation ranges from 0.0002-0.56 µg/L, with a median of 0.015 µg/L. Thus, compared to the single extrapolation, the two-step extrapolation produces median effects about a factor of five lower and prediction intervals about an order of magnitude wider.

Comparisons of Figs. 5.3, 5.4, and 5.5 suggests that, as is true in extrapolation of MATC's (Section 4), in extrapolation of concentration-response functions the acute-chronic extrapolation is dominant source of uncertainty. As a means of confirming this inference, we examined the importance of uncertainty concerning β in determining the widths of prediction intervals obtained in the single-step extrapolation (Fig. 5.4). Figure 5.6 presents a

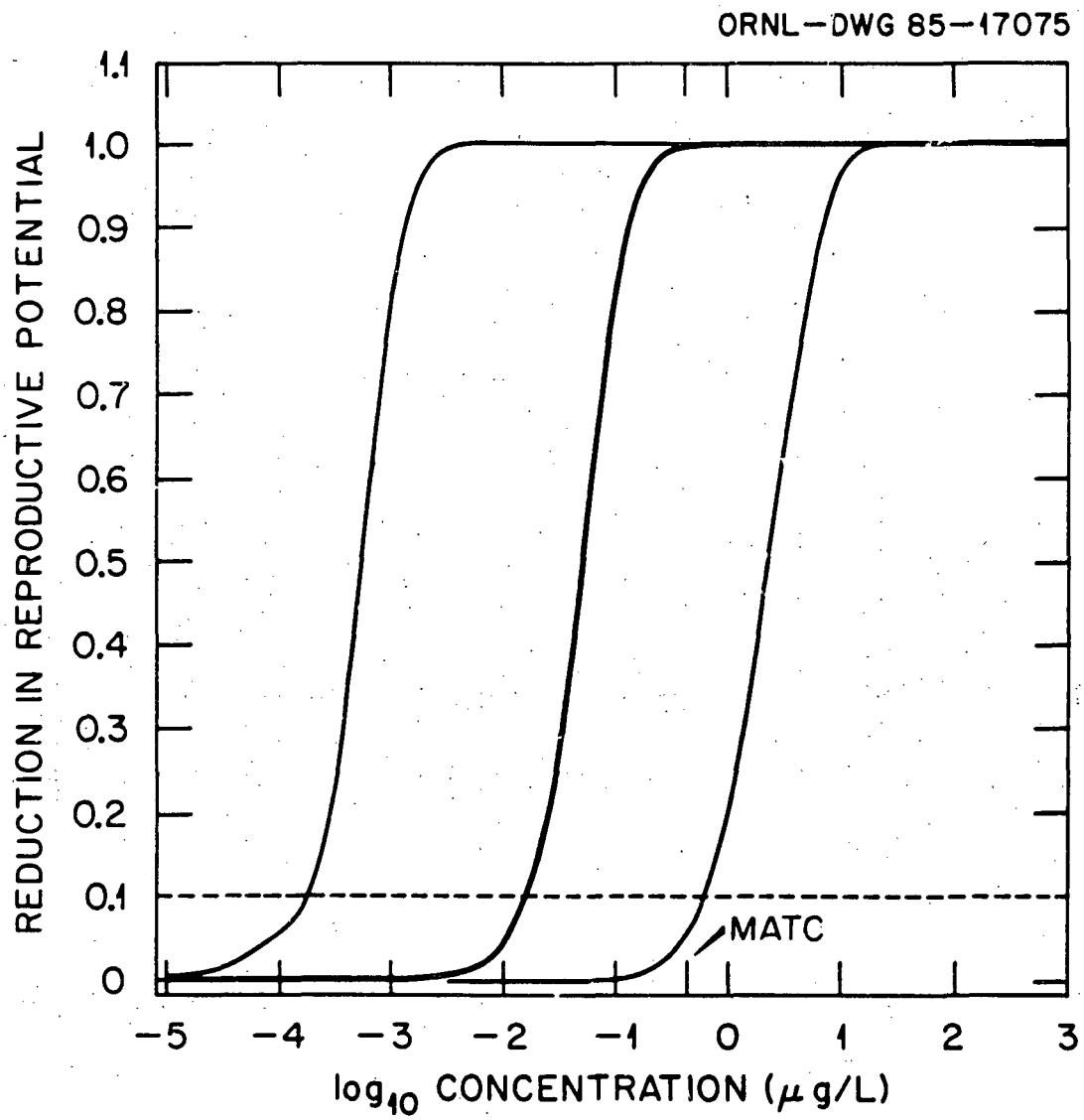

Fig. 5.5. Synthetic concentration-response function and uncertainty band for the reduction in female reproductive potential of rainbow trout (Salmo gairdneri) exposed to methylmercuric chloride. Chronic LC_{25}s for the three life stages were obtained by two-step extrapolation from an acute LC_{50} for fathead minnow (Pimephales promelas).

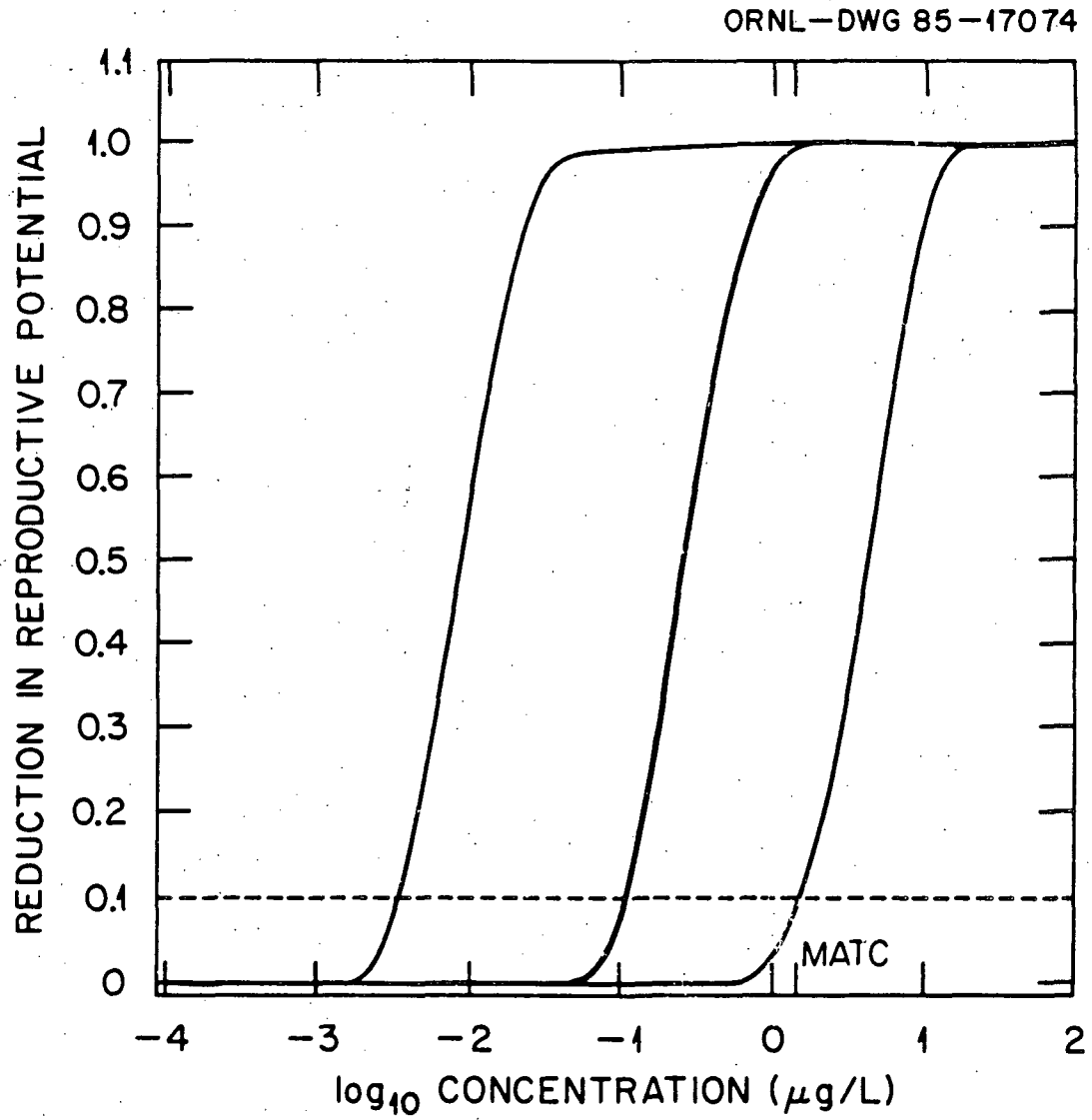

Fig. 5.6. Synthetic concentration-response function and uncertainty band for the reduction in female reproductive potential of rainbow trout (Salmo gairdneri) exposed methylmercuric chloride. Chronic LC_{25}s were obtained as in Fig. 5.4. Uncertainty concerning the curvature of the function was eliminated by setting the curvature parameter (β) constant at its median value.

concentration-response function constructed similarly to Fig. 5.4, but assuming the value of β to be constant at its median value. Because β is constant, the width of the prediction interval in Fig. 5.6 is determined solely by the confidence intervals around the extrapolated LC_{25}s for the three life stages. Within the effects interval of 10 to 90%, Figs. 5.4 and 5.6 are nearly identical. Thus, within this range, uncertainty accumulated in the acute-chronic extrapolation dominates all other sources.

5.5.2 Comparison of Extrapolated Concentration-Response Functions and Prediction Intervals for Different Species

Figures 5.7 and 5.8 show extrapolated concentration-response functions and uncertainty bands for rainbow trout and largemouth bass exposed to cadmium. For rainbow trout, a single extrapolation was required, from rainbow trout acute LC_{50} to chronic LC_{25}. A double extrapolation, including a genus-level taxonomic extrapolation from *Lepomis spp.* to *Micropterus spp.* and an acute-chronic extrapolation was necessary for largemouth bass. Despite the double extrapolation, the uncertainty band for largemouth bass is noticeably narrower than the uncertainty band for rainbow trout. The explanation for this result is the relatively high sensitivity of salmonids to cadmium. The rainbow trout acute LC_{50} is near the low end of the range of LC_{50}s (Appendix A) used in the acute-chronic regression; as in all linear regression models, prediction intervals for extrapolated chronic LC_{25}s increase in width with increasing distance from the mean LC_{50}. Otherwise, the two sets of bands are qualitatively similar.

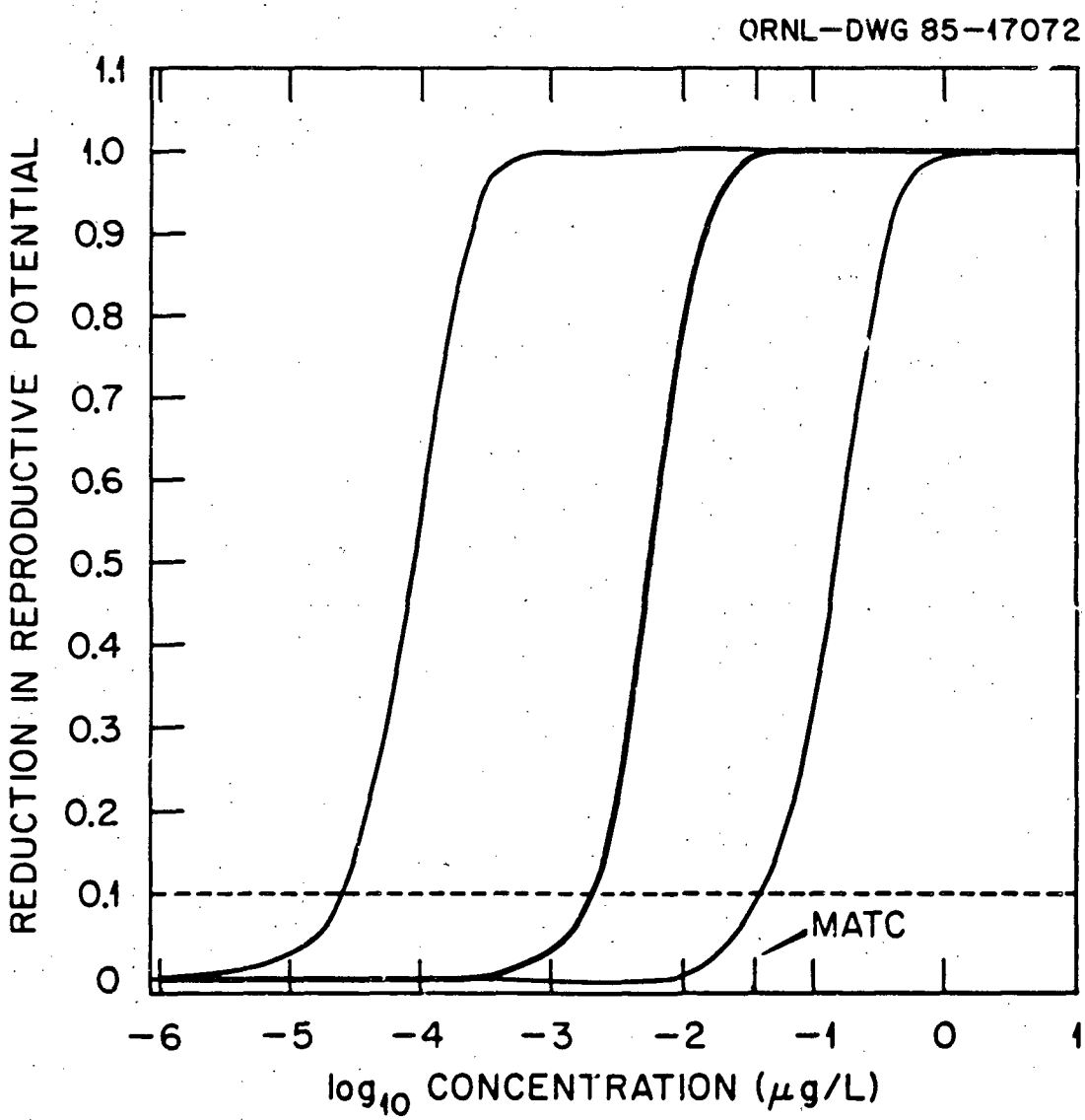

Fig. 5.7. Synthetic concentration-response function and uncertainty band for the reduction in female reproductive potential of rainbow trout (Salmo gairdneri) exposed to cadmium. Chronic LC_{25}s were obtained by single-step extrapolation from an acute LC_{50} for rainbow trout.

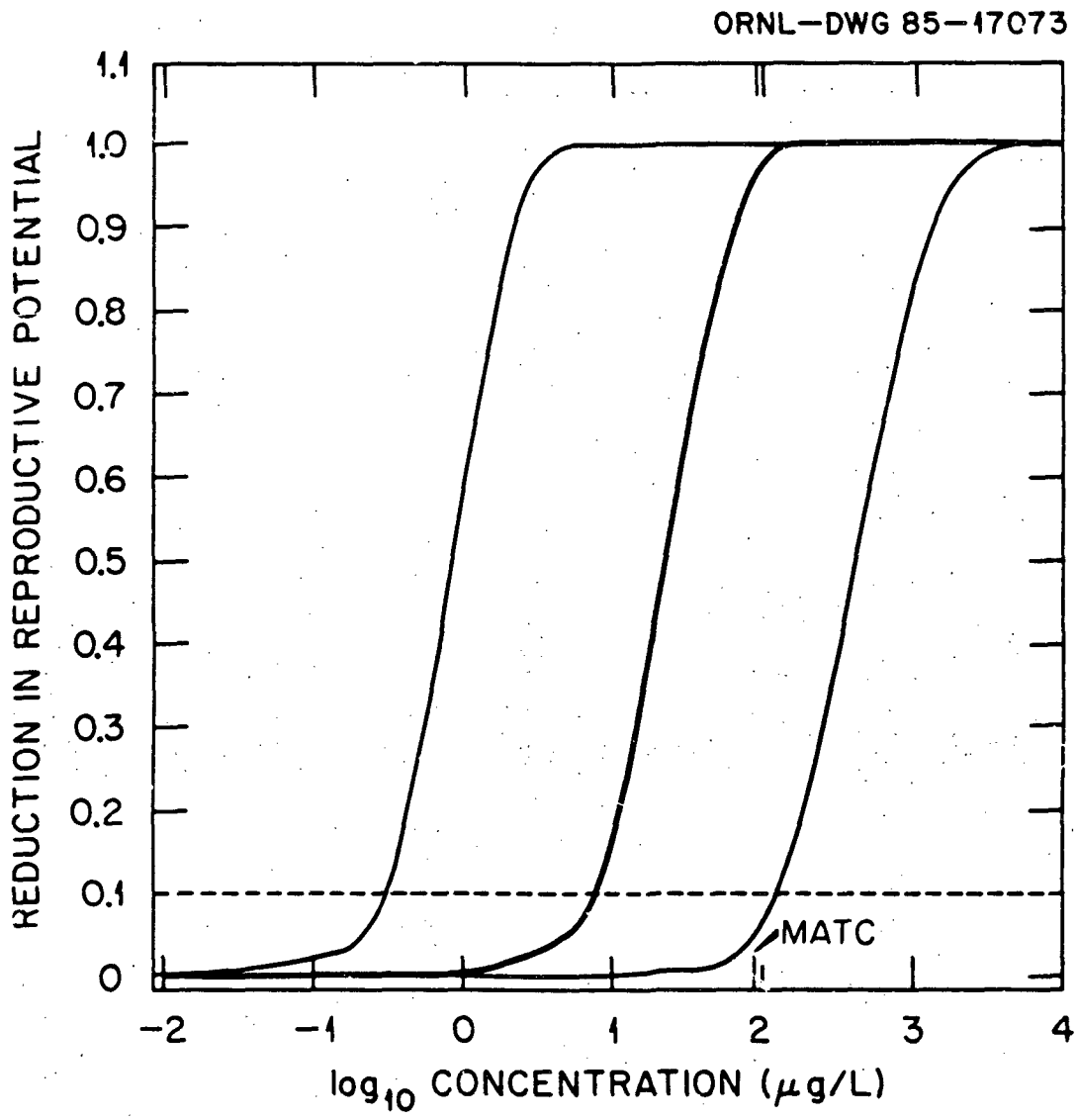

Fig. 5.8. Synthetic concentration-response function and uncertainty band for the reduction in female reproductive potential of largemouth bass (*Micropterus salmoides*) exposed to cadmium. Chronic LC_{25}s were obtained by two-step extrapolation from an acute LC_{50} for bluegill (*Lepomis macrochirus*).

For both species, the range of cadmium exposure concentrations can be divided fairly precisely into three segments: a region of no significant reduction, a region of certain extinction, and a region of indeterminate reduction. The curves defining the upper and lower limits of the predicted responses are quite steep. The upper limit of the predicted response, for example, falls to near zero at concentrations only a factor of 2 lower than the lower limit of the EC_{10}. Similarly, the lower limit of the predicted response rises to a 100% reduction within an order of magnitude of the upper limit of the EC_{10}. These limits provide useful operational definitions for qualitative identification of low, high, and indeterminate impacts. For example, based on Fig. 5.8 it might be concluded that a long-term average cadmium exposure concentration of 0.01 ug/L would have no impact on a largemouth bass population, because, at that level, the upper limit of the predicted response interval is less than 1%. However, no inference could be made regarding the effect of this same concentration on rainbow trout, because the predicted response interval at 0.01 µg/L spans the full range from 0 to 100%.

For both species, cadmium MATCs correspond to predicted reductions in reproductive potential ranging from 10 to 100%. In fact, for all Figs. 5.4 through 5.8, the MATC's fall within the range of maximum uncertainty concerning population response. In Fig. 5.3, the MATC corresponds to a 60 to 80% reduction in female reproductive potential. This result is especially noteworthy because the concentration-response function and confidence bands plotted in Fig. 5.3 were obtained without taxonomic or acute-chronic extrapolation by fitting the logistic model

to the same data set used to estimate the MATC for brook trout. Although no firm conclusions are possible from the limited number of comparisons presented here, the consistent pattern displayed suggests that it may inappropriate to interpret the MATC, either calculated or extrapolated, as a chronic effects threshold for fish.

5.6 DISCUSSION

Waller et al. (1971) and Wallis (1975) proposed the use of fisheries-derived population models for quantifying the effects of contaminants on populations, although experimental or observational data on model applicability was not provided. We do not propose that the methods described in this report can be used to directly predict the long-term responses of fish populations to toxic contaminants. We have noted elsewhere (Barnthouse et al. in press) that fisheries scientists are still unable to predict the long-term effects of exploitation on fish populations to an accuracy and precision that would be useful for management decisions. However, we believe it is feasible to use population-level assessment methods to perform risk assessments in the same way that these methods are used by fisheries managers: as indicators of stress to be supplemented by expert judgment. We consider three applications to be currently feasible: (1) identification of data collection priorities, (2) setting of water quality standards, and (3) quantitative comparison of contaminant-related risks to risks associated with fishing or other environmental stresses.

We noted in Section 5.5.1 that the dominant source of uncertainty in estimating reductions in female reproductive potential (due to toxic

contaminants) is the uncertainty accumulated in extrapolating from acute LC_{50}s to chronic LC_{25}s. This result, and the fact that only acute data are available for most chemicals, suggests the great importance of obtaining a better understanding of relationships between acute and chronic effects in risk assessment. The sensitivity of population-level indices to estimates of contaminant effects on adult fish in iteroparous species, noted in Section 5.4, indicates the need to evaluate the effects of contaminants on older fish, at least to the extent of testing the hypothesis that mortality is restricted primarily to early life stages.

Currently, water quality criteria are derived from MATCs, the geometric means of no observed effects and lowest observed effects concentrations (NOECs and LOECs). A NOEC is the highest concentration used in a toxicity test at which no statistically significant (conventional 95% confidence level) difference is observed between experimental and control mortality and the LOEC is the next higher concentration in the dilution series. As noted by Gelber et al. (1985), NOECs have the undesirable property that the likelihood of observing an effect at a given concentration is as much a function of experimental design as of contaminant toxicity. In particular, NOECs are nonconservative in that factors resulting in lower test precision (e.g., low number of organisms per replicate, low number of replicates, and high between-replicate variability) tend to increase the observed NOEC and reduce the level of environmental protection afforded by water criteria derived from the NOEC. In Section 5.5.2, it was shown that MATCs for rainbow trout and largemouth bass are consistently greater

than estimated population-level EC_{10}s, even when the logistic model is fitted directly to the same concentration-response data used to derive the MATC. It seems possible, if the results in Section 5.5.2 are confirmed by further research, that an approach to water quality criteria based on concentration-response relationships would be superior to one based on MATCs. In this connection, it is significant that, when concentrations are plotted logarithmically, all of the concentration- response functions developed in this section approximate step functions. When uncertainty bands are considered, the plots can be divided into nearly rectangular regions of no expected effect, high expected effect, and indeterminate effect. If this observation is generally true of concentration-response relationships for toxic chemicals, then the response regions could be used to define ambient water quality criteria that reflect the degree of scientific uncertainty concerning concentrations having adverse effects on populations.

Expression of the effects of toxic contaminants in the same units used to assess other forms of mortality permits comparison of the effects of contaminants with the effects of exploitation by fishermen. Many coastal fish stocks, for example, are subject both to intense fishing pressure and to environmental pollution. Successful management of these populations depends on determining the relative importance of these stresses. The reproductive potential index used in Section 5 is similar to indices that have been used to compare the entrainment and impingement by power plants to the impact of fishing (Goodyear 1977, Dew 1981), thus, the index appears suitable for this purpose.

The utility of comparing/combining estimates of effects of contaminants and of exploitation depends on whether populations exposed to toxic contaminants respond in a manner similar to exploited populations. Some evidence exists that these responses are at least qualitatively similar. In a review of the effects of exploitation on fish populations, McFadden (1977) concluded that exploitation typically causes increased growth and fecundity and sometimes causes decreased maturation time. These responses have the effect of compensating for the increased mortality associated with fishing, thus allowing the populations to persist and sustain exploitation. MacFarlane and Franzin (1978) noted these same changes in a population of white suckers (Catastomus commersoni) in a metal-contaminated lake. Jensen and Marshall (1983) noted that laboratory populations of Daphnia galeata mendotae exhibit responses to cadmium stress that are qualitatively similar to the responses described by McFadden. They proposed that effects of toxic contaminants on zooplankton populations could be quantified using models developed to describe fisheries.

At least for fish populations, population-level risk assessment models appear to have several important uses. We believe that the reproductive potential index used in this report is the simplest such index that integrates data on effects of toxic contaminants on all life stages; however, it is by no means the only possible index that could be used. Several authors, notably Gentile et al. (1983) and Daniels and Allan (1981), have used the intrinsic rate of natural increase (r) to integrate data on mortality, growth, and reproduction obtained from chronic toxicity tests for zooplankton. Models of growth could be used

to assess the effects of contaminants on biomass production, where the primary effect of chemicals is reduced growth rather than increased mortality. All of these approaches are applicable to invertebrate populations as well as to fish. The extent to which the use of population-level risk assessment models can supplement or supplant currently used individual-level approaches remains to be determined.

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6. ECOSYSTEM LEVEL RISK ASSESSMENT

R. V. O'Neill, S. M. Bartell, and R. H. Gardner

6.1 INTRODUCTION

Environmental toxicology is in a period of rapid transition. The need to predict toxic effects in natural ecosystems is pressing, yet our ability to extrapolate from laboratory to field is limited by our inability to describe mechanisms controlling natural systems. Thus, the science is experiencing rapid evolution in laboratory measurements and in methods for extrapolation to the field.

Particularly critical is the need to predict higher-order effects at concentrations well below acute toxicity (LC_{50}). Synergistic effects result from biotic interactions, such as competition and predation, and abiotic constraints, such as temperature and limited nutrients. These processes alter the response of organisms in the ecosystem and cause effects that would not be anticipated from laboratory measurements of single species.

Development of a credible predictive ability logically begins with the extrapolation of toxicological data collected in the laboratory to more complicated systems. O'Neill et al. (1982) introduced ecosystem uncertainty analysis (EUA) as one potential method for extrapolating toxicity data in aquatic systems. The objective of this section is (1) to review the methodology that has been developed, (2) to illustrate results obtained with EUA using the Standard Water Column Model (SWACOM), and (3) to briefly discuss the methodology with regard to future modifications and refinements.

6.2 ECOSYSTEM RISK METHODS

Because most of our work has centered on SWACOM, it is convenient to begin by describing this model. This will permit us to describe the methods in the context in which they were developed and permit us to use SWACOM to illustrate methodological details.

6.2.1 Description of the Standard Water Column Model (SWACOM)

SWACOM was modified from an earlier model known as CLEAN (Park et al. 1974). The model (Fig. 6.1) is designed to mimic the pelagic portions of a lake ecosystem, including ten phytoplankton populations, five zooplankton populations, three planktivorous fish, and a top carnivore. The populations within a trophic level are described by similar equations but with different parameter values. Thus, each phytoplankton population is characterized by its maximum photosynthetic rate, light saturation constant, Michaelis-Menten constant, temperature optimum, and susceptibility to grazing.

The abiotic driving variables mimic the environment of a northern dimictic lake (Fig. 6.2). The temperature describes an annual sinusoidal curve with lake turnover occurring at 4°C in the spring and fall. Radiant energy follows a similar curve, with light greatly reduced under ice cover. External sources add nutrients each day of the year. Remineralized nutrients are added to the water column from the hypolimnion at spring and fall overturn.

Phytoplankton grow in response to light, temperature, and available nutrients. Self-shading effects are accounted for by integrating photosynthesis over the 10-m deep euphotic zone. Each phytoplankton

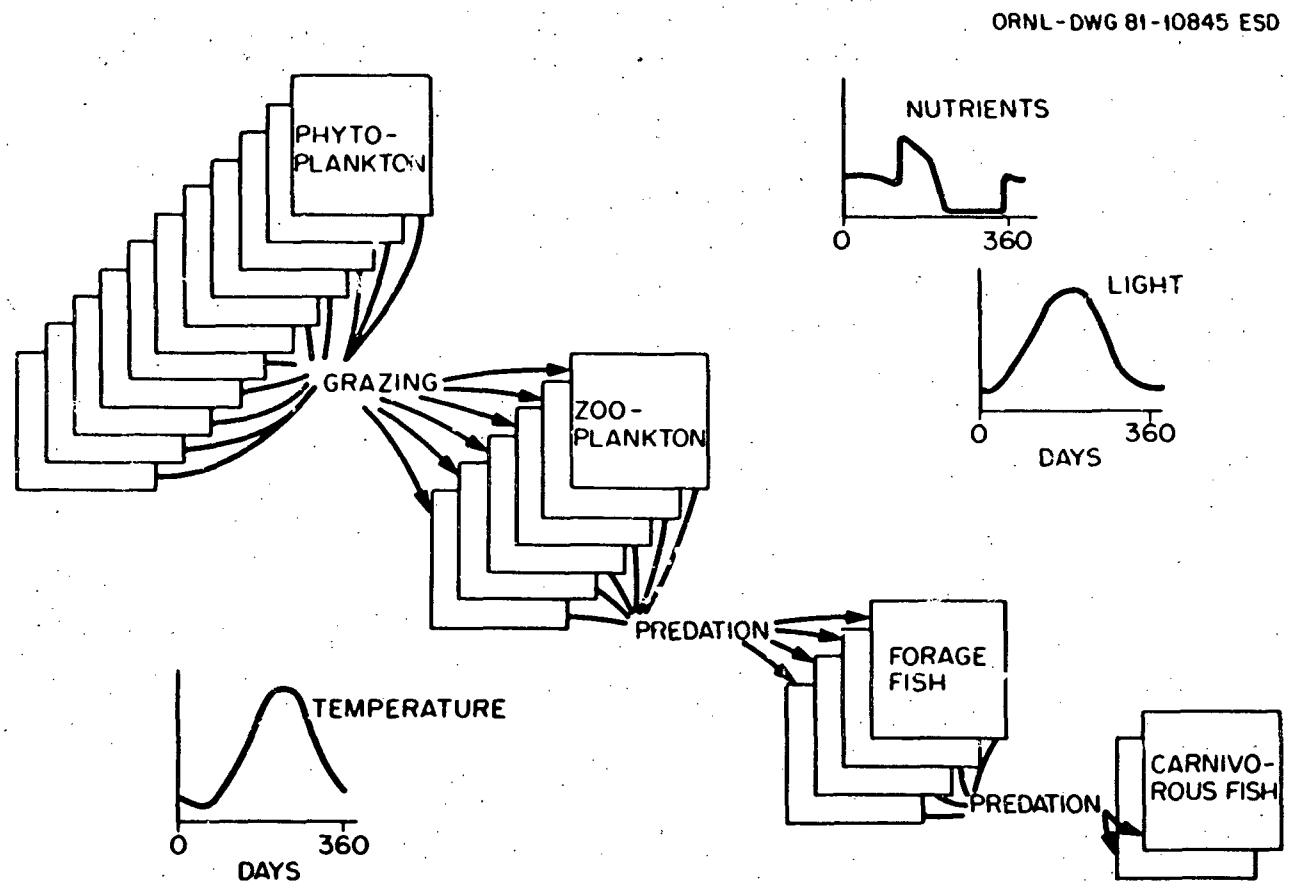

Fig. 6.1. A schematic illustration of SWACOM (Standard Water Column Model). Daily levels of nutrients, light, and temperature serve as model input. SWACOM considers the trophic relationships of 10 phytoplankton, 5 zooplankton, 3 forage fish, and a single carnivorous fish population (From O'Neill et al. 1982).

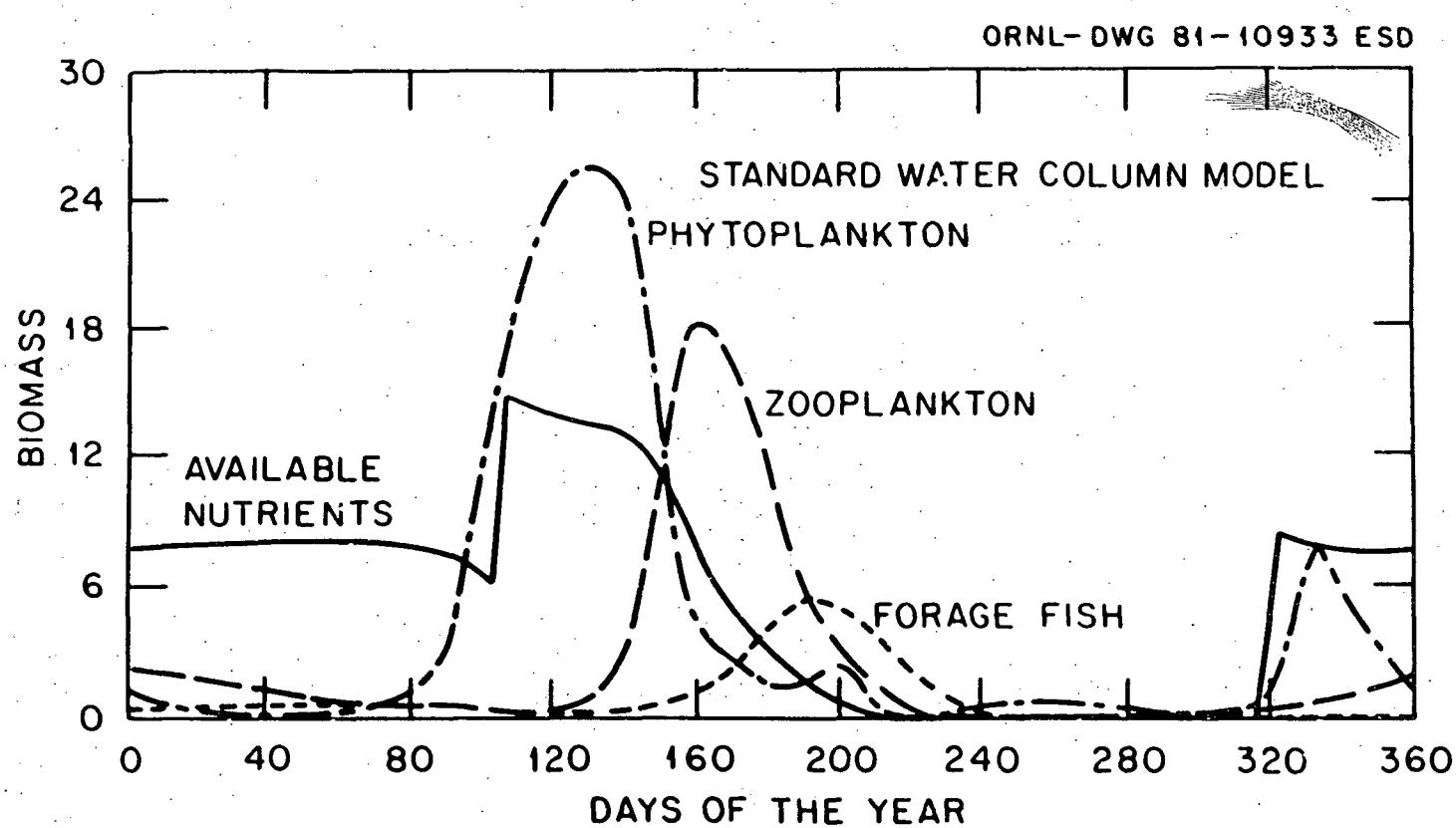

Fig. 6.2. A typical simulation of SWACOM showing seasonal dynamics of phytoplankton, zooplankton, and forage fish. Values shown on the graph are summed over the component populations (from O'Neill et al. 1982).

population has an optimal temperature at which its photosynthetic rate is maximum. Total fixation of biomass is primarily limited by available nutrients that are exhausted in periods of rapid growth.

Grazing and predation are described by a nonlinear interaction function (DeAngelis et al. 1975). This function considers both limited food supply and competition with other grazers. The consumer populations are limited by their individual metabolic and mortality rates and by predation. Both grazing and respiration rates are affected by temperature, with each population characterized by an optimal temperature.

SWACOM can describe a number of higher-order effects. Effects on one population can be altered by competition with other populations in the same trophic level. For example, stress on one phytoplankton population permits other phytoplankton populations to increase until the nutrient pool limits growth. Effects of a toxicant on one trophic level can precipitate effects elsewhere in the system. For example, increased mortality in the forage fishes releases zooplankton from predation, which results in increased grazing on phytoplankton. Effects on all populations are influenced by seasonal variations in light, temperature and available nutrients. All these indirect effects are consequences of the dynamic relationships included in SWACOM.

6.2.2 Organizing Toxicity Data

Ecosystem uncertainty analysis was derived to extrapolate toxic chemical effects measured on laboratory populations to likely effects on ecological production in aquatic systems. Laboratory test species

are not comprehensive in their representation of inhabitants of aquatic environments. Thus, an important aspect of performing EUA lies in associating assay species with their ecological equivalents as expressed in SWACOM.

The first step in implementing EUA is to select of appropriate toxicity data and to associate that data with specific components of SWACOM. Toxicity data on phytoplankton are sparse. It is possible to find values for green algae, such as Selenastrum capricornutum, and these data are used for all ten algal populations if no other information is available. If data are available on diatoms and bluegreens, then a further division is possible based on physiological parameters in the model and past experience with SWACOM. Like diatoms, species 1 to 3 appear early in the spring and are associated with low temperatures and high nutrient concentrations. Species 4 to 7 dominate the spring bloom and are associated with intermediate temperatures and light. Species 8 to 10 appear in the summer and are tolerant of high temperatures and low nutrient concentrations.

The identification of zooplankton is more tenuous. Based on model behavior and physiological parameters, species 12 and 13 are identified with Cladocerans. The ubiquitous data for Daphnia magna are used for species 12. When data are available for Daphnia pulex, they are used for species 13. The remaining zooplankters (species 11, 14 and 15, and species 12 when no data were available for D. pulex) are simply identified as crustaceans. Of the available data, the smallest LC_{50} is assigned to 15 and the largest to 11. Species 14 (and 13 when necessary) is assigned an intermediate value between these extremes.

To assume species 15 to be the most sensitive is conservative. Since an increase in bluegreen algae is one of our end points, we assign the greatest sensitivity to the consumer (i.e., 15), which is most abundant during the summer of the simulated year.

Acute toxicity data for fathead minnow (Pimephales promelas), bluegill (Lepomis macrochirus), and guppy (Poecilia reticulata) are assigned to forage fish (species 16, 17, and 18). When data on these species are not available, others are substituted, such as goldfish or mosquitofish. The top carnivore or game fish (species 19) is usually identified as rainbow trout (Salmo gairdneri).

The general paucity of acute toxicity data can complicate the assignment of SWACOM populations to assay species. Therefore, it has been prudent to determine the sensitivity of risk estimates to different patterns of assigning assay species to model populations (O'Neill et at. 1983).

6.2.3 General Stress Syndrome

Typical toxicity data provide information on mortality (or similar end point) but provide little insight on the mode of action of the chemicals. Thus, some assumptions must be made about how the toxicant affects the physiological processes in SWACOM. In an application that focuses on a single chemical, it may be possible to obtain detailed information on modes of action. However, in general, such information is not available, and it is necessary to make a single overall assumption.

We assumed that organisms respond to all toxicants in a uniform manner, that is, the General Stress Syndrome (GSS). For phytoplankton, this involved decreased maximum photosynthetic rates (Ps), an increased Michaelis-Menten constant (Xk), increased susceptibility to grazing (W), and decreased light saturation (Si). For zooplankton, forage fish, and game fish, the syndrome involved increased respiration (R), decreased grazing rates (G), increased susceptibility to predation (W), and decreased assimilation (A).

The GSS defines the direction of change of each parameter in SWACOM. It is also necessary to make an assumption about the relative change in each parameter. We have assumed that all parameters are changed by the same percentage.

To test the effects of the GSS on estimates of risk, the signs on the growth parameters were systematically varied, and EUA was performed for two chemicals characterized by very different patterns of sensitivity among assay species: naphthalene and mercury. The signs on the effects parameters for photosynthesis and consumption must be negative or no toxic effects are possible. Results of biologically reasonable variation in the remaining growth parameters showed the GSS to be conservative in its estimation of the risk of blue green algal production (Table 6.1). Effects syndromes other than the GSS always produced greater estimates of risk to game fish. However, these syndromes involved a decrease in optimal temperatures for growth in response to toxicant exposure, for which little experimental evidence is likely to be available from current bioassays. If information concerning the physiological mode of chemical action is available for a

Table 6.1. Risks of increased algal production and decreased game fish production in systematic alteration of the General Stress Syndrome. The optimal temperature for growth (To), prey preference (W), assimilation efficiency (A), and grazing rate (G) were either increased (+), decreased (-), or unchanged (0) in the associated estimates of risk for exposure to naphthalene (0.0468 mg/L).

To	W	A	G	Algae increase	Game fish decrease
0	+	-	-[a]	43.6	1.6
0	-	+	+	0.4	0
0	0	0	0	9.4	4.0
-	-	-	-	0.2	31.0
+	+	+	+	9.4	0
+	+	+	-	7.0	0.2
+	+	-	+	0	13.2
+	+	-	-	42.4	1.0
+	-	+	+	0	0
+	-	+	-	0	0.2
+	-	-	+	0	14.8
+	-	-	-	0	1.6
-	+	+	+	11.2	0
-	+	+	-	14.4	1.8
-	+	-	+	0	30.6
-	+	-	-	31.6	33.8
-	-	+	+	0	0
-	-	-	+	0	29.2
-	-	+	-	1.8	0.4

[a]Used in the General Stress Syndrome

specific toxicant, the GSS may be appropriately modified. For example, chemicals with a narcotizing effect could be represented by decreasing respiration in the GSS. Similarly, photosynthetic enhancers or inhibitors can be more explicitly depicted. The development of alternative stress syndromes is limited only by the basic bioenergetic formulation of the growth equations in SWACOM.

In the absence of information that details the mode of action, the GSS appears as a conservative choice in the application of EUA for evaluating the likely effects of potentially toxic chemicals.

6.2.4 Microcosm Simulations

The key to changing parameters in the model is simulation of the experiments used to generate toxicity data. This involved simulating the production dynamics of each species in isolation, as it might occur in a laboratory under ideal constant conditions. The parameters of that species were then altered to duplicate the end point used in the original experiment. Thus, for an LC_{50} of 96 h, we would find the percentage change that halved the population in 4 d.

At the conclusion of the MICROCOSM simulations, we have the percentage change in the parameters that matches the experimental end point; that is, we can match the response of the population to the specific concentration that represents the LC_{50} and EC_{50}. We must now make an additional assumption to arrive at the level of response to be expected for other concentrations that lie below the LC_{50} or EC_{50}. We assumed a linear concentration-response relationship. Thus, an environmental concentration one-fifth of the LC_{50} would

cause a 10% reduction in the population over the same time interval as the original test. MICROCOSM simulations are then repeated with this new end point to arrive at the percentage change in the parameter resulting in a 10% reduction. The linear assumption can be removed if a concentration-response curve is available for the toxicant. Because most concentration-response curves are concave, our assumption should result in choosing a level of effect larger than would actually result if the test were conducted at that concentration. Therefore, the linear assumption is conservative. In addition, EUA emphasizes the implications of interacting ecosystem components on modeling the response of the system to toxicant exposure. It is not the intent to model concentration-response relationships for individual organisms.

6.3 UNCERTAINTIES ASSOCIATED WITH EXTRAPOLATION

To implement EUA, it is necessary to know not only the percentage change in parameters but also the uncertainty to be associated with this change. Monte Carlo simulation (Sect. 6.5) is used to translate uncertainties regarding individual parameters into uncertainty regarding system responses. We have assumed that all parameter changes have an associated uncertainty of plus or minus 100%. This assumption seemed sufficiently conservative. In a specific assessment, one might wish to adopt a more complex strategy that would combine greater information on modes of action with statistical extrapolation procedures (Sect. 4) or a survey of experienced researchers to arrive at more specific estimates of uncertainty.

Because of the relatively large uncertainties, the possibility exists that risks are due to the uncertainties rather than the actual effect of the chemicals. In such a case, the risk is due to our ignorance of the system rather than the potential toxic effect of the chemicals.

To test for the effect of large uncertainties, we analyzed the deterministic response of the model to several toxic substances. The deterministic response assumes no uncertainties in the parameters. This response is approximately the average response of the system to that level of toxicant. The response can be expressed as the percentage change in the mean population relative to the "no toxicant" case. If the percentage change is close to zero, then the risk can be attributed to uncertainty alone. If the mean populations are significantly changed, the risks are attributed to toxic effect plus uncertainty.

Analysis of the deterministic solution for nine chemicals associated with the production of synthetic fuels from direct (Table 3.3.2 in Suter et al. 1984) and indirect (Table 3.3.2 in Barnthouse et al. 1985) coal liquefaction indicates that the toxicity of mercury, cadmium, nickel, ammonia, naphthalene, and phenol contributes significantly to estimates of risk. Risks posed by arsenic and lead result more from uncertainties in extrapolation in these particular applications.

6.4 RESULTS OF ECOSYSTEM RISK ASSESSMENTS

Having described the methods to be used in setting up EUA, we will now present four example applications. Our primary purpose is to

demonstrate the utility of the method in routine assessments. However, we will also make it a point to show how the results of EUA differ from population-oriented assessments.

6.4.1 Risk Assessment for Direct and Indirect Liquefaction

The results of risk assessments for real liquefaction technologies are shown in Fig. 6.3 (Suter et al. 1984). Two end points were considered: A quadrupling of the peak biomass of noxious bluegreen algae and a 25% decrease in game fish biomass. These end points were chosen as indicative of minimal effects that could be noticed in the field. Risk values i.e., probabilities of exceeding the above end points, were calculated across a range of environmental concentrations. The range of exposures for each technology is shown at the bottom of the figure.

Results for naphthalene are shown in Fig. 6.3. There is an upturn in the risk curves, showing significant risks at the higher concentrations reached by at least one of the technologies. The increased risk to game fish populations seems intuitively reasonable. However, the increasing risk of a bluegreen algal bloom with increasing concentration is counterintuitive. This is an example of the indirect effects that EUA is capable of showing. Even though each of the chemicals is toxic to the algae, the reduction in sensitive grazing organisms more than compensates for the direct effect on phytoplankton.

Ecosystem uncertainty analysis can be used to compare risks estimated for different classes of chemicals for different direct liquefaction technologies (Fig. 6.4). Here the four technologies all

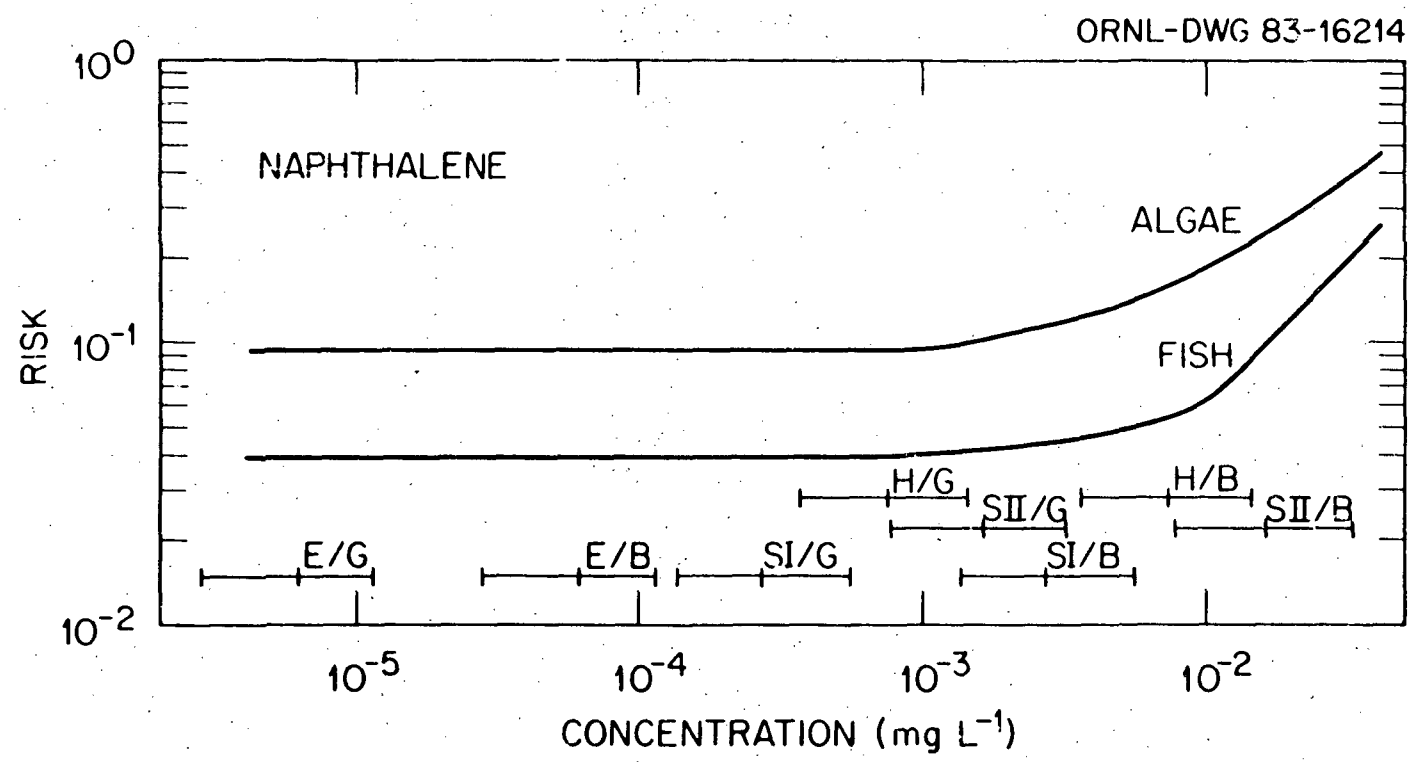

Fig. 6.3. Risk estimates for naphthalene over a range of environmental concentrations. The 5th percentile, mean, and 95th percentile concentrations associated with four direct coal liquefaction technologies are shown at the bottom of the graph. The notations /B and /G refer to two alternative wastewater treatment options. The plotted values are the probability of a fourfold increase in algal biomass and a 25% reduction in game fish biomass (From Suter et al. 1984).

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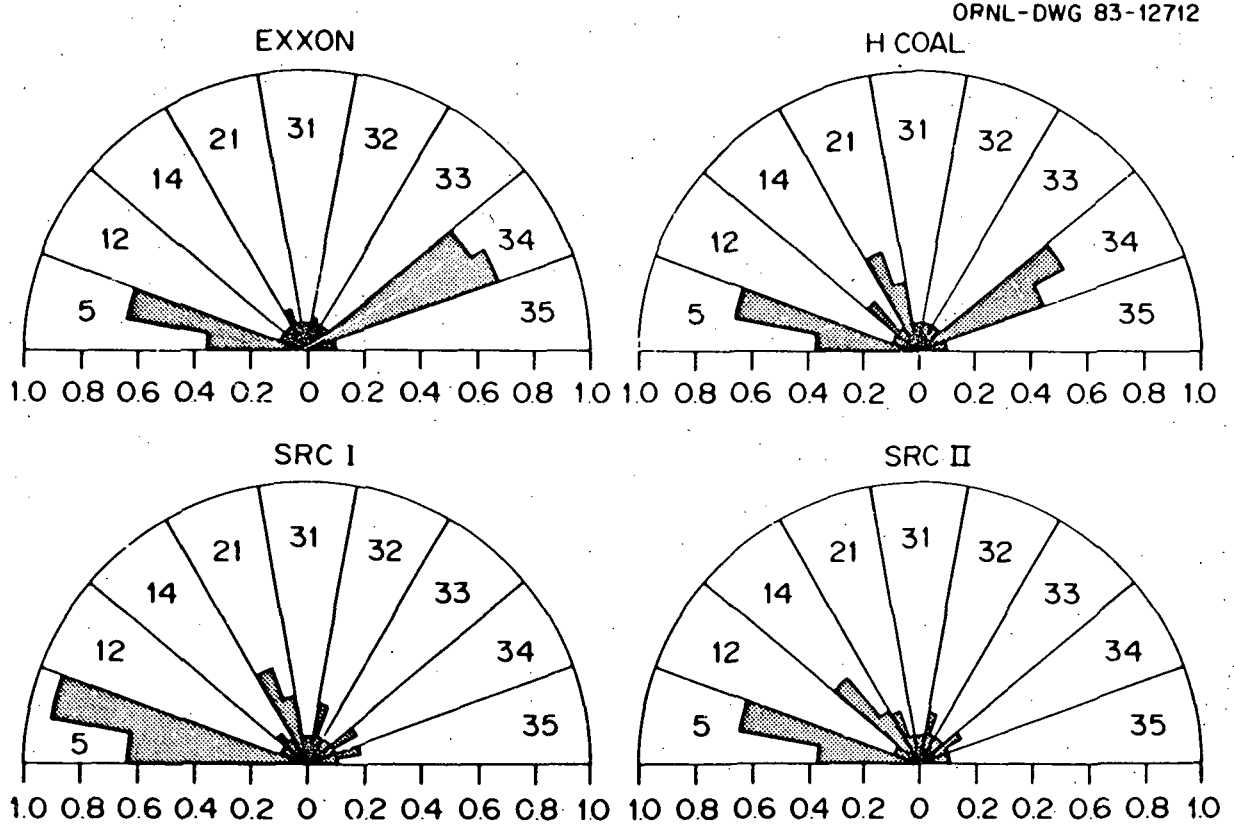

Fig. 6.4. Comparison of risks among direct coal liquefaction technologies. Risks at the 95th percentile concentration are shown first for algae and then for game fish for each of nine contaminant categories (5 = ammonia, 12 = benzene, 14 = mono- and diaromatic hydrocarbons, 21 = phenols, 31 = arsenic, 32 = cadmium, 33 = nickel, 34 = mercury, and 35 = lead; from Suter et al. 1984).

show considerable risks of increased algal production for chemical class 5 (ammonia). The Exxon and H coal processes also suggest similar risks associated with class 34 (cadmium). Other similarities and differences among the technologies are readily apparent from these presentations. Risks posed by chemical classes 5 and 34 are also notable for indirect liquefactor technologies (Fig. 6.5).

6.4.2 Risk Assessment of Chloroparaffins

SWACOM has also been applied (Bartell 1984) in an assessment of risk for chloroparaffins (CPs). In this case, the risk of increased algal production is 14 to 33% at concentrations of 0.0001 mg/L. These risks increase at intermediate exposure concentrations and then decrease to near zero at the highest concentrations tested.

The risk of decreased production of zooplankton, forage fish, and game fish increase monotonically with exposure concentrations. At the highest test concentrations, the likelihood of a 50% decrease in forage fish and game fish approaches 1.0. The highest estimates of risk to game fish result at exposure concentrations that lie at the upper range of expected ambient concentrations (Zapotsky et al. 1981).

Risks of decreased game fish biomass appear to result from the combined direct toxic effects and the effects of decreases in zooplankton and forage fish biomass at intermediate chloroparaffin concentrations.

The relative importance of direct and indirect effects on the responses of each trophic level to chloroparaffins was analyzed. The

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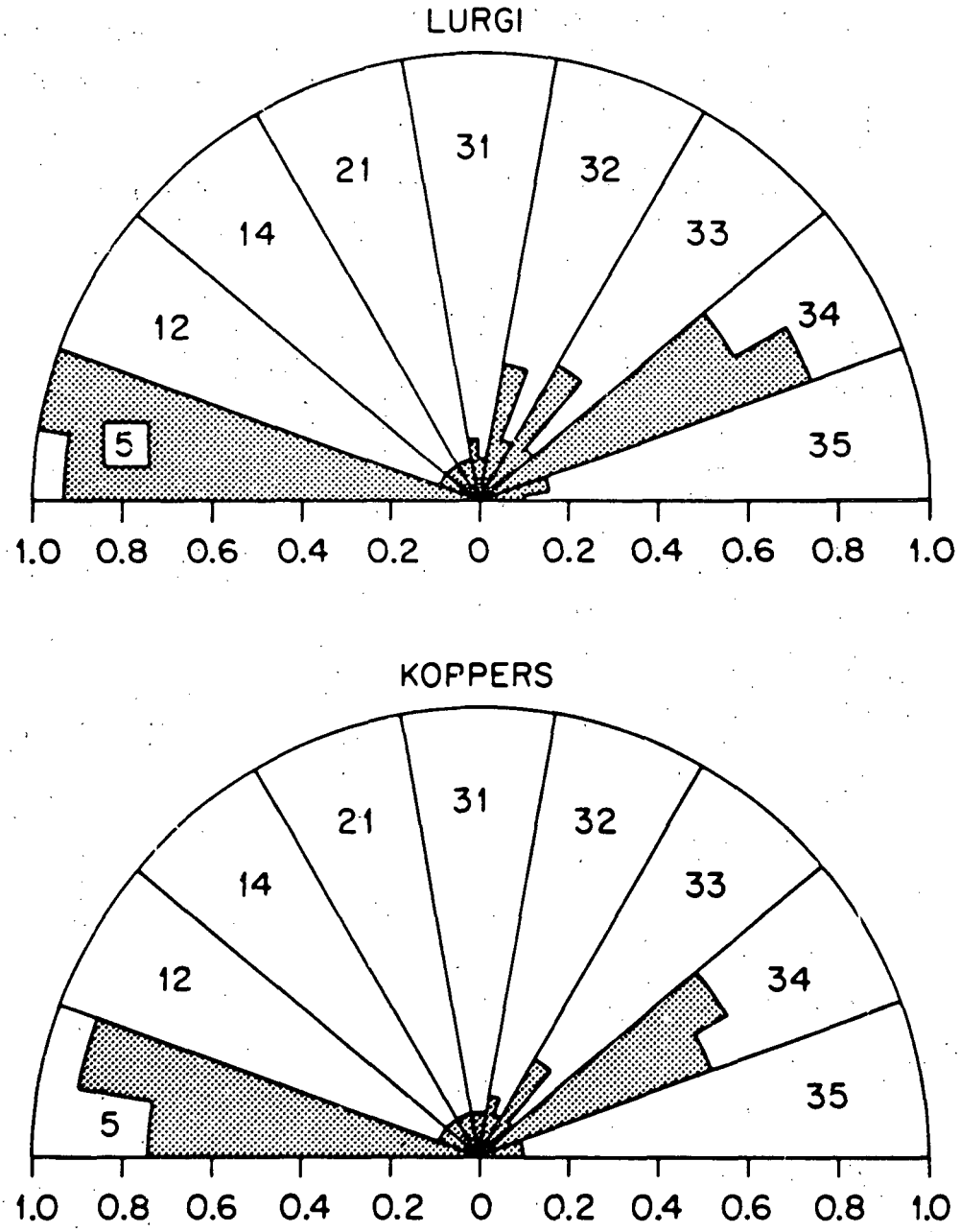

Fig. 6.5. Comparison of risks for two indirect coal liquefaction technologies. Risks and contaminant categories defined as in Fig. 6-4 (from Suter et al. 1984).

results indicated that indirect effects contribute more to risk that do direct effects on individual growth processes within trophic levels.

At exposure concentrations that approach the highest measured concentrations of CPs, the risk of a 100% increase in bluegreen algae blooms ranges from 70 to 76%. At this concentration, the risks of a 50% decrease in forage fish or game fish might reasonably be expected.

6.4.3 Patterns of Toxicological Effects in SWACOM

In another study (O'Neill et al. 1983), SWACOM was used to investigate how different aggregations of ecosystem components might alter conclusions drawn from laboratory data. We compiled data for cadmium, as shown in Table 6.2. The distribution of sensitivities in the first column of Table 6.2 will be referred to as the standard or "population" pattern.

The first step was to remove the differences in sensitivity among populations in the same trophic level. The standard approach would be to take the geometric means of LC_{50}s; however, the data represent a variety of test durations and end points (e.g., EC_{50}s and EC_{20}s). To correct for differences in test conditions, we assumed a simple mortality process described by $x(t) = x(0) \exp(-dt)$, where $x(0)$ is the initial population size, $x(t)$ is the size at time t, and d is the mortality rate. We assume that mortality is a function of concentration, $d = aC$. We know the fraction, $F_1 = x(t)/x(0)$, that survives at one concentration, C_1, measured over one time period, t_1. Since $\ln F_1/C_1t_1 = -a = \ln F_2/C_2t_2$, we can then estimate the concentration, C_2, that would result in a different

Table 6.2. Toxicological data used in examination of patterns of effects for cadmium

Model populations		LC_{50}/EC_{50}, µg/L		
		Population pattern	Trophic pattern	No pattern
Phytoplankton	1-3	0.16	0.050	0.025
	4.7	0.06	0.050	0.025
	8-10	0.06	0.050	0.025
Zooplankton	11	0.50	0.057	0.025
	12	0.0099	0.057	0.025
	13	0.14	0.057	0.025
	14	0.25	0.057	0.025
	15	0.0035	0.057	0.025
Forage fish	16	0.63	1.2	0.025
	17	1.9	1.2	0.025
	18	1.6	1.2	0.025
Game fish	19	0.002	0.002	0.025

fraction, P_2, measured over a different time period, t_2. By simple rearrangement we find

$$C_2 = (C_1 t_1 \ln F_2)/(t_2 \ln F_1) \quad . \tag{6.1}$$

Using Eq. 6.1 we arrived at a single LC_{50} for each trophic level. The distribution of sensitivities shown in the second column of Table 6.2 will be referred to as the "trophic" pattern. In addition, we applied this approach once again to equate the trophic value and arrived at a single LC_{50} that removes even the trophic pattern. This value is shown in the last column of Table 6.2 and will be referred to as "no-pattern." By beginning with the no-pattern case, we can progressively add elements of toxic pattern into the simulations. In this way, we can analyze for the effect of the pattern of differential sensitivities.

Comparing the trophic with the no-pattern case, the upper half of Table 6.3 shows the percent difference in annual biomass of each trophic level. The results indicate the kind of indirect effect that one could reasonably expect to find in the ecosystem. The game fish is more sensitive than the no-pattern LC_{50} would indicate. The other trophic levels are relatively insensitive. Therefore, the toxicant reduces game fish population and has relatively less direct effect on other organisms. Because game fish are reduced, the forage fish experience less predation and show an increase. Because there are more forage fish, there are fewer zooplankton. Because there is less grazing, the phytoplankton increase.

Table 5.3. Comparisons of responses to different patterns of sensitivity to cadmium

Trophic vs no pattern	Percent difference
Phytoplankton	19.
Zooplankton	-19.
Forage fish	25.
Game fish	-33.
<u>Population vs trophic pattern</u>	
Phytoplankton	1.0
Zooplankton	-6.0
Forage fish	-4.0
Game fish	-4.0

The next step is to compare the trophic pattern with the full population pattern of toxic sensitivities. The percent difference between trophic and population response is shown in the lower portion of Table 6.3. The average phytoplankton population is larger, and the consumer trophic levels are always smaller when population-specific patterns of toxic sensitivity are ignored. Thus, the interactions that occur among differentially sensitive populations within a trophic level can affect the way the system responds to chemical stress.

Biotic interactions are important determinants of how the ecosystem will respond to stress. The results emphasize that predator-prey and competitive interactions are important determinants of system response to toxicants. Ignoring the way ecosystem processes interact with toxic stress can bias estimates of environmental risk.

6.4.4 Using SWACOM to Extrapolate Bioassays

An alternative to standard algal bioassay methods measures short-term effects on physiological processes. Photosynthesis can be measured simply and precisely and is more sensitive to low concentrations of some toxicants than population growth. In the study described here (Giddings et al. 1983), photosynthetic inhibition in algae was extrapolated to the ecosystem level using SWACOM to illustrate the potential risk of photosynthetic inhibition for the ecosystem as a whole. We considered a toxic impact of 7-d duration, introduced at various times during the year. On each date, we simulated a toxicant that caused a 50% reduction in the maximum photosynthetic rate and a 10% mortality on all consumer populations.

Mortality alone had little effect on the simulated pelagic ecosystem. When 50% inhibition was included in the deterministic solution of the model, the effects were much more pronounced with average changes approaching 25% if the stress began in day 170. Thus, the model indicates that even a temporary inhibition of photosynthesis can have an important effect on other populations in the ecosystem. The exercise demonstrates that the interdependence of populations in an ecosystem makes it possible for even temporary inhibition of algal photosynthesis to have a measurable impact on other organisms, particularly if the other organisms are also experiencing toxic effects.

Another implication of the ecosystem simulation is that the net effects of releasing a toxicant into the whole ecosystem depend on the state of the ecosystem at the time of release. The authors also infer that the effects on a population are, to a large extent, functions of the ecosystem of which the populations are a part. A single toxicological response may have a variety of expressions, depending on the ecosystem context. For example, the death of a fraction of a population may be inconsequential if the growth of the population is limited by intraspecific competition; reduced competition may compensate for the additional mortality. Conversely, a slight toxic effect may lead to complete elimination of the population by increasing its vulnerability to predators or reducing its ability to compete with other populations.

6.5 MONTE CARLO METHODS AND ANALYSIS

The essential feature of the ecosystem approach to risk analysis is to use models such as SWACOM to extrapolate information on toxic substances to the ecosystem level. There are many numerical techniques available to quantify the effect of uncertainties associated with such extrapolations (Rose and Swartzman 1981). Monte Carlo methods are particularly useful because they are easily implemented, and they provide the necessary information to estimate confidence intervals (Gardner et al. 1983).

Monte Carlo methods involve the iterative selection of random values for model parameters from specified frequency distributions, simulation of the model for each set of parameters, and analysis of the combined set of inputs and outputs (McGrath et al. 1975, Rubinstein 1981). Systematic sampling methods are more efficient than simple random sampling. We use quasi-orthogonal stratified random sampling methods (referred to as Latin Hypercube sampling) because (1) the estimates of output parameters (e.g., mean, median, and mode) are more precise (see McKay et al. 1979), (2) low rates of spurious relationships between randomly generated values are ensured (Iman and Conover 1982), and (3) computer codes exist for generating values from a variety of distributions.

We have implemented a program, PRISM (Gardner et al. 1983), especially written to perform Monte Carlo simulations for the estimation of risk indices. The program requires a FORTRAN subroutine of the model and an input file listing model parameters and their frequency distributions (e.g., normal, uniform, lognormal, etc.).

Multiple regression analysis of the Monte Carlo results provides an analysis of how the index is affected by assumptions required in extrapolating from laboratory to the ecosystem level (Downing et al. 1985). The contribution of each parameter to the regression sum of squares (i.e., the amount of the variability of y explained by a particular parameter) divided by the total sum of squares and multiplied by 100 forms an index, U, representing the percent variability of the model prediction explained by each parameter. The values of U range from 0.0 to 1.0, thus allowing a comparison between parameters. The adequacy of each index can be determined by comparison and by inspection of the R^2 statistic.

The classical sensitivity index, S (Tomovic 1963) analytically examines the relationships between model predictions and model parameters. This approach is limited by the difficulty of obtaining an analytical solution for many models and by its assumption of small instantaneous changes (Gardner et al. 1981). These difficulties have resulted in the proliferation of numerical and statistical approaches to uncertainty analysis (Hoffman and Gardner 1983).

If a single parameter is randomly varied from a prespecified probability distribution, then the slope of the regression of the model prediction on the parameter is the least-squares estimate of S if the parameter perturbations are very small (Gardner et al. 1981). If several parameters are simultaneously and independently varied, then a multiple regression on all the parameters simultaneously estimates all the sensitivities. The adequacy of this method of estimating linear relationships between model predictions and parameters can be evaluated

by inspection of R^2, the ratio of regression sum of squares to total sum of squares. If R^2 is nearly 1.0, then linear methods are adequate to describe the relationship between parameters and predictions. The divergency of R^2 from 1.0 indicates that nonlinear effects and interactions between parameters are important.

Any analysis that relates the importance of an input to a prediction without first removing the effects of the variability of other inputs (e.g., simple regression or correlations) is not very useful. Partial sum of squares (Draper and Smith 1966) determined by regression techniques are particularly useful because they quantitatively express relationships between each model input and output, with the effects of the variability of the remaining inputs statistically removed.

The partial sum of squares (PSS) represents the unique effect of each input on each prediction after correction of the total sum of squares because of the variability in all the other input variables. The PSS has the property that (1) the estimated effect does not involve other model inputs, (2) the estimates are invariant to the ordering of the calculation, and (3) the sums of squares calculated in this way do not add up to the total regression sum of squares, unless the inputs are orthogonal to each other.

If there are a large number of inputs, it is natural to ask if these could be replaced by a smaller number of inputs or some linear function of them, with a minimal loss of information in explaining the output. This problem was first investigated by Rao (1964) and termed principal components of instrumental variables.

Principal components of instrumental variables reduce to multiple regression in the case where there is only one main variable to predict. The coefficients of the multiple regression equation, when the variables are standardized, can be looked upon as importance coefficients, indicating which input variables are most important in influencing the output. Principal components are thus an extension of the multiple regression techniques when more than one output is examined simultaneously. The coefficients of the eigenvector indicate which input variables are most important, and the size of the eigenvalue determines how important that eigenvector is in explaining the variation we observe in the outputs.

6.6 DISCUSSION

The physiological process formulation of the growth equations in SWACOM provides the framework for extrapolation of acute toxicity data to estimates of likely effects of chemicals in aquatic ecosystems. Translation of mortality measurements to reductions in biomass production through the use of the General Stress Syndrome permits investigation of the implications of sublethal chemical effects on population dynamics calculated in an ecosystem context. The role of competitive and predator-prey interactions in mitigating or amplifying chemical effects can be examined through EUA (O'Neill et al. 1982, 1983). Statistical analyses of simulations used to estimate risk can identify the relative importance of direct vs indirect chemical effects as components of risk. Application of the methods to date encourage further evaluation and refinement of EUA.

Several areas for improvement in EUA are evident from our results. A more comprehensive collection of acute toxicity data could aid in the refinement of risk estimation. An examination of the relative contributions to risk identifies physiological processes that determine risk in specific applications. Risk estimates could be refined if bioassay protocols were modified to measure effects on physiological processes. For example, modification of acute assays for Daphnia, fathead minnows, or bluegills to measure changes in oxygen consumption during the course of the assay would provide direct data to test the GSS and estimate corresponding effects parameters for SWACOM.

The accuracy of risks estimated with EUA is a function of the applicability of SWACOM or other models to the systems of interest. SWACOM was designed to mimic the behavior of a northern dimictic lake. As the particular system of interest departs in its characteristics from those of a lake, SWACOM becomes less appropriate for risk estimation. In the case of chloroparaffins (CPs), low estimates of risk might underestimate the potential hazard of these chemicals. The propensity of CPs to accumulate in sediments might pose potential effects to benthic populations. SWACOM does not directly consider benthic populations or sediments. Again, SWACOM can be replaced with a more site-specific model to further refine estimates of risk. Even though absolute magnitudes of risk might be in error when the system of interest deviates substantially from a dimictic lake, SWACOM might still be used to compare relative risks for several different chemicals.

In EUA, risk is a function of both toxicity and the uncertainty in extrapolation from bioassay to natural systems. In the cases we have examined, the toxic effect has been more important than the uncertainty associated with the effects parameters (Bartell 1984). Nevertheless, the analyses would be considerably improved if more information were available on the field effects of toxicants. Future emphasis should focus on reducing the uncertainties associated with extrapolation so that attention can focus on the risks involved in ecosystem effects due directly to the toxicants.

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7. GENERAL DISCUSSION

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Combining exposure and effects estimates and interpreting the results requires considerable judgment on the part of the analyst. Among the key issues are matching spatiotemporal scales of exposure and effects models, interpreting uncertainties, and identifying "significant" risks. We cannot provide explicit procedures for addressing these issues because they will vary with each application. A discussion of how issues were addressed in the synfuels risk assessments should, however, provide some useful guidance. In addition to discussing the application of our approach in technology assessment, this section presents our views on (1) other potential applications to regulatory and resource management problems, and (2) critical research needs for the future development of ecological risk assessment.

7.1 SPATIOTEMPORAL SCALE IN THE INTEGRATION OF EXPOSURE AND EFFECTS

Superficially, integrating exposure and effects models appears to be a simple matter of estimating an environmental concentration and then comparing it with a toxicological benchmark or a concentration-response curve. However, the risk assessment may be meaningless if the spatiotemporal scale of the exposure assessment is improperly matched to the scale of the ecological effects of interest (and vice versa). Both short-term and long-term exposure assessments were used in synfuels risk assessments to address, respectively, acute effects and chronic effects of contaminant releases. A stochastic surface water fate model (Sect. 2) was used to estimate frequency distributions of

contaminant concentrations as functions of daily variability in important hydrological parameters. To assess risks of acute mortality during high-concentration episodes, 96-h LC_{50}s (both measured and extrapolated) were compared with 95th percentile contaminant concentrations (i.e., concentrations expected to be exceeded on 5% of days). To assess risks of chronic toxicity, MATCs and ecosystem risk functions were compared to seasonal average contaminant concentrations. In a site-specific assessment, seasonal dilution volumes could be matched to chronic benchmarks for the species and life stages present at the site.

Spatial scaling was not a significant problem in the synfuels risk assessments we performed. In the absence of detailed information on the spatial distribution of vulnerable resources, it was appropriate to use spatially homogeneous exposure and effects models. In site-specific risk assessments, however, spatial scales of both exposure estimates (deposition rates, surface concentrations) and effects measures (number or fraction of organisms affected, reduction in system productivity) must match the spatial resolution of distributional data for the exposed organisms. For reasons of scale, the models used in the synfuels risk assessment project may not be appropriate for site-specific assessments.

7.2 INTERPRETING UNCERTAINTY

As noted in Section 1, a major objective of risk assessment is to identify and quantify the uncertainties involved in extrapolating from experimental data on the environmental chemistry and toxicology of

contaminants to expected fate and effects in the field. We could not quantify all of these uncertainties. In risk assessment, there must always be a trade-off between uncertainties that are explicitly modeled and uncertainties that are consigned to expert judgment. At one extreme, it is possible to base assessments on simple toxicity quotients and safety factors without explicit treatment of uncertainty (Sect. 3). Although feasible, this approach provides no information about either the reliability of the assessment or the feasibility of improving it through research. At the other extreme, one can imagine developing an explicit model of all the physicochemical, physiological, and ecological processes that determine the fate and effects of a chemical and then assigning parameter distributions to each. We have argued elsewhere (Barnthouse et al. 1984, Suter et al. 1985, Barnthouse et al. in press) that current scientific understanding of natural populations and ecosystems is insufficient to support such an approach. In the synfuels risk assessment project, we attempted to identify the major classes of uncertainties involved in ecological risk assessment and to develop methods of addressing them without exceeding the limits of feasibility or scientific credibility.

We distinguish three qualitatively distinct sources of uncertainty in ecological risk assessment: inherent variability, parameter uncertainty, and model error. It is important to distinguish between these three sources, because they differ with respect to (1) feasibility of quantification and (2) degree of possible reduction through research or environmental monitoring.

7.2.1 Inherent Variability

Limits on the precision with which variable properties of the environment can be quantified limit the precision with which it is possible to predict the ecological effects of stress. The concentration of a contaminant in air or water varies unpredictably in space and time because of essentially unpredictable variation in meteorological parameters such as precipitation and wind direction. The spatiotemporal distributions and sensitivities to stress of organisms in nature are similarly variable. This variability can be quantified for many characteristics of the physical environment that influence the environmental fate of contaminants. For the synfuels risk assessment project, long-term hydrological records were used to estimate frequency distributions of contaminant concentrations in rivers (Sect. 2) as functions of daily variability in stream discharge, sediment load, and temperature.

Variable biological aspects of the environment are more difficult to quantify. Little is typically known, for example, about the variability of sensitivities among individuals in natural populations, and long-term records of variations in the abundance and distribution of organisms are uncommon. We did not quantify biological variability among individual organisms for the synfuels risk assessment project.

7.2.2 Parameter Uncertainty

Errors in parameter estimates introduce additional uncertainties into ecological risk estimates. Parameter values of interest may have to be estimated from structure-activity relationships (e.g., Kenaga and

Goring 1980, Veith et al. 1984) or from taxonomic correlations (e.g., Suter et al. 1983, Calabrese 1984). Even direct laboratory measurements are subject to errors (e.g., confidence limits on LC_{50}s and variation between replicate tests), although these are often unreported. Major efforts in the synfuels risk assessment project were devoted to quantifying uncertainties from this source. The methods described in Sections 4 and 5, for example, were specifically developed to quantify uncertainty due to (1) variations in sensitivity between taxonomic groups of organisms and (2) the variable relationship between acute and chronic toxicity. The ecosystem uncertainty analysis described in Section 6 was designed to translate uncertainties concerning effects of contaminants on individual species into uncertainties regarding ultimate ecological effects.

Unlike inherent variability, uncertainties due to parameter error can be reduced by increasing the precision of measurements or by replacing extrapolated parameter estimates with direct measurements. Comparisons of the relative contributions of different uncertainties to overall risk estimates provide guidance as to which parameters should be refined. The analyses described in Sections 4 and 5 show, for example, that uncertainty accumulated in predicting chronic effects of contaminants from acute LC_{50}s is far more important than is uncertainty resulting from interspecies extrapolation of acute LC_{50}s.

7.2.3 Model Error

Model errors constitute the least tractable source of uncertainty in risk assessment. Major types of model errors that have been

identified include (1) using a small number of variables to represent a large number of complex phenomena (termed aggregation error), (2) choosing incorrect functional forms for interactions among variables, and (3) setting inappropriate boundaries for the components of the world to be included in the model. The most serious problem associated with model error is that these errors frequently involve systematic biases whose magnitudes and directions may be difficult to determine One might naively think that the solution to model error is to disaggregate variables and increase the boundaries of the system until errors are eliminated. However, as has been noted by O'Neill (1973), there is a trade-off between model error and parameter error such that, the more variables and processes represented in a model, the greater the cost of data aquisition and the greater the opportunity for parameter error. For any model, a point is reached where adding additional variables and parameters reduces, rather than increases, the accuracy of model predictions.

Although model errors can never be completely eliminated, they can be bounded and reduced. The most straightforward method is to test the model against independent field data. However, the data necessary to perform such tests are difficult to collect and, when collected, are difficult to interpret. No matter how well a model performs for one set of environmental conditions, it is never possible to predict with certainty its applicability to a new set of conditions.

Empirical testing, although crucial in the long run for improving the models used in risk assessment (Mankin et al. 1975, National Research Council 1981), is unsuitable as a routine method of assessing

model errors. However, it is still possible to evaluate model assumptions by comparing of different models (Gardner et al. 1980). By comparing models that use different sets of assumptions, it is possible to assess how assumptions alter model output. This was the principal rationale for developing both statistical (Sects. 4 and 5) and ecological process (Sect. 6) models for the synfuels risk assessment project. Although this procedure does not ensure that model results will correspond to effects in the field, it can be used to distinguish between predictions that are robust to model assumptions and predictions that are highly sensitive to assumptions, and therefore susceptible to serious model errors (Levins 1966, Gardner et al. 1980). The strategy of comparing different risk models was used to identify potentially hazardous contaminants in the environmental risk assessments for indirect (Barnthouse et al. 1985a) and direct (Suter et al. 1984) coal liquefaction (see Sect. 7.3).

7.3 INTERPRETING ECOLOGICAL SIGNIFICANCE

The question of how large an ecological impact is significant has statistical, ecological, and societal components (Beanlands and Duinker 1983). In the synfuels risk assessment project, we considered statistical and societal components, respectively, by using probabilistic risk models and by defining end points in terms of societally valued environmental attributes. No generally applicable definition of ecological significance has ever been formulated (Beanlands and Duinker 1983); therefore, definitions must be developed

in the context of particular assessment objectives. We developed operational definitions of ecological significance based on the primary objective of the project, that is, the identification of synfuels-related contaminant classes having the greatest potential for adverse ecological effects. Our strategy for assessing significance involved (1) defining, for each effects method used, a criterion below which risks would be considered insignificant, (2) counting, for each contaminant class studied, the number of methods by which it was judged "significant"; and (3) explaining, where possible, the failures of the three methods to agree.

For the quotient method (Sect. 3), the significance criterion used was an acute-effects quotient greater than 0.01, that is, a lowest observed LC_{50} less than two orders of magnitude greater than the estimated environmental concentration. This criterion has sometimes been used in hazard assessments for toxic chemicals. For analysis of extrapolation error, potential ecological effects of a contaminant were considered significant if the risk that the environmental concentration may exceed the MATC of one or more reference fish species is greater than 0.1. This value was chosen to avoid (1) being overly conservative and (2) relying on risk estimates obtained from the tails of the probability distributions for MATCs, where the reliability of extrapolation is most questionable. For ecosystem uncertainty analysis, contaminants were considered to pose significant risks if the risk of a 25% reduction in game fish biomass is greater than 0.1. This value was selected on the basis that risks should be at least twice as high as

the background risk resulting from environmental variability incorporated in SWACOM (about 0.04) before they are considered significant.

Assessments of the aquatic end points in indirect coal liquefaction (Barnthouse et al. 1985a) provide an illustration of our procedure (only toxicity quotients were used to assess terrestrial end points). For the fish end point, comparisons between risk estimates obtained from all three risk methods were possible. Using at least one of the three methods (Table 7.1), nine contaminant categories were determined to pose potential risks to fish populations. The nine were identified as the classes most appropriate for refined risk assessments and/or further research. Four contaminant classes, all trace elements or conventional industrial pollutants (hydrogen sulfide and ammonia), were found significant by two or more methods and identified as the contaminants of greatest concern.

For the phytoplankton end point, only nickel and cadmium were judged significant using toxicity quotients. However, using ecosystem uncertainty analysis, these elements, along with three other heavy metals, and ammonia were all judged significant This result required explanation in that, although all of the contaminants studied are potentially toxic to phytoplankton, the end point in ecosystem uncertainty analysis is defined as a fourfold increase in peak phytoplankton biomass. An inspection of the model output revealed that indirect effects of contaminants on fish and zooplankton, rather than direct effects on phytoplankton, were responsible for the results.

Table 7.1. Contaminant classes determined to pose potentially significant risks to fish populations by one or more of three risk analysis methods: quotient method (QM), analysis of extrapolation error (AEE), and ecosystem uncertainty analysis (EUA). Separate lists were developed for treated aqueous waste streams from two indirect coal liquefaction processes. From Barnthouse et al. (1985)

Lurgi/Fischer-Tropsch process	Koppers-Totzek/Fischer-Tropsch process
(acid gases) - QM, AEE	(acid gases) - QM, AEE
(alkaline gases) - QM, AEE, EUA	(alkaline gases) - QM, AEE, EUA
(volatile carboxylic acids) - AEE	(volatile carboxylic acids) - QM, AEE
(carboxylic acids, excluding volatiles) - AEE	(cadmium) - QM, AEE, EUA
(arsenic) - AEE	
(mercury) - AEE, EUA	
(nickel) - EUA	
(cadmium) - QM, AEE, EUA	

7.4 OTHER APPLICATIONS OF ECOLOGICAL RISK ASSESSMENT

We have not claimed to accurately predict the magnitudes of ecological risks associated with toxic chemicals, whether or not associated with synfuels production. However, even without such predictions, applications of the concept of risk and, in some cases, the methods described in this report can substantially improve current approaches to environmental decision-making. By (1) emphasizing probabilities and frequencies of events and (2) explicitly quantifying uncertainty, risk assessment can provide a more rational basis for decisions that may otherwise be highly subjective.

For example, frequency distributions of ambient contaminant concentrations can be used to forecast water quality impacts or compliance with standards. For any given benchmark concentration (e.g., an ambient air or water quality criterion), the probability of exceeding the benchmark can be read from the cumulative distribution function in Fig. 7.1(a). The presentation of such functions would enhance the quality of environmental impact assessments, which commonly are based on worst-case analyses (e.g., 7-d, 10-year low flow) of questionable ecological significance. If the benchmark concentration is an action level above which contaminant discharges are not permitted, then Fig. 7.1(a) could be used to estimate the frequency of days on which action would be required. Probabilistic environmental fate models that could be used for this purpose already exist (e.g., Parkhurst et al. 1981, Travis et al. 1983).

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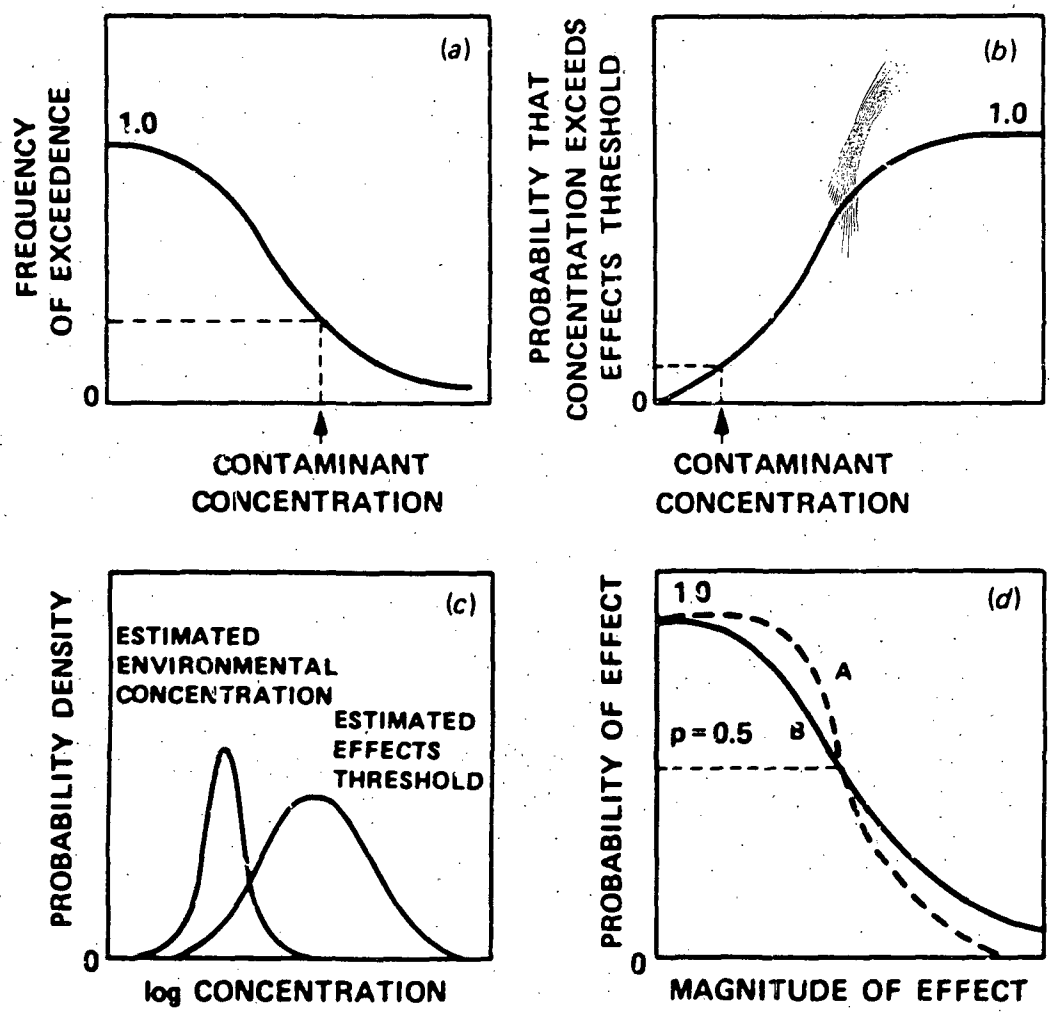

Fig. 7.1. Four applications of ecological risk functions. In (a), a cumulative frequency function is used to estimate the frequency with which the environmental concentration of a contaminant will exceed an "action" concentration. In (b), a cumulative probability function for the effects threhsold of a hypothetical organism is used to select an action concentration with a 5% chance of exceeding the true effects threshold. In (c), probability density functions for two components of a risk estimate are compared to identify the component with the greater uncertainty. In (d), the risks of adverse effects of different magnitudes are compared for two alternative facility designs. The expected effects of the two alternatives are the same, but alternative B presents greater risks of severe adverse effects.

Risk estimates could also be used to set standards based on probabilities of exceeding effects thresholds. Section 4 of this report describes a method for calculating probability distributions for acute LC_{50}s and MATCs. Figure 7.1(b) presents such a distribution plotted as a cumulative probability function. Using this curve, the allowable ambient concentration of a contaminant might be set so that the risk of exceeding the threshold level is 5%. Figure 7.1(b) could also be used to define the decision points in tiered hazard assessment schemes. In this application, the decision to perform further tests on a chemical would be determined by the risk of exceeding an LC_{50} or MATC, and by the reduction in uncertainty expected to result from acquisition of additional test data.

If the contributions to total uncertainty of different components of a risk estimate can be compared, then research effort can be concentrated on the component(s) contributing the greatest uncertainty. For example, in Fig. 7.1(c), uncertainty about the environmental concentration of a contaminant is compared with uncertainty concerning its effects threshold. The relative variances of the two distributions correspond roughly to the variances estimated by Suter et al. (1983) for largemouth bass exposed to mercury released from a hypothetical indirect coal liquefaction plant. Barnthouse et al. (1985b) used comparisons between variances of MATCs and of environmental concentrations estimated for 23 synfuels-related contaminants to argue that, in general, uncertainty concerning effects thresholds for contaminants is much larger than uncertainty concerning environmental fate.

Decisions concerning alternative plant sites and mitigating technologies could be facilitated by using risk curves like those shown in Fig. 7.1(d). Such curves provide information about both the expected effects of an action (e.g., building a plant or licensing a chemical) and the risk of extremely large effects. Risk curves are commonly used to assess safety-related risks (e.g., comparing automobile travel to airplanes or earthquakes to nuclear power plant accidents); we see no reason why they could not also be used to assess ecological risks.

7.5 CRITICAL RESEARCH NEEDS

Given the immaturity of the art of risk assessment, it would be possible to list dozens of research topics that would enhance our capabilities. Through the application of risk assessment concepts to synfuels technologies, we have identified four deficiencies that we think are especially critical: (1) insufficient understanding of chronic effects of toxic chemicals, (2) insufficient data on effects of contaminants on invertebrates, (3) poor standardization of toxicity test systems for aquatic and terrestrial plants, and (4) insufficient validation of ecological risk models.

Most exposures of organisms to toxic contaminants are chronic rather than acute. However, most research and toxicity testing to date has been directed at acute exposures. We have shown in Sections 4 and 5 of this report that, at least for fish and probably also for aquatic invertebrates, it is possible to extrapolate from acute effects to

MATCs and even to population-level effects of chronic exposures. The uncertainties associated with this extrapolation are very large, presumably because the relationship between effective concentrations for acute vs chronic effects is highly variable. Significant reductions in uncertainty could be obtained if more effort were devoted to chronic toxicity testing and to understanding the physiological mechanisms responsible for chronic toxicity. In contrast, acute effects of contaminants on fish are well studied, and our research (Sect. 4) has shown that acute effects of contaminants on one fish species can be extrapolated to other fish species with a relatively low degree of uncertainty (i.e., within an order of magnitude).

A redressing of the imbalance in testing effort between fish and invertebrates is needed. Modeling studies performed using SWACOM (Sect. 6) suggest that differences in sensitivity between and within trophic levels in aquatic ecosystems can cause responses that are qualitatively different from those predicted on the basis of a few standard species. Although invertebrates are both taxonomically and physiologically more diverse than fish, more aquatic toxicity data is available for fish than for invertebrates. Moreover, most testing of invertebrate responses is restricted to a small set of standard organisms (e.g., *Daphnia magna*).

Lack of comparability of test systems limits the possibility of any meaningful risk assessments for plants and especially terrestrial vegetation. Suitable test systems for phytoplankton are available, all that is required is a standardization of end points. For terrestrial plants, interpretability is an even greater problem than comparability.

Many systems are of severely limited utility for risk assessment because of the near impossibility of relating the test end points (e.g., reductions in root elongation rates) to meaningful ecological end points. Readily interpretable data are available only for major combustion products, such as ozone and SO_x.

Lack of validation of ecological risk models, especially ecosystem models, is perhaps the greatest single limitation on the future development of ecological risk assessment. The Standard Water Column Model, a model of the pelagic zone of a northern dimictic lake, was used to develop ecosystem uncertainty analysis (Sect. 6), not because such lakes are relevant to synfuels risk assessment, but because northern dimictic lakes are by far the best understood aquatic ecosystems. The model itself has not been rigorously validated, but the functional components of the model have been validated through more than a century of limnological research. Because of the great expense and difficulty of site-specific modeling efforts, it is likely that ecosystem-level risk assessments will always be limited primarily to site-independent purposes, such as identifying particular contaminants or contaminant classes with the potential for causing indirect ecological effects. Even for this more limited purpose, validation studies are needed. At a minimum, the existing case studies on ecological effects of toxic chemicals should be synthesized to determine how frequently indirect effects have been observed and to identify the ecological processes (e.g., prey switching or reductions in primary production) responsible.

Ecological risk assessment methods inevitably represent a compromise between the ideal and the possible. Ideally, we would like to quantify effects of toxic contaminants on valued ecosystem components in any environment of interest, based on an understanding of fundamental chemical, physiological, and ecological processes. Statistical models and generic ecosystem models, such as those described in this report, would then be unnecessary. Until breakthroughs in fundamental understanding are achieved, however, we believe that the most appropriate strategy for improving our capability in ecological risk assessment is the strategy pursued in the synfuels risk assessment project, that is, incremental extension of the existing state of the art in ecotoxicology and ecology.

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APPENDIX A

Acute and Chronic Effects Data Used in Analysis of Extrapolation Error

Table A.1. LC_{50}/MATC data set (units are µg/L)

OBS	CHEMICAL	SOURCE	SPECIES	CLASS	TYPE	LC50	NOEC	LOEC	MATC
1	AC 222,705	SPEHAR ET AL. 1983	FM	PY	ELS	0.22	0.03	0.07	0.0
2	ACENAPHTHENE	CAIRNS AND NEBEKER 1982	FM	PA	ELS	608	345	495	413.2
3	ACENAPHTHENE	LEMKE ET AL. 1983	FM	PA	ELS		139.5	274	195.5
4	ACROLEIN	MACEK ET AL. 1976C	FM	HC	LC	84	11.4	41.7	21.8
5	AG	DAVIES ET AL. 1978	RT	M	ELS	6.5	0.09	0.17	0.1
6	AG	NEBEKER ET AL. 1983	RT	M	ELS	9.2	<0.1		
7	AG SULFIDE GELL	LEBLANC ET AL. 1984	FM		ELS	>240		>11000	
8	AG THIOSULFATE COMPLEX	LEBLANC ET AL. 1984	FM		ELS	>280	16000	35000	23664.3
9	ALACHLOR	CALL ET AL. 1983	FM	OC	ELS	5000	520	1100	756.3
10	ALDICARB	PICKERING AND GILIAM 1982	FM	CB	ELS	1370	78	156	110.3
11	AROCLOR1242	NEBEKER ET AL. 1974	FM	OC	LC	300	5.4	15	9.0
12	AROCLOR1248	DEFOE ET AL. 1978	FM	OC	LC		0.1	0.4	0.2
13	AROCLOR1248	NEBEKER ET AL. 1974	FF	OC	LC		2.2	5.1	3.3
14	AROCLOR1254	NEBEKER ET AL. 1974	FM	OC	LC	>33	0.52	1.8	1.0
15	AROCLOR1260	DEFOE ET AL. 1978	FM	OC	LC		<0.1		
16	AS	BIDDINGER 1981	JM		LC	30200	2500	5000	3535.5
17	AS	CALL ET AL. 1983B	FF		ELS	14400	2130	4120	2962.4
18	AS	CALL ET AL. 1983B	FM		ELS	14200	2130	4300	3026.4
19	ATRAZINE	MACEK ET AL. 1976B	BG	ON	LC	6700	95	500	217.9
20	ATRAZINE	MACEK ET AL. 1976B	BT	ON	LC	4900	65	120	88.3
21	ATRAZINE	MACEK ET AL. 1976B	FM	ON	LC	15000	213	870	430.5
22	BENZOPHENONE	CALL ET AL. 1985	FM	N	ELS	14800	540	990	731.2
23	BROMACIL	CALL ET AL. 1983	FM	ON	ELS	182000	<1000		
24	CAPTAN	HERMANUTZ ET AL. 1973	FM	OS	LC	65	16.5	39.5	25.5
25	CARBARYL	CARLSON 1971	FM		LC	9000	210	680	377.9
26	CD	BENOIT ET AL. 1976	BT	M	LC		1.7	3.4	2.4
27	CD	CARLSON ET AL. 1982	FF	M	LC		3.3	7.4	4.9
28	CD	EATON ET AL. 1978	BNT	M	ELS		3.8	11.7	6.7
29	CD	EATON ET AL. 1978	BT	M	ELS		1.1	3.8	2.0
30	CD	EATON ET AL. 1978	COS	M	ELS		4.1	12.5	7.2
31	CD	EATON ET AL. 1978	LT	M	ELS		4.4	12.3	7.4
32	CD	EATON ET AL. 1978	MP	M	ELS		4.2	12.9	7.4
33	CD	EATON ET AL. 1978	SB	M	ELS		4.3	12.7	7.4
34	CD	EATON ET AL. 1978	WS	M	ELS		4.2	12.0	7.1
35	CD	EATON 1974	BG	M	LC	21100	31	80	49.8
36	CD	PICKERING AND GAST 1972	FM	M	LC	7200	37	57	45.9
37	CD	SAUTER ET AL. 1976	BT	M	ELS		1	3	1.7
38	CD	SAUTER ET AL. 1976	CC	M	ELS		11	17	13.7
39	CD	SAUTER ET AL. 1976	WE	M	ELS		9	25	15.0
40	CD	SPEHAR 1976	FF	M	LC	2500	4.1	8.1	5.8
41	CHLORAMINE	ARTHUR AND EATON 1971	FM		LC	114	16	35	23.7
42	CHLORDANE	CARDWELL ET AL. 1977	BG	OC	LC	59	1.22	2.20	1.6
43	CHLORDANE	CARDWELL ET AL. 1977	BT	OC	LC	47	<0.32		
44	CN	LEDUC 1978	AS		ELS		<0.01		
45	CN	SMITH ET AL. 1979	BG		LC	120	<5.2		

Table A.1 (Continued)

OBS	CHEMICAL	SOURCE	SPECIES	CLASS	TYPE	LC50	NOEC	LOEC	MATC
46	CN	SMITH ET AL. 1979	BT		PLC	68.3	5.7	11.2	8.0
47	CN	SMITH ET AL. 1979	FM		LC	129	12.9	19.6	15.9
48	CNSO4	HAZEL AND MEITH 1970	CHS		ELS		<0.02		
49	CR	BENOIT 1976	BT	M	LC	59000	200	350	264.6
50	CR	BENOIT 1976	RT	M	LC	69000	200	350	264.6
51	CR	PICKERING 1980	FM	M	LC	36900	1000	3950	1987.5
52	CR	SAUTER ET AL. 1976	BG	M	ELS		522	1122	765.3
53	CR	SAUTER ET AL. 1976	CC	M	ELS		150	305	213.9
54	CR	SAUTER ET AL. 1976	LT	M	ELS		105	194	142.7
55	CR	SAUTER ET AL. 1976	NP	M	ELS		538	963	719.8
56	CR	SAUTER ET AL. 1976	RT	M	ELS		51	105	73.2
57	CR	SAUTER ET AL. 1976	WE	M	ELS			>2167	
58	CR	SAUTER ET AL. 1976	WS	M	ELS		290	538	395.0
59	CR	STEVENS AND CHAPMAN 1984	RT	M	ELS	4400	48	89	65.4
60	CU	BENOIT 1975	BG	M	LC	1100	21	40	29.0
61	CU	HORNING AND NEIHCISEL 1979	BM	M	LC	230	4.3	18	8.8
62	CU	MCKIM AND BENOIT 1971	BT	M	LC	100	9.5	17.4	12.9
63	CU	MCKIM AND BENOIT 1974	BT	M	LC			>9.4	
64	CU	MCKIM ET AL. 1978	BNT	M	ELS		22.3	44.5	31.5
65	CU	MCKIM ET AL. 1978	BT	M	ELS		21.5	43.5	30.6
66	CU	MCKIM ET AL. 1978	LT	M	ELS		22.0	42.3	30.5
67	CU	MCKIM ET AL. 1978	NP	M	ELS		34.9	104.4	60.4
68	CU	MCKIM ET AL. 1978	RT	M	ELS		11.4	31.7	19.0
69	CU	MCKIM ET AL. 1978	WS	M	ELS		12.9	33.8	20.9
70	CU	MOUNT AND STEPHAN 1969	FM	M	LC	75	10.6	18.4	14.0
71	CU	MOUNT 1968	FM	M	LC	470	14.5	33	21.9
72	CU	PICKERING ET AL. 1977	FM	M	LC	460	38	60	47.7
73	CU	SAUTER ET AL. 1976	BT	M	ELS		3	5	3.9
74	CU	SAUTER ET AL. 1976	CC	M	ELS		12	18	14.7
75	CU	SAUTER ET AL. 1976	WE	M	ELS		13	21	16.5
76	CU	SEIM ET AL. 1984	RT	M	ELS	80	16	31	22.3
77	DDT	JARVINEN ET AL. 1977	FM	OC	LC	48	0.5	2.0	1.0
78	DI-N-BUTYL PHTHALATE	MCCARTHY AND WHITMORE 1985	FM	N	ELS		560	1000	748.3
79	DI-N-OCTYL PHTHALATE	MCCARTHY AND WHITMORE 1985	FM	N	ELS		3200	10000	5656.9
80	DIAZINON	ALLISON AND HERMANUTZ 1977	BT	OP	PLC	770	<0.55		
81	DIAZINON	ALLISON AND HERMANUTZ 1977	FM		LC	7800	3.2	13.5	6.6
82	DIAZINON	JARVINEN AND TANNER 1982	FM	OP	ELS	690	50	90	67.1
83	DINOSEB	CALL ET AL. 1983	FM	ON	ELS	700	14.5	48.5	26.5
84	DINOSEB	WOODWARD 1976	LT	ON	NS	79	<0.5		
85	DIUROM	CALL ET AL. 1983	FM	ON	ELS	14200	33.4	78	51.0
86	DTDMAC	LEWIS AND WEE 1983	FM	S	ELS		53	90	69.1
87	DURSBAN	JARVINEN AND TANNER 1982	FM	OP	ELS	140	1.6	3.2	2.3
88	ENDOSULFAN	CARLSON ET AL. 1982	FM	OC		0.86			
89	ENDOSULFAN	MACEK ET AL. 1976C	FM	OC	LC	0.86	0.2	0.4	0.3
90	ENDRIN	CARLSON ET AL. 1982	FM	OC	NS				

Table A.1 (Continued)

OBS	CHEMICAL	SOURCE	SPECIES	CLASS	TYPE	LC50	NOEC	LOEC	MATC
91	ENDRIN	HERMANUTZ 1978	FF	OC	LC	0.85	0.22	0.3	0.3
92	ENDRIN	JARVINEN AND TYO 1978	FM	OC	LC		<0.17		
93	ETHYLBENZENE	EPA 1980A	FM	N	ELS	45300		>440	
94	FENITROTHION	KLEINER ET AL. 1984	FM	CX	ELS		130	300	197.5
95	FONOFOS	PICKERING AND GILLIAM 1982	FM	OP	ELS	1090	16	33	23.0
96	FURAN	CALL ET AL. 1985	FM	N	ELS	60676	8270	12200	10044.6
97	GUTHION	ADELMAN ET AL. 1976	FM	OP	LC		0.33	0.51	0.4
98	HEPTACHLOR	MACEK ET AL. 1976C	FM		LC	7	0.86	1.84	1.3
99	HEXACHLOROBUTADIENE	BENOIT ET AL. 1982	FM	OC	ELS	102	6.5	13	9.2
100	HEXACHLOROCYCLOHEXANE	MACEK ET AL. 1976A	BG	N	LC	30	9.1	12.5	10.7
101	HEXACHLOROCYCLOHEXANE	MACEK ET AL. 1976A	BT	N	LC	26	8.8	16.6	12.1
102	HEXACHLOROCYCLOHEXANE	MACEK ET AL. 1976A	FM	N	LC	69	9.1	23.5	14.6
103	HEXACHLOROETHANE	AHMED ET AL. 1984	FM	N	ELS	1510	69	207	119.5
104	HEXACHLOROPENTADIENE	EPA 1980B	FM	N	ELS	7.0	3.7	7.3	5.2
105	HG	CALL ET AL. 1983B	FM	M	ELS	150	<0.23		
106	HG	SNARSKI AND OLSON 1982	FM	M	LC	168	<0.26		
107	ISOPHORONE	CAIRNS AND NEBEKER 1982	FM	HC	ELS	145000	56000	112000	79196.0
108	ISOPHORONE	LEMKE ET AL. 1983	FM	HC	ELS	145000	8535	15610	11542.6
109	KELTHANE	SPEHAR ET AL. 1982	FM	OC	ELS		19	39	27.2
110	KEPONE	BUCKLER ET AL. 1981	FM	OC	LC	340	1.2	3.1	1.9
111	LAS MIXTURE	PICKERING AND THATCHER 1970	FM	S	LC	4350	630	1200	869.5
112	LAS 11.2	HOLMAN AND MACEK 1980	FM	S	ELS	12300	5100	8400	6545.2
113	LAS 11.7	HOLMAN AND MACEK 1980	FM	S	LC	4100	480	490	485.0
114	LAS 13.3	HOLMAN AND MACEK 1980	FM	S	LC	860	110	250	165.8
115	MALATHION	EATON 1970	BG	OP	LC	110	3.6	7.4	5.2
116	MALATHION	EATON 1970	FM	OP	LC	10500	200	580	340.6
117	MALATHION	HERMANUTZ 1978	FF		LC	349	8.6	10.9	9.7
118	METHYL PARATHION	JARVINEN AND TANNER 1982	FM	OP	ELS		310	380	343.2
119	METHYLMERCURIC CHLORIDE	MCKIM ET AL. 1976	BT	OM	LC	75	0.29	0.93	0.5
120	METHYLMERCURIC CHLORIDE	MCKIM 1977	FF	OM	LC	240	0.17	0.33	0.2
121	METHYLMERCURIC CHLORIDE	MCKIM 1977	FM	OM	LC	65	0.07	0.13	0.1
122	MIREX	BUCKLER ET AL. 1981	FM	OC	LC	750	7	13	9.5
123	NAPTHALENE	DEGRAEVE ET AL. 1982	FM	HC	ELS	7900	450	850	618.5
124	NI	PICKERING 1974	FM	M	LC	27000	380	730	526.7
125	PB	DAVIES ET AL. 1976	RT	M	ELS	1170	4.1	7.6	5.6
126	PB	HOLCOMBE ET AL. 1976	BT	M	LC	4100	58	119	83.1
127	PB	MCKIM 1977	FF	M	LC	2750	31.2	62.5	44.2
128	PB	SAUTER ET AL. 1976	BG	M	ELS		70	120	91.7
129	PB	SAUTER ET AL. 1976	CC	M	ELS		75	136	101.0
130	PB	SAUTER ET AL. 1976	LT	M	ELS		48	83	63.1
131	PB	SAUTER ET AL. 1976	NP	M	ELS		253	483	349.6
132	PB	SAUTER ET AL. 1976	RT	M	ELS		71	146	101.8
133	PB	SAUTER ET AL. 1976	WS	M	ELS		119	253	173.5
134	PENTACHLOROETHANE	AHMED ET AL. 1984	FM	N	ELS	7340	900	1400	1122.5
135	PENTACHLOROPHENOL	HOLCOMBE ET AL. 1982	FM	OC	ELS		44.9	73.0	57.3

Table A.1 (Continued)

OBS	CHEMICAL	SOURCE	SPECIES	CLASS	TYPE	LC50	NOEC	LOEC	MATC
136	PERMETHRIN	SPEHAR ET AL. 1983	FM	PY	ELS	15.6	0.66	1.4	1.0
137	PHENOL	DEGRAEVE ET AL. 1980	FM	HC	ELS	24900	750	2500	1369.3
138	PHENOL	DEGRAEVE ET AL. 1980	RT	HC	ELS	8900	<200		
139	PHENOL	HOLCOMBE ET AL. 1982	FM	HC	ELS		1830	3570	2556.0
140	PHENOLS	DAUBLE ET AL. 1993	FM	HC	ELS,R		130	250	180.3
141	PHENOLS	DAUBLE ET AL. 1983	RT	HC	ELS		<130		
142	PICLORAM	WOODWARD 1976	LT	CX	ELS	1850	<35		
143	PROPANIL	CALL ET AL. 1983	FM	DN	ELS	8600	0.4	0.6	0.5
144	PYDRIN	SPEHAR ET AL. 1982	FM	PY	ELS		.19	.33	0.3
145	SODIUM NITRILOTRIACETATE	ARTHUR ET AL. 1974	FM	S	LC	114000		>54000	
146	T-1,2-DICHLOROCYCLOHEXANE	CALL ET AL. 1985	FM	N	ELS	18400	610	980	773.2
147	TETRACHLOROETHYLENE	AHMED ET AL. 1984	FM	N	ELS	13400	1400	2800	1979.9
148	TETRAHYDROFURAN	CALL ET AL. 1985	FM	N	ELS	2160000	216000	367000	281552.8
149	TOXAPHENE	MAYER ET AL. 1975	BT	OC	LC	10.8	<0.039		
150	TOXAPHENE	MAYER ET AL. 1977	CC	OC	LC	16.5	0.129	0.299	0.2
151	TOXAPHENE	MAYER ET AL. 1977	FM		LC	7.2	0.025	0.054	0.0
152	TRIFLURALIN	MACEK ET AL. 1976C	FM	ON	LC	115	1.95	5.1	3.2
153	VANADIUM	HOLDWAY AND SPRAGUE 1979	FF	M	LC	11200	80	170	116.6
154	ZEOLITE, TYPE A	MAKI AND MACEK 1978	FM		ELS	>860000		>86700	
155	ZN	BENOIT AND HOLCOMBE 1978	FM	M	LC	600	78	145	106.3
156	ZN	BRUNGS 1969	FM	M	LC	9200	30	180	73.5
157	ZN	HOLCOMBE ET AL. 1979	BT	M	LC	2000	534	1360	852.2
158	ZN	PIERSON 1981	G	M	LC	5800	<113		
159	ZN	SINLEY ET AL. 1974	RT	M	LC	430	140	260	190.8
160	ZN	SPEHAR 1976	FF	M	LC	1500	26	51	36.4
161	1,1,2-TRICHLOROETHANE	AHMED ET AL. 1984	FM	N	ELS	81600	6000	14800	9423.4
162	1,1,2,2-TETRACHLOROETHANE	AHMED ET AL. 1984	FM	N	ELS	20400	1400	4000	2366.4
163	1,2-DICHLOROBENZENE	EPA 1980C	FM	N	ELS		1600	2500	2000.0
164	1,2-DICHLOROETHANE	BENOIT ET AL. 1982	FM	N	ELS	118000	29000	59000	41364.2
165	1,2-DICHLOROPROPANE	BENOIT ET AL. 1982	FM	N	ELS	139000	6000	11000	8124.0
166	1,2,3,4-TETRACHLOROBENZE	AHMED ET AL. 1984	FM	N	ELS	1070	245	412	317.7
167	1,2,4-TRICHLOROBENZE	AHMED ET AL. 1984	FM	N	ELS	2760	499	1001	706.8
168	1,3-DICHLOROBENZENE	AHMED ET AL. 1984	FM	N	ELS	7190	2263	1000	1505.7
169	1,3-DICHLOROPROPANE	BENOIT ET AL. 1982	FM	N	ELS	131000	8000	16000	11313.7
170	1,3-DICHLOROPROPENE	EPA 1980D	FM	N	ELS		180	330	243.7
171	1,4-DICHLOROBENZENE	AHMED ET AL. 1984	FM	N	ELS	4160	565	1040	766.6
172	1,4-DIMETHOXYBENZENE	CALL ET AL. 1985	FM	N	ELS	117600	16600	27400	21327.0
173	2,4-DICHLOROPHENOL	HOLCOMBE ET AL. 1982	FM	OC	ELS		290	460	365.2
174	2,4-DIMETHYLPHENOL	HOLCOMBE ET AL. 1982	FM	HC	ELS		1970	3110	2475.2
175	3,4-DICHLOROTOLUENE	CALL ET AL. 1985	FM	N	ELS	2910	78	148	107.4
176	4-BROMOPHENYLPHENYL ETHER	EPA 1980E	FM	N	ELS		89	167	121.9
177	4-METHYL-2-PENTANONE	CALL ET AL. 1985	FM	N	ELS	505000	57000	105000	77362.8

SPECIES = Species of test organism: AS = atlantic salmon, BG = bluegill, BM = bluntnose minnow, BNT = brown trout, BT = brook trout, CC = channel catfish, CNS = chinook salmon, COS = coho salmon, FF = flagfish, FM = fathead minnow, G = guppy, JM - Japanese medaka, LT = lake trout, NP = northern pike, RT = rainbow trout, SB = smallmouth bass, WE = walleye, and WS = white sucker.
CLASS = Chemical class : CB = carbamate pesticide, CX = carboxylate herbicide, HC = hydrocarbon, M = metal, N = narcotic, OC = organochloride, OP = organophosphate pesticide, OS = organosulfur, PA = polycyclic aromatic hydrocarbon, and PY = pyrethyroid pesticide.
TYPE = The types of tests included: LC = life-cycle or partial life cycle, ELS = early life stage.
LC_{50} = A 96-h median lethal concentration determined in the same study as the corresponding MATC, or at least in the same laboratory using the same water.
NOEC = No observed effects concentration.
LOEC = Lowest observed effects concentration.

APPENDIX B

Concentration-Response Data Sets from Chronic Toxicity Experiments

Table B.1 Concentration-Response Data Set

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1	ACENAPHTHENE	FM	MORTS	0.00	30	6			CAIRNS AND NEBEKER 1982
2	ACENAPHTHENE	FM	MORTS	197.00	37	5			CAIRNS AND NEBEKER 1982
3	ACENAPHTHENE	FM	MORTS	345.00	33	4			CAIRNS AND NEBEKER 1982
4	ACENAPHTHENE	FM	MORTS	509.00	32	9			CAIRNS AND NEBEKER 1982
5	ACENAPHTHENE	FM	MORTS	682.00	33	18			CAIRNS AND NEBEKER 1982
6	ACENAPHTHENE	FM	MORTS	1153.00	33	32			CAIRNS AND NEBEKER 1982
7	ACENAPHTHENE	FM	WEIGHT	0.00				0.02	CAIRNS AND NEBEKER 1982
8	ACENAPHTHENE	FM	WEIGHT	197.00				0.02	CAIRNS AND NEBEKER 1982
9	ACENAPHTHENE	FM	WEIGHT	345.00				0.02	CAIRNS AND NEBEKER 1982
10	ACENAPHTHENE	FM	WEIGHT	509.00				0.02	CAIRNS AND NEBEKER 1982
11	ACENAPHTHENE	FM	WEIGHT	682.00				0.01	CAIRNS AND NEBEKER 1982
12	ACENAPHTHENE	FM	WEIGHT	1153.00				0.00	CAIRNS AND NEBEKER 1982
13	ACENAPHTHENE	FM	WEIGHT	0.00				0.20	LEMKE ET AL 1983
14	ACENAPHTHENE	FM	WEIGHT	69.50				0.18	LEMKE ET AL 1983
15	ACENAPHTHENE	FM	WEIGHT	139.50				0.19	LEMKE ET AL 1983
16	ACENAPHTHENE	FM	WEIGHT	274.00				0.15	LEMKE ET AL 1983
17	ACENAPHTHENE	FM	WEIGHT	533.50				0.13	LEMKE ET AL 1983
18	ACENAPHTHENE	FM	WEIGHT	1025.50				0.08	LEMKE ET AL 1983
19	ACROLEIN	FM	HATCH	0.00	500	44			MACEK ET AL 1976C
20	ACROLEIN	FM	HATCH	4.60	750	118			MACEK ET AL 1976C
21	ACROLEIN	FM	HATCH	6.40	600	76			MACEK ET AL 1976C
22	ACROLEIN	FM	HATCH	11.40	600	114			MACEK ET AL 1976C
23	ACROLEIN	FM	HATCH	41.70	250	48			MACEK ET AL 1976C
24	ACROLEIN	FM	MORT1	0.00	30	2			MACEK ET AL 1976C
25	ACROLEIN	FM	MORT1	4.60	30	4			MACEK ET AL 1976C
26	ACROLEIN	FM	MORT1	6.40	30	7			MACEK ET AL 1976C
27	ACROLEIN	FM	MORT1	11.40	30	2			MACEK ET AL 1976C
28	ACROLEIN	FM	MORT1	20.80	15	5			MACEK ET AL 1976C
29	ACROLEIN	FM	MORT1	41.70	30	2			MACEK ET AL 1976C
30	ACROLEIN	FM	MORT2	0.00	160	77			MACEK ET AL 1976C
31	ACROLEIN	FM	MORT2	4.60	160	76			MACEK ET AL 1976C
32	ACROLEIN	FM	MORT2	6.40	160	56			MACEK ET AL 1976C
33	ACROLEIN	FM	MORT2	11.40	160	108			MACEK ET AL 1976C
34	ACROLEIN	FM	MORT2	41.70	80	78			MACEK ET AL 1976C
35	AC222,705	FM	HATCH	0.00	100	9			SPEHAR ET AL 1983
36	AC222,705	FM	HATCH	0.02	100	4			SPEHAR ET AL 1983
37	AC222,705	FM	HATCH	0.03	100	4			SPEHAR ET AL 1983
38	AC222,705	FM	HATCH	0.07	100	8			SPEHAR ET AL 1983
39	AC222,705	FM	HATCH	0.13	100	100			SPEHAR ET AL 1983
40	AC222,705	FM	HATCH	0.29	100	100			SPEHAR ET AL 1983
41	AC222,705	FM	MORT2	0.00	60	5			SPEHAR ET AL 1983
42	AC222,705	FM	MORT2	0.02	60	8			SPEHAR ET AL 1983
43	AC222,705	FM	MORT2	0.03	60	9			SPEHAR ET AL 1983
44	AC222,705	FM	MORT2	0.07	60	15			SPEHAR ET AL 1983
45	AC222,705	FM	MORT2	0.13	60	59			SPEHAR ET AL 1983
46	AC222,705	FM	MORT2	0.29	60	60			SPEHAR ET AL 1983
47	AC222,705	FM	WEIGHT	0.00				0.13	SPEHAR ET AL 1983
48	AC222,705	FM	WEIGHT	0.02				0.13	SPEHAR ET AL 1983
49	AC222,705	FM	WEIGHT	0.03				0.13	SPEHAR ET AL 1983
50	AC222,705	FM	WEIGHT	0.07				0.13	SPEHAR ET AL 1983
51	AC222,705	FM	WEIGHT	0.13				0.11	SPEHAR ET AL 1983
52	AC222,705	FM	WEIGHT	0.29				0.00	SPEHAR ET AL 1983
53	AG	RT	MORT2	0.00	123	23			NEBEKER ET AL 1983
54	AG	RT	MORT2	0.10	77	17			NEBEKER ET AL 1983

Table 8.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
55	AG	RT	MORT2	0.13	62	11			NEBEKER ET AL 1983
56	AG	RT	MORT2	0.20	52	5			NEBEKER ET AL 1983
57	AG	RT	MORT2	0.24	46	5			NEBEKER ET AL 1983
58	AG	RT	MORT2	0.36	39	13			NEBEKER ET AL 1983
59	AG	RT	MORT2	0.51	36	14			NEBEKER ET AL 1983
60	AG	RT	MORT2	0.70	44	21			NEBEKER ET AL 1983
61	AG	RT	MORT2	1.06	61	39			NEBEKER ET AL 1983
62	AG	RT	MORT2	1.32	33	33			NEBEKER ET AL 1983
63	AG	RT	MORT2	1.95	38	36			NEBEKER ET AL 1983
64	AG	RT	WEIGHT	0.00				31.70	NEBEKER ET AL 1983
65	AG	RT	WEIGHT	0.10				29.50	NEBEKER ET AL 1983
66	AG	RT	WEIGHT	0.13				29.40	NEBEKER ET AL 1983
67	AG	RT	WEIGHT	0.20				30.00	NEBEKER ET AL 1983
68	AG	RT	WEIGHT	0.24				29.80	NEBEKER ET AL 1983
69	AG	RT	WEIGHT	0.36				28.60	NEBEKER ET AL 1983
70	AG	RT	WEIGHT	0.51				28.90	NEBEKER ET AL 1983
71	AG	RT	WEIGHT	0.70				28.10	NEBEKER ET AL 1983
72	AG	RT	WEIGHT	1.06				24.70	NEBEKER ET AL 1983
73	AG	RT	WEIGHT	1.32					NEBEKER ET AL 1983
74	AG	RT	WEIGHT	1.95					NEBEKER ET AL 1983
75	AG THIOSULFATE COMPL	FM	HATCH	0.00	120	13			LEBLANC ET AL 1984
76	AG THIOSULFATE COMPL	FM	HATCH	10.00	120	7			LEBLANC ET AL 1984
77	AG THIOSULFATE COMPL	FM	HATCH	16.00	120	6			LEBLANC ET AL 1984
78	AG THIOSULFATE COMPL	FM	HATCH	35.00	120	10			LEBLANC ET AL 1984
79	AG THIOSULFATE COMPL	FM	HATCH	64.00	120	12			LEBLANC ET AL 1984
80	AG THIOSULFATE COMPL	FM	HATCH	140.00	120	102			LEBLANC ET AL 1984
81	AG THIOSULFATE COMPL	FM	MORT2	0.00	80	5			LEBLANC ET AL 1984
82	AG THIOSULFATE COMPL	FM	MORT2	10.00	80	5			LEBLANC ET AL 1984
83	AG THIOSULFATE COMPL	FM	MORT2	16.00	80	5			LEBLANC ET AL 1984
84	AG THIOSULFATE COMPL	FM	MORT2	35.00	80	10			LEBLANC ET AL 1984
85	AG THIOSULFATE COMPL	FM	MORT2	64.00	80	58			LEBLANC ET AL 1984
86	AG THIOSULFATE COMPL	FM	MORT2	140.00	80	80			LEBLANC ET AL 1984
87	AG THIOSULFATE COMPL	FM	WEIGHT	0.00				0.10	LEBLANC ET AL 1984
88	AG THIOSULFATE COMPL	FM	WEIGHT	10.00				0.12	LEBLANC ET AL 1984
89	AG THIOSULFATE COMPL	FM	WEIGHT	16.00				0.12	LEBLANC ET AL 1984
90	AG THIOSULFATE COMPL	FM	WEIGHT	35.00				0.08	LEBLANC ET AL 1984
91	AG THIOSULFATE COMPL	FM	WEIGHT	64.00				0.04	LEBLANC ET AL 1984
92	AG THIOSULFATE COMPL	FM	WEIGHT	140.00					LEBLANC ET AL 1984
93	ALACHLOR	FM	HATCH	0.00	200	58			CALL ET AL 1983
94	ALACHLOR	FM	HATCH	60.00	200	60			CALL ET AL 1983
95	ALACHLOR	FM	HATCH	140.00	200	68			CALL ET AL 1983
96	ALACHLOR	FM	HATCH	260.00	200	51			CALL ET AL 1983
97	ALACHLOR	FM	HATCH	520.00	200	48			CALL ET AL 1983
98	ALACHLOR	FM	HATCH	1100.00	200	53			CALL ET AL 1983
99	ALACHLOR	FM	MORT2	0.00	60	11			CALL ET AL 1983
100	ALACHLOR	FM	MORT2	60.00	60	7			CALL ET AL 1983
101	ALACHLOR	FM	MORT2	140.00	60	4			CALL ET AL 1983
102	ALACHLOR	FM	MORT2	260.00	60	4			CALL ET AL 1983
103	ALACHLOR	FM	MORT2	520.00	60	1			CALL ET AL 1983
104	ALACHLOR	FM	MORT2	1100.00	60	10			CALL ET AL 1983
105	ALACHLOR	FM	WEIGHT	0.00				0.48	CALL ET AL 1983
106	ALACHLOR	FM	WEIGHT	60.00				0.43	CALL ET AL 1983
107	ALACHLOR	FM	WEIGHT	140.00				0.42	CALL ET AL 1983
108	ALACHLOR	FM	WEIGHT	260.00				0.40	CALL ET AL 1983

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
109	ALACHLOR	FM	WEIGHT	520.00				0.42	CALL ET AL 1983
110	ALACHLOR	FM	WEIGHT	1100.00				0.32	CALL ET AL 1983
111	ALDICARB	FM	HATCH	0.00	100	5			PICKERING AND GILIAM 1982
112	ALDICARB	FM	HATCH	20.00	100	3			PICKERING AND GILIAM 1982
113	ALDICARB	FM	HATCH	38.00	100	4			PICKERING AND GILIAM 1982
114	ALDICARB	FM	HATCH	78.00	100	4			PICKERING AND GILIAM 1982
115	ALDICARB	FM	HATCH	156.00	100	3			PICKERING AND GILIAM 1982
116	ALDICARB	FM	HATCH	340.00	100	3			PICKERING AND GILIAM 1982
117	ALDICARB	FM	MORT2	0.00	80	7			PICKERING AND GILIAM 1982
118	ALDICARB	FM	MORT2	20.00	60	9			PICKERING AND GILIAM 1982
119	ALDICARB	FM	MORT2	38.00	80	8			PICKERING AND GILIAM 1982
120	ALDICARB	FM	MORT2	78.00	80	7			PICKERING AND GILIAM 1982
121	ALDICARB	FM	MORT2	156.00	80	47			PICKERING AND GILIAM 1982
122	ALDICARB	FM	MORT2	340.00	80	64			PICKERING AND GILIAM 1982
123	ALDICARB	FM	WEIGHT	0.00				0.15	PICKERING AND GILIAM 1982
124	ALDICARB	FM	WEIGHT	20.00				0.14	PICKERING AND GILIAM 1982
125	ALDICARB	FM	WEIGHT	38.00				0.14	PICKERING AND GILIAM 1982
126	ALDICARB	FM	WEIGHT	78.00				0.14	PICKERING AND GILIAM 1982
127	ALDICARB	FM	WEIGHT	156.00				0.12	PICKERING AND GILIAM 1982
128	ALDICARB	FM	WEIGHT	340.00				0.08	PICKERING AND GILIAM 1982
129	AROCLOR1242	FM	EGGS	0.00			442		NEBEKER ET AL 1974
130	AROCLOR1242	FM	EGGS	2.90			283		NEBEKER ET AL 1974
131	AROCLOR1242	FM	EGGS	5.40			152		NEBEKER ET AL 1974
132	AROCLOR1242	FM	EGGS	15.00			0		NEBEKER ET AL 1974
133	AROCLOR1242	FM	EGGS	51.00			0		NEBEKER ET AL 1974
134	AROCLOR1242	FM	MORT4	0.00	20	0			NEBEKER ET AL 1974
135	AROCLOR1242	FM	MORT4	0.86	20	2			NEBEKER ET AL 1974
136	AROCLOR1242	FM	MORT4	2.90	20	0			NEBEKER ET AL 1974
137	AROCLOR1242	FM	MORT4	5.40	20	3			NEBEKER ET AL 1974
138	AROCLOR1242	FM	MORT4	15.00	20	13			NEBEKER ET AL 1974
139	AROCLOR1242	FM	MORT4	51.00	20	20			NEBEKER ET AL 1974
140	AROCLOR1248	FM	WEIGHT	0.00				0.15	DEFOE ET AL 1978
141	AROCLOR1248	FM	WEIGHT	0.10				0.14	DEFOE ET AL 1978
142	AROCLOR1248	FM	WEIGHT	0.40				0.12	DEFOE ET AL 1978
143	AROCLOR1248	FM	WEIGHT	1.10				0.11	DEFOE ET AL 1978
144	AROCLOR1248	FM	WEIGHT	3.00				0.10	DEFOE ET AL 1978
145	AROCLOR1248	FF	MORT2	0.00	20	0			NEBEKER ET AL 1974
146	AROCLOR1248	FF	MORT2	0.18	20	2			NEBEKER ET AL 1974
147	AROCLOR1248	FF	MORT2	0.54	20	0			NEBEKER ET AL 1974
148	AROCLOR1248	FF	MORT2	2.20	20	3			NEBEKER ET AL 1974
149	AROCLOR1248	FF	MORT2	5.10	20	13			NEBEKER ET AL 1974
150	AROCLOR1248	FF	MORT2	18.00	20	20			NEBEKER ET AL 1974
151	AROCLOR1248	FF	WEIGHT	0.00				4.33	NEBEKER ET AL 1974
152	AROCLOR1248	FF	WEIGHT	0.18				3.90	NEBEKER ET AL 1974
153	AROCLOR1248	FF	WEIGHT	0.54				4.47	NEBEKER ET AL 1974
154	AROCLOR1248	FF	WEIGHT	2.20				3.02	NEBEKER ET AL 1974
155	AROCLOR1248	FF	WEIGHT	5.10				0.60	NEBEKER ET AL 1974
156	AROCLOR1249	FF	WEIGHT	18.00					NEBEKER ET AL 1974
157	AROCLOR1254	FM	EGGS	0.00			254		NEBEKER ET AL 1974
158	AROCLOR1254	FM	EGGS	0.23			222		NEBEKER ET AL 1974
159	AROCLOR1254	FM	EGGS	0.52			557		NEBEKER ET AL 1974
160	AROCLOR1254	FM	EGGS	1.80			107		NEBEKER ET AL 1974
161	AROCLOR1254	FM	EGGS	4.60			0		NEBEKER ET AL 1974
162	AROCLOR1254	FM	EGGS	15.00			0		NEBEKER ET AL 1974

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
163	AROCLOR1254	FM	HATCH	0.00	400	103			NEBEKER ET AL 1974
164	AROCLOR1254	FM	HATCH	0.23	272	122			NEBEKER ET AL 1974
165	AROCLOR1254	FM	HATCH	0.52	720	264			NEBEKER ET AL 1974
166	AROCLOR1254	FM	HATCH	1.80	350	116			NEBEKER ET AL 1974
167	AS	FF	MORT2	0.00	40	9			CALL ET AL 1983B
168	AS	FF	MORT2	1240.00	40	6			CALL ET AL 1983B
169	AS	FF	MORT2	2130.00	40	8			CALL ET AL 1983B
170	AS	FF	MORT2	4120.00	40	2			CALL ET AL 1983B
171	AS	FF	MORT2	7570.00	40	7			CALL ET AL 1983B
172	AS	FF	MORT2	16300.00	40	10			CALL ET AL 1983B
173	AS	FF	WEIGHT	0.00				0.06	CALL ET AL 1983B
174	AS	FF	WEIGHT	1240.00				0.05	CALL ET AL 1983B
175	AS	FF	WEIGHT	2130.00				0.05	CALL ET AL 1983B
176	AS	FF	WEIGHT	4120.00				0.04	CALL ET AL 1983B
177	AS	FF	WEIGHT	7570.00				0.03	CALL ET AL 1983B
178	AS	FF	WEIGHT	16300.00				0.03	CALL ET AL 1983B
179	AS	FM	HATCH	0.00	200	34			CALL ET AL 1983B
180	AS	FM	HATCH	1060.00	200	27			CALL ET AL 1983B
181	AS	FM	HATCH	2130.00	200	40			CALL ET AL 1983B
182	AS	FM	HATCH	4300.00	200	25			CALL ET AL 1983B
183	AS	FM	HATCH	7370.00	200	40			CALL ET AL 1983B
184	AS	FM	HATCH	16500.00	200	44			CALL ET AL 1983B
185	AS	FM	MORT2	0.00	40	2			CALL ET AL 1983B
186	AS	FM	MORT2	1060.00	40	12			CALL ET AL 1983B
187	AS	FM	MORT2	2130.00	40	4			CALL ET AL 1983B
188	AS	FM	MORT2	4300.00	40	9			CALL ET AL 1983B
189	AS	FM	MORT2	7370.00	40	1			CALL ET AL 1983B
190	AS	FM	MORT2	16500.00	40	31			CALL ET AL 1983B
191	AS	FM	WEIGHT	0.00				0.06	CALL ET AL 1983B
192	AS	FM	WEIGHT	1060.00				0.06	CALL ET AL 1983B
193	AS	FM	WEIGHT	2130.00				0.05	CALL ET AL 1983B
194	AS	FM	WEIGHT	4300.00				0.04	CALL ET AL 1983B
195	AS	FM	WEIGHT	7370.00				0.03	CALL ET AL 1983B
196	AS	FM	WEIGHT	16500.00				0.01	CALL ET AL 1983B
197	ATRAZINE	BG	EGGS	0.00			8735		MACEK ET AL 1976A
198	ATRAZINE	BG	EGGS	8.00			15254		MACEK ET AL 1976A
199	ATRAZINE	BG	EGGS	14.00			7460		MACEK ET AL 1976A
200	ATRAZINE	BG	EGGS	25.00			5153		MACEK ET AL 1976A
201	ATRAZINE	BG	EGGS	49.00			7331		MACEK ET AL 1976A
202	ATRAZINE	BG	EGGS	95.00			7676		MACEK ET AL 1976A
203	ATRAZINE	BG	HATCH	0.00	1400	224			MACEK ET AL 1976A
204	ATRAZINE	BG	HATCH	8.00	600	204			MACEK ET AL 1976A
205	ATRAZINE	BG	HATCH	14.00	2400	456			MACEK ET AL 1976A
206	ATRAZINE	BG	HATCH	25.00	1200	156			MACEK ET AL 1976A
207	ATRAZINE	BG	HATCH	49.00	600	60			MACEK ET AL 1976A
208	ATRAZINE	BG	HATCH	95.00	800	72			MACEK ET AL 1976A
209	ATRAZINE	BG	MORT1	0.00	20	1			MACEK ET AL 1976A
210	ATRAZINE	BG	MORT1	8.00	20	3			MACEK ET AL 1976A
211	ATRAZINE	BG	MORT1	14.00	20	0			MACEK ET AL 1976A
212	ATRAZINE	BG	MORT1	25.00	20	1			MACEK ET AL 1976A
213	ATRAZINE	BG	MORT1	49.00	20	1			MACEK ET AL 1976A
214	ATRAZINE	BG	MORT1	95.00	20	3			MACEK ET AL 1976A
215	ATRAZINE	BG	MORT2	0.00	100	78			MACEK ET AL 1976A
216	ATRAZINE	BG	MORT2	8.00	100	57			MACEK ET AL 1976A

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
217	ATRAZINE	BG	MORT2	14.00	200	130			MACEK ET AL 1976A
218	ATRAZINE	BG	MORT2	25.00	100	58			MACEK ET AL 1976A
219	ATRAZINE	BG	MORT2	49.00	50	40			MACEK ET AL 1976A
220	ATRAZINE	BG	MORT2	95.00	50	41			MACEK ET AL 1976A
221	ATRAZINE	BT	EGGS	0.00			327		MACEK ET AL 1976A
222	ATRAZINE	BT	EGGS	65.00			400		MACEK ET AL 1976A
223	ATRAZINE	BT	EGGS	120.00			389		MACEK ET AL 1976A
224	ATRAZINE	BT	EGGS	240.00			437		MACEK ET AL 1976A
225	ATRAZINE	BT	EGGS	450.00			168		MACEK ET AL 1976A
226	ATRAZINE	BT	EGGS	720.00			259		MACEK ET AL 1976A
227	ATRAZINE	BT	HATCH	0.00	100	49			MACEK ET AL 1976A
228	ATRAZINE	BT	HATCH	65.00	100	70			MACEK ET AL 1976A
229	ATRAZINE	BT	HATCH	120.00	100	30			MACEK ET AL 1976A
230	ATRAZINE	BT	HATCH	240.00	100	54			MACEK ET AL 1976A
231	ATRAZINE	BT	HATCH	450.00	50	26			MACEK ET AL 1976A
232	ATRAZINE	BT	HATCH	720.00	100	67			MACEK ET AL 1976A
233	ATRAZINE	BT	MORT2	0.00	100	49			MACEK ET AL 1976A
234	ATRAZINE	BT	MORT2	65.00	100	58			MACEK ET AL 1976A
235	ATRAZINE	BT	MORT2	120.00	100	60			MACEK ET AL 1976A
236	ATRAZINE	BT	MORT2	240.00	100	80			MACEK ET AL 1976A
237	ATRAZINE	BT	MORT2	450.00	100	72			MACEK ET AL 1976A
238	ATRAZINE	BT	MORT2	720.00	100	90			MACEK ET AL 1976A
239	ATRAZINE	FM	HATCH	0.00	3800	642			MACEK ET AL 1976A
240	ATRAZINE	FM	HATCH	15.00	1650	308			MACEK ET AL 1976A
241	ATRAZINE	FM	HATCH	54.00	1550	254			MACEK ET AL 1976A
242	ATRAZINE	FM	HATCH	112.00	2450	510			MACEK ET AL 1976A
243	ATRAZINE	FM	HATCH	213.00	1600	369			MACEK ET AL 1976A
244	ATRAZINE	FM	MORT1	0.00	30	2			MACEK ET AL 1976A
245	ATRAZINE	FM	MORT1	15.00	30	5			MACEK ET AL 1976A
246	ATRAZINE	FM	MORT1	33.00	30	5			MACEK ET AL 1976A
247	ATRAZINE	FM	MORT1	54.00	30	6			MACEK ET AL 1976A
248	ATRAZINE	FM	MORT1	112.00	30	7			MACEK ET AL 1976A
249	ATRAZINE	FM	MORT1	213.00	30	6			MACEK ET AL 1976A
250	ATRAZINE	FM	MORT2	0.00	200	55			MACEK ET AL 1976A
251	ATRAZINE	FM	MORT2	15.00	240	110			MACEK ET AL 1976A
252	ATRAZINE	FM	MORT2	54.00	160	72			MACEK ET AL 1976A
253	ATRAZINE	FM	MORT2	112.00	240	77			MACEK ET AL 1976A
254	ATRAZINE	FM	MORT2	213.00	160	43			MACEK ET AL 1976A
255	BROMACIL	FM	HATCH	0.00	200	76			CALL ET AL 1983
256	BROMACIL	FM	HATCH	1000.00	200	72			CALL ET AL 1983
257	BROMACIL	FM	HATCH	1900.00	200	92			CALL ET AL 1983
258	BROMACIL	FM	HATCH	4400.00	200	93			CALL ET AL 1983
259	BROMACIL	FM	HATCH	12000.00	200	90			CALL ET AL 1983
260	BROMACIL	FM	HATCH	29000.00	200	72			CALL ET AL 1983
261	BROMACIL	FM	MORT2	0.00	60	7			CALL ET AL 1983
262	BROMACIL	FM	MORT2	1000.00	60	3			CALL ET AL 1983
263	BROMACIL	FM	MORT2	1900.00	60	1			CALL ET AL 1983
264	BROMACIL	FM	MORT2	4400.00	60	1			CALL ET AL 1983
265	BROMACIL	FM	MORT2	12000.00	60	5			CALL ET AL 1983
266	BROMACIL	FM	MORT2	29000.00	60	7			CALL ET AL 1983
267	BROMACIL	FM	WEIGHT	0.00				0.47	CALL ET AL 1983
268	BROMACIL	FM	WEIGHT	1000.00				0.41	CALL ET AL 1983
269	BROMACIL	FM	WEIGHT	1900.00				0.42	CALL ET AL 1983
270	BROMACIL	FM	WEIGHT	4400.00				0.38	CALL ET AL 1983

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
271	BROMACIL	FM	WEIGHT	12000.00				0.37	CALL ET AL 1983
272	BROMACIL	FM	WEIGHT	29000.00				0.33	CALL ET AL 1983
273	CAPTAN	FM	EGGS	0.00			1853		HERMANUTZ ET AL 1973
274	CAPTAN	FM	EGGS	3.30			1024		HERMANUTZ ET AL 1973
275	CAPTAN	FM	EGGS	7.40			795		HERMANUTZ ET AL 1973
276	CAPTAN	FM	EGGS	16.80			422		HERMANUTZ ET AL 1973
277	CAPTAN	FM	EGGS	39.50			40		HERMANUTZ ET AL 1973
278	CAPTAN	FM	EGGS	63.50					HERMANUTZ ET AL 1973
279	CAPTAN	FM	HATCH	0.00	1900	531			HERMANUTZ ET AL 1973
280	CAPTAN	FM	HATCH	3.30	1350	347			HERMANUTZ ET AL 1973
281	CAPTAN	FM	HATCH	.40	1150	173			HERMANUTZ ET AL 1973
282	CAPTAN	FM	HATCH	16.80	800	95			HERMANUTZ ET AL 1973
283	CAPTAN	FM	HATCH	39.50	150	26			HERMANUTZ ET AL 1973
284	CAPTAN	FM	HATCH	63.50	400	125			HERMANUTZ ET AL 1973
285	CAPTAN	FM	MORT1	0.00	30	1			HERMANUTZ ET AL 1973
286	CAPTAN	FM	MORT1	3.30	30	1			HERMANUTZ ET AL 1973
287	CAPTAN	FM	MORT1	7.40	30	0			HERMANUTZ ET AL 1973
288	CAPTAN	FM	MORT1	16.80	30	1			HERMANUTZ ET AL 1973
289	CAPTAN	FM	MORT1	39.50	30	7			HERMANUTZ ET AL 1973
290	CAPTAN	FM	MORT1	63.50	30	30			HERMANUTZ ET AL 1973
291	CAPTAN	FM	MORT2	0.00	320	93			HERMANUTZ ET AL 1973
292	CAPTAN	FM	MORT2	3.30	320	128			HERMANUTZ ET AL 1973
293	CAPTAN	FM	MORT2	7.40	320	143			HERMANUTZ ET AL 1973
294	CAPTAN	FM	MORT2	16.80	320	118			HERMANUTZ ET AL 1973
295	CAPTAN	FM	MORT2	39.50	240	164			HERMANUTZ ET AL 1973
296	CAPTAN	FM	MORT2	63.50	320	320			HERMANUTZ ET AL 1973
297	CARBARYL	FM	EGGS	0.00			683		CARLSON 1971
298	CARBARYL	FM	EGGS	8.00			1070		CARLSON 1971
299	CARBARYL	FM	EGGS	17.00			624		CARLSON 1971
300	CARBARYL	FM	EGGS	62.00			265		CARLSON 1971
301	CARBARYL	FM	EGGS	210.00			723		CARLSON 1971
302	CARBARYL	FM	EGGS	680.00			11		CARLSON 1971
303	CARBARYL	FM	HATCH	0.00	1360	484			CARLSON 1971
304	CARBARYL	FM	HATCH	8.00	1120	553			CARLSON 1971
305	CARBARYL	FM	HATCH	17.00	1360	539			CARLSON 1971
306	CARBARYL	FM	HATCH	62.00	920	348			CARLSON 1971
307	CARBARYL	FM	HATCH	210.00	1920	1268			CARLSON 1971
308	CARBARYL	FM	HATCH	680.00	320	320			CARLSON 1971
309	CARBARYL	FM	MORT2	0.00	100	8			CARLSON 1971
310	CARBARYL	FM	MORT2	8.00	100	54			CARLSON 1971
311	CARBARYL	FM	MORT2	17.00	100	18			CARLSON 1971
312	CARBARYL	FM	MORT2	62.00	100	34			CARLSON 1971
313	CARBARYL	FM	MORT2	210.00	100	13			CARLSON 1971
314	CARBARYL	FM	MORT2	680.00	100	60			CARLSON 1971
315	CARBARYL	FM	MORT4	0.00	20	6			CARLSON 1971
316	CARBARYL	FM	MORT4	8.00	20	7			CARLSON 1971
317	CARBARYL	FM	MORT4	17.00	20	4			CARLSON 1971
318	CARBARYL	FM	MORT4	62.00	20	4			CARLSON 1971
319	CARBARYL	FM	MORT4	210.00	20	7			CARLSON 1971
320	CARBARYL	FM	MORT4	680.00	20	10			CARLSON 1971
321	CD	BT	EGGS	0.06			502		BENOIT ET AL 1976
322	CD	BT	EGGS	0.50			244		BENOIT ET AL 1976
323	CD	BT	EGGS	0.90			454		BENOIT ET AL 1976
324	CD	BT	EGGS	1.70			260		BENOIT ET AL 1976

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
325	CD	BT	EGGS	3.40			98		BENOIT ET AL 1976
326	CD	BT	MORT1	0.06	5	0			BENOIT ET AL 1976
327	CD	BT	MORT1	0.50	10	0			BENOIT ET AL 1976
328	CD	BT	MORT1	0.85	10	0			BENOIT ET AL 1976
329	CD	BT	MORT1	1.65	10	0			BENOIT ET AL 1976
330	CD	BT	MORT1	3.40	10	5			BENOIT ET AL 1976
331	CD	BT	MORT1	6.35	10	10			BENOIT ET AL 1976
332	CD	BT	WEIGHT	0.06				3.63	BENOIT ET AL 1976
333	CD	BT	WEIGHT	0.50				3.32	BENOIT ET AL 1976
334	CD	BT	WEIGHT	0.90				3.42	BENOIT ET AL 1976
335	CD	BT	WEIGHT	1.70				3.81	BENOIT ET AL 1976
336	CD	BT	WEIGHT	3.40				1.80	BENOIT ET AL 1976
337	CD	FF	EGGS	0.00			1086		CARLSON ET AL 1982
338	CD	FF	EGGS	1.80			912		CARLSON ET AL 1982
339	CD	FF	EGGS	3.70			890		CARLSON ET AL 1982
340	CD	FF	EGGS	7.50			636		CARLSON ET AL 1982
341	CD	FF	EGGS	15.00			23		CARLSON ET AL 1982
342	CD	FF	MORT1	0.00	14	1			CARLSON ET AL 1982
343	CD	FF	MORT1	1.80	14	2			CARLSON ET AL 1982
344	CD	FF	MORT1	3.70	14	6			CARLSON ET AL 1982
345	CD	FF	MORT1	7.50	14	0			CARLSON ET AL 1982
346	CD	FF	MORT1	15.00	8	6			CARLSON ET AL 1982
347	CD	FF	MORT1	30.00	1	1			CARLSON ET AL 1982
348	CD	FF	MORT2	0.00	40	7			CARLSON ET AL 1982
349	CD	FF	MORT2	1.80	40	3			CARLSON ET AL 1982
350	CD	FF	MORT2	3.70	40	3			CARLSON ET AL 1982
351	CD	FF	MORT2	7.50	40	4			CARLSON ET AL 1982
352	CD	FF	MORT2	15.00	11	2			CARLSON ET AL 1982
353	CD	FF	WEIGHT	0.00				17.40	CARLSON ET AL 1982
354	CD	FF	WEIGHT	1.80				25.30	CARLSON ET AL 1982
355	CD	FF	WEIGHT	3.70				22.70	CARLSON ET AL 1982
356	CD	FF	WEIGHT	7.50				30.50	CARLSON ET AL 1982
357	CD	FF	WEIGHT	15.00				17.50	CARLSON ET AL 1982
358	CD	BG	HATCH	2.30	300	19			EATON 1974
359	CD	BG	HATCH	31.00	100	7			EATON 1974
360	CD	BG	HATCH	80.00	550	41			EATON 1974
361	CD	BG	HATCH	239.00	150	54			EATON 1974
362	CD	BG	HATCH	2140.00	100	20			EATON 1974
363	CD	BG	MORT1	2.30	18	0			EATON 1974
364	CD	BG	MORT1	31.00	18	0			EATON 1974
365	CD	BG	MORT1	80.00	18	9			EATON 1974
366	CD	BG	MORT1	239.00	18	16			EATON 1974
367	CD	BG	MORT1	757.00	18	18			EATON 1974
368	CD	BG	MORT1	2140.00	18	18			EATON 1974
369	CD	BG	MORT2	2.30	100	22			EATON 1974
370	CD	BG	MORT2	31.00	100	40			EATON 1974
371	CD	BG	MORT2	80.00	100	90			EATON 1974
372	CD	BG	MORT2	239.00	100	100			EATON 1974
373	CD	BG	WEIGHT	2.30				0.40	EATON 1974
374	CD	BG	WEIGHT	31.00				0.54	EATON 1974
375	CD	BG	WEIGHT	80.00				0.01	EATON 1974
376	CD	BG	WEIGHT	239.00				0.00	EATON 1974
377	CD	FM	EGGS	1.00			1468		PICKERING AND GAST 1972
378	CD	FM	EGGS	7.80			1704		PICKERING AND GAST 1972

Table 8.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
379	CD	FM	EGGS	14.00			4606		PICKERING AND GAST 1972
380	CD	FM	EGGS	27.00			1448		PICKERING AND GAST 1972
381	CD	FM	EGGS	57.00			962		PICKERING AND GAST 1972
382	CD	FM	EGGS	110.00			403		PICKERING AND GAST 1972
383	CD	FM	HATCH	1.00	100	5			PICKERING AND GAST 1972
384	CD	FM	HATCH	7.80	100	4			PICKERING AND GAST 1972
385	CD	FM	HATCH	14.00	100	5			PICKERING AND GAST 1972
386	CD	FM	HATCH	27.00	100	6			PICKERING AND GAST 1972
387	CD	FM	HATCH	57.00	100	22			PICKERING AND GAST 1972
388	CD	FM	MORT1	1.00	80	24			PICKERING AND GAST 1972
389	CD	FM	MORT1	7.80	80	25			PICKERING AND GAST 1972
390	CD	FM	MORT1	14.00	80	33			PICKERING AND GAST 1972
391	CD	FM	MORT1	27.00	80	30			PICKERING AND GAST 1972
392	CD	FM	MORT1	57.00	80	30			PICKERING AND GAST 1972
393	CD	FM	MORT1	110.00	80	66			PICKERING AND GAST 1972
394	CD	FM	MORT2	1.20	50	11			PICKERING AND GAST 1972
395	CD	FM	MORT2	6.80	50	17			PICKERING AND GAST 1972
396	CD	FM	MORT2	15.00	50	2			PICKERING AND GAST 1972
397	CD	FM	MORT2	29.00	50	25			PICKERING AND GAST 1972
398	CD	FM	MORT2	57.00	50	16			PICKERING AND GAST 1972
399	CD	FM	MORT2	110.00	50	42			PICKERING AND GAST 1972
400	CD	BT	MORT2	0.00	400	0			SAUTER ET AL 1976
401	CD	BT	MORT2	1.00	400	105			SAUTER ET AL 1976
402	CD	BT	MORT2	3.00	400	82			SAUTER ET AL 1976
403	CD	BT	MORT2	6.00	400	243			SAUTER ET AL 1976
404	CD	BT	MORT2	10.00	400	320			SAUTER ET AL 1976
405	CD	BT	MORT2	24.00	400	352			SAUTER ET AL 1976
406	CD	BT	MORT2	47.00	400	392			SAUTER ET AL 1976
407	CD	BT	WEIGHT	0.00				0.24	SAUTER ET AL 1976
408	CD	BT	WEIGHT	1.00				0.23	SAUTER ET AL 1976
409	CD	BT	WEIGHT	3.00				0.19	SAUTER ET AL 1976
410	CD	BT	WEIGHT	6.00				0.14	SAUTER ET AL 1976
411	CD	BT	WEIGHT	10.00				0.13	SAUTER ET AL 1976
412	CD	BT	WEIGHT	24.00				0.14	SAUTER ET AL 1976
413	CD	BT	WEIGHT	47.00				0.13	SAUTER ET AL 1976
414	CD	FF	EGGS	0.11			665		SPEHAR 1976
415	CD	FF	EGGS	0.17			768		SPEHAR 1976
416	CD	FF	EGGS	4.10			660		SPEHAR 1976
417	CD	FF	EGGS	8.10			283		SPEHAR 1976
418	CD	FF	EGGS	16.00			50		SPEHAR 1976
419	CD	FF	EGGS	31.00			0		SPEHAR 1976
420	CD	FF	HATCH	0.11	40	14			SPEHAR 1976
421	CD	FF	HATCH	1.70	40	14			SPEHAR 1976
422	CD	FF	HATCH	4.10	40	11			SPEHAR 1976
423	CD	FF	HATCH	8.10	40	14			SPEHAR 1976
424	CD	FF	HATCH	16.00	40	13			SPEHAR 1976
425	CD	FF	MORT1	0.11	60	2			SPEHAR 1976
426	CD	FF	MORT1	1.70	60	1			SPEHAR 1976
427	CD	FF	MORT1	4.10	60	6			SPEHAR 1976
428	CD	FF	MORT1	8.10	60	8			SPEHAR 1976
429	CD	FF	MORT1	16.00	60	14			SPEHAR 1976
430	CD	FF	MORT1	31.00	60	36			SPEHAR 1976
431	CHLORAMINE	FM	MORT1	0.00	10	3			ARTHUR AND EATON 1971
432	CHLORAMINE	FM	MORT1	6.60	10	1			ARTHUR AND EATON 1971

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
433	CHLORAMINE	FM	MORT1	16.00	10	0			ARTHUR AND EATON 1971
434	CHLORAMINE	FM	MORT1	42.00	10	0			ARTHUR AND EATON 1971
435	CHLORAMINE	FM	MORT1	85.00	10	7			ARTHUR AND EATON 1971
436	CHLORAMINE	FM	MORT1	154.00	10	10			ARTHUR AND EATON 1971
437	CHLORAMINE	FM	MORT2	0.00	49	14			ARTHUR AND EATON 1971
438	CHLORAMINE	FM	MORT2	3.80	44	1			ARTHUR AND EATON 1971
439	CHLORAMINE	FM	MORT2	17.00	34	8			ARTHUR AND EATON 1971
440	CHLORAMINE	FM	MORT2	40.00	37	12			ARTHUR AND EATON 1971
441	CHLORAMINE	FM	MORT2	108.00	24	15			ARTHUR AND EATON 1971
442	CHLORDANE	BG	EGGS	0.00			1136		CARDWELL ET AL 1977
443	CHLORDANE	BG	EGGS	0.25			1979		CARDWELL ET AL 1977
444	CHLORDANE	BG	EGGS	0.54			2759		CARDWELL ET AL 1977
445	CHLORDANE	BG	EGGS	1.22			131		CARDWELL ET AL 1977
446	CHLORDANE	BG	EGGS	2.20			0		CARDWELL ET AL 1977
447	CHLORDANE	BG	EGGS	5.17			0		CARDWELL ET AL 1977
448	CHLORDANE	BG	MORT1	0.00	40	5			CARDWELL ET AL 1977
449	CHLORDANE	BG	MORT1	0.25	40	1			CARDWELL ET AL 1977
450	CHLORDANE	BG	MORT1	0.54	40	5			CARDWELL ET AL 1977
451	CHLORDANE	BG	MORT1	1.22	40	1			CARDWELL ET AL 1977
452	CHLORDANE	BG	MORT1	2.20	40	7			CARDWELL ET AL 1977
453	CHLORDANE	BG	MORT1	5.17	40	27			CARDWELL ET AL 1977
454	CHLORDANE	BT	EGGS	0.00			190		CARDWELL ET AL 1977
455	CHLORDANE	BT	EGGS	0.32			231		CARDWELL ET AL 1977
456	CHLORDANE	BT	EGGS	0.66			184		CARDWELL ET AL 1977
457	CHLORDANE	BT	EGGS	1.29			192		CARDWELL ET AL 1977
458	CHLORDANE	BT	EGGS	2.21			38		CARDWELL ET AL 1977
459	CHLORDANE	BT	EGGS	5.80			16		CARDWELL ET AL 1977
460	CHLORDANE	BT	HATCH	0.00	450	37			CARDWELL ET AL 1977
461	CHLORDANE	BT	HATCH	0.32	300	121			CARDWELL ET AL 1977
462	CHLORDANE	BT	HATCH	0.66	50	5			CARDWELL ET AL 1977
463	CHLORDANE	BT	HATCH	1.29	50	13			CARDWELL ET AL 1977
464	CHLORDANE	BT	HATCH	2.21	0				CARDWELL ET AL 1977
465	CHLORDANE	BT	HATCH	5.80	0	0			CARDWELL ET AL 1977
466	CHLORDANE	BT	MORT1	0.00	18	3			CARDWELL ET AL 1977
467	CHLORDANE	BT	MORT1	0.32	18	3			CARDWELL ET AL 1977
468	CHLORDANE	BT	MORT1	0.66	18	2			CARDWELL ET AL 1977
469	CHLORDANE	BT	MORT1	1.29	18	3			CARDWELL ET AL 1977
470	CHLORDANE	BT	MORT1	2.21	16	13			CARDWELL ET AL 1977
471	CHLORDANE	BT	MORT1	5.80	12	12			CARDWELL ET AL 1977
472	CHLORDANE	BT	WEIGHT	0.00				0.61	CARDWELL ET AL 1977
473	CHLORDANE	BT	WEIGHT	0.32				0.91	CARDWELL ET AL 1977
474	CHLORDANE	BT	WEIGHT	0.66				0.80	CARDWELL ET AL 1977
475	CHLORDANE	BT	WEIGHT	1.29				0.85	CARDWELL ET AL 1977
476	CHLORDANE	BT	WEIGHT	2.21					CARDWELL ET AL 1977
477	CHLORDANE	BT	WEIGHT	5.80					CARDWELL ET AL 1977
478	CN	AS	HATCH	0.00	1827	113			LEDUC 1978
479	CN	AS	HATCH	10.00	855	221			LEDUC 1978
480	CN	AS	HATCH	20.00	915	346			LEDUC 1978
481	CN	AS	HATCH	40.00	1041	359			LEDUC 1978
482	CN	AS	HATCH	80.00	1012	399			LEDUC 1978
483	CN	AS	HATCH	100.00	978	631			LEDUC 1978
484	CN	AS	MORT2	0.00	200	26			LEDUC 1978
485	CN	AS	MORT2	10.00	100	3			LEDUC 1978
486	CN	AS	MORT2	20.00	100	2			LEDUC 1978

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
487	CN	AS	MORT2	40.00	100	2			LEDUC 1978
488	CN	AS	MORT2	80.00	100	5			LEDUC 1978
489	CN	AS	MORT2	100.00	100	12			LEDUC 1978
490	CN	AS	WEIGHT	0.00				14.80	LEDUC 1978
491	CN	AS	WEIGHT	10.00				16.20	LEDUC 1978
492	CN	AS	WEIGHT	20.00				17.20	LEDUC 1978
493	CN	AS	WEIGHT	40.00				16.90	LEDUC 1978
494	CN	AS	WEIGHT	80.00				15.50	LEDUC 1978
495	CN	AS	WEIGHT	100.00				13.60	LEDUC 1978
496	CN	BG	EGGS	0.00			62		SMITH ET AL 1979
497	CN	BG	EGGS	5.20			0		SMITH ET AL 1979
498	CN	BG	EGGS	9.80			0		SMITH ET AL 1979
499	CN	BG	EGGS	20.50			0		SMITH ET AL 1979
500	CN	BG	EGGS	30.00			0		SMITH ET AL 1979
501	CN	BG	EGGS	39.70			0		SMITH ET AL 1979
502	CN	BG	EGGS	50.20			0		SMITH ET AL 1979
503	CN	BG	EGGS	65.60			0		SMITH ET AL 1979
504	CN	BG	EGGS	80.00			0		SMITH ET AL 1979
505	CN	BG	MORT1	0.00	30	0			SMITH ET AL 1979
506	CN	BG	MORT1	5.20	15	0			SMITH ET AL 1979
507	CN	BG	MORT1	9.80	15	0			SMITH ET AL 1979
508	CN	BG	MORT1	20.50	15	1			SMITH ET AL 1979
509	CN	BG	MORT1	30.00	15	1			SMITH ET AL 1979
510	CN	BG	MORT1	39.70	15	2			SMITH ET AL 1979
511	CN	BG	MORT1	50.20	15	1			SMITH ET AL 1979
512	CN	BG	MORT1	65.60	15	6			SMITH ET AL 1979
513	CN	BG	MORT1	80.00	15	9			SMITH ET AL 1979
514	CN	BT	MORT2	0.00	60	1			SMITH ET AL 1979
515	CN	BT	MORT2	5.60	40	0			SMITH ET AL 1979
516	CN	BT	MORT2	11.30	40	0			SMITH ET AL 1979
517	CN	BT	MORT2	21.85	40	2			SMITH ET AL 1979
518	CN	BT	MORT2	33.30	40	0			SMITH ET AL 1979
519	CN	BT	MORT2	43.55	40	0			SMITH ET AL 1979
520	CN	BT	MORT2	55.30	40	6			SMITH ET AL 1979
521	CN	BT	MORT2	67.15	40	11			SMITH ET AL 1979
522	CN	BT	MORT2	77.20	40	28			SMITH ET AL 1979
523	CN	FM	EGGS	0.00			3476		SMITH ET AL 1979
524	CN	FM	EGGS	5.80			2512		SMITH ET AL 1979
525	CN	FM	EGGS	12.90			1845		SMITH ET AL 1979
526	CN	FM	EGGS	19.60			1467		SMITH ET AL 1979
527	CN	FM	EGGS	27.20			1366		SMITH ET AL 1979
528	CN	FM	EGGS	35.80			1009		SMITH ET AL 1979
529	CN	FM	EGGS	44.20			1124		SMITH ET AL 1979
530	CN	FM	EGGS	63.50			72		SMITH ET AL 1979
531	CN	FM	EGGS	72.80			318		SMITH ET AL 1979
532	CN	FM	EGGS	80.60			242		SMITH ET AL 1979
533	CN	FM	EGGS	96.10			0		SMITH ET AL 1979
534	CN	FM	EGGS	105.40			0		SMITH ET AL 1979
535	CN	FM	HATCH	0.00	250	77			SMITH ET AL 1979
536	CN	FM	HATCH	5.80	100	39			SMITH ET AL 1979
537	CN	FM	HATCH	12.90	100	19			SMITH ET AL 1979
538	CN	FM	HATCH	19.60	100	44			SMITH ET AL 1979
539	CN	FM	HATCH	27.30	100	61			SMITH ET AL 1979
540	CN	FM	HATCH	35.80	100	50			SMITH ET AL 1979

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
541	CN	FM	HATCH	44.20	100	87			SMITH ET AL 1979
542	CN	FM	HATCH	63.50	100	79			SMITH ET AL 1979
543	CN	FM	HATCH	72.80	100	81			SMITH ET AL 1979
544	CN	FM	HATCH	80.60	100	90			SMITH ET AL 1979
545	CN	FM	HATCH	96.10	100	100			SMITH ET AL 1979
546	CN	FM	HATCH	105.40	100	100			SMITH ET AL 1979
547	CN	FM	MORT1	0.00	240	88			SMITH ET AL 1979
548	CN	FM	MORT1	5.90	80	16			SMITH ET AL 1979
549	CN	FM	MORT1	11.40	80	33			SMITH ET AL 1979
550	CN	FM	MORT1	17.90	80	33			SMITH ET AL 1979
551	CN	FM	MORT1	24.70	80	39			SMITH ET AL 1979
552	CN	FM	MORT1	32.80	80	43			SMITH ET AL 1979
553	CN	FM	MORT1	40.50	80	33			SMITH ET AL 1979
554	CN	FM	MORT1	57.50	80	42			SMITH ET AL 1979
555	CN	FM	MORT1	66.80	80	46			SMITH ET AL 1979
556	CN	FM	MORT1	75.30	80	59			SMITH ET AL 1979
557	CN	FM	MORT1	88.90	80	68			SMITH ET AL 1979
558	CN	FM	MORT1	98.10	80	71			SMITH ET AL 1979
559	CN	FM	WEIGHT	0.00				0.29	SMITH ET AL 1979
560	CN	FM	WEIGHT	5.90				0.20	SMITH ET AL 1979
561	CN	FM	WEIGHT	11.40				0.27	SMITH ET AL 1979
562	CN	FM	WEIGHT	17.90				0.27	SMITH ET AL 1979
563	CN	FM	WEIGHT	24.70				0.30	SMITH ET AL 1979
564	CN	FM	WEIGHT	32.80				0.38	SMITH ET AL 1979
565	CN	FM	WEIGHT	40.50				0.27	SMITH ET AL 1979
566	CN	FM	WEIGHT	57.50				0.19	SMITH ET AL 1979
567	CN	FM	WEIGHT	66.80				0.22	SMITH ET AL 1979
568	CN	FM	WEIGHT	75.30				0.26	SMITH ET AL 1979
569	CN	FM	WEIGHT	88.90				0.20	SMITH ET AL 1979
570	CN	FM	WEIGHT	98.10				0.19	SMITH ET AL 1979
571	CNSO4	CHS	HATCH	0.00	267	53			HAZEL AND MEITH 1970
572	CNSO4	CHS	HATCH	21.00	377	90			HAZEL AND MEITH 1970
573	CNSO4	CHS	HATCH	40.00	357	65			HAZEL AND MEITH 1970
574	CNSO4	CHS	HATCH	80.00	404	90			HAZEL AND MEITH 1970
575	CNSO4	CHS	MORT2	0.00	214	49			HAZEL AND MEITH 1970
576	CNSO4	CHS	MORT2	21.00	286	94			HAZEL AND MEITH 1970
577	CNSO4	CHS	MORT2	40.00	292	276			HAZEL AND MEITH 1970
578	CNSO4	CHS	MORT2	80.00	314	314			HAZEL AND MEITH 1970
579	CNSO4	CHS	WEIGHT	0.00				0.38	HAZEL AND MEITH 1970
580	CNSO4	CHS	WEIGHT	21.00				0.33	HAZEL AND MEITH 1970
581	CNSO4	CHS	WEIGHT	40.00				0.30	HAZEL AND MEITH 1970
582	CNSO4	CHS	WEIGHT	80.00				0.00	HAZEL AND MEITH 1970
583	CR	FM	HATCH	0.00	525	26			PICKERING 1980
584	CR	FM	HATCH	18.00	547	22			PICKERING 1980
585	CR	FM	HATCH	66.00	364	25			PICKERING 1980
586	CR	FM	HATCH	260.00	625	44			PICKERING 1980
587	CR	FM	HATCH	1000.00	600	30			PICKERING 1980
588	CR	FM	HATCH	3950.00	135	19			PICKERING 1980
589	CR	FM	MORT1	0.00	35	0			PICKERING 1980
590	CR	FM	MORT1	18.00	35	1			PICKERING 1980
591	CR	FM	MORT1	66.00	35	1			PICKERING 1980
592	CR	FM	MORT1	260.00	35	5			PICKERING 1980
593	CR	FM	MORT1	1000.00	35	2			PICKERING 1980
594	CR	FM	MORT1	3950.00	35	22			PICKERING 1980

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
595	CR	FM	MORT2	0.00	50	14			PICKERING 1980
596	CR	FM	MORT2	18.00	50	10			PICKERING 1980
597	CR	FM	MORT2	66.00	50	9			PICKERING 1980
598	CR	FM	MORT2	260.00	50	3			PICKERING 1980
599	CR	FM	MORT2	1000.00	50	1			PICKERING 1980
600	CR	FM	MORT2	3950.00	50	44			PICKERING 1980
601	CR	BG	WEIGHT	0.00				0.30	SAUTER ET AL 1976
602	CR	BG	WEIGHT	57.00				0.29	SAUTER ET AL 1976
603	CR	BG	WEIGHT	70.00				0.25	SAUTER ET AL 1976
604	CR	BG	WEIGHT	140.00				0.29	SAUTER ET AL 1976
605	CR	BG	WEIGHT	265.00				0.20	SAUTER ET AL 1976
606	CR	BG	WEIGHT	522.00				0.24	SAUTER ET AL 1976
607	CR	BG	WEIGHT	1122.00				0.13	SAUTER ET AL 1976
608	CR	CC	WEIGHT	0.00				0.33	SAUTER ET AL 1976
609	CR	CC	WEIGHT	39.00				0.33	SAUTER ET AL 1976
610	CR	CC	WEIGHT	73.00				0.34	SAUTER ET AL 1976
611	CR	CC	WEIGHT	150.00				0.27	SAUTER ET AL 1976
612	CR	CC	WEIGHT	305.00				0.23	SAUTER ET AL 1976
613	CR	CC	WEIGHT	570.00				0.12	SAUTER ET AL 1976
614	CR	CC	WEIGHT	1290.00				0.00	SAUTER ET AL 1976
615	CR	LT	WEIGHT	0.00				0.21	SAUTER ET AL 1976
616	CR	LT	WEIGHT	1400.00				0.09	SAUTER ET AL 1976
617	CR	LT	WEIGHT	2900.00				0.09	SAUTER ET AL 1976
618	CR	LT	WEIGHT	6000.00				0.06	SAUTER ET AL 1976
619	CR	LT	WEIGHT	11600.00				0.09	SAUTER ET AL 1976
620	CR	LT	WEIGHT	24400.00				0.00	SAUTER ET AL 1976
621	CR	LT	WEIGHT	50700.00				0.00	SAUTER ET AL 1976
622	CR	NP	WEIGHT	0.00				1.03	SAUTER ET AL 1976
623	CR	NP	WEIGHT	123.00				0.88	SAUTER ET AL 1976
624	CR	NP	WEIGHT	290.00				1.47	SAUTER ET AL 1976
625	CR	NP	WEIGHT	538.00				0.76	SAUTER ET AL 1976
626	CR	NP	WEIGHT	963.00				0.44	SAUTER ET AL 1976
627	CR	NP	WEIGHT	1975.00				0.34	SAUTER ET AL 1976
628	CR	RT	HATCH	0.00	400	94			SAUTER ET AL 1976
629	CR	RT	HATCH	1600.00	400	72			SAUTER ET AL 1976
630	CR	RT	HATCH	3200.00	400	126			SAUTER ET AL 1976
631	CR	RT	HATCH	6100.00	400	164			SAUTER ET AL 1976
632	CR	RT	HATCH	12200.00	400	338			SAUTER ET AL 1976
633	CR	RT	HATCH	26700.00	400	400			SAUTER ET AL 1976
634	CR	RT	HATCH	49700.00	400	400			SAUTER ET AL 1976
635	CR	RT	MORT2	0.00	200	21			SAUTER ET AL 1976
636	CR	RT	MORT2	1600.00	200	186			SAUTER ET AL 1976
637	CR	RT	MORT2	3200.00	200	200			SAUTER ET AL 1976
638	CR	RT	MORT2	6100.00	200	200			SAUTER ET AL 1976
639	CR	RT	MORT2	12200.00	200	200			SAUTER ET AL 1976
640	CR	RT	MORT2	26700.00	200	200			SAUTER ET AL 1976
641	CR	RT	MORT2	49700.00	200	200			SAUTER ET AL 1976
642	CR	RT	WEIGHT	0.00				0.47	SAUTER ET AL 1976
643	CR	RT	WEIGHT	1600.00				0.25	SAUTER ET AL 1976
644	CR	RT	WEIGHT	3200.00				0.00	SAUTER ET AL 1976
645	CR	RT	WEIGHT	6100.00				0.00	SAUTER ET AL 1976
646	CR	RT	WEIGHT	12200.00				0.00	SAUTER ET AL 1976
647	CR	RT	WEIGHT	26700.00				0.00	SAUTER ET AL 1976
648	CR	RT	WEIGHT	49700.00				0.00	SAUTER ET AL 1976

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
649	CR	WS	WEIGHT	0.00				0.24	SAUTER ET AL 1976
650	CR	WS	WEIGHT	123.00				0.19	SAUTER ET AL 1976
651	CR	WS	WEIGHT	290.00				0.22	SAUTER ET AL 1976
652	CR	WS	WEIGHT	538.00				0.17	SAUTER ET AL 1976
653	CR	WS	WEIGHT	963.00				0.11	SAUTER ET AL 1976
654	CR	WS	WEIGHT	1975.00				0	UTER ET AL 1976
655	CR	RT	HATCH	0.00	267	4			TEVENS AND CHAPMAN 1984
656	CR	RT	HATCH	9.00	146	3			STEVENS AND CHAPMAN 1984
657	CR	RT	HATCH	13.00	141	1			STEVENS AND CHAPMAN 1984
658	CR	RT	HATCH	19.00	146	4			STEVENS AND CHAPMAN 1984
659	CR	RT	HATCH	30.00	134	3			STEVENS AND CHAPMAN 1984
660	CR	RT	HATCH	48.00	136	3			STEVENS AND CHAPMAN 1984
661	CR	RT	HATCH	89.00	140	18			STEVENS AND CHAPMAN 1984
662	CR	RT	HATCH	157.00	137	77			STEVENS AND CHAPMAN 1984
663	CR	RT	HATCH	271.00	145	141			STEVENS AND CHAPMAN 1984
664	CR	RT	HATCH	495.00	139	139			STEVENS AND CHAPMAN 1984
665	CR	RT	MORT2	0.00	243	10			STEVENS AND CHAPMAN 1984
666	CR	RT	MORT2	9.00	143	11			STEVENS AND CHAPMAN 1984
667	CR	RT	MORT2	13.00	140	10			STEVENS AND CHAPMAN 1984
668	CR	RT	MORT2	19.00	142	6			STEVENS AND CHAPMAN 1984
669	CR	RT	MORT2	30.00	131	12			STEVENS AND CHAPMAN 1984
670	CR	RT	MORT2	48.00	133	12			STEVENS AND CHAPMAN 1984
671	CR	RT	MORT2	89.00	122	2			STEVENS AND CHAPMAN 1984
672	CR	RT	MORT2	157.00	60	7			STEVENS AND CHAPMAN 1984
673	CR	RT	MORT2	271.00	4	1			STEVENS AND CHAPMAN 1984
674	CR	RT	MORT2	495.00	0	0			STEVENS AND CHAPMAN 1984
675	CR	RT	WEIGHT	0.00				0.35	STEVENS AND CHAPMAN 1984
676	CR	RT	WEIGHT	9.00				0.33	STEVENS AND CHAPMAN 1984
677	CR	RT	WEIGHT	13.00				0.32	STEVENS AND CHAPMAN 1984
678	CR	RT	WEIGHT	19.00				0.38	STEVENS AND CHAPMAN 1984
679	CR	RT	WEIGHT	30.00				0.31	STEVENS AND CHAPMAN 1984
680	CR	RT	WEIGHT	48.00				0.30	STEVENS AND CHAPMAN 1984
681	CR	RT	WEIGHT	89.00				0.31	STEVENS AND CHAPMAN 1984
682	CR	RT	WEIGHT	157.00				0.32	STEVENS AND CHAPMAN 1984
683	CR	RT	WEIGHT	271.00				0.28	STEVENS AND CHAPMAN 1984
684	CR	RT	WEIGHT	495.00					STEVENS AND CHAPMAN 1984
685	CU	BG	EGGS	3.00			51906		BENOIT 1975
686	CU	BG	EGGS	12.00			46953		BENOIT 1975
687	CU	BG	EGGS	21.00			25354		BENOIT 1975
688	CU	BG	EGGS	40.00			4403		BENOIT 1975
689	CU	BG	EGGS	77.00			33300		BENOIT 1975
690	CU	BG	EGGS	162.00			0		BENOIT 1975
691	CU	BG	MORT1	3.00	20	1			BENOIT 1975
692	CU	BG	MORT1	12.00	20	1			BENOIT 1975
693	CU	BG	MORT1	21.00	20	1			BENOIT 1975
694	CU	BG	MORT1	40.00	20	1			BENOIT 1975
695	CU	BG	MORT1	77.00	20	4			BENOIT 1975
696	CU	BG	MORT1	162.00	20	12			BENOIT 1975
697	CU	BG	MORT2	3.00	100	61			BENOIT 1975
698	CU	BG	MORT2	12.00	100	51			BENOIT 1975
699	CU	BG	MORT2	21.00	100	56			BENOIT 1975
700	CU	BG	MORT2	40.00	100	83			BENOIT 1975
701	CU	BG	MORT2	77.00	100	91			BENOIT 1975
702	CU	BG	MORT2	162.00	100	100			BENOIT 1975

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
703	CU	BT	EGGS	1.90			328		MCKIM AND BENOIT 1971
704	CU	BT	EGGS	3.40			364		MCKIM AND BENOIT 1971
705	CU	BT	EGGS	5.70			296		MCKIM AND BENOIT 1971
706	CU	BT	EGGS	9.50			209		MCKIM AND BENOIT 1971
707	CU	BT	EGGS	17.40			315		MCKIM AND BENOIT 1971
708	CU	BT	EGGS	32.50			158		MCKIM AND BENOIT 1971
709	CU	BT	HATCH	1.90	200	38			MCKIM AND BENOIT 1971
710	CU	BT	HATCH	3.40	200	2			MCKIM AND BENOIT 1971
711	CU	BT	HATCH	5.70	200	30			MCKIM AND BENOIT 1971
712	CU	BT	HATCH	9.50	200	4			MCKIM AND BENOIT 1971
713	CU	BT	HATCH	17.40	200	10			MCKIM AND BENOIT 1971
714	CU	BT	HATCH	32.50	200	148			MCKIM AND BENOIT 1971
715	CU	BT	MORT1	1.90	14	1			MCKIM AND BENOIT 1971
716	CU	BT	MORT1	5.70	14	4			MCKIM AND BENOIT 1971
717	CU	BT	MORT1	9.50	28	4			MCKIM AND BENOIT 1971
718	CU	BT	MORT1	17.40	14	3			MCKIM AND BENOIT 1971
719	CU	BT	MORT1	32.50	14	8			MCKIM AND BENOIT 1971
720	CU	BT	MORT2	1.90	50	4			MCKIM AND BENOIT 1971
721	CU	BT	MORT2	3.40	50	4			MCKIM AND BENOIT 1971
722	CU	BT	MORT2	5.70	50	10			MCKIM AND BENOIT 1971
723	CU	BT	MORT2	9.50	50	11			MCKIM AND BENOIT 1971
724	CU	BT	MORT2	17.40	50	50			MCKIM AND BENOIT 1971
725	CU	BT	MORT2	32.50	50	50			MCKIM AND BENOIT 1971
726	CU	FM	EGGS	4.40			504		MOUNT AND STEPHAN 1969
727	CU	FM	EGGS	5.00			748		MOUNT AND STEPHAN 1969
728	CU	FM	EGGS	7.70			186		MOUNT AND STEPHAN 1969
729	CU	FM	EGGS	10.60			766		MOUNT AND STEPHAN 1969
730	CU	FM	EGGS	18.40			0		MOUNT AND STEPHAN 1969
731	CU	FM	HATCH	4.40	250	80			MOUNT AND STEPHAN 1969
732	CU	FM	HATCH	5.00	500	175			MOUNT AND STEPHAN 1969
733	CU	FM	HATCH	7.70	400	212			MOUNT AND STEPHAN 1969
734	CU	FM	HATCH	10.60	650	195			MOUNT AND STEPHAN 1969
735	CU	FM	MORT1	4.40	40	8			MOUNT AND STEPHAN 1969
736	CU	FM	MORT1	5.00	40	2			MOUNT AND STEPHAN 1969
737	CU	FM	MORT1	7.70	40	2			MOUNT AND STEPHAN 1969
738	CU	FM	MORT1	10.60	40	6			MOUNT AND STEPHAN 1969
739	CU	FM	MORT1	18.40	40	20			MOUNT AND STEPHAN 1969
740	CU	FM	MORT2	4.40	50	27			MOUNT AND STEPHAN 1969
741	CU	FM	MORT2	5.00	50	3			MOUNT AND STEPHAN 1969
742	CU	FM	MORT2	7.70	50	23			MOUNT AND STEPHAN 1969
743	CU	FM	MORT2	10.60	50	28			MOUNT AND STEPHAN 1969
744	CU	FM	EGGS	4.40			524		MOUNT 1968
745	CU	FM	EGGS	5.30			397		MOUNT 1968
746	CU	FM	EGGS	6.30			481		MOUNT 1968
747	CU	FM	EGGS	15.00			201		MOUNT 1968
748	CU	FM	EGGS	14.00			528		MOUNT 1968
749	CU	FM	EGGS	32.00			0		MOUNT 1968
750	CU	FM	EGGS	34.00			0		MOUNT 1968
751	CU	FM	EGGS	95.00			0		MOUNT 1968
752	CU	FM	HATCH	4.40	200	15			MOUNT 1968
753	CU	FM	HATCH	5.30	200	35			MOUNT 1968
754	CU	FM	HATCH	6.30	200	11			MOUNT 1968
755	CU	FM	HATCH	14.00	200	11			MOUNT 1968
756	CU	FM	HATCH	15.00	200	12			MOUNT 1968

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
757	CU	FM	MORT1	4.40	10	1			MOUNT 1968
758	CU	FM	MORT1	5.30	10	1			MOUNT 1968
759	CU	FM	MORT1	6.30	10	0			MOUNT 1968
760	CU	FM	MORT1	14.00	10	1			MOUNT 1968
761	CU	FM	MORT1	15.00	10	1			MOUNT 1968
762	CU	FM	MORT1	32.00	10	3			MOUNT 1968
763	CU	FM	MORT1	34.00	10	2			MOUNT 1968
764	CU	FM	MORT1	95.00	20	9			MOUNT 1968
765	CU	BT	HATCH	0.00	400	96			SAUTER ET AL 1976
766	CU	BT	HATCH	5.00	400	102			SAUTER ET AL 1976
767	CU	BT	HATCH	7.00	400	130			SAUTER ET AL 1976
768	CU	BT	HATCH	13.00	400	264			SAUTER ET AL 1976
769	CU	BT	HATCH	27.00	400	380			SAUTER ET AL 1976
770	CU	BT	HATCH	51.00	400	386			SAUTER ET AL 1976
771	CU	BT	HATCH	95.00	400	400			SAUTER ET AL 1976
772	CU	BT	MORT2	0.00	200	6			SAUTER ET AL 1976
773	CU	BT	MORT2	5.00	200	14			SAUTER ET AL 1976
774	CU	BT	MORT2	7.00	200	6			SAUTER ET AL 1976
775	CU	BT	MORT2	13.00	200	55			SAUTER ET AL 1976
776	CU	BT	MORT2	27.00	200	198			SAUTER ET AL 1976
777	CU	BT	MORT2	51.00	200	200			SAUTER ET AL 1976
778	CU	BT	MORT2	95.00	200	200			SAUTER ET AL 1976
779	CU	BT	WEIGHT	0.00				0.22	SAUTER ET AL 1976
780	CU	BT	WEIGHT	5.00				0.15	SAUTER ET AL 1976
781	CU	BT	WEIGHT	7.00				0.13	SAUTER ET AL 1976
782	CU	BT	WEIGHT	13.00				0.11	SAUTER ET AL 1976
783	CU	BT	WEIGHT	27.00				0.09	SAUTER ET AL 1976
784	CU	BT	WEIGHT	51.00				0.00	SAUTER ET AL 1976
785	CU	BT	WEIGHT	95.00				0.00	SAUTER ET AL 1976
786	CU	CC	WEIGHT	0.00				0.37	SAUTER ET AL 1976
787	CU	CC	WEIGHT	3.00				0.29	SAUTER ET AL 1976
788	CU	CC	WEIGHT	6.00				0.32	SAUTER ET AL 1976
789	CU	CC	WEIGHT	7.00				0.34	SAUTER ET AL 1976
790	CU	CC	WEIGHT	12.00				0.32	SAUTER ET AL 1976
791	CU	CC	WEIGHT	18.00				0.20	SAUTER ET AL 1976
792	CU	CC	WEIGHT	24.00				0.00	SAUTER ET AL 1976
793	CU	RT	HATCH	3.00	240	6			SEIM ET AL 1984
794	CU	RT	HATCH	6.00	240	3			SEIM ET AL 1984
795	CU	RT	HATCH	9.00	240	5			SEIM ET AL 1984
796	CU	RT	HATCH	16.00	240	8			SEIM ET AL 1984
797	CU	RT	HATCH	31.00	240	6			SEIM ET AL 1984
798	CU	RT	HATCH	57.00	240	3			SEIM ET AL 1984
799	CU	RT	HATCH	121.00	240	183			SEIM ET AL 1984
800	CU	RT	MORT2	3.00	100	3			SEIM ET AL 1984
801	CU	RT	MORT2	6.00	100	0			SEIM ET AL 1984
802	CU	RT	MORT2	9.00	100	0			SEIM ET AL 1984
803	CU	RT	MORT2	16.00	100	1			SEIM ET AL 1984
804	CU	RT	MORT2	31.00	100	5			SEIM ET AL 1984
805	CU	RT	MORT2	57.00	100	16			SEIM ET AL 1984
806	CU	RT	MORT2	121.00	37	37			SEIM ET AL 1984
807	CU	RT	WEIGHT	3.00				0.13	SEIM ET AL 1984
808	CU	RT	WEIGHT	6.00				0.14	SEIM ET AL 1984
809	CU	RT	WEIGHT	9.00				0.15	SEIM ET AL 1984
810	CU	RT	WEIGHT	16.00				0.15	SEIM ET AL 1984

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
811	CU	RT	WEIGHT	31.00				0.11	SEIM ET AL 1984
812	CU	RT	WEIGHT	57.00				0.05	SEIM ET AL 1984
813	CU	RT	WEIGHT	121.00				0.00	SEIM ET AL 1984
814	DI-N-BUTYL PHTHALATE	FM	HATCH	0.00	100	31			MCCARTHY AND WHITMORE 1984
815	DI-N-BUTYL PHTHALATE	FM	HATCH	100.00	100	34			MCCARTHY AND WHITMORE 1984
816	DI-N-BUTYL PHTHALATE	FM	HATCH	180.00	100	31			MCCARTHY AND WHITMORE 1984
817	DI-N-BUTYL PHTHALATE	FM	HATCH	320.00	100	32			MCCARTHY AND WHITMORE 1984
818	DI-N-BUTYL PHTHALATE	FM	HATCH	560.00	100	45			MCCARTHY AND WHITMORE 1984
819	DI-N-BUTYL PHTHALATE	FM	HATCH	1000.00	100	72			MCCARTHY AND WHITMORE 1984
820	DI-N-BUTYL PHTHALATE	FM	HATCH	1800.00	100	100			MCCARTHY AND WHITMORE 1984
821	DI-N-BUTYL PHTHALATE	FM	MORT2	0.00	69	4			MCCARTHY AND WHITMORE 1984
822	DI-N-BUTYL PHTHALATE	FM	MORT2	100.00	66	11			MCCARTHY AND WHITMORE 1984
823	DI-N-BUTYL PHTHALATE	FM	MORT2	180.00	69	9			MCCARTHY AND WHITMORE 1984
824	DI-N-BUTYL PHTHALATE	FM	MORT2	320.00	68	4			MCCARTHY AND WHITMORE 1984
825	DI-N-BUTYL PHTHALATE	FM	MORT2	560.00	55	8			MCCARTHY AND WHITMORE 1984
826	DI-N-BUTYL PHTHALATE	FM	MORT2	1000.00	28	22			MCCARTHY AND WHITMORE 1984
827	DI-N-BUTYL PHTHALATE	FM	MORT2	1800.00	0				MCCARTHY AND WHITMORE 1984
828	DI-N-OCTYL PHTHALATE	FM	HATCH	0.00	100	1			MCCARTHY AND WHITMORE 1984
829	DI-N-OCTYL PHTHALATE	FM	HATCH	100.00	100	0			MCCARTHY AND WHITMORE 1984
830	DI-N-OCTYL PHTHALATE	FM	HATCH	320.00	100	1			MCCARTHY AND WHITMORE 1984
831	DI-N-OCTYL PHTHALATE	FM	HATCH	1000.00	100	5			MCCARTHY AND WHITMORE 1984
832	DI-N-OCTYL PHTHALATE	FM	HATCH	3200.00	100	0			MCCARTHY AND WHITMORE 1984
833	DI-N-OCTYL PHTHALATE	FM	HATCH	10000.00	100	35			MCCARTHY AND WHITMORE 1984
834	DIAZINON	BT	EGGS	0.00			490		ALLISON AND HERMANUTZ 1977
835	DIAZINON	BT	EGGS	0.55			334		ALLISON AND HERMANUTZ 1977
836	DIAZINON	BT	EGGS	1.10			807		ALLISON AND HERMANUTZ 1977
837	DIAZINON	BT	EGGS	2.40			593		ALLISON AND HERMANUTZ 1977
838	DIAZINON	BT	EGGS	4.80			402		ALLISON AND HERMANUTZ 1977
839	DIAZINON	BT	EGGS	9.60			220		ALLISON AND HERMANUTZ 1977
840	DIAZINON	BT	HATCH	0.00	250	92			ALLISON AND HERMANUTZ 1977
841	DIAZINON	BT	HATCH	0.80	300	28			ALLISON AND HERMANUTZ 1977
842	DIAZINON	BT	HATCH	1.40	500	145			ALLISON AND HERMANUTZ 1977
843	DIAZINON	BT	HATCH	2.70	200	77			ALLISON AND HERMANUTZ 1977
844	DIAZINON	BT	HATCH	5.60	50	26			ALLISON AND HERMANUTZ 1977
845	DIAZINON	BT	HATCH	11.10	250	15			ALLISON AND HERMANUTZ 1977
846	DIAZINON	BT	MORT1	0.00	24	0			ALLISON AND HERMANUTZ 1977
847	DIAZINON	BT	MORT1	0.55	24	0			ALLISON AND HERMANUTZ 1977
848	DIAZINON	BT	MORT1	1.10	24	0			ALLISON AND HERMANUTZ 1977
849	DIAZINON	BT	MORT1	2.40	24	1			ALLISON AND HERMANUTZ 1977
850	DIAZINON	BT	MORT1	4.80	24	1			ALLISON AND HERMANUTZ 1977
851	DIAZINON	BT	MORT1	9.60	24	6			ALLISON AND HERMANUTZ 1977
852	DIAZINON	BT	MORT2	0.00	100	8			ALLISON AND HERMANUTZ 1977
853	DIAZINON	BT	MORT2	0.80	100	28			ALLISON AND HERMANUTZ 1977
854	DIAZINON	BT	MORT2	1.40	100	23			ALLISON AND HERMANUTZ 1977
855	DIAZINON	BT	MORT2	2.70	93	4			ALLISON AND HERMANUTZ 1977
856	DIAZINON	BT	MORT2	5.60	25	9			ALLISON AND HERMANUTZ 1977
857	DIAZINON	BT	MORT2	11.10	75	13			ALLISON AND HERMANUTZ 1977
858	DIAZINON	FM	EGGS	0.00			361		ALLISON AND HERMANUTZ 1977
859	DIAZINON	FM	EGGS	3.20			505		ALLISON AND HERMANUTZ 1977
860	DIAZINON	FM	EGGS	6.90			137		ALLISON AND HERMANUTZ 1977
861	DIAZINON	FM	EGGS	13.50			76		ALLISON AND HERMANUTZ 1977
862	DIAZINON	FM	EGGS	28.00			1		ALLISON AND HERMANUTZ 1977
863	DIAZINON	FM	EGGS	60.30			0		ALLISON AND HERMANUTZ 1977
864	DIAZINON	FM	HATCH	0.00	1100	88			ALLISON AND HERMANUTZ 1977

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
865	DIAZINON	FM	HATCH	3.20	900	288			ALLISON AND HERMANUTZ 1977
866	DIAZINON	FM	HATCH	6.90	150	36			ALLISON AND HERMANUTZ 1977
867	DIAZINON	FM	HATCH	28.00	200	12			ALLISON AND HERMANUTZ 1977
868	DIAZINON	FM	HATCH	60.30	500	35			ALLISON AND HERMANUTZ 1977
869	DIAZINON	FM	MORT1	0.00	100	28			ALLISON AND HERMANUTZ 1977
870	DIAZINON	FM	MORT1	3.20	100	15			ALLISON AND HERMANUTZ 1977
871	DIAZINON	FM	MORT1	6.90	100	36			ALLISON AND HERMANUTZ 1977
872	DIAZINON	FM	MORT1	13.50	100	18			ALLISON AND HERMANUTZ 1977
873	DIAZINON	FM	MORT1	28.00	100	34			ALLISON AND HERMANUTZ 1977
874	DIAZINON	FM	MORT1	60.30	100	66			ALLISON AND HERMANUTZ 1977
875	DIAZINON	FM	MORT2	0.00	400	134			ALLISON AND HERMANUTZ 1977
876	DIAZINON	FM	MORT2	3.30	320	83			ALLISON AND HERMANUTZ 1977
877	DIAZINON	FM	MORT2	6.80	40	18			ALLISON AND HERMANUTZ 1977
878	DIAZINON	FM	MORT2	28.00	280	99			ALLISON AND HERMANUTZ 1977
879	DIAZINON	FM	MORT2	62.60	320	77			ALLISON AND HERMANUTZ 1977
880	DINOSEB	FM	HATCH	0.00	200	55			CALL ET AL 1983
881	DINOSEB	FM	HATCH	0.40	200	31			CALL ET AL 1983
882	DINOSEB	FM	HATCH	1.70	200	33			CALL ET AL 1983
883	DINOSEB	FM	HATCH	4.30	200	46			CALL ET AL 1983
884	DINOSEB	FM	HATCH	14.50	200	62			CALL ET AL 1983
885	DINOSEB	FM	HATCH	48.50	200	43			CALL ET AL 1983
886	DINOSEB	FM	MORT2	0.00	60	7			CALL ET AL 1983
887	DINOSEB	FM	MORT2	0.40	60	13			CALL ET AL 1983
888	DINOSEB	FM	MORT2	1.70	60	11			CALL ET AL 1983
889	DINOSEB	FM	MORT2	4.30	60	8			CALL ET AL 1983
890	DINOSEB	FM	MORT2	14.50	60	28			CALL ET AL 1983
891	DINOSEB	FM	MORT2	48.50	60	55			CALL ET AL 1983
892	DINOSEB	FM	WEIGHT	0.00				0.60	CALL ET AL 1983
893	DINOSEB	FM	WEIGHT	0.40				0.68	CALL ET AL 1983
894	DINOSEB	FM	WEIGHT	1.70				0.73	CALL ET AL 1983
895	DINOSEB	FM	WEIGHT	4.30				0.65	CALL ET AL 1983
896	DINOSEB	FM	WEIGHT	14.50				0.68	CALL ET AL 1983
897	DINOSEB	FM	WEIGHT	48.50				0.52	CALL ET AL 1983
898	DINOSEB	LT	WEIGHT	0.00				378.00	WOODWARD 1976
899	DINOSEB	LT	WEIGHT	0.50				247.00	WOODWARD 1976
900	DINOSEB	LT	WEIGHT	1.60				241.00	WOODWARD 1976
901	DINOSEB	LT	WEIGHT	2.30				244.00	WOODWARD 1976
902	DINOSEB	LT	WEIGHT	4.90				208.00	WOODWARD 1976
903	DINOSEB	LT	WEIGHT	10.00				152.00	WOODWARD 1976
904	DIURON	FM	HATCH	0.00	200	67			CALL ET AL 1983
905	DIURON	FM	HATCH	2.60	200	45			CALL ET AL 1983
906	DIURON	FM	HATCH	6.10	200	52			CALL ET AL 1983
907	DIURON	FM	HATCH	14.50	200	61			CALL ET AL 1983
908	DIURON	FM	HATCH	33.40	200	75			CALL ET AL 1983
909	DIURON	FM	HATCH	78.00	200	88			CALL ET AL 1983
910	DIURON	FM	MORT2	0.00	60	11			CALL ET AL 1983
911	DIURON	FM	MORT2	2.60	60	7			CALL ET AL 1983
912	DIURON	FM	MORT2	6.10	60	4			CALL ET AL 1983
913	DIURON	FM	MORT2	14.50	60	17			CALL ET AL 1983
914	DIURON	FM	MORT2	33.40	60	15			CALL ET AL 1983
915	DIURON	FM	MORT2	78.00	60	45			CALL ET AL 1983
916	DIURON	FM	WEIGHT	0.00				0.57	CALL ET AL 1983
917	DIURON	FM	WEIGHT	2.60				0.57	CALL ET AL 1983
918	DIURON	FM	WEIGHT	6.10				0.56	CALL ET AL 1983

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
919	DIURON	FM	WEIGHT	14.50				0.62	CALL ET AL 1983
920	DIURON	FM	WEIGHT	33.40				0.56	CALL ET AL 1983
921	DIURON	FM	WEIGHT	78.00				0.50	CALL ET AL 1983
922	DTDMAC	FM	WEIGHT	0.00				0.08	LEWIS AND WEE 1983
923	DTDMAC	FM	WEIGHT	6.00				0.08	LEWIS AND WEE 1983
924	DTDMAC	FM	WEIGHT	13.00				0.08	LEWIS AND WEE 1983
925	DTDMAC	FM	WEIGHT	24.00				0.07	LEWIS AND WEE 1983
926	DTDMAC	FM	WEIGHT	53.00				0.08	LEWIS AND WEE 1983
927	DTDMAC	FM	WEIGHT	90.00				0.03	LEWIS AND WEE 1983
928	ENDOSULFAN	FM	HATCH	0.00	1900	325			CARLSON ET AL 1982
929	ENDOSULFAN	FM	HATCH	0.04	200	28			CARLSON ET AL 1982
930	ENDOSULFAN	FM	HATCH	0.06	1850	231			CARLSON ET AL 1982
931	ENDOSULFAN	FM	HATCH	0.10	1150	161			CARLSON ET AL 1982
932	ENDOSULFAN	FM	HATCH	0.20	1850	425			CARLSON ET AL 1982
933	ENDOSULFAN	FM	HATCH	0.40	150	148			CARLSON ET AL 1982
934	ENDOSULFAN	FM	MORT1	0.00	30	8			CARLSON ET AL 1982
935	ENDOSULFAN	FM	MORT1	0.04	30	18			CARLSON ET AL 1982
936	ENDOSULFAN	FM	MORT1	0.06	30	6			CARLSON ET AL 1982
937	ENDOSULFAN	FM	MORT1	0.10	30	5			CARLSON ET AL 1982
938	ENDOSULFAN	FM	MORT1	0.20	30	13			CARLSON ET AL 1982
939	ENDOSULFAN	FM	MORT1	0.40	15	15			CARLSON ET AL 1982
940	ENDOSULFAN	FM	MORT2	0.00	360	77			CARLSON ET AL 1982
941	ENDOSULFAN	FM	MORT2	0.04	80	21			CARLSON ET AL 1982
942	ENDOSULFAN	FM	MORT2	0.06	320	83			CARLSON ET AL 1982
943	ENDOSULFAN	FM	MORT2	0.10	320	73			CARLSON ET AL 1982
944	ENDOSULFAN	FM	MORT2	0.20	280	70			CARLSON ET AL 1982
945	ENDRIN	FF	MORT2	0.00	90	1			CARLSON ET AL 1982
946	ENDRIN	FF	MORT2	0.04	90	3			CARLSON ET AL 1982
947	ENDRIN	FF	MORT2	0.07	90	4			CARLSON ET AL 1982
948	ENDRIN	FF	MORT2	0.15	90	2			CARLSON ET AL 1982
949	ENDRIN	FF	MORT2	0.30	90	12			CARLSON ET AL 1982
950	ENDRIN	FF	MORT2	0.60	90	90			CARLSON ET AL 1982
951	FENITROTHION	FM	MORT2	0.00	60	15			KLEINER ET AL 1984
952	FENITROTHION	FM	MORT2	20.00	60	10			KLEINER ET AL 1984
953	FENITROTHION	FM	MORT2	60.00	60	17			KLEINER ET AL 1984
954	FENITROTHION	FM	MORT2	130.00	60	14			KLEINER ET AL 1984
955	FENITROTHION	FM	MORT2	300.00	60	24			KLEINER ET AL 1984
956	FENITROTHION	FM	MORT2	740.00	60	43			KLEINER ET AL 1984
957	FENITROTHION	FM	WEIGHT	0.00				0.14	KLEINER ET AL 1984
958	FENITROTHION	FM	WEIGHT	20.00				0.14	KLEINER ET AL 1984
959	FENITROTHION	FM	WEIGHT	60.00				0.15	KLEINER ET AL 1984
960	FENITROTHION	FM	WEIGHT	130.00				0.14	KLEINER ET AL 1984
961	FENITROTHION	FM	WEIGHT	300.00				0.10	KLEINER ET AL 1984
962	FENITROTHION	FM	WEIGHT	740.00				0.06	KLEINER ET AL 1984
963	FONOFOS	FM	HATCH	0.00	100	6			PICKERING AND GILIAM 1982
964	FONOFOS	FM	HATCH	4.90	100	5			PICKERING AND GILIAM 1982
965	FONOFOS	FM	HATCH	9.20	100	3			PICKERING AND GILIAM 1982
966	FONOFOS	FM	HATCH	16.00	100	4			PICKERING AND GILIAM 1982
967	FONOFOS	FM	HATCH	33.00	100	7			PICKERING AND GILIAM 1982
968	FONOFOS	FM	HATCH	66.00	100	5			PICKERING AND GILIAM 1982
969	FONOFOS	FM	MORT2	0.00	60	5			PICKERING AND GILIAM 1982
970	FONOFOS	FM	MORT2	4.90	60	5			PICKERING AND GILIAM 1982
971	FONOFOS	FM	MORT2	9.20	60	4			PICKERING AND GILIAM 1982
972	FONOFOS	FM	MORT2	16.00	60	5			PICKERING AND GILIAM 1982

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
973	FONOFOS	FM	MORT2	33.00	60	20			PICKERING AND GILLIAM 1982
974	FONOFOS	FM	MORT2	66.00	60	40			PICKERING AND GILLIAM 1982
975	FONOFOS	FM	WEIGHT	0.00				0.17	PICKERING AND GILLIAM 1982
976	FONOFOS	FM	WEIGHT	4.90				0.20	PICKERING AND GILLIAM 1982
977	FONOFOS	FM	WEIGHT	9.20				0.18	PICKERING AND GILLIAM 1982
978	FONOFOS	FM	WEIGHT	16.00				0.15	PICKERING AND GILLIAM 1982
979	FONOFOS	FM	WEIGHT	33.00				0.12	PICKERING AND GILLIAM 1982
980	FONOFOS	FM	WEIGHT	66.00				0.04	PICKERING AND GILLIAM 1982
981	GUTHION	FM	EGGS	0.04	15		1691		ADELMAN ET AL 1976
982	GUTHION	FM	EGGS	0.10			1220		ADELMAN ET AL 1976
983	GUTHION	FM	EGGS	0.16			1611		ADELMAN ET AL 1976
984	GUTHION	FM	EGGS	0.24			1239		ADELMAN ET AL 1976
985	GUTHION	FM	EGGS	0.33			1718		ADELMAN ET AL 1976
986	GUTHION	FM	EGGS	0.51			256		ADELMAN ET AL 1976
987	GUTHION	FM	EGGS	0.72			782		ADELMAN ET AL 1976
988	HEPTACHLOR	FM	EGGS	0.00			772		MACEK ET AL 1976A
989	HEPTACHLOR	FM	EGGS	0.11			385		MACEK ET AL 1976A
990	HEPTACHLOR	FM	EGGS	0.20			697		MACEK ET AL 1976A
991	HEPTACHLOR	FM	EGGS	0.43			733		MACEK ET AL 1976A
992	HEPTACHLOR	FM	EGGS	0.86			1558		MACEK ET AL 1976A
993	HEPTACHLOR	FM	EGGS	1.84			0		MACEK ET AL 1976A
994	HEPTACHLOR	FM	HATCH	0.11	650	91			MACEK ET AL 1976A
995	HEPTACHLOR	FM	HATCH	0.20	900	112			MACEK ET AL 1976A
996	HEPTACHLOR	FM	HATCH	0.43	1550	276			MACEK ET AL 1976A
997	HEPTACHLOR	FM	HATCH	0.86	2350	245			MACEK ET AL 1976A
998	HEPTACHLOR	FM	MORT1	0.00	30	6			MACEK ET AL 1976A
999	HEPTACHLOR	FM	MORT1	0.11	30	13			MACEK ET AL 1976A
1000	HEPTACHLOR	FM	MORT1	0.20	30	6			MACEK ET AL 1976A
1001	HEPTACHLOR	FM	MORT1	0.43	30	9			MACEK ET AL 1976A
1002	HEPTACHLOR	FM	MORT1	0.86	30	13			MACEK ET AL 1976A
1003	HEPTACHLOR	FM	MORT1	1.84	30	30			MACEK ET AL 1976A
1004	HEPTACHLOR	FM	MORT2	0.00	320	107			MACEK ET AL 1976A
1005	HEPTACHLOR	FM	MORT2	0.11	320	77			MACEK ET AL 1976A
1006	HEPTACHLOR	FM	MORT2	0.20	320	198			MACEK ET AL 1976A
1007	HEPTACHLOR	FM	MORT2	0.43	320	54			MACEK ET AL 1976A
1008	HEPTACHLOR	FM	MORT2	0.86	320	114			MACEK ET AL 1976A
1009	HEXACHLOROBUTADIENE	FM	HATCH	0.08	120	25			BENOIT ET AL 1982
1010	HEXACHLOROBUTADIENE	FM	HATCH	1.70	120	40			BENOIT ET AL 1982
1011	HEXACHLOROBUTADIENE	FM	HATCH	3.20	120	39			BENOIT ET AL 1982
1012	HEXACHLOROBUTADIENE	FM	HATCH	6.50	120	43			BENOIT ET AL 1982
1013	HEXACHLOROBUTADIENE	FM	HATCH	13.00	120	42			BENOIT ET AL 1982
1014	HEXACHLOROBUTADIENE	FM	HATCH	27.00	120	34			BENOIT ET AL 1982
1015	HEXACHLOROBUTADIENE	FM	MORT2	0.08	60	0			BENOIT ET AL 1982
1016	HEXACHLOROBUTADIENE	FM	MORT2	1.70	60	1			BENOIT ET AL 1982
1017	HEXACHLOROBUTADIENE	FM	MORT2	3.20	60	2			BENOIT ET AL 1982
1018	HEXACHLOROBUTADIENE	FM	MORT2	6.50	60	9			BENOIT ET AL 1982
1019	HEXACHLOROBUTADIENE	FM	MORT2	13.00	60	28			BENOIT ET AL 1982
1020	HEXACHLOROBUTADIENE	FM	MORT2	27.00	60	27			BENOIT ET AL 1982
1021	HEXACHLOROBUTADIENE	FM	WEIGHT	0.08				0.13	BENOIT ET AL 1982
1022	HEXACHLOROBUTADIENE	FM	WEIGHT	1.70				0.13	BENOIT ET AL 1982
1023	HEXACHLOROBUTADIENE	FM	WEIGHT	3.20				0.13	BENOIT ET AL 1982
1024	HEXACHLOROBUTADIENE	FM	WEIGHT	6.50				0.13	BENOIT ET AL 1982
1025	HEXACHLOROBUTADIENE	FM	WEIGHT	13.00				0.10	BENOIT ET AL 1982
1026	HEXACHLOROBUTADIENE	FM	WEIGHT	27.00				.03	BENOIT ET AL 1982

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1027	HEXACHLOROCYCLOHEXAN	BG	HATCH	0.60	600	60			MACEK ET AL 1976B
1028	HEXACHLOROCYCLOHEXAN	BG	HATCH	1.10	200	24			MACEK ET AL 1976B
1029	HEXACHLOROCYCLOHEXAN	BG	HATCH	2.30	2200	770			MACEK ET AL 1976B
1030	HEXACHLOROCYCLOHEXAN	BG	HATCH	4.40	400	120			MACEK ET AL 1976B
1031	HEXACHLOROCYCLOHEXAN	BG	MORT1	0.00	20	3			MACEK ET AL 1976B
1032	HEXACHLOROCYCLOHEXAN	BG	MORT1	0.60	20	1			MACEK ET AL 1976B
1033	HEXACHLOROCYCLOHEXAN	BG	MORT1	1.10	20	3			MACEK ET AL 1976B
1034	HEXACHLOROCYCLOHEXAN	BG	MORT1	2.30	20	5			MACEK ET AL 1976B
1035	HEXACHLOROCYCLOHEXAN	BG	MORT1	4.40	20	4			MACEK ET AL 1976B
1036	HEXACHLOROCYCLOHEXAN	BG	MORT1	9.10	20	3			MACEK ET AL 1976B
1037	HEXACHLOROCYCLOHEXAN	BG	MORT2	0.60	30	30			MACEK ET AL 1976B
1038	HEXACHLOROCYCLOHEXAN	BG	MORT2	1.10	30	26			MACEK ET AL 1976B
1039	HEXACHLOROCYCLOHEXAN	BG	MORT2	2.30	120	49			MACEK ET AL 1976B
1040	HEXACHLOROCYCLOHEXAN	BG	MORT2	4.40	30	26			MACEK ET AL 1976B
1041	HEXACHLOROCYCLOHEXAN	BT	HATCH	0.00	100	75			MACEK ET AL 1976B
1042	HEXACHLOROCYCLOHEXAN	BT	HATCH	1.10	50	7			MACEK ET AL 1976B
1043	HEXACHLOROCYCLOHEXAN	BT	HATCH	2.10	200	6			MACEK ET AL 1976B
1044	HEXACHLOROCYCLOHEXAN	BT	HATCH	4.10	150	53			MACEK ET AL 1976B
1045	HEXACHLOROCYCLOHEXAN	BT	HATCH	8.80	50	12			MACEK ET AL 1976B
1046	HEXACHLOROCYCLOHEXAN	BT	HATCH	16.60	50	36			MACEK ET AL 1976B
1047	HEXACHLOROCYCLOHEXAN	BT	MORT2	0.00	50	23			MACEK ET AL 1976B
1048	HEXACHLOROCYCLOHEXAN	BT	MORT2	1.10	50	49			MACEK ET AL 1976B
1049	HEXACHLOROCYCLOHEXAN	BT	MORT2	2.10	50	25			MACEK ET AL 1976B
1050	HEXACHLOROCYCLOHEXAN	BT	MORT2	4.10	50	34			MACEK ET AL 1976B
1051	HEXACHLOROCYCLOHEXAN	BT	MORT2	8.80	50	39			MACEK ET AL 1976B
1052	HEXACHLOROCYCLOHEXAN	BT	MORT2	16.60	25	23			MACEK ET AL 1976B
1053	HEXACHLOROCYCLOHEXAN	FM	HATCH	0.00	200	26			MACEK ET AL 1976B
1054	HEXACHLOROCYCLOHEXAN	FM	HATCH	1.40	900	81			MACEK ET AL 1976B
1055	HEXACHLOROCYCLOHEXAN	FM	HATCH	2.40	1600	192			MACEK ET AL 1976B
1056	HEXACHLOROCYCLOHEXAN	FM	HATCH	5.60	1600	176			MACEK ET AL 1976B
1057	HEXACHLOROCYCLOHEXAN	FM	HATCH	9.10	1550	186			MACEK ET AL 1976B
1058	HEXACHLOROCYCLOHEXAN	FM	HATCH	23.40	1350	189			MACEK ET AL 1976B
1059	HEXACHLOROCYCLOHEXAN	FM	MORT1	0.00	15	1			MACEK ET AL 1976B
1060	HEXACHLOROCYCLOHEXAN	FM	MORT1	1.40	15	0			MACEK ET AL 1976B
1061	HEXACHLOROCYCLOHEXAN	FM	MORT1	2.40	15	0			MACEK ET AL 1976B
1062	HEXACHLOROCYCLOHEXAN	FM	MORT1	5.60	15	1			MACEK ET AL 1976B
1063	HEXACHLOROCYCLOHEXAN	FM	MORT1	9.10	15	1			MACEK ET AL 1976B
1064	HEXACHLOROCYCLOHEXAN	FM	MORT1	23.50	15	4			MACEK ET AL 1976B
1065	HEXACHLOROCYCLOHEXAN	FM	MORT2	0.00	40	10			MACEK ET AL 1976B
1066	HEXACHLOROCYCLOHEXAN	FM	MORT2	1.40	160	26			MACEK ET AL 1976B
1067	HEXACHLOROCYCLOHEXAN	FM	MORT2	2.40	160	48			MACEK ET AL 1976B
1068	HEXACHLOROCYCLOHEXAN	FM	MORT2	5.60	160	53			MACEK ET AL 1976B
1069	HEXACHLOROCYCLOHEXAN	FM	MORT2	9.10	80	24			MACEK ET AL 1976B
1070	HEXACHLOROCYCLOHEXAN	FM	MORT2	23.40	80	14			MACEK ET AL 1976B
1071	HEXACHLOROETHANE	FM	MORT2	0.90	120	15			AHMED ET AL 1984
1072	HEXACHLOROETHANE	FM	MORT2	28.00	120	39			AHMED ET AL 1984
1073	HEXACHLOROETHANE	FM	MORT2	69.00	120	30			AHMED ET AL 1984
1074	HEXACHLOROETHANE	FM	MORT2	207.00	120	21			AHMED ET AL 1984
1075	HEXACHLOROETHANE	FM	MORT2	608.00	120	12			AHMED ET AL 1984
1076	HEXACHLOROETHANE	FM	MORT2	1604.00	120	120			AHMED ET AL 1984
1077	HEXACHLOROETHANE	FM	WEIGHT	0.90				0.17	AHMED ET AL 1984
1078	HEXACHLOROETHANE	FM	WEIGHT	28.00				0.19	AHMED ET AL 1984
1079	HEXACHLOROETHANE	FM	WEIGHT	69.00				0.16	AHMED ET AL 1984
1080	HEXACHLOROETHANE	FM	WEIGHT	207.00				0.12	AHMED ET AL 1984

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1081	HEXACHLOROETHANE	FM	WEIGHT	608.00				0.04	AHMED ET AL 1984
1082	HEXACHLOROETHANE	FM	WEIGHT	1604.00				0.00	AHMED ET AL 1984
1083	HG	FM	HATCH	0.01	200	71			CALL ET AL 1983B
1084	HG	FM	HATCH	0.23	200	61			CALL ET AL 1983B
1085	HG	FM	HATCH	0.48	200	66			CALL ET AL 1983B
1086	HG	FM	HATCH	1.85	200	88			CALL ET AL 1983B
1087	HG	FM	HATCH	0.87	200	54			CALL ET AL 1983B
1088	HG	FM	HATCH	0.67	200	200			CALL ET AL 1983B
1089	HG	FM	MORT2	0.01	60	0			CALL ET AL 1983B
1090	HG	FM	MORT2	0.23	60	0			CALL ET AL 1983B
1091	HG	FM	MORT2	0.48	60	0			CALL ET AL 1983B
1092	HG	FM	MORT2	0.87	60	0			CALL ET AL 1983B
1093	HG	FM	MORT2	1.85	60	26			CALL ET AL 1983B
1094	HG	FM	MORT2	3.70	60	53			CALL ET AL 1983B
1095	HG	FM	WEIGHT	0.01				0.21	CALL ET AL 1983B
1096	HG	FM	WEIGHT	0.23				0.19	CALL ET AL 1983B
1097	HG	FM	WEIGHT	0.48				0.19	CALL ET AL 1983B
1098	HG	FM	WEIGHT	0.87					CALL ET AL 1983B
1099	HG	FM	WEIGHT	1.85					CALL ET AL 1983B
1100	HG	FM	WEIGHT	3.70				0.01	CALL ET AL 1983B
1101	HG	FM	EGGS	0.00			1204		SNARSKI AND OLSON 1982
1102	HG	FM	EGGS	0.26			557		SNARSKI AND OLSON 1982
1103	HG	FM	EGGS	0.50			646		SNARSKI AND OLSON 1982
1104	HG	FM	EGGS	1.02			0		SNARSKI AND OLSON 1982
1105	HG	FM	EGGS	2.01			0		SNARSKI AND OLSON 1982
1106	HG	FM	EGGS	3.69			0		SNARSKI AND OLSON 1982
1107	HG	FM	WEIGHT	0.00				0.26	SNARSKI AND OLSON 1982
1108	HG	FM	WEIGHT	0.26				0.19	SNARSKI AND OLSON 1982
1109	HG	FM	WEIGHT	0.50				0.23	SNARSKI AND OLSON 1982
1110	HG	FM	WEIGHT	1.02				0.19	SNARSKI AND OLSON 1982
1111	HG	FM	WEIGHT	2.01				0.15	SNARSKI AND OLSON 1982
1112	HG	FM	WEIGHT	3.69				0.09	SNARSKI AND OLSON 1982
1113	ISOPHORONE	FM	MORT5	0.00	31	4			CAIRNS AND NEBEKER 1982
1114	ISOPHORONE	FM	MORT5	11.00	33	5			CAIRNS AND NEBEKER 1982
1115	ISOPHORONE	FM	MORT5	19.00	37	5			CAIRNS AND NEBEKER 1982
1116	ISOPHORONE	FM	MORT5	30.00	33	6			CAIRNS AND NEBEKER 1982
1117	ISOPHORONE	FM	MORT5	56.00	32	8			CAIRNS AND NEBEKER 1982
1118	ISOPHORONE	FM	MORT5	112.00	32	29			CAIRNS AND NEBEKER 1982
1119	ISOPHORONE	FM	WEIGHT	0.00				0.03	CAIRNS AND NEBEKER 1982
1120	ISOPHORONE	FM	WEIGHT	11000.00				0.02	CAIRNS AND NEBEKER 1982
1121	ISOPHORONE	FM	WEIGHT	19000.00				0.02	CAIRNS AND NEBEKER 1982
1122	ISOPHORONE	FM	WEIGHT	30000.00				0.01	CAIRNS AND NEBEKER 1982
1123	ISOPHORONE	FM	WEIGHT	56000.00				0.01	CAIRNS AND NEBEKER 1982
1124	ISOPHORONE	FM	WEIGHT	0.00				0.17	LEMKE ET AL 1983
1125	ISOPHORONE	FM	WEIGHT	2160.00				0.18	LEMKE ET AL 1983
1126	ISOPHORONE	FM	WEIGHT	4165.00				0.17	LEMKE ET AL 1983
1127	ISOPHORONE	FM	WEIGHT	8535.00				0.16	LEMKE ET AL 1993
1128	ISOPHORONE	FM	WEIGHT	15610.00				0.15	LEMKE ET AL 1983
1129	ISOPHORONE	FM	WEIGHT	25145.00				0.14	LEMKE ET AL 1983
1130	KELTHANE	FM	MORT2	0.00	30	0			SPEHAR ET AL 1982
1131	KELTHANE	FM	MORT2	8.90	30	6			SPEHAR ET AL 1982
1132	KELTHANE	FM	MORT2	19.00	30	6			SPEHAR ET AL 1982
1133	KELTHANE	FM	MORT2	39.00	30	16			SPEHAR ET AL 1982
1134	KELTHANE	FM	MORT2	73.00	30	30			SPEHAR ET AL 1982

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1135	KELTHANE	FM	MORT2	125.00	15	15			SPEHAR ET AL 1982
1136	KEPONE	FM	EGGS	0.00			386		BUCKLER ET AL 1981
1137	KEPONE	FM	EGGS	0.01			293		BUCKLER ET AL 1981
1138	KEPONE	FM	EGGS	0.07			212		BUCKLER ET AL 1981
1139	KEPONE	FM	EGGS	0.17			259		BUCKLER ET AL 1981
1140	KEPONE	FM	EGGS	0.33			319		BUCKLER ET AL 1981
1141	KEPONE	FM	EGGS	0.31			581		BUCKLER ET AL 1981
1142	KEPONE	FM	EGGS	0.31			581		BUCKLER ET AL 1991
1143	KEPONE	FM	HATCH	0.00	2950	1062			BUCKLER ET AL 1981
1144	KEPONE	FM	HATCH	0.01	2750	825			BUCKLER ET AL 1981
1145	KEPONE	FM	HATCH	0.03	2850	1083			BUCKLER ET AL 1981
1146	KEPONE	FM	HATCH	0.07	1950	566			BUCKLER ET AL 1981
1147	KEPONE	FM	HATCH	0.17	2250	652			BUCKLER ET AL 1981
1148	KEPONE	FM	HATCH	0.31	4200	2016			BUCKLER ET AL 1981
1149	KEPONE	FM	MORT1	0.00	68	4			BUCKLER ET AL 1981
1150	KEPONE	FM	MORT1	0.01	71	2			BUCKLER ET AL 1981
1151	KEPONE	FM	MORT1	0.03	71	0			BUCKLER ET AL 1981
1152	KEPONE	FM	MORT1	0.07	62	0			BUCKLER ET AL 1981
1153	KEPONE	FM	MORT1	0.17	60	7			BUCKLER ET AL 1981
1154	KEPONE	FM	MORT1	0.31	66	2			BUCKLER ET AL 1981
1155	KEPONE	FM	MORT2	0.00	80	19			BUCKLER ET AL 1981
1156	KEPONE	FM	MORT2	0.01	80	30			BUCKLER ET AL 1981
1157	KEPONE	FM	MORT2	0.03	80	18			BUCKLER ET AL 1981
1158	KEPONE	FM	MORT2	0.07	80	14			BUCKLER ET AL 1981
1159	KEPONE	FM	MORT2	0.17	80	35			BUCKLER ET AL 1981
1160	KEPONE	FM	MORT2	0.31	80	27			BUCKLER ET AL 1981
1161	LAS MIXTURE	FM	EGGS	0.00			2496		PICKERING AND THATCHER 1970
1162	LAS MIXTURE	FM	EGGS	340.00			3811		PICKERING AND THATCHER 1970
1163	LAS MIXTURE	FM	EGGS	630.00			2583		PICKERING AND THATCHER 1970
1164	LAS MIXTURE	FM	EGGS	1200.00			2168		PICKERING AND THATCHER 1970
1165	LAS MIXTURE	FM	EGGS	2700.00			1710		PICKERING AND THATCHER 1970
1166	LAS MIXTURE	FM	HATCH	0.00	400	16			PICKERING AND THATCHER 1970
1167	LAS MIXTURE	FM	HATCH	340.00	400	22			PICKERING AND THATCHER 1970
1168	LAS MIXTURE	FM	HATCH	630.00	400	16			PICKERING AND THATCHER 1970
1169	LAS MIXTURE	FM	HATCH	1200.00	400	23			PICKERING AND THATCHER 1970
1170	LAS MIXTURE	FM	HATCH	2700.00	400	46			PICKERING AND THATCHER 1970
1171	LAS MIXTURE	FM	MORT2	0.00	400	68			PICKERING AND THATCHER 1970
1172	LAS MIXTURE	FM	MORT2	340.00	400	60			PICKERING AND THATCHER 1970
1173	LAS MIXTURE	FM	MORT2	630.00	400	82			PICKERING AND THATCHER 1970
1174	LAS MIXTURE	FM	MORT2	1200.00	400	240			PICKERING AND THATCHER 1970
1175	LAS MIXTURE	FM	MORT2	2700.00	400	341			PICKERING AND THATCHER 1970
1176	LAS 11.2	FM	HATCH	0.00	100	17			HOLMAN AND MACEK 1980
1177	LAS 11.2	FM	HATCH	2500.00	100	11			HOLMAN AND MACEK 1980
1178	LAS 11.2	FM	HATCH	3000.00	100	19			HOLMAN AND MACEK 1980
1179	LAS 11.2	FM	HATCH	4400.00	100	21			HOLMAN AND MACEK 1980
1180	LAS 11.2	FM	HATCH	5100.00	100	34			HOLMAN AND MACEK 1980
1181	LAS 11.2	FM	HATCH	8400.00	100	64			HOLMAN AND MACEK 1980
1182	LAS 11.2	FM	HATCH	9800.00	100	59			HOLMAN AND MACEK 1980
1183	LAS 11.2	FM	HATCH	14200.00	100	94			HOLMAN AND MACEK 1980
1184	LAS 11.2	FM	MORT2	0.00	80	29			HOLMAN AND MACEK 1980
1185	LAS 11.2	FM	MORT2	2500.00	80	41			HOLMAN AND MACEK 1980
1186	LAS 11.2	FM	MORT2	3000.00	80	42			HOLMAN AND MACEK 1980
1187	LAS 11.2	FM	MORT2	4400.00	80	32			HOLMAN AND MACEK 1980
1188	LAS 11.2	FM	MORT2	5100.00	80	50			HOLMAN AND MACEK 1980

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1189	LAS 11.2	FM	MORT2	8400.00	80	29			HOLMAN AND MACEK 1980
1190	LAS 11.2	FM	MORT2	9800.00	80	58			HOLMAN AND MACEK 1980
1191	LAS 11.2	FM	MORT2	14200.00	80	80			HOLMAN AND MACEK 1980
1192	LAS 11.7	FM	HATCH	0.00	150	17			HOLMAN AND MACEK 1980
1193	LAS 11.7	FM	HATCH	200.00	150	9			HOLMAN AND MACEK 1980
1194	LAS 11.7	FM	HATCH	220.00	150	5			HOLMAN AND MACEK 1980
1195	LAS 11.7	FM	HATCH	310.00	150	11			HOLMAN AND MACEK 1980
1196	LAS 11.7	FM	HATCH	480.00	150	6			HOLMAN AND MACEK 1980
1197	LAS 11.7	FM	HATCH	490.00	150	5			HOLMAN AND MACEK 1980
1198	LAS 11.7	FM	HATCH	570.00	150	6			HOLMAN AND MACEK 1980
1199	LAS 11.7	FM	HATCH	740.00	150	5			HOLMAN AND MACEK 1980
1200	LAS 11.7	FM	MORT1	0.00	30	1			HOLMAN AND MACEK 1980
1201	LAS 11.7	FM	MORT1	60.00	30	6			HOLMAN AND MACEK 1980
1202	LAS 11.7	FM	MORT1	120.00	30	10			HOLMAN AND MACEK 1980
1203	LAS 11.7	FM	MORT1	250.00	30	10			HOLMAN AND MACEK 1980
1204	LAS 11.7	FM	MORT1	530.00	30	16			HOLMAN AND MACEK 1980
1205	LAS 11.7	FM	MORT1	1090.00	30	5			HOLMAN AND MACEK 1980
1206	LAS 11.7	FM	MORT2	0.00	80	1			HOLMAN AND MACEK 1980
1207	LAS 11.7	FM	MORT2	200.00	80	6			HOLMAN AND MACEK 1980
1208	LAS 11.7	FM	MORT2	220.00	80	0			HOLMAN AND MACEK 1980
1209	LAS 11.7	FM	MORT2	310.00	80	9			HOLMAN AND MACEK 1980
1210	LAS 11.7	FM	MORT2	480.00	80	16			HOLMAN AND MACEK 1980
1211	LAS 11.7	FM	MORT2	490.00	80	44			HOLMAN AND MACEK 1980
1212	LAS 11.7	FM	MORT2	570.00	80	22			HOLMAN AND MACEK 1980
1213	LAS 11.7	FM	MORT2	740.00	80	42			HOLMAN AND MACEK 1980
1214	LAS 13.3	FM	EGGS	0.00			530		HOLMAN AND MACEK 1980
1215	LAS 13.3	FM	EGGS	20.00			221		HOLMAN AND MACEK 1980
1216	LAS 13.3	FM	EGGS	33.00			72		HOLMAN AND MACEK 1980
1217	LAS 13.3	FM	EGGS	56.00			346		HOLMAN AND MACEK 1980
1218	LAS 13.3	FM	EGGS	106.00			135		HOLMAN AND MACEK 1980
1219	LAS 13.3	FM	EGGS	252.00			7		HOLMAN AND MACEK 1980
1220	LAS 13.3	FM	MORT1	0.00	30	4			HOLMAN AND MACEK 1980
1221	LAS 13.3	FM	MORT1	20.00	30	11			HOLMAN AND MACEK 1980
1222	LAS 13.3	FM	MORT1	33.00	30	9			HOLMAN AND MACEK 1980
1223	LAS 13.3	FM	MORT1	56.00	30	9			HOLMAN AND MACEK 1980
1224	LAS 13.3	FM	MORT1	106.00	30	7			HOLMAN AND MACEK 1980
1225	LAS 13.3	FM	MORT1	252.00	30	9			HOLMAN AND MACEK 1980
1226	MALATHION	FF	MORT2	0.00	80	16			HERMANUTZ 1978
1227	MALATHION	FF	MORT2	5.80	80	8			HERMANUTZ 1978
1228	MALATHION	FF	MORT2	8.60	80	9			HERMANUTZ 1978
1229	MALATHION	FF	MORT2	10.90	80	16			HERMANUTZ 1978
1230	MALATHION	FF	MORT2	15.00	80	39			HERMANUTZ 1978
1231	MALATHION	FF	MORT2	19.30	80	9			HERMANUTZ 1978
1232	MALATHION	FF	MORT2	24.70	80	15			HERMANUTZ 1978
1233	MALATHION	FF	MORT2	31.50	80	47			HERMANUTZ 1978
1234	MALATHION	FF	MORT4	0.00	40	0			HERMANUTZ 1978
1235	MALATHION	FF	MORT4	5.80	40	0			HERMANUTZ 1978
1236	MALATHION	FF	MORT4	8.60	40	1			HERMANUTZ 1978
1237	MALATHION	FF	MORT4	10.90	40	2			HERMANUTZ 1978
1238	MALATHION	FF	MORT4	15.00	40	4			HERMANUTZ 1978
1239	MALATHION	FF	MORT4	19.30	40	5			HERMANUTZ 1978
1240	MALATHION	FF	MORT4	24.70	40	17			HERMANUTZ 1978
1241	MALATHION	FF	MORT4	31.50	40	14			HERMANUTZ 1978
1242	METHYLMERCURIC CHLOR	BT	EGGS	0.00			506		MCKIM ET AL 1976

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1243	METHYLMERCURIC CHLOR	BT	EGGS	0.03			299		MCKIM ET AL 1976
1244	METHYLMERCURIC CHLOR	BT	EGGS	0.09			430		MCKIM ET AL 1976
1245	METHYLMERCURIC CHLOR	BT	EGGS	0.29			191		MCKIM ET AL 1976
1246	METHYLMERCURIC CHLOR	BT	EGGS	0.93			368		MCKIM ET AL 1976
1247	METHYLMERCURIC CHLOR	BT	EGGS	2.93			0		MCKIM ET AL 1976
1248	METHYLMERCURIC CHLOR	BT	HATCH	0.00	200	6			MCKIM ET AL 1976
1249	METHYLMERCURIC CHLOR	BT	HATCH	0.03	200	26			MCKIM ET AL 1976
1250	METHYLMERCURIC CHLOR	BT	HATCH	0.09	200	1			MCKIM ET AL 1976
1251	METHYLMERCURIC CHLOR	BT	HATCH	0.29	100	2			MCKIM ET AL 1976
1252	METHYLMERCURIC CHLOR	BT	HATCH	0.93	200	116			MCKIM ET AL 1976
1253	METHYLMERCURIC CHLOR	BT	MORT1	0.00	12	1			MCKIM ET AL 1976
1254	METHYLMERCURIC CHLOR	BT	MORT1	0.03	12	2			MCKIM ET AL 1976
1255	METHYLMERCURIC CHLOR	BT	MORT1	0.09	12	2			MCKIM ET AL 1976
1256	METHYLMERCURIC CHLOR	BT	MORT1	0.29	6	1			MCKIM ET AL 1976
1257	METHYLMERCURIC CHLOR	BT	MORT1	0.91	6	5			MCKIM ET AL 1976
1258	METHYLMERCURIC CHLOR	BT	MORT2	0.00	100	4			MCKIM ET AL 1976
1259	METHYLMERCURIC CHLOR	BT	MORT2	0.03	100	6			MCKIM ET AL 1976
1260	METHYLMERCURIC CHLOR	BT	MORT2	0.09	100	3			MCKIM ET AL 1976
1261	METHYLMERCURIC CHLOR	BT	MORT2	0.29	100	1			MCKIM ET AL 1976
1262	METHYLMERCURIC CHLOR	BT	MORT2	0.93	100	55			MCKIM ET AL 1976
1263	MIREX	FM	EGGS	0.00			395		BUCKLER ET AL 1981
1264	MIREX	FM	EGGS	2.00			283		BUCKLER ET AL 1981
1265	MIREX	FM	EGGS	3.00			104		BUCKLER ET AL 1981
1266	MIREX	FM	EGGS	7.00			272		BUCKLER ET AL 1981
1267	MIREX	FM	EGGS	13.00			128		BUCKLER ET AL 1981
1268	MIREX	FM	EGGS	34.00			84		BUCKLER ET AL 1981
1269	MIREX	FM	HATCH	0.00	2900	1015			BUCKLER ET AL 1981
1270	MIREX	FM	HATCH	2.00	2400	360			BUCKLER ET AL 1981
1271	MIREX	FM	HATCH	3.00	900	117			BUCKLER ET AL 1981
1272	MIREX	FM	HATCH	7.00	2300	368			BUCKLER ET AL 1981
1273	MIREX	FM	HATCH	13.00	1050	284			BUCKLER ET AL 1981
1274	MIREX	FM	HATCH	34.00	1000	370			BUCKLER ET AL 1981
1275	MIREX	FM	MORT1	0.00	70	4			BUCKLER ET AL 1981
1276	MIREX	FM	MORT1	2.00	72	11			BUCKLER ET AL 1981
1277	MIREX	FM	MORT1	3.00	69	7			BUCKLER ET AL 1981
1278	MIREX	FM	MORT1	7.00	72	20			BUCKLER ET AL 1981
1279	MIREX	FM	MORT1	13.00	63	13			BUCKLER ET AL 1981
1280	MIREX	FM	MORT1	34.00	67	18			BUCKLER ET AL 1981
1281	MIREX	FM	MORT2	0.00	80	9			BUCKLER ET AL 1981
1282	MIREX	FM	MORT2	2.00	80	9			BUCKLER ET AL 1981
1283	MIREX	FM	MORT2	3.00	80	18			BUCKLER ET AL 1981
1284	MIREX	FM	MORT2	7.00	80	11			BUCKLER ET AL 1981
1285	MIREX	FM	MORT2	13.00	80	29			BUCKLER ET AL 1981
1286	MIREX	FM	MORT2	34.00	80	18			BUCKLER ET AL 1981
1287	NAPTHALENE	FM	HATCH	0.00	500	48			DEGRAEVE ET AL 1982
1288	NAPTHALENE	FM	HATCH	130.00	500	78			DEGRAEVE ET AL 1982
1289	NAPTHALENE	FM	HATCH	210.00	500	55			DEGRAEVE ET AL 1982
1290	NAPTHALENE	FM	HATCH	450.00	500	68			DEGRAEVE ET AL 1982
1291	NAPTHALENE	FM	HATCH	850.00	500	114			DEGRAEVE ET AL 1982
1292	NAPTHALENE	FM	HATCH	1840.00	500	57			DEGRAEVE ET AL 1982
1293	NAPTHALENE	FM	HATCH	4380.00	500	171			DEGRAEVE ET AL 1982
1294	NAPTHALENE	FM	HATCH	8510.00	500	317			DEGRAEVE ET AL 1982
1295	NI	FM	EGGS	0.00			1603		PICKERING 1974
1296	NI	FM	EGGS	82.00			1104		PICKERING 1974

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1297	NI	FM	EGGS	180.00			1320		PICKERING 1974
1298	NI	FM	EGGS	380.00			1398		PICKERING 1974
1299	NI	FM	EGGS	730.00			498		PICKERING 1974
1300	NI	FM	EGGS	1600.00			36		PICKERING 1974
1301	NI	FM	HATCH	0.00	1000	72			PICKERING 1974
1302	NI	FM	HATCH	82.00	1100	45			PICKERING 1974
1303	NI	FM	HATCH	180.00	1200	50			PICKERING 1974
1304	NI	FM	HATCH	380.00	1300	75			PICKERING 1974
1305	NI	FM	HATCH	730.00	2300	1325			PICKERING 1974
1306	NI	FM	MORT2	0.00	50	7			PICKERING 1974
1307	NI	FM	MORT2	82.00	50	4			PICKERING 1974
1308	NI	FM	MORT2	180.00	50	3			PICKERING 1974
1309	NI	FM	MORT2	380.00	50	4			PICKERING 1974
1310	NI	FM	MORT2	730.00	50	3			PICKERING 1974
1311	PB	BT	EGGS	0.85			479		HOLCOMBE ET AL 1976
1312	PB	BT	EGGS	33.40			497		HOLCOMBE ET AL 1976
1313	PB	BT	EGGS	57.60			233		HOLCOMBE ET AL 1976
1314	PB	BT	EGGS	119.20			480		HOLCOMBE ET AL 1976
1315	PB	BT	EGGS	235.20			555		HOLCOMBE ET AL 1976
1316	PB	BT	EGGS	475.40			183		HOLCOMBE ET AL 1976
1317	PB	BT	HATCH	0.90	724	13			HOLCOMBE ET AL 1976
1318	PB	BT	HATCH	34.00	710	140			HOLCOMBE ET AL 1976
1319	PB	BT	HATCH	58.00	250	52			HOLCOMBE ET AL 1976
1320	PB	BT	HATCH	119.00	687	99			HOLCOMBE ET AL 1976
1321	PB	BT	HATCH	235.00	792	264			HOLCOMBE ET AL 1976
1322	PB	BT	HATCH	474.00	262	189			HOLCOMBE ET AL 1976
1323	PB	BT	MORT1	0.85	10	3			HOLCOMBE ET AL 1976
1324	PB	BT	MORT1	33.45	10	0			HOLCOMBE ET AL 1976
1325	PB	BT	MORT1	57.90	5	0			HOLCOMBE ET AL 1976
1326	PB	BT	MORT1	119.20	10	3			HOLCOMBE ET AL 1976
1327	PB	BT	MORT1	235.00	10	2			HOLCOMBE ET AL 1976
1328	PB	BT	MORT1	472.60	10	2			HOLCOMBE ET AL 1976
1329	PB	BT	MORT2	0.90	200	31			HOLCOMBE ET AL 1976
1330	PB	BT	MORT2	34.00	200	23			HOLCOMBE ET AL 1976
1331	PB	BT	MORT2	58.00	150	9			HOLCOMBE ET AL 1976
1332	PB	BT	MORT2	119.00	150	3			HOLCOMBE ET AL 1976
1333	PB	BT	MORT2	235.00	100	6			HOLCOMBE ET AL 1976
1334	PB	BT	MORT2	474.00	50	40			HOLCOMBE ET AL 1976
1335	PB	BG	WEIGHT	0.00				0.38	SAUTER ET AL 1976
1336	PB	BG	WEIGHT	12.00				0.42	SAUTER ET AL 1976
1337	PB	BG	WEIGHT	33.00				0.41	SAUTER ET AL 1976
1338	PB	BG	WEIGHT	70.00				0.49	SAUTER ET AL 1976
1339	PB	BG	WEIGHT	120.00				0.25	SAUTER ET AL 1976
1340	PB	BG	WEIGHT	277.00				0.00	SAUTER ET AL 1976
1341	PB	BG	WEIGHT	447.00				0.00	SAUTER ET AL 1976
1342	PB	CC	WEIGHT	0.00				0.24	SAUTER ET AL 1976
1343	PB	CC	WEIGHT	17.00				0.23	SAUTER ET AL 1976
1344	PB	CC	WEIGHT	33.00				0.24	SAUTER ET AL 1976
1345	PB	CC	WEIGHT	75.00				0.23	SAUTER ET AL 1976
1346	PB	CC	WEIGHT	136.00				0.15	SAUTER ET AL 1976
1347	PB	CC	WEIGHT	280.00				0.00	SAUTER ET AL 1976
1348	PB	CC	WEIGHT	460.00				0.00	SAUTER ET AL 1976
1349	PB	LT	WEIGHT	0.00				0.18	SAUTER ET AL 1976
1350	PB	LT	WEIGHT	48.00				0.19	SAUTER ET AL 1976

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1351	PB	LT	WEIGHT	83.00				0.16	SAUTER ET AL 1976
1352	PB	LT	WEIGHT	120.00				0.15	SAUTER ET AL 1976
1353	PB	LT	WEIGHT	198.00				0.13	SAUTER ET AL 1976
1354	PB	LT	WEIGHT	404.00				0.00	SAUTER ET AL 1976
1355	PB	LT	WEIGHT	483.00				0.00	SAUTER ET AL 1976
1356	PB	RT	HATCH	0.00	400	62			SAUTER ET AL 1976
1357	PB	RT	HATCH	49.00	400	26			SAUTER ET AL 1976
1358	PB	RT	HATCH	71.00	400	46			SAUTER ET AL 1976
1359	PB	RT	HATCH	146.00	400	34			SAUTER ET AL 1976
1360	PB	RT	HATCH	250.00	400	50			SAUTER ET AL 1976
1361	PB	RT	HATCH	443.00	400	34			SAUTER ET AL 1976
1362	PB	RT	HATCH	672.00	400	286			SAUTER ET AL 1976
1363	PB	RT	MORT2	0.00	200	20			SAUTER ET AL 1976
1364	PB	RT	MORT2	49.00	200	24			SAUTER ET AL 1976
1365	PB	RT	MORT2	71.00	200	24			SAUTER ET AL 1976
1366	PB	RT	MORT2	146.00	200	109			SAUTER ET AL 1976
1367	PB	RT	MORT2	250.00	200	199			SAUTER ET AL 1976
1368	PB	RT	MORT2	443.00	200	200			SAUTER ET AL 1976
1369	PB	RT	MORT2	677.00	200	200			SAUTER ET AL 1976
1370	PB	RT	WEIGHT	0.00				0.71	SAUTER ET AL 1976
1371	PB	RT	WEIGHT	49.00				0.67	SAUTER ET AL 1976
1372	PB	RT	WEIGHT	71.00				0.73	SAUTER ET AL 1976
1373	PB	RT	WEIGHT	146.00				0.70	SAUTER ET AL 1976
1374	PB	RT	WEIGHT	250.00				0.70	SAUTER ET AL 1976
1375	PB	RT	WEIGHT	443.00				0.00	SAUTER ET AL 1976
1376	PB	RT	WEIGHT	672.00				0.00	SAUTER ET AL 1976
1377	PB	WS	WEIGHT	0.00				0.19	SAUTER ET AL 1976
1378	PB	WS	WEIGHT	33.00				0.26	SAUTER ET AL 1976
1379	PB	WS	WEIGHT	67.00				0.19	SAUTER ET AL 1976
1380	PB	WS	WEIGHT	119.00				0.18	SAUTER ET AL 1976
1381	PB	WS	WEIGHT	253.00				0.07	SAUTER ET AL 1976
1382	PB	WS	WEIGHT	483.00				0.00	SAUTER ET AL 1976
1383	PENTACHLOROETHANE	FM	MORT2	10.00	120	18			AHMED ET AL 1984
1384	PENTACHLOROETHANE	FM	MORT2	900.00	120	21			AHMED ET AL 1984
1385	PENTACHLOROETHANE	FM	MORT2	1400.00	120	27			AHMED ET AL 1984
1386	PENTACHLOROETHANE	FM	MORT2	2900.00	120	9			AHMED ET AL 1984
1387	PENTACHLOROETHANE	FM	MORT2	4100.00	120	66			AHMED ET AL 1984
1388	PENTACHLOROETHANE	FM	MORT2	13900.00	120	120			AHMED ET AL 1984
1389	PENTACHLOROETHANE	FM	WEIGHT	10.00				0.22	AHMED ET AL 1984
1390	PENTACHLOROETHANE	FM	WEIGHT	900.00				0.23	AHMED ET AL 1984
1391	PENTACHLOROETHANE	FM	WEIGHT	1400.00				0.15	AHMED ET AL 1984
1392	PENTACHLOROETHANE	FM	WEIGHT	2900.00				0.09	AHMED ET AL 1984
1393	PENTACHLOROETHANE	FM	WEIGHT	4100.00				0.05	AHMED ET AL 1984
1394	PENTACHLOROETHANE	FM	WEIGHT	13900.00				0.00	AHMED ET AL 1984
1395	PENTACHLOROPHENOL	FM	HATCH	0.00	200	73			HOLCOMBE ET AL 1982
1396	PENTACHLOROPHENOL	FM	HATCH	27.20	200	73			HOLCOMBE ET AL 1982
1397	PENTACHLOROPHENOL	FM	HATCH	44.90	200	65			HOLCOMBE ET AL 1982
1398	PENTACHLOROPHENOL	FM	HATCH	73.00	200	81			HOLCOMBE ET AL 1982
1399	PENTACHLOROPHENOL	FM	HATCH	128.00	200	74			HOLCOMBE ET AL 1982
1400	PENTACHLOROPHENOL	FM	HATCH	223.00	200	200			HOLCOMBE ET AL 1982
1401	PENTACHLOROPHENOL	FM	MORT2	0.00	100	6			HOLCOMBE ET AL 1982
1402	PENTACHLOROPHENOL	FM	MORT2	27.20	100	8			HOLCOMBE ET AL 1982
1403	PENTACHLOROPHENOL	FM	MORT2	44.90	100	8			HOLCOMBE ET AL 1982
1404	PENTACHLOROPHENOL	FM	MORT2	73.00	100	13			HOLCOMBE ET AL 1982

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1405	PENTACHLOROPHENOL	FM	MORT2	128.00	100	79			HOLCOMBE ET AL 1982
1406	PENTACHLOROPHENOL	FM	MORT2	223.00	100	100			HOLCOMBE ET AL 1982
1407	PENTACHLOROPHENOL	FM	WEIGHT	0.00	100			0.13	HOLCOMBE ET AL 1982
1408	PENTACHLOROPHENOL	FM	WEIGHT	27.20	100			0.14	HOLCOMBE ET AL 1982
1409	PENTACHLOROPHENOL	FM	WEIGHT	44.90	100			0.13	HOLCOMBE ET AL 1982
1410	PENTACHLOROPHENOL	FM	WEIGHT	73.00	100			0.11	HOLCOMBE ET AL 1982
1411	PENTACHLOROPHENOL	FM	WEIGHT	128.00	100			0.11	HOLCOMBE ET AL 1982
1412	PENTACHLOROPHENOL	FM	WEIGHT	223.00	100			0.00	HOLCOMBE ET AL 1982
1413	PERMETHRIN	FM	HATCH	0.00	100	10			SPEHAR ET AL 1983
1414	PERMETHRIN	FM	HATCH	0.11	100	3			SPEHAR ET AL 1983
1415	PERMETHRIN	FM	HATCH	0.18	100	8			SPEHAR ET AL 1983
1416	PERMETHRIN	FM	HATCH	0.33	100	10			SPEHAR ET AL 1983
1417	PERMETHRIN	FM	HATCH	0.66	100	14			SPEHAR ET AL 1983
1418	PERMETHRIN	FM	HATCH	1.40	100	10			SPEHAR ET AL 1983
1419	PERMETHRIN	FM	MORT2	0.00	60	5			SPEHAR ET AL 1983
1420	PERMETHRIN	FM	MORT2	0.11	60	2			SPEHAR ET AL 1983
1421	PERMETHRIN	FM	MORT2	0.18	60	2			SPEHAR ET AL 1983
1422	PERMETHRIN	FM	MORT2	0.33	60	2			SPEHAR ET AL 1983
1423	PERMETHRIN	FM	MORT2	0.66	60	4			SPEHAR ET AL 1983
1424	PERMETHRIN	FM	MORT2	1.40	60	59			SPEHAR ET AL 1983
1425	PERMETHRIN	FM	WEIGHT	0.00				0.10	SPEHAR ET AL 1983
1426	PERMETHRIN	FM	WEIGHT	0.11				0.09	SPEHAR ET AL 1983
1427	PERMETHRIN	FM	WEIGHT	0.18				0.10	SPEHAR ET AL 1983
1428	PERMETHRIN	FM	WEIGHT	0.33				0.09	SPEHAR ET AL 1983
1429	PERMETHRIN	FM	WEIGHT	0.66				0.09	SPEHAR ET AL 1983
1430	PERMETHRIN	FM	WEIGHT	1.40				0.11	SPEHAR ET AL 1983
1431	PHENOL	FM	HATCH	0.00	500	91			DEGRAEVE ET AL 1980
1432	PHENOL	FM	HATCH	230.00	500	87			DEGRAEVE ET AL 1980
1433	PHENOL	FM	HATCH	750.00	500	93			DEGRAEVE ET AL 1980
1434	PHENOL	FM	HATCH	2500.00	500	109			DEGRAEVE ET AL 1980
1435	PHENOL	FM	HATCH	6100.00	500	114			DEGRAEVE ET AL 1980
1436	PHENOL	FM	HATCH	14500.00	500	139			DEGRAEVE ET AL 1980
1437	PHENOL	FM	HATCH	33200.00	500	111			DEGRAEVE ET AL 1980
1438	PHENOL	FM	HATCH	68500.00	500	274			DEGRAEVE ET AL 1980
1439	PHENOL	FM	MORT2	0.00	30	14			DEGRAEVE ET AL 1980
1440	PHENOL	FM	MORT2	230.00	30	21			DEGRAEVE ET AL 1980
1441	PHENOL	FM	MORT2	750.00	30	17			DEGRAEVE ET AL 1980
1442	PHENOL	FM	MORT2	2500.00	30	15			DEGRAEVE ET AL 1980
1443	PHENOL	FM	MORT2	6100.00	30	16			DEGRAEVE ET AL 1980
1444	PHENOL	FM	MORT2	14500.00	30	22			DEGRAEVE ET AL 1980
1445	PHENOL	FM	MORT2	33200.00	30	30			DEGRAEVE ET AL 1980
1446	PHENOL	FM	MORT2	68500.00	30	30			DEGRAEVE ET AL 1980
1447	PHENOL	FM	WEIGHT	0.00				0.27	DEGRAEVE ET AL 1980
1448	PHENOL	FM	WEIGHT	230.00				0.18	DEGRAEVE ET AL 1980
1449	PHENOL	FM	WEIGHT	750.00				0.25	DEGRAEVE ET AL 1980
1450	PHENOL	FM	WEIGHT	2500.00				0.19	DEGRAEVE ET AL 1980
1451	PHENOL	FM	WEIGHT	6100.00				0.15	DEGRAEVE ET AL 1980
1452	PHENOL	FM	WEIGHT	14500.00				0.18	DEGRAEVE ET AL 1980
1453	PHENOL	FM	WEIGHT	33200.00					DEGRAEVE ET AL 1980
1454	PHENOL	FM	WEIGHT	68500.00					DEGRAEVE ET AL 1980
1455	PHENOL	RT	MORT2	0.00	200	19			DEGRAEVE ET AL 1980
1456	PHENOL	RT	MORT2	340.00	200	23			DEGRAEVE ET AL 1980
1457	PHENOL	RT	MORT2	540.00	200	14			DEGRAEVE ET AL 1980
1458	PHENOL	RT	MORT2	1100.00	200	69			DEGRAEVE ET AL 1980

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1459	PHENOL	RT	MORT2	2800.00	200	134			DEGRAEVE ET AL 1980
1460	PHENOL	RT	MORT2	5900.00	200	94			DEGRAEVE ET AL 1980
1461	PHENOL	RT	MORT2	13800.00	200	200			DEGRAEVE ET AL 1980
1462	PHENOL	RT	WEIGHT	0.00				1.57	DEGRAEVE ET AL 1980
1463	PHENOL	RT	WEIGHT	340.00				1.31	DEGRAEVE ET AL 1980
1464	PHENOL	RT	WEIGHT	540.00				1.18	DEGRAEVE ET AL 1980
1465	PHENOL	RT	WEIGHT	1100.00				0.96	DEGRAEVE ET AL 1980
1466	PHENOL	RT	WEIGHT	2800.00				0.91	DEGRAEVE ET AL 1980
1467	PHENOL	RT	WEIGHT	5900.00				0.46	DEGRAEVE ET AL 1980
1468	PHENOL	RT	WEIGHT	13800.00					DEGRAEVE ET AL 1980
1469	PHENOL	FM	HATCH	0.00	200	23			HOLCOMBE ET AL 1982
1470	PHENOL	FM	HATCH	240.00	200	17			HOLCOMBE ET AL 1982
1471	PHENOL	FM	HATCH	450.00	200	15			HOLCOMBE ET AL 1982
1472	PHENOL	FM	HATCH	910.00	200	23			HOLCOMBE ET AL 1982
1473	PHENOL	FM	HATCH	1830.00	200	19			HOLCOMBE ET AL 1982
1474	PHENOL	FM	HATCH	3570.00	200	14			HOLCOMBE ET AL 1982
1475	PHENOL	FM	MORT2	0.00	100	21			HOLCOMBE ET AL 1982
1476	PHENOL	FM	MORT2	240.00	100	25			HOLCOMBE ET AL 1982
1477	PHENOL	FM	MORT2	450.00	100	26			HOLCOMBE ET AL 1982
1478	PHENOL	FM	MORT2	910.00	100	27			HOLCOMBE ET AL 1982
1479	PHENOL	FM	MORT2	1830.00	100	26			HOLCOMBE ET AL 1982
1480	PHENOL	FM	MORT2	3570.00	100	13			HOLCOMBE ET AL 1982
1481	PHENOL	FM	WEIGHT	0.00	100			0.10	HOLCOMBE ET AL 1982
1482	PHENOL	FM	WEIGHT	240.00	100			0.10	HOLCOMBE ET AL 1982
1483	PHENOL	FM	WEIGHT	450.00	100			0.10	HOLCOMBE ET AL 1982
1484	PHENOL	FM	WEIGHT	910.00	100			0.10	HOLCOMBE ET AL 1982
1485	PHENOL	FM	WEIGHT	1830.00	100			0.10	HOLCOMBE ET AL 1982
1486	PHENOL	FM	WEIGHT	3570.00	100			0.08	HOLCOMBE ET AL 1982
1487	PHENOLS	FM	EGGS	0.00			270		DAUBLE ET AL 1983
1488	PHENOLS	FM	EGGS	60.00			182		DAUBLE ET AL 1983
1489	PHENOLS	FM	EGGS	130.00			91		DAUBLE ET AL 1983
1490	PHENOLS	FM	EGGS	250.00			202		DAUBLE ET AL 1983
1491	PHENOLS	FM	EGGS	560.00			50		DAUBLE ET AL 1983
1492	PHENOLS	FM	EGGS	1210.00			0		DAUBLE ET AL 1983
1493	PHENOLS	FM	WEIGHT	0.00				20.40	DAUBLE ET AL 1983
1494	PHENOLS	FM	WEIGHT	60.00				16.80	DAUBLE ET AL 1983
1495	PHENOLS	FM	WEIGHT	130.00				23.10	DAUBLE ET AL 1983
1496	PHENOLS	FM	WEIGHT	250.00				11.50	DAUBLE ET AL 1983
1497	PHENOLS	FM	WEIGHT	560.00				13.60	DAUBLE ET AL 1983
1498	PHENOLS	FM	WEIGHT	1210.00				6.80	DAUBLE ET AL 1983
1499	PICLORAM	LT	WEIGHT	0.00				373.00	WOODWARD 1976
1500	PICLORAM	LT	WEIGHT	35.00				233.00	WOODWARD 1976
1501	PICLORAM	LT	WEIGHT	75.00				154.00	WOODWARD 1976
1502	PICLORAM	LT	WEIGHT	240.00				117.00	WOODWARD 1976
1503	PICLORAM	LT	WEIGHT	500.00					WOODWARD 1976
1504	PICLORAM	LT	WEIGHT	1000.00					WOODWARD 1976
1505	PROPANIL	FM	HATCH	0.00	200	53			CALL ET AL 1983
1506	PROPANIL	FM	HATCH	0.40	200	48			CALL ET AL 1983
1507	PROPANIL	FM	HATCH	0.60	200	74			CALL ET AL 1983
1508	PROPANIL	FM	HATCH	1.20	200	85			CALL ET AL 1983
1509	PROPANIL	FM	HATCH	2.40	200	89			CALL ET AL 1983
1510	PROPANIL	FM	HATCH	3.80	200	161			CALL ET AL 1983
1511	PROPANIL	FM	MORT2	0.00	60	4			CALL ET AL 1983
1512	PROPANIL	FM	MORT2	0.40	60	16			CALL ET AL 1983

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1513	PROPANIL	FM	MORT2	0.60	60	30			CALL ET AL 1983
1514	PROPANIL	FM	MORT2	1.20	60	50			CALL ET AL 1983
1515	PROPANIL	FM	MORT2	2.40	60	60			CALL ET AL 1983
1516	PROPANIL	FM	MORT2	3.80	60	60			CALL ET AL 1983
1517	PROPANIL	FM	WEIGHT	0.00				0.59	CALL ET AL 1983
1518	PROPANIL	FM	WEIGHT	0.40				0.56	CALL ET AL 1983
1519	PROPANIL	FM	WEIGHT	0.60				0.49	CALL ET AL 1983
1520	PROPANIL	FM	WEIGHT	1.20				0.45	CALL ET AL 1983
1521	PROPANIL	FM	WEIGHT	2.40					CALL ET AL 1983
1522	PROPANIL	FM	WEIGHT	3.80					CALL ET AL 1983
1523	PYDRIN	FM	MORT2	0.00	30	3			SPEHAR ET AL 1982
1524	PYDRIN	FM	MORT2	0.14	30	8			SPEHAR ET AL 1982
1525	PYDRIN	FM	MORT2	0.17	30	3			SPEHAR ET AL 1982
1526	PYDRIN	FM	MORT2	0.19	30	2			SPEHAR ET AL 1982
1527	PYDRIN	FM	MORT2	0.33	30	7			SPEHAR ET AL 1982
1528	PYDRIN	FM	MORT2	0.43	30	22			SPEHAR ET AL 1982
1529	TETRACHLOROETHYLENE	FM	MORT2	0.00	120	6			AHMED ET AL 1984
1530	TETRACHLOROETHYLENE	FM	MORT2	1400.00	120	20			AHMED ET AL 1984
1531	TETRACHLOROETHYLENE	FM	MORT2	2800.00	120	74			AHMED ET AL 1984
1532	TETRACHLOROETHYLENE	FM	MORT2	4100.00	120	120			AHMED ET AL 1984
1533	TETRACHLOROETHYLENE	FM	MORT2	8600.00	120	120			AHMED ET AL 1984
1534	TETRACHLOROETHYLENE	FM	WEIGHT	0.00				0.26	AHMED ET AL 1984
1535	TETRACHLOROETHYLENE	FM	WEIGHT	500.00				0.25	AHMED ET AL 1984
1536	TETRACHLOROETHYLENE	FM	WEIGHT	1400.00				0.18	AHMED ET AL 1984
1537	TETRACHLOROETHYLENE	FM	WEIGHT	2800.00				0.12	AHMED ET AL 1984
1538	TETRACHLOROETHYLENE	FM	WEIGHT	4100.00				0.00	AHMED ET AL 1984
1539	TETRACHLOROETHYLENE	FM	WEIGHT	8600.00				0.00	AHMED ET AL 1984
1540	TOXAPHENE	BT	EGGS	0.00			855		MAYER ET AL 1975
1541	TOXAPHENE	BT	EGGS	0.04			541		MAYER ET AL 1975
1542	TOXAPHENE	BT	EGGS	0.07			516		MAYER ET AL 1975
1543	TOXAPHENE	BT	EGGS	0.13			542		MAYER ET AL 1975
1544	TOXAPHENE	BT	EGGS	0.27			462		MAYER ET AL 1975
1545	TOXAPHENE	BT	EGGS	0.50			617		MAYER ET AL 1975
1546	TOXAPHENE	BT	MORT1	0.00	24	0			MAYER ET AL 1975
1547	TOXAPHENE	BT	MORT1	0.04	24	2			MAYER ET AL 1975
1548	TOXAPHENE	BT	MORT1	0.07	24	2			MAYER ET AL 1975
1549	TOXAPHENE	BT	MORT1	0.13	24	2			MAYER ET AL 1975
1550	TOXAPHENE	BT	MORT1	0.27	24	12			MAYER ET AL 1975
1551	TOXAPHENE	BT	MORT1	0.50	24	24			MAYER ET AL 1975
1552	TOXAPHENE	BT	MORT2	0.00	200	128			MAYER ET AL 1975
1553	TOXAPHENE	BT	MORT2	0.04	200	166			MAYER ET AL 1975
1554	TOXAPHENE	BT	MORT2	0.07	200	156			MAYER ET AL 1975
1555	TOXAPHENE	BT	MORT2	0.13	200	164			MAYER ET AL 1975
1556	TOXAPHENE	BT	MORT2	0.27	200	200			MAYER ET AL 1975
1557	TOXAPHENE	BT	MORT2	0.50	200	200			MAYER ET AL 1975
1558	TOXAPHENE	BT	WEIGHT	0.00				0.70	MAYER ET AL 1975
1559	TOXAPHENE	BT	WEIGHT	0.04				0.37	MAYER ET AL 1975
1560	TOXAPHENE	BT	WEIGHT	0.07				0.51	MAYER ET AL 1975
1561	TOXAPHENE	BT	WEIGHT	0.13				0.40	MAYER ET AL 1975
1562	TOXAPHENE	BT	WEIGHT	0.27				0.00	MAYER ET AL 1975
1563	TOXAPHENE	BT	WEIGHT	0.50				0.00	MAYER ET AL 1975
1564	TOXAPHENE	CC	HATCH	0.00	1800	126			MAYER ET AL 1977
1565	TOXAPHENE	CC	HATCH	0.05	1500	75			MAYER ET AL 1977
1566	TOXAPHENE	CC	HATCH	0.07	1200	84			MAYER ET AL 1977

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1567	TOXAPHENE	CC	HATCH	0.13	1800	180			MAYER ET AL 1977
1568	TOXAPHENE	CC	HATCH	0.30	1200	108			MAYER ET AL 1977
1569	TOXAPHENE	CC	HATCH	0.63	1200	300			MAYER ET AL 1977
1570	TOXAPHENE	CC	MORT1	0.00	8	0			MAYER ET AL 1977
1571	TOXAPHENE	CC	MORT1	0.05	8	1			MAYER ET AL 1977
1572	TOXAPHENE	CC	MORT1	0.07	8	1			MAYER ET AL 1977
1573	TOXAPHENE	CC	MORT1	0.13	8	1			MAYER ET AL 1977
1574	TOXAPHENE	CC	MORT1	0.30	8	0			MAYER ET AL 1977
1575	TOXAPHENE	CC	MORT1	0.63	8	2			MAYER ET AL 1977
1576	TOXAPHENE	CC	WEIGHT	0.00				0.13	MAYER ET AL 1977
1577	TOXAPHENE	CC	WEIGHT	0.05				0.11	MAYER ET AL 1977
1578	TOXAPHENE	CC	WEIGHT	0.07				0.13	MAYER ET AL 1977
1579	TOXAPHENE	CC	WEIGHT	0.13				0.11	MAYER ET AL 1977
1580	TOXAPHENE	CC	WEIGHT	0.30				0.09	MAYER ET AL 1977
1581	TOXAPHENE	CC	WEIGHT	0.63				0.10	MAYER ET AL 1977
1582	TOXAPHENE	FM	EGGS	0.00			256		MAYER ET AL 1977
1583	TOXAPHENE	FM	EGGS	0.01			125		MAYER ET AL 1977
1584	TOXAPHENE	FM	EGGS	0.02			165		MAYER ET AL 1977
1585	TOXAPHENE	FM	EGGS	0.05			604		MAYER ET AL 1977
1586	TOXAPHENE	FM	EGGS	0.10			301		MAYER ET AL 1977
1587	TOXAPHENE	FM	EGGS	0.17			258		MAYER ET AL 1977
1588	TOXAPHENE	FM	HATCH	0.00	50	11			MAYER ET AL 1977
1589	TOXAPHENE	FM	HATCH	0.01	50	5			MAYER ET AL 1977
1590	TOXAPHENE	FM	HATCH	0.02	50	11			MAYER ET AL 1977
1591	TOXAPHENE	FM	HATCH	0.05	50	11			MAYER ET AL 1977
1592	TOXAPHENE	FM	HATCH	0.10	50	6			MAYER ET AL 1977
1593	TOXAPHENE	FM	HATCH	0.17	50	9			MAYER ET AL 1977
1594	TOXAPHENE	FM	MORT1	0.00	20	1			MAYER ET AL 1977
1595	TOXAPHENE	FM	MORT1	0.01	20	3			MAYER ET AL 1977
1596	TOXAPHENE	FM	MORT1	0.02	20	1			MAYER ET AL 1977
1597	TOXAPHENE	FM	MORT1	0.05	20	5			MAYER ET AL 1977
1598	TOXAPHENE	FM	MORT1	0.10	20	2			MAYER ET AL 1977
1599	TOXAPHENE	FM	MORT1	0.17	20	1			MAYER ET AL 1977
1600	TOXAPHENE	FM	WEIGHT	0.00				0.17	MAYER ET AL 1977
1601	TOXAPHENE	FM	WEIGHT	0.01				0.16	MAYER ET AL 1977
1602	TOXAPHENE	FM	WEIGHT	0.02				0.17	MAYER ET AL 1977
1603	TOXAPHENE	FM	WEIGHT	0.05				0.16	MAYER ET AL 1977
1604	TOXAPHENE	FM	WEIGHT	0.10				0.15	MAYER ET AL 1977
1605	TOXAPHENE	FM	WEIGHT	0.17				0.15	MAYER ET AL 1977
1606	TRIFLURALIN	FM	HATCH	0.00	100	9			MACEK ET AL 1976C
1607	TRIFLURALIN	FM	HATCH	1.90	100	15			MACEK ET AL 1976C
1608	TRIFLURALIN	FM	HATCH	5.10	100	19			MACEK ET AL 1976C
1609	TRIFLURALIN	FM	MORT1	0.00	30	5			MACEK ET AL 1976C
1610	TRIFLURALIN	FM	MORT1	1.50	30	8			MACEK ET AL 1976C
1611	TRIFLURALIN	FM	MORT1	1.90	30	8			MACEK ET AL 1976C
1612	TRIFLURALIN	FM	MORT1	5.10	30	21			MACEK ET AL 1976C
1613	TRIFLURALIN	FM	MORT1	8.20	30	30			MACEK ET AL 1976C
1614	TRIFLURALIN	FM	MORT1	16.50	30	30			MACEK ET AL 1976C
1615	TRIFLURALIN	FM	MORT2	0.00	80	13			MACEK ET AL 1976C
1616	TRIFLURALIN	FM	MORT2	1.90	120	53			MACEK ET AL 1976C
1617	TRIFLURALIN	FM	MORT2	5.10	160	46			MACEK ET AL 1976C
1618	VANADIUM	FF	WEIGHT	0.00				0.00	HOLDWAY AND SPRAGUE 1979
1619	VANADIUM	FF	WEIGHT	41.00				0.01	HOLDWAY AND SPRAGUE 1979
1620	VANADIUM	FF	WEIGHT	170.00				0.00	HOLDWAY AND SPRAGUE 1979

Table B.1 (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1621	VANADIUM	FF	WEIGHT	480.00				0.00	HOLDWAY AND SPRAGUE 1979
1622	VANADIUM	FF	WEIGHT	1500.00				0.00	HOLDWAY AND SPRAGUE 1979
1623	ZN	FM	HATCH	2.00	16863	981			BENOIT AND HOLCOMBE 1978
1624	ZN	FM	HATCH	44.00	14341	620			BENOIT AND HOLCOMBE 1978
1625	ZN	FM	HATCH	78.00	12973	921			BENOIT AND HOLCOMBE 1978
1626	ZN	FM	HATCH	145.00	2158	455			BENOIT AND HOLCOMBE 1978
1627	ZN	FM	HATCH	295.00	694	512			BENOIT AND HOLCOMBE 1978
1628	ZN	FM	MORT2	2.00	100	2			BENOIT AND HOLCOMBE 1978
1629	ZN	FM	MORT2	44.00	100	2			BENOIT AND HOLCOMBE 1978
1630	ZN	FM	MORT2	78.00	100	2			BENOIT AND HOLCOMBE 1978
1631	ZN	FM	MORT2	145.00	100	18			BENOIT AND HOLCOMBE 1978
1632	ZN	FM	MORT2	295.00	100	82			BENOIT AND HOLCOMBE 1978
1633	ZN	FM	EGGS	30.00			1532		BRUNGS 1969
1634	ZN	FM	EGGS	180.00			263		BRUNGS 1969
1635	ZN	FM	EGGS	350.00			34		BRUNGS 1969
1636	ZN	FM	EGGS	670.00			9		BRUNGS 1969
1637	ZN	FM	EGGS	1300.00			12		BRUNGS 1969
1638	ZN	FM	EGGS	2800.00			0		BRUNGS 1969
1639	ZN	FM	HATCH	30.00	442	76			BRUNGS 1969
1640	ZN	FM	HATCH	180.00	345	27			BRUNGS 1969
1641	ZN	FM	HATCH	660.00	425	33			BRUNGS 1969
1642	ZN	FM	HATCH	1300.00	408	27			BRUNGS 1969
1643	ZN	FM	HATCH	2800.00	475	0			BRUNGS 1969
1644	ZN	FM	MORT2	30.00	366	42			BRUNGS 1969
1645	ZN	FM	MORT2	180.00	318	31			BRUNGS 1969
1646	ZN	FM	MORT2	660.00	352	28			BRUNGS 1969
1647	ZN	FM	MORT2	1300.00	381	232			BRUNGS 1969
1648	ZN	BT	MORT2	2.60	100	4			HOLCOMBE ET AL 1979
1649	ZN	BT	MORT2	39.00	100	10			HOLCOMBE ET AL 1979
1650	ZN	BT	MORT2	69.00	100	3			HOLCOMBE ET AL 1979
1651	ZN	BT	MORT2	144.00	100	11			HOLCOMBE ET AL 1979
1652	ZN	BT	MORT2	266.00	100	5			HOLCOMBE ET AL 1979
1653	ZN	BT	MORT2	534.00	100	2			HOLCOMBE ET AL 1979
1654	ZN	G	WEIGHT	0.00				0.03	PIERSON 1981
1655	ZN	G	WEIGHT	173.00				0.02	PIERSON 1981
1656	ZN	G	WEIGHT	328.00				0.02	PIERSON 1981
1657	ZN	G	WEIGHT	607.00				0.01	PIERSON 1981
1658	ZN	RT	HATCH	2.00	50	2			SINLEY ET AL 1974
1659	ZN	RT	HATCH	11.00	48	1			SINLEY ET AL 1974
1660	ZN	RT	HATCH	36.00	48	2			SINLEY ET AL 1974
1661	ZN	RT	HATCH	71.00	48	1			SINLEY ET AL 1974
1662	ZN	RT	HATCH	140.00	48	1			SINLEY ET AL 1974
1663	ZN	RT	HATCH	260.00	48	2			SINLEY ET AL 1974
1664	ZN	RT	HATCH	547.00	48	2			SINLEY ET AL 1974
1665	ZN	RT	MORT2	2.00	48	6			SINLEY ET AL 1974
1666	ZN	RT	MORT2	11.00	47	4			SINLEY ET AL 1974
1667	ZN	RT	MORT2	36.00	46	6			SINLEY ET AL 1974
1668	ZN	RT	MORT2	71.00	46	5			SINLEY ET AL 1974
1669	ZN	RT	MORT2	140.00	46	5			SINLEY ET AL 1974
1670	ZN	RT	MORT2	260.00	46	9			SINLEY ET AL 1974
1671	ZN	RT	MORT2	547.00	46	25			SINLEY ET AL 1974
1672	ZN	FF	EGGS	10.00			484		SPEHAR 1976
1673	ZN	FF	EGGS	28.00			280		SPEHAR 1976
1674	ZN	FF	EGGS	47.00			422		SPEHAR 1976

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1675	ZN	FF	EGGS	75.00			296		SPEHAR 1976
1676	ZN	FF	EGGS	139.00			36		SPEHAR 1976
1677	ZN	FF	HATCH	10.00	40	12			SPEHAR 1976
1678	ZN	FF	HATCH	28.00	40	10			SPEHAR 1976
1679	ZN	FF	HATCH	47.00	40	11			SPEHAR 1976
1680	ZN	FF	HATCH	75.00	40	16			SPEHAR 1976
1681	ZN	FF	HATCH	137.00	40	11			SPEHAR 1976
1682	ZN	FF	MORT1	10.00	60	6			SPEHAR 1976
1683	ZN	FF	MORT1	28.00	60	8			SPEHAR 1976
1684	ZN	FF	MORT1	47.00	60	3			SPEHAR 1976
1685	ZN	FF	MORT1	75.00	60	1			SPEHAR 1976
1686	ZN	FF	MORT1	139.00	60	15			SPEHAR 1976
1687	ZN	FF	MORT1	267.00	60	57			SPEHAR 1976
1688	1,1,2-TRICHLOROETHAN	FM	MORT2	50.00	120	0			AHMED ET AL 1984
1689	1,1,2-TRICHLOROETHAN	FM	MORT2	2000.00	120	0			AHMED ET AL 1984
1690	1,1,2-TRICHLOROETHAN	FM	MORT2	6000.00	120	6			AHMED ET AL 1984
1691	1,1,2-TRICHLOROETHAN	FM	MORT2	14800.00	120	0			AHMED ET AL 1984
1692	1,1,2-TRICHLOROETHAN	FM	MORT2	48000.00	120	27			AHMED ET AL 1984
1693	1,1,2-TRICHLOROETHAN	FM	MORT2	147000.00	120	120			AHMED ET AL 1984
1694	1,1,2-TRICHLOROETHAN	FM	WEIGHT	50.00				0.14	AHMED ET AL 1984
1695	1,1,2-TRICHLOROETHAN	FM	WEIGHT	2000.00				0.15	AHMED ET AL 1984
1696	1,1,2-TRICHLOROETHAN	FM	WEIGHT	6000.00				0.14	AHMED ET AL 1984
1697	1,1,2-TRICHLOROETHAN	FM	WEIGHT	14800.00				0.12	AHMED ET AL 1984
1698	1,1,2-TRICHLOROETHAN	FM	WEIGHT	48000.00				0.04	AHMED ET AL 1984
1699	1,1,2-TRICHLOROETHAN	FM	WEIGHT	147000.00				0.00	AHMED ET AL 1984
1700	1,1,2,2-TETRACHLOROE	FM	MORT2	12.00	120	6			AHMED ET AL 1984
1701	1,1,2,2-TETRACHLOROE	FM	MORT2	1400.00	120	0			AHMED ET AL 1984
1702	1,1,2,2-TETRACHLOROE	FM	MORT2	4000.00	120	6			AHMED ET AL 1984
1703	1,1,2,2-TETRACHLOROE	FM	MORT2	6800.00	120	6			AHMED ET AL 1984
1704	1,1,2,2-TETRACHLOROE	FM	MORT2	13700.00	120	105			AHMED ET AL 1984
1705	1,1,2,2-TETRACHLOROE	FM	MORT2	28400.00	120	120			AHMED ET AL 1984
1706	1,1,2,2-TETRACHLOROE	FM	WEIGHT	12.00				0.19	AHMED ET AL 1984
1707	1,1,2,2-TETRACHLOROE	FM	WEIGHT	1400.00				0.19	AHMED ET AL 1984
1708	1,1,2,2-TETRACHLOROE	FM	WEIGHT	4000.00				0.15	AHMED ET AL 1984
1709	1,1,2,2-TETRACHLOROE	FM	WEIGHT	6800.00				0.14	AHMED ET AL 1984
1710	1,1,2,2-TETRACHLOROE	FM	WEIGHT	13700.00				0.02	AHMED ET AL 1984
1711	1,1,2,2-TETRACHLOROE	FM	WEIGHT	28400.00				0.00	AHMED ET AL 1984
1712	1,2-DICHLOROETHANE	FM	HATCH	300.00	120	23			BENOIT ET AL 1982
1713	1,2-DICHLOROETHANE	FM	HATCH	4000.00	120	23			BENOIT ET AL 1982
1714	1,2-DICHLOROETHANE	FM	HATCH	7000.00	120	27			BENOIT ET AL 1982
1715	1,2-DICHLOROETHANE	FM	HATCH	14000.00	120	33			BENOIT ET AL 1982
1716	1,2-DICHLOROETHANE	FM	HATCH	29000.00	120	25			BENOIT ET AL 1982
1717	1,2-DICHLOROETHANE	FM	HATCH	59000.00	120	25			BENOIT ET AL 1982
1718	1,2-DICHLOROETHANE	FM	MORT2	300.00	60	5			BENOIT ET AL 1982
1719	1,2-DICHLOROETHANE	FM	MORT2	4000.00	60	3			BENOIT ET AL 1982
1720	1,2-DICHLOROETHANE	FM	MORT2	7000.00	60	5			BENOIT ET AL 1982
1721	1,2-DICHLOROETHANE	FM	MORT2	14000.00	60	5			BENOIT ET AL 1982
1722	1,2-DICHLOROETHANE	FM	MORT2	29000.00	60	2			BENOIT ET AL 1982
1723	1,2-DICHLOROETHANE	FM	MORT2	59000.00	60	6			BENOIT ET AL 1982
1724	1,2-DICHLOROETHANE	FM	WEIGHT	300.00				0.13	BENOIT ET AL 1982
1725	1,2-DICHLOROETHANE	FM	WEIGHT	4000.00				0.13	BENOIT ET AL 1982
1726	1,2-DICHLOROETHANE	FM	WEIGHT	7000.00				0.13	BENOIT ET AL 1982
1727	1,2-DICHLOROETHANE	FM	WEIGHT	14000.00				0.13	BENOIT ET AL 1982
1728	1,2-DICHLOROETHANE	FM	WEIGHT	29000.00				0.12	BENOIT ET AL 1982

Table B.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1729	1,2-DICHLOROETHANE	FM	WEIGHT	59000.00				0.05	BENOIT ET AL 1982
1730	1,2-DICHLOROPROPANE	FM	HATCH	100.00	120	4			BENOIT ET AL 1982
1731	1,2-DICHLOROPROPANE	FM	HATCH	6000.00	120	5			BENOIT ET AL 1982
1732	1,2-DICHLOROPROPANE	FM	HATCH	11000.00	120	3			BENOIT ET AL 1982
1733	1,2-DICHLOROPROPANE	FM	HATCH	25000.00	120	3			BENOIT ET AL 1982
1734	1,2-DICHLOROPROPANE	FM	HATCH	51000.00	120	43			BENOIT ET AL 1982
1735	1,2-DICHLOROPROPANE	FM	HATCH	110000.00	120	120			BENOIT ET AL 1982
1736	1,2-DICHLOROPROPANE	FM	MORT2	100.00	60	3			BENOIT ET AL 1982
1737	1,2-DICHLOROPROPANE	FM	MORT2	6000.00	60	5			BENOIT ET AL 1982
1738	1,2-DICHLOROPROPANE	FM	MORT2	11000.00	60	3			BENOIT ET AL 1982
1739	1,2-DICHLOROPROPANE	FM	MORT2	25000.00	60	25			BENOIT ET AL 1982
1740	1,2-DICHLOROPROPANE	FM	MORT2	51000.00	60	44			BENOIT ET AL 1982
1741	1,2-DICHLOROPROPANE	FM	MORT2	110000.00	120	120			BENOIT ET AL 1982
1742	1,2-DICHLOROPROPANE	FM	WEIGHT	100.00				0.14	BENOIT ET AL 1982
1743	1,2-DICHLOROPROPANE	FM	WEIGHT	6000.00				0.14	BENOIT ET AL 1982
1744	1,2-DICHLOROPROPANE	FM	WEIGHT	11000.00				0.13	BENOIT ET AL 1982
1745	1,2-DICHLOROPROPANE	FM	WEIGHT	25000.00				0.08	BENOIT ET AL 1982
1746	1,2-DICHLOROPROPANE	FM	WEIGHT	51000.00				0.02	BENOIT ET AL 1982
1747	1,2-DICHLOROPROPANE	FM	WEIGHT	110000.00				0.00	BENOIT ET AL 1982
1748	1,2,3,4-TETRACHLOROB	FM	MORT2	0.35	120	10			AHMED ET AL 1984
1749	1,2,3,4-TETRACHLOROB	FM	MORT2	19.00	120	20			AHMED ET AL 1984
1750	1,2,3,4-TETRACHLOROB	FM	MORT2	39.00	120	12			AHMED ET AL 1984
1751	1,2,3,4-TETRACHLOROB	FM	MORT2	110.00	120	8			AHMED ET AL 1984
1752	1,2,3,4-TETRACHLOROB	FM	MORT2	245.00	120	22			AHMED ET AL 1984
1753	1,2,3,4-TETRACHLOROB	FM	MORT2	412.00	120	48			AHMED ET AL 1984
1754	1,2,3,4-TETRACHLOROB	FM	WEIGHT	0.35				0.11	AHMED ET AL 1984
1755	1,2,3,4-TETRACHLOROB	FM	WEIGHT	19.00				0.11	AHMED ET AL 1984
1756	1,2,3,4-TETRACHLOROB	FM	WEIGHT	39.00				0.11	AHMED ET AL 1984
1757	1,2,3,4-TETRACHLOROB	FM	WEIGHT	110.00				0.10	AHMED ET AL 1984
1758	1,2,3,4-TETRACHLOROB	FM	WEIGHT	245.00				0.10	AHMED ET AL 1984
1759	1,2,3,4-TETRACHLOROB	FM	WEIGHT	412.00				0.06	AHMED ET AL 1984
1760	1,2,4-TRICHLOROBENZE	FM	MORT2	15.00	120	10			AHMED ET AL 1984
1761	1,2,4-TRICHLOROBENZE	FM	MORT2	75.00	120	20			AHMED ET AL 1984
1762	1,2,4-TRICHLOROBENZE	FM	MORT2	134.00	120	10			AHMED ET AL 1984
1763	1,2,4-TRICHLOROBENZE	FM	MORT2	304.00	120	10			AHMED ET AL 1984
1764	1,2,4-TRICHLOROBENZE	FM	MORT2	499.00	120	14			AHMED ET AL 1984
1765	1,2,4-TRICHLOROBENZE	FM	MORT2	1001.00	120	46			AHMED ET AL 1984
1766	1,2,4-TRICHLOROBENZE	FM	WEIGHT	15.00				0.09	AHMED ET AL 1984
1767	1,2,4-TRICHLOROBENZE	FM	WEIGHT	75.00				0.10	AHMED ET AL 1984
1768	1,2,4-TRICHLOROBENZE	FM	WEIGHT	134.00				0.09	AHMED ET AL 1984
1769	1,2,4-TRICHLOROBENZE	FM	WEIGHT	304.00				0.08	AHMED ET AL 1984
1770	1,2,4-TRICHLOROBENZE	FM	WEIGHT	499.00				0.09	AHMED ET AL 1984
1771	1,2,4-TRICHLOROBENZE	FM	WEIGHT	1001.00				0.07	AHMED ET AL 1984
1772	1,3-DICHLOROBENZENE	FM	MORT2	31.00	120	4			AHMED ET AL 1984
1773	1,3-DICHLOROBENZENE	FM	MORT2	304.00	120	2			AHMED ET AL 1984
1774	1,3-DICHLOROBENZENE	FM	MORT2	555.00	120	4			AHMED ET AL 1984
1775	1,3-DICHLOROBENZENE	FM	MORT2	1000.00	120	6			AHMED ET AL 1984
1776	1,3-DICHLOROBENZENE	FM	MORT2	2267.00	120	8			AHMED ET AL 1984
1777	1,3-DICHLOROBENZENE	FM	MORT2	3913.00	120	112			AHMED ET AL 1984
1778	1,3-DICHLOROBENZENE	FM	WEIGHT	31.00				0.10	AHMED ET AL 1984
1779	1,3-DICHLOROBENZENE	FM	WEIGHT	304.00				0.10	AHMED ET AL 1984
1780	1,3-DICHLOROBENZENE	FM	WEIGHT	555.00				0.10	AHMED ET AL 1984
1781	1,3-DICHLOROBENZENE	FM	WEIGHT	1000.00				0.10	AHMED ET AL 1984
1782	1,3-DICHLOROBENZENE	FM	WEIGHT	2267.00				0.07	AHMED ET AL 1984

Table 8.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1783	1,3-DICHLOROBENZENE	FM	WEIGHT	3913.00				0.01	AHMED ET AL 1984
1784	1,3-DICHLOROPROPANE	FM	HATCH	200.00	120	20			BENOIT ET AL 1982
1785	1,3-DICHLOROPROPANE	FM	HATCH	4000.00	120	29			BENOIT ET AL 1982
1786	1,3-DICHLOROPROPANE	FM	HATCH	8000.00	120	21			BENOIT ET AL 1982
1787	1,3-DICHLOROPROPANE	FM	HATCH	16000.00	120	26			BENOIT ET AL 1982
1788	1,3-DICHLOROPROPANE	FM	HATCH	32000.00	120	22			BENOIT ET AL 1982
1789	1,3-DICHLOROPROPANE	FM	HATCH	65000.00	120	19			BENOIT ET AL 1982
1790	1,3-DICHLOROPROPANE	FM	MORT2	200.00	60	4			BENOIT ET AL 1982
1791	1,3-DICHLOROPROPANE	FM	MORT2	4000.00	60	1			BENOIT ET AL 1982
1792	1,3-DICHLOROPROPANE	FM	MORT2	8000.00	60	4			BENOIT ET AL 1982
1793	1,3-DICHLOROPROPANE	FM	MORT2	16000.00	60	2			BENOIT ET AL 1982
1794	1,3-DICHLOROPROPANE	FM	MORT2	32000.00	60	1			BENOIT ET AL 1982
1795	1,3-DICHLOROPROPANE	FM	MORT2	65000.00	60	31			BENOIT ET AL 1982
1796	1,3-DICHLOROPROPANE	FM	WEIGHT	200.00				0.13	BENOIT ET AL 1982
1797	1,3-DICHLOROPROPANE	FM	WEIGHT	4000.00				0.11	BENOIT ET AL 1982
1798	1,3-DICHLOROPROPANE	FM	WEIGHT	8000.00				0.11	BENOIT ET AL 1982
1799	1,3-DICHLOROPROPANE	FM	WEIGHT	16000.00				0.10	BENOIT ET AL 1982
1800	1,3-DICHLOROPROPANE	FM	WEIGHT	32000.00				0.08	BENOIT ET AL 1982
1801	1,3-DICHLOROPROPANE	FM	WEIGHT	65000.00				0.02	BENOIT ET AL 1982
1802	1,4-DICHLOROBENZENE	FM	MORT2	19.00	120	6			AHMED ET AL 1984
1803	1,4-DICHLOROBENZENE	FM	MORT2	565.00	120	8			AHMED ET AL 1984
1804	1,4-DICHLOROBENZENE	FM	MORT2	1040.00	120	26			AHMED ET AL 1984
1805	1,4-DICHLOROBENZENE	FM	MORT2	2000.00	120	120			AHMED ET AL 1984
1806	1,4-DICHLOROBENZENE	FM	MORT2	4090.00	120	120			AHMED ET AL 1984
1807	1,4-DICHLOROBENZENE	FM	MORT2	8720.00	120	120			AHMED ET AL 1984
1808	1,4-DICHLOROBENZENE	FM	WEIGHT	19.00				0.10	AHMED ET AL 1984
1809	1,4-DICHLOROBENZENE	FM	WEIGHT	565.00				0.10	AHMED ET AL 1984
1810	1,4-DICHLOROBENZENE	FM	WEIGHT	1040.00				0.09	AHMED ET AL 1984
1811	1,4-DICHLOROBENZENE	FM	WEIGHT	2000.00					AHMED ET AL 1984
1812	1,4-DICHLOROBENZENE	FM	WEIGHT	4090.00					AHMED ET AL 1984
1813	1,4-DICHLOROBENZENE	FM	WEIGHT	8720.00					AHMED ET AL 1984
1814	2,4-DICHLOROPHENOL	FM	HATCH	0.00	200	37			HOLCOMBE ET AL 1982
1815	2,4-DICHLOROPHENOL	FM	HATCH	150.00	200	28			HOLCOMBE ET AL 1982
1816	2,4-DICHLOROPHENOL	FM	HATCH	290.00	200	36			HOLCOMBE ET AL 1982
1817	2,4-DICHLOROPHENOL	FM	HATCH	460.00	200	48			HOLCOMBE ET AL 1982
1818	2,4-DICHLOROPHENOL	FM	HATCH	770.00	200	41			HOLCOMBE ET AL 1982
1819	2,4-DICHLOROPHENOL	FM	HATCH	1240.00	200	40			HOLCOMBE ET AL 1982
1820	2,4-DICHLOROPHENOL	FM	MORT2	0.00	100	25			HOLCOMBE ET AL 1982
1821	2,4-DICHLOROPHENOL	FM	MORT2	150.00	100	31			HOLCOMBE ET AL 1982
1822	2,4-DICHLOROPHENOL	FM	MORT2	290.00	100	30			HOLCOMBE ET AL 1982
1823	2,4-DICHLOROPHENOL	FM	MORT2	460.00	100	58			HOLCOMBE ET AL 1982
1824	2,4-DICHLOROPHENOL	FM	MORT2	770.00	100	78			HOLCOMBE ET AL 1982
1825	2,4-DICHLOROPHENOL	FM	MORT2	1240.00	100	94			HOLCOMBE ET AL 1982
1826	2,4-DICHLOROPHENOL	FM	WEIGHT	0.00	100			0.09	HOLCOMBE ET AL 1982
1827	2,4-DICHLOROPHENOL	FM	WEIGHT	150.00	100			0.09	HOLCOMBE ET AL 1982
1828	2,4-DICHLOROPHENOL	FM	WEIGHT	290.00	100			0.09	HOLCOMBE ET AL 1982
1829	2,4-DICHLOROPHENOL	FM	WEIGHT	460.00	100			0.11	HOLCOMBE ET AL 1982
1830	2,4-DICHLOROPHENOL	FM	WEIGHT	770.00	100			0.08	HOLCOMBE ET AL 1982
1831	2,4-DICHLOROPHENOL	FM	WEIGHT	1240.00	100			0.02	HOLCOMBE ET AL 1982
1832	2,4-DIMETHYLPHENOL	FM	HATCH	0.00	200	35			HOLCOMBE ET AL 1982
1833	2,4-DIMETHYLPHENOL	FM	HATCH	900.00	200	23			HOLCOMBE ET AL 1982
1834	2,4-DIMETHYLPHENOL	FM	HATCH	1360.00	200	25			HOLCOMBE ET AL 1982
1835	2,4-DIMETHYLPHENOL	FM	HATCH	1970.00	200	25			HOLCOMBE ET AL 1982
1836	2,4-DIMETHYLPHENOL	FM	HATCH	3100.00	200	25			HOLCOMBE ET AL 1982

Table 8.1. (Continued)

OBS	CHEMICAL	SPECIES	PARAM	DOSE	NTESTED	RESPONSE	EGGS	WEIGHT	SOURCE
1837	2,4-DIMETHYLPHENOL	FM	HATCH	5130.00	200	40			HOLCOMBE ET AL 1982
1838	2,4-DIMETHYLPHENOL	FM	MORT2	0.00	100	10			HOLCOMBE ET AL 1982
1839	2,4-DIMETHYLPHENOL	FM	MORT2	900.00	100	22			HOLCOMBE ET AL 1982
1840	2,4-DIMETHYLPHENOL	FM	MORT2	1360.00	100	22			HOLCOMBE ET AL 1982
1841	2,4-DIMETHYLPHENOL	FM	MORT2	1970.00	100	25			HOLCOMBE ET AL 1982
1842	2,4-DIMETHYLPHENOL	FM	MORT2	3110.00	100	27			HOLCOMBE ET AL 1982
1843	2,4-DIMETHYLPHENOL	FM	MORT2	5130.00	100	44			HOLCOMBE ET AL 1982
1844	2,4-DIMETHYLPHENOL	FM	WEIGHT	0.00				0.07	HOLCOMBE ET AL 1982
1845	2,4-DIMETHYLPHENOL	FM	WEIGHT	900.00				0.08	HOLCOMBE ET AL 1982
1846	2,4-DIMETHYLPHENOL	FM	WEIGHT	1360.00				0.08	HOLCOMBE ET AL 1982
1847	2,4-DIMETHYLPHENOL	FM	WEIGHT	1970.00				0.07	HOLCOMBE ET AL 1982
1848	2,4-DIMETHYLPHENOL	FM	WEIGHT	3110.00				0.06	HOLCOMBE ET AL 1982
1849	2,4-DIMETHYLPHENOL	FM	WEIGHT	5130.00				0.05	HOLCOMBE ET AL 1982

SPECIES = Species of test organism: AS = atlantic salmon, BG = bluegill, BM = bluntnose minnow, BNT = brown trout, BT = brook trout, CC = channel catfish, CHS = chinook salmon, COS = coho salmon, FF = flagfish, FM = fathead minnow, G = guppy, JM = Japanese medaka, LT = lake trout, NP = northern pike, RT = rainbow trout, SB = smallmouth bass, WE = walleye, and WS = white sucker.
PARAM = Response parameter: MORT1 = mortality of parental fish, EGGS = number of eggs per female, HATCH = proportion of eggs failing to produce normal larvae, MORT2 = mortality of larval fish, and WEIGHT = mean weight of individual fish at the end of larval exposure.
DOSE = Exposure concentration.
NTESTED = Number of test organisms per concentration.
RESPONSE = Number of organisms per concentration.
EGGS = Number of eggs per female.
WEIGHT = Mean weight of individual fish at the end of larval exposure in grams.

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