Risk Assessment Pilot Study. Phase 3
Naval Construction Battalion Center, Davisville, Rhode Island

Science Applications International Corp., Narragansett, RI

Prepared for:

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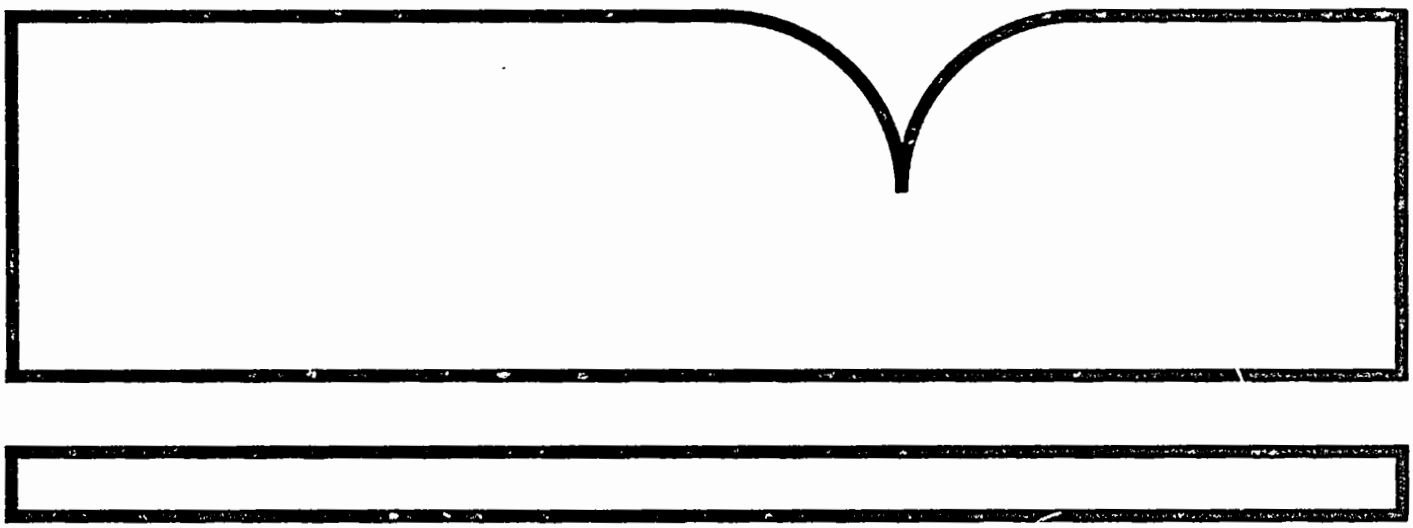

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To undertake a marine ecological risk assessment at the Naval Construction Battalion Center (NCBC) Davisville, Rhode Island to determine the effect of hazardous waste disposal on Allen Harbor and Narragansett Bay. Allen Harbor, located in Narragansett Bay at NCBC Davisville, was closed to shellfishing by the Rhode Island Department of Environmental Management because of suspected hazardous waste contamination from a landfill and disposal area adjacent to the harbor. NCBC Davisville was added to the National Priority List in November 1989. Between 1946 and 1972, the 15-acre landfill received a wide variety of wastes, including sewage sludge, solvents, paints, chromic acid, PCB-contaminated waste oils, preservatives, blasting grit, and other municipal and industrial wastes generated at NCBC Davisville and at the Naval Air Station Quonset Point. Another site, also adjacent to Allen Harbor on Calf Pasture Point, was used for disposal of calcium hypochlorite decontaminating solution and chlorides.-

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NAVAL CONSTRUCTION BATTALION CENTER
DAVISVILLE, RHODE ISLAND

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ABBREVIATIONS AND ACRONYMS

AA	-- atomic absorption
ANOVA	-- analysis of variance
BAF	-- bioaccumulation factor
BHC	-- hexachlorocyclohexane
CERCLA	-- Comprehensive Environmental Response Compensation and Liability Act of 1980
CLIS	-- Central Long Island Sound
DANC	-- Decontaminating Agent Non-Corrosive (1,3-dichloro-5,5-dimethylhydantoin)
DDD	-- dichlorodiphenyldichloroethane
DDE	-- dichlorodiphenyldichloroethene
DDT	-- dichlorodiphenyltrichloroethene
DMSO	-- dimethylsulfoxide
ECD	-- electron capture detection
EPA	-- United States Environmental Protection Agency
ERA	-- environmental risk assessment
ERLN	-- Environmental Research Laboratory, Narragansett, RI
GC	-- gas chromatograph
HCB	-- hexachlorobenzene
H-DANC	-- 5,5-dimethylhydantoin
HGA	-- heated graphite atomization
Hn	-- hematopoietic neoplasia
ICP	-- inductively coupled plasma spectrometer
MBT	-- monobutyltin
MOA	-- Memorandum of Agreement
MS	-- mass spectrometer
NAS	-- Naval Air Station
NCCOSC	-- Naval Command, Control and Ocean Surveillance Center, San Diego, CA
NCBC	-- Naval Construction Battalion Center
ND	-- not detected
NM	-- not measured
NPL	-- National Priorities List

NSW	-- Narragansett Bay seawater
PAH	-- polycyclic aromatic hydrocarbon
PCA	-- principal component analysis
PCB	-- polychlorinated biphenyl
ppb	-- parts per billion
ppm	-- parts per million
pptr	-- parts per trillion
RAPS	-- Allen Harbor Risk Assessment Pilot Study
RCRA	-- Resource Conservation and Recovery Act of 1976
RIDEM	-- Rhode Island Department of Environmental Management
RI/FS	-- Remedial Investigation/Feasibility Study
RQ	-- risk quotient
QA/QC	-- quality assurance/quality control
SAIC	-- Science Applications International Corporation
SARA	-- Superfund Amendment and Reauthorization Act of 1986
SFG	-- Scope for Growth
SOP	-- Standard Operating Procedure
ΣTU	-- sum of toxic units
TBT	-- tributyltin
TOC	-- total organic carbon
TU	-- toxic unit
VOC	-- volatile organic compound

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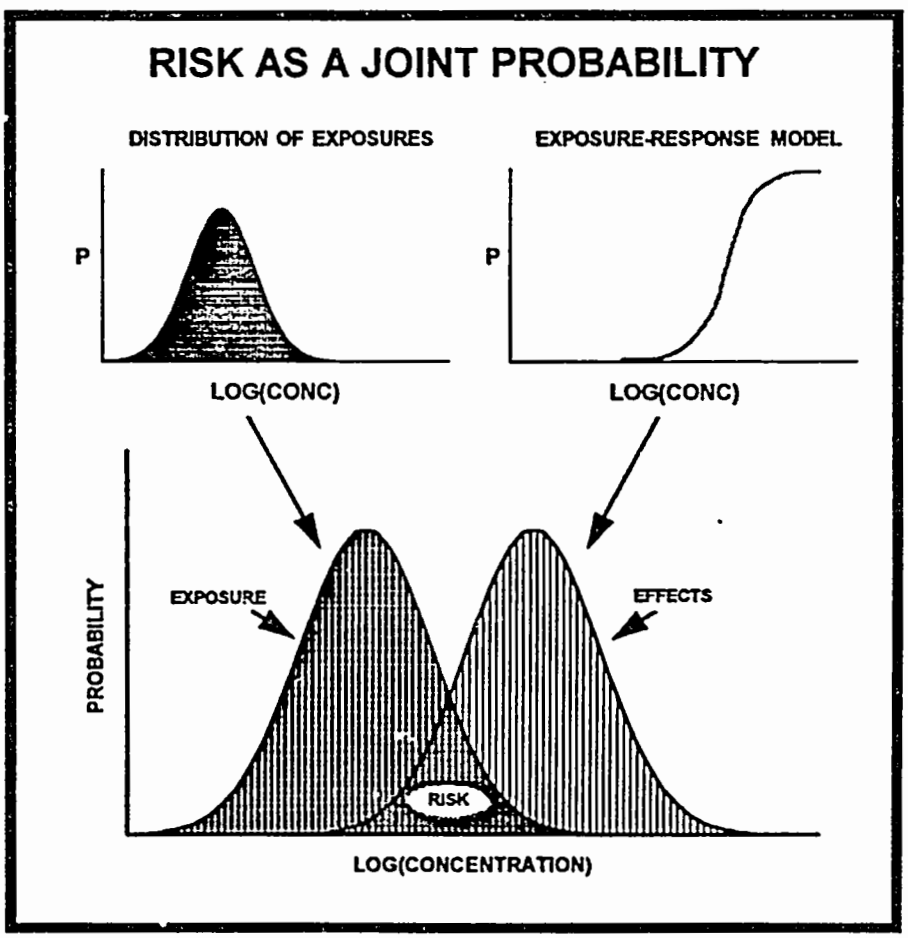

EXECUTIVE SUMMARY

OBJECTIVE

To undertake a marine ecological risk assessment at the Naval Construction Battalion Center (NCBC) Davisville, Rhode Island to determine the effect of hazardous waste disposal on Allen Harbor and Narragansett Bay. Allen Harbor, located in Narragansett Bay at NCBC Davisville, was closed to shellfishing by the Rhode Island Department of Environmental Management because of suspected hazardous waste contamination from a landfill and disposal area adjacent to the harbor. NCBC Davisville was added to the National Priority List in November 1989. Between 1946 and 1972, the 15-acre landfill received a wide variety of wastes, including sewage sludge, solvents, paints, chromic acid, PCB-contaminated waste oils, preservatives, blasting grit, and other municipal and industrial wastes generated at NCBC Davisville and at the Naval Air Station Quonset Point. Another site, also adjacent to Allen Harbor on Calf Pasture Point, was used for disposal of calcium hypochlorite decontaminating solution and chlorides.

APPROACH

A phased approach was developed to assess the ecological risks to Allen Harbor and Narragansett Bay posed by these hazardous waste sites. This report covers Phase III, a quantification of biological effects and ecological risks directly associated with the NCBC landfill. Exposure-response assays were conducted of landfill seep water, sediments, and sediment extracts using a variety of marine species and endpoints. Resulting data were used to develop models describing biological response as a function of exposure concentration. These models were used to quantify risks posed by the landfill to pelagic and benthic ecological systems in Allen Harbor using a joint probability method. A laboratory evaluation of the relationship between landfill contaminants and neoplasia development in the soft-shell clam also was conducted.

RESULTS

This Phase III study provided quantitative information useful in describing the ecological risks to Allen Harbor. Exposure-response models were developed successfully for landfill seep water and sediment extracts using data obtained for a number of species and short-term toxicological endpoints. Using a joint probability method, upper-bound probabilities of risk ranging between 0.24 and 0.69 were estimated for landfill seep water, with similar values calculated for storm runoff sources. Whole landfill sediments were not toxic to organisms tested in the laboratory, but sediment extract models suggested risks up to 0.75 to benthic organisms with contaminant bioavailability taken into account. No statistical relationships were observed between landfill exposure media and soft-shell clam neoplasia, although the experiment was not conclusive because conditions may have compromised treatment effects.

INTRODUCTION

BACKGROUND

In 1988, the U.S. Environmental Protection Agency (U.S. EPA) Environmental Research Laboratory at Narragansett, Rhode Island (ERLN), and the U.S. Navy Naval Command, Control and Ocean Surveillance Center (NCCOSC; formerly the Naval Ocean Systems Center), entered into a Memorandum of Agreement (MOA) to develop cooperative research and monitoring activities for conducting ecological risk assessments (ERAs). Under this agreement case studies were developed to characterize the risk of Navy hazardous waste disposal sites which could potentially impact aquatic ecosystems. This joint research supports the Navy's response to the requirements of the Comprehensive Environmental Response Compensation and Liability Act of 1980 (CERCLA), as amended by the Superfund Amendment and Reauthorization Act of 1986 (SARA), and the Resource Conservation and Recovery Act of 1976 (RCRA). Additionally, the agreement afforded the opportunity for ERLN to develop and refine methodologies for examining ecological risks associated with anthropogenic wastes in the marine environment through their application in specific case studies.

The first case study developed under the MOA was the Allen Harbor Risk Assessment Pilot Study (RAPS) conducted at the Naval Construction Battalion Center (NCBC) Davisville, Rhode Island. Allen Harbor is a small embayment of Narragansett Bay located adjacent to NCBC Davisville, a facility added to the National Priorities List (NPL) in 1989. Two sites at NCBC Davisville were of particular concern with respect to potential negative impacts on Allen Harbor: a 15-acre landfill situated next to Allen Harbor, and Calf Pasture Point, which separates Allen Harbor from the West Passage of Narragansett Bay (Figure 1).

The primary objective of the RAPS was to determine the presence and extent of adverse ecological impacts in Allen Harbor and Narragansett Bay potentially related to NCBC Davisville. A phased approach was developed for this study. These phases, modified somewhat from those reported in earlier documents (Munns *et al.* 1991) are:

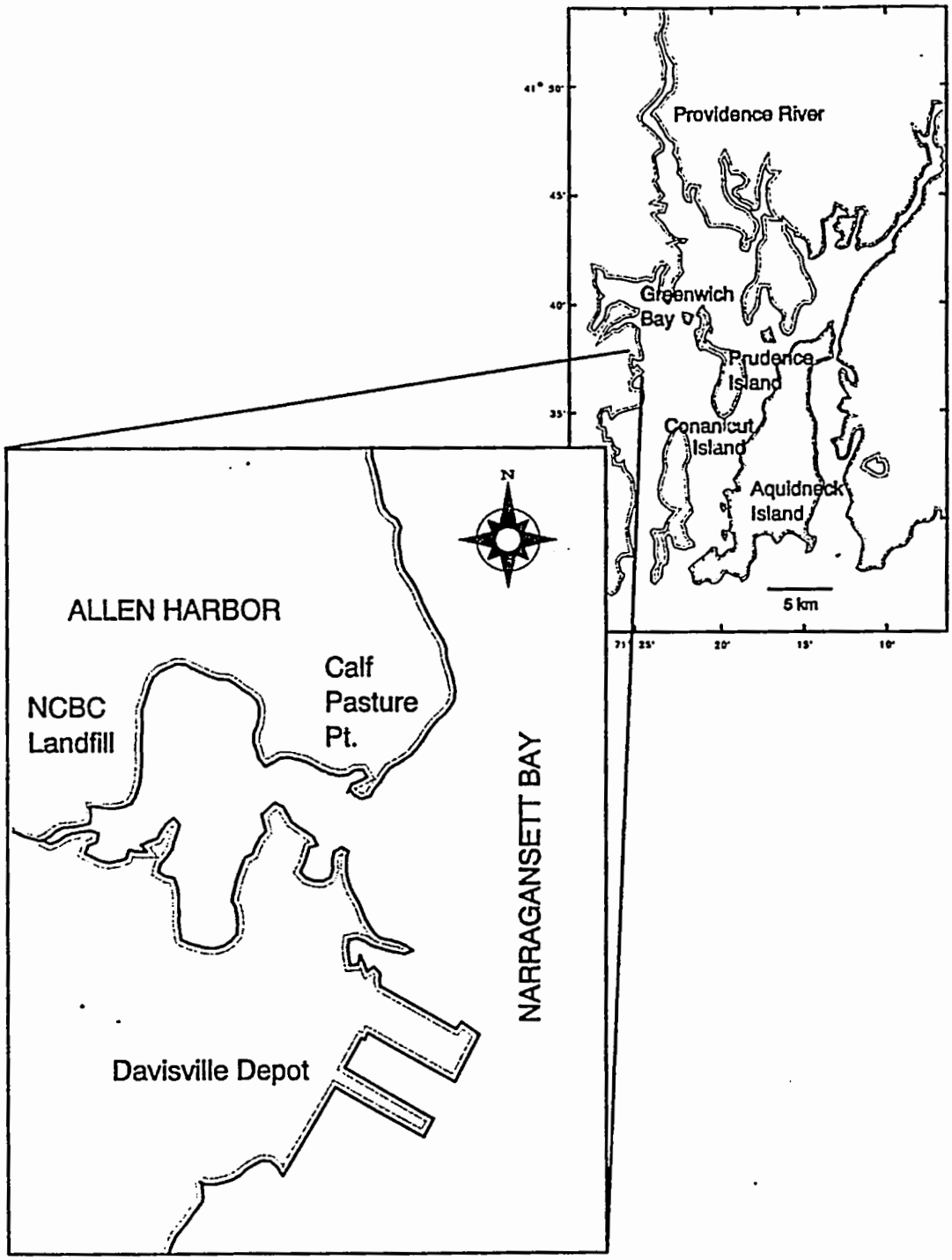

Figure 1. Allen Harbor and the locations of the landfill and Calf Pasture Point.

Phase I - **Information Gathering** -- To determine the existence, nature, and extent of adverse impact in Allen Harbor and Narragansett Bay resulting from contaminants originating from NCBC Davisville. The specific activities involved in this step included identification and collation of existing data and information relevant to the ecology of Allen Harbor, characterization of sediments and the water column in the harbor and nearby areas of Narragansett Bay, evaluation of the natural resources of Allen Harbor relative to nearby areas of Narragansett Bay, and development of a preliminary ERA of Allen Harbor.

Phase II - **Verification and Quantification of Toxicological Effects** -- To verify the lack of adverse environmental impact (Option I), or to determine the nature and extent of contaminant impact on the marine system (Option II). Option I was indicated, so studies were to be conducted to confirm the lack of negative impact. Information obtained from this phase was used to further evaluate marine risks at NCBC Davisville. If Option II had been indicated, characterizations of contaminant source and movement were to occur. Further, criteria were to be identified for evaluation of remedial alternatives, and development of a monitoring plan capable of evaluating remedial activities would be developed.

Phase III - **Quantification of Ecological Risks** -- To quantify ecological risks to Allen Harbor associated with waste sites of concern. The primary activities of this phase included conduct of laboratory assays and experiments to characterize toxicological impacts to biota, representative of those living in Allen Harbor, in the form of exposure-response models. Together with the information collected in Phases I and II, these models formed the basis of the Final Marine Ecological Risk Assessment for NCBC Davisville. This information could be used to develop a monitoring program for continuous verification of environmental safety.

Detailed descriptions of the activities and findings of Phases I, completed in 1990, and Phase II, completed in 1991, are given in Munns *et al.* (1991, 1993). Summaries of Phases I and II also are presented below. The approach taken to address the objectives of Phase III, and the results of those activities, are the primary subject of this report.

PHASE I SUMMARY

Phase I involved the collection and collation of empirical environmental data characterizing contaminant input to Allen Harbor (Waste Characterization), the resulting exposure field relative to contaminant levels in Narragansett Bay (Exposure Assessment), and the status and responses of biota residing in the harbor (Ecosystem Characterization and Effects Assessment). This information was synthesized into a preliminary characterization of ecological risk to Allen Harbor (Munns *et al.* 1991). The specific findings of these characterization and assessment activities are described briefly below.

Waste Site Characterization

Information regarding contaminants associated with the NCBC Davisville landfill and Calf Pasture Point was obtained from past reports and from chemical analyses of seep and ground water, and of sediments. Historic information indicated a range of waste materials to have been disposed in the landfill, including complex organic and inorganic wastes such as jet fuel, waste oils, and coal ash, as well as organic solvents, asbestos, and sewage sludge. Chemical analysis of seep, test pit, and well water samples indicated high levels of several chlorinated solvents, including *cis*- and *trans*-1,2-dichloroethene, 1,2-dichloroethane, chlorobenzene, and benzene. High levels (up to 1.49 ppb) of total polychlorinated biphenyls (PCBs) were measured in seep samples from the south face of the landfill, and trace metal concentrations were high enough to violate the U.S. EPA Water Quality Criteria (WQC) for Cu, Cd, and Pb. Petroleum hydrocarbons were also present (up to 100-200 ppb) in some samples. Pesticides were typically not detected in the ground water.

Exposure Assessment

Contaminant exposure conditions in Allen Harbor were assessed through chemical analyses of sediments, large volume water samples, and of indigenous and deployed biota obtained at a total of 29 intertidal and subtidal stations in Allen Harbor and Narragansett Bay. Allen Harbor displayed some of the highest chemical concentrations observed among stations. Highest concentrations within the harbor generally occurred at a station at the

southern end of the harbor, removed from the landfill. Little evidence of contaminant migration from the landfill or from Calf Pasture Point was observed.

Tissue residue levels in benthic organisms provided an additional measure of sediment exposure conditions. Significant differences were often observed among stations in quahog (*Mercenaria mercenaria*) tissue residues, and Allen Harbor clams often grouped with those exhibiting the highest mean concentrations. In concordance with sediment chemistry levels, tissue residues of quahogs within Allen Harbor were typically highest at the southern end of the harbor, away from the landfill and Calf Pasture Point. Tissue concentrations in *Mya arenaria* were elevated relative to other stations for the pesticide γ-hexachlorocyclohexane (BHC) and for butyltin species. Tributyltin (TBT) residues were extremely high (8,800 ppb dry weight) in Allen Harbor *Mya*. Again, contaminant residues were generally higher at the south end of the landfill than at other stations within Allen Harbor.

Characterization of water column exposure conditions through direct water sampling and analysis was limited to organic compounds. Concentrations of pesticides in both dissolved and particulate phases were generally below detection both within and outside of the harbor. PCBs were observed in the particulate phase at concentrations in the 1-2 parts per trillion (pptr) range, with somewhat higher levels in Allen Harbor than in Narragansett Bay. Generally, however, water-bourne contaminant levels were similar to background levels observed in relatively clean areas. Tissue residues of selected contaminants in deployed *Mytilus edulis*, used as a surrogate for water chemistry, were somewhat elevated relative to mussels from reference areas in Narragansett Bay, but were fairly typical of clean areas elsewhere in the northeast United States.

Effects Assessment

The ecological impacts of contaminants within Allen Harbor were evaluated through a combination of field sampling, field experimentation, and laboratory assays. These activities involved evaluation of a number of biological endpoints which have been shown to be sensitive to contaminant insult, and whose relationship with ecological status are fairly well established. Native *Mercenaria mercenaria*, *Mya arenaria*, and *Crassostrea virginica* were

sampled for population abundance, individual condition, and histopathological effects. The blue mussel, *Mytilus edulis*, was deployed at several stations to address the effects of water quality on physiological condition and growth. Finally, the toxicity of sediments within Allen Harbor and at stations in Narragansett Bay was determined in the laboratory using both standard amphipod (*Ampelisca abdita*) bioassays and biomarker tests under development at ERLN. These later tests utilized field exposed organisms or laboratory exposed cell cultures to investigate the modes and mechanisms of contaminant impact on cellular and subcellular biological processes.

Despite the closure of Allen Harbor to shellfishing in 1984, *Mercenaria* in Allen Harbor were significantly smaller than those found at Narragansett Bay stations. Reduced shell size may reflect some impact of sediment or water quality, or may simply be the result of a lack of fishing pressure (thereby increasing intraspecific competition) due to the harbor shellfish closure. Condition Index followed a pattern among stations similar to that of shell length. Proximity to the landfill had no discernible effect on *Mercenaria* length or condition. No significant pathologies were observed in Allen Harbor animals. Densities of *Mya* were higher in Allen Harbor than at Narragansett Bay stations, likely reflecting the lack of recreational clamming in the harbor. No clear pattern in *Mya* shell length emerged in station-wise comparisons, although Allen Harbor animals were typically larger than those collected outside of the harbor. A number of pathological conditions were observed in *Mya* from all stations throughout this study. These included pathologies commonly associated with soft shell clams, such as atypical cell hyperplasia in the gills and kidney, and inflammatory responses. Neoplastic lesions associated with the heart and hematopoietic system (Hn) were found in clams collected in Allen Harbor and at nearby Marsh Point. Within Allen Harbor proper, the highest prevalence of Hn was found near Calf Pasture Point. *Crassostrea* in Allen Harbor were both larger and in better condition than those at a reference station in Narragansett Bay. Differences in shellfishing pressure may explain these differences. Histological examination of oysters revealed no pathology. All organisms were in good to excellent health.

Allen Harbor subtidal sediments exhibited uniformly low toxicity, as measured by the

acute mortality response of the benthic amphipod *Ampelisca abdita*, indicating little impact from the landfill or Calf Pasture Point. In contrast, extreme mortality was associated with material collected from the north and middle faces of the landfill. Although suggestive of landfill-associated contaminant effects, material from these sites was composed primarily of very course-grained material, thereby confounding toxicological analyses through a grain size effect. Extracts of Allen Harbor sediment produced significant mutagenic effects under certain conditions as determined by the biomarker Sister Chromatid Exchange assay. No significant response was observed in the V79/Metabolic Cooperation biomarker assay for the presence of tumor promoters. Sediment extracts also affected fertilization, growth (length) and survival of the sea urchin *Arbacia*, although equivalent responses were observed with extracts of sediments obtained from a reference site.

Mytilus edulis deployed in Allen Harbor in May-June 1989 showed both lower clearance and higher respiration rates than did mussels deployed at the Narragansett Bay stations. When integrated into the Scope for Growth (SFG) index, these rates indicated significantly reduced physiological condition for Allen Harbor mussels. Chemical analysis of the soft tissues of these animals were equivocal with respect to the causes of the observed differences in physiological response. Mussels exposed during a fall deployment in September-October of that year also exhibited differences in clearance and respiration rates with respect to station. These differences translate into SFG estimates which were depressed in Allen Harbor and immediately outside the harbor, relative to that in lower Narragansett Bay. The consistently low clearance rate and SFG integration observed in Allen Harbor mussels indicates a harbor water quality problem. No differences were observed in the *in vivo* immunological response of *Mytilus* deployed in Allen Harbor and in lower Narragansett Bay, nor was pathology observed in animals deployed at any site.

Preliminary Characterization of Ecological Risk

Information collected during the Waste Site Characterization, and Exposure and Effects Assessments were synthesized into a preliminary characterization of ecological risk to Allen Harbor. Two approaches were used to assess risk. The first involved calculation of

risk quotients as the ratio of contaminant-specific exposure concentrations to benchmark effect concentrations for single contaminants. In this process, field measurements of sediment and water column contaminant concentrations were compared with published measures of sediment and water quality. The second approach compared the results of all biological and chemical assessments conducted for Allen Harbor with those obtained for stations in Narragansett Bay proper. The intent behind this latter approach was to evaluate conditions in Allen Harbor within the context of the larger bay system as a whole. Such an evaluation might identify potential influences of the land-based hazardous waste sites on the ecology of Allen Harbor.

Risk quotients (RQs) calculated for Allen Harbor sediments ranged in magnitude from much less than 0.1, to as high as 47 for the maximum level observed of the pesticide DDT. Classes of contaminants were identified as falling into three levels of concern: those with quotients less than 0.1 (no risk presumed), those with RQs greater than 0.1 but less than 1 (moderate risk presumed), and those with RQs greater than 1 (risk presumed). Although the actual quotient values for specific contaminants varied with the particular ecological benchmark used, the major risk to benthic systems derived primarily from pesticides, PCBs, selected metals, and polycyclic aromatic hydrocarbons (PAHs). There was, however, no clear association of this risk with the land-based hazardous waste sites located at NCBC Davisville.

Based upon the small number of RQs calculated for Allen Harbor surface waters, the ecological risks associated with water-borne contaminants appeared to be minimal. This contrasted with the *Mytilus* SFG results observed during Effects Assessment activities. It may be that contaminants for which toxicological benchmarks do not exist, or which were not quantified in this study, played some role in reducing harbor water quality.

A more subjective, but equally useful approach to assessing ecological risks associated with the landfill and Calf Pasture Point was to compare the results of all assessment activities in Allen Harbor with those obtained for the bay stations. At a gross level, differences observed in such a comparison might reasonably be attributed to the unique association of Allen Harbor with the hazardous waste sites. Confounding this assessment were the other unique attributes of the harbor, such as its enclosed nature, and the high level of boating

activity present therein. At this level of analysis, there was a fairly strong indication that both sediment and water quality were impacted in Allen Harbor relative to the bay proper. However, other sites within the bay also appeared to be impacted to some degree. The causes of these suggested risks are not at all clear, as none of the sites exhibited untoward contamination.

Results obtained during Phase I suggested no major environmental problems unique to Allen Harbor, but did call into question some aspects of the quality of water column and sediment conditions. Most notably, mussels deployed in the harbor consistently exhibited reduced physiological condition relative to those exposed at other stations in Narragansett Bay. Impacts were observed on sea urchin early life stage processes and in biomarkers assays, and an increased incidence of hematopoietic neoplasia in *Mya* was associated with proximity to Allen Harbor. Appreciation of the meaning of the observed responses within an ecological context is confounded by the general lack of impact observed at higher levels of biological organization: *in situ* populations of benthic organisms seemed reasonably healthy with respect to those in other areas of the Bay.

PHASE II SUMMARY

The findings of Phase I were equivocal with respect to the degree of impact in Allen Harbor, and to the extent of ecological risks posed by the land-based waste sites associated with NCBC Davisville. Because some impact was observed, a modified version of Option II, as described above, was followed in developing the objectives and activities of Phase II (see Munns *et al.* 1993).

Although the results of Waste Characterization activities indicated the landfill to be a potential source of toxicologically important contaminants to the harbor, there was no clear association of observed impacts with proximity to the landfill. Of particular interest were the observations that contaminant exposures and biological effects were often most severe at the southern end of the harbor, farthest removed from the landfill and Calf Pasture Point. Other potential sources of contamination of the harbor were known to exist. For instance, the area immediately south of Allen Harbor currently is used as a staging area for automobiles off-

loaded from transport ships. Large areas of land are paved over with asphalt, serving as holding lots in preparation for over-land distribution. The community of Mount View, RI is located to the immediate north of the harbor, and contains a golf course in addition to residential housing. Surface water runoff from these areas was viewed as a potential contaminant source. Additionally, Allen Harbor supports an active marina for the Town of North Kingstown, RI on its eastern shore, and is a popular anchoring spot for day trips by local boaters. A second marina, serving the Quonset Davisville Yacht Club, is located on the southwest shore of the harbor. Fuel leakage, dispersion of hull antifoulant paints, and septic wastes resulting from this intense boating and marina activity were suspected potentially to impact harbor quality. To clarify the role of NCBC-associated waste sites on the observed impacts to Allen Harbor, activities in Phase II focused on partitioning contamination and toxicity among these three potential sources: the NCBC landfill, surface runoff from the surrounding land, and from boating and marina activities conducted within the harbor. The approach taken involved implementation of a temporal and spatial sampling plan which took advantage of the seasonal nature of boating activities in Allen Harbor. This was accomplished through collection of water, sediment, and biota samples, and subsequent quantification of contaminant levels and biological effects.

A second component of Phase II involved further examination of the hemopoietic neoplasia observed in Allen Harbor *Mya arenaria*. Because harbor *Mya* displayed high rates of Hn relative to Narragansett Bay stations, the possibility exists that Allen Harbor may be acting as a source of the disease. To address this question, a one-time survey of *Mya* neoplasia was conducted throughout the West Passage of Narragansett Bay. Samples of *Mya* were collected at 20 stations, and were scored in the laboratory for rate of infliction within each subpopulation. Additionally, research was conducted to identify chemical compounds which could potentially be used to identify and quantify sources of contaminant input to Allen Harbor. This effort involved a survey of existing inventories of chemicals disposed in the landfill, and analyses of selected sediment samples to evaluate potential input from sewage, runoff, atmospheric, and petroleum sources in addition to the landfill. The specific findings of these activities are described briefly below.

Exposure and Effects Partitioning

Environmental samples representing the three potential source inputs to Allen Harbor were collected prior to the onset of major boating activity in Spring, at the height of the boating season in Summer, and at the conclusion of the season in Fall. Replicate sampling stations were established to characterize each source: three at active seeps along the face of the Landfill, three in association with surface water Runoff (two at the mouths of major creeks and one at a storm drain), and two within the areas of significant boating and Marina activities in the harbor. Two additional stations in the West Passage of Narragansett Bay (one located at mid-bay, one in the southern part of the bay) provided Reference information. Samples were obtained of input water, sediments (as proximal receptors of water-bourne contaminant input), and biota to assess exposure conditions through chemical and microbial analyses, and to evaluate potential biological effects through performance of laboratory bioassays and biomonitoring activities. Data analyses focused upon comparisons among sources and seasons to address the central questions of Phase II, and among individual stations to identify targets of potential remediation.

Chemical analyses of water and receiving sediments indicated significant contributions of PCB, PAHs, pesticides, and trace metals by the NCBC Davisville landfill and surface water runoff sources. Although large variation in water-bourne contaminant levels confounded analyses of relative source strengths, the Spink Neck (SN) storm drain (located at the southern end of the harbor) and a seep located along the middle face of the landfill (LANDM) were identified as the major contributors. Volatile organic compound (VOC) concentrations were ubiquitously low throughout the study. Tissue chemistry of deployed *Mytilus edulis*, measured to evaluate Marina water column exposure conditions, indicated higher PCB and PAH levels in the harbor relative to Narragansett Bay reference stations, but lower concentrations of metals such as Cr and Pb. Typically, residues in both areas were highest in Spring, prior to the onset of intense boating activities. Patterns of tissue chemistry of native *Mercenaria*, another water column suspension feeder, reflected those observed in *Mytilus*.

Highest densities in water samples of fecal coliforms, indicators of sewage contamination and exposure to pathogens, were observed at the Runoff station North Creek

(NC). Levels also were elevated at West Creek (WC), but were relatively low at Marina and Reference stations. Peak concentrations typically occurred in Summer at all stations. These patterns were generally reflected in the concentrations of coliforms in indigenous and deployed bivalves: highest levels were observed in ribbed mussels (*Modiolus demissus*) collected from the Runoff stations NC and WC in Summer and Fall, and levels of indicator bacteria in *Mercenaria* and deployed *Mytilus* tended to be lower than those in ribbed mussels.

Statistical comparisons of receiving sediment chemistry indicated Runoff and Landfill sources to be the largest contributors of contaminant input, whereas concentrations associated with Marina stations were indistinguishable from Reference. SN and LANDM were again implicated as the major contributors of chemical stressors: PAHs and trace metals were highest at SN, while PCBs and the pesticide DDE were highest at LANDM. Sediment chemistry was correlated with that of the source water input for metals, but not for organic compounds. Tissue chemistry of indigenous *Modiolus* indicated overall higher levels of PCBs, DDT, and Cr at Landfill stations relative to animals from Runoff stations, although PAH concentrations were highest at SN. Contaminant residues were correlated with organic chemistry in sediments, but were statistically unrelated to trace metal sediment chemistry or to source water chemistry.

No overall differences in water toxicity, as measured by sea urchin fertilization success, were observed between Landfill and Runoff sources or among seasons. However, toxicity of water collected from SN was statistically higher than those of all other stations. LANDM samples also displayed some toxicity. Fairly strong negative correlations were observed between fertilization success and the concentrations of PAHs and metals in source waters. Marina waters caused statistically higher 7-day mortality in mysid shrimp (*Mysidopsis bahia*) laboratory exposures, while differences between sources in mysid reproduction and growth were lacking. Mortality was highest in Early Summer (an additional sampling event conducted immediately after onset of boating activities), but reproduction and growth were highest in Summer and Fall, respectively. As evaluated by these endpoints, Spring water quality was relatively poor: mortality was high, and reproduction and growth were low. No differences were observed in endpoints among

stations. Experimental field deployments of *Mysidopsis* were attempted in during all four seasons, but were successful in Summer and Fall only. No source or seasonal differences were observed in mortality, but both reproduction and growth were higher at Marina stations than at Reference stations. In all seasons except Early Summer (when no differences occurred), the SFG index of *Mytilus* was lower for mussels deployed at Marina stations. Reduced SFG in Spring suggested deterioration of harbor water quality prior to the boating season. SFG was negatively correlated with *Mytilus* PCB, PAH, DDT, and Cu tissue residues.

Statistically significant source and seasonal effects were absent in the acute mortality response of *Ampelisca*, although Spring mortality tended to be lower at most stations. Biologically significant rates of mortality were observed at LANDM, WC, and SN in Summer and Fall. Toxicity was correlated with sediment concentrations of DDE, CU, Ni, Pb, and Zn.

The assessments conducted during Phase II strongly implicated the NCBC landfill and surface water runoff as important contributors to contaminant exposure and biological effects in Allen Harbor. The design utilized in that phase did not permit evaluation of the absolute magnitude of these contributions, nor of their relative importance. However, seasonal and spatial patterns in exposure and effects associated with boating and marina activities suggested this source to be relatively unimportant to the environmental quality of Allen Harbor.

Mya Neoplasia Survey

A semi-synoptic survey of neoplastic disease in *Mya arenaria* was conducted at 20 stations in Allen Harbor and the West Passage of Narragansett Bay during the spring of 1990 to provide correlative information regarding the role of NCBC contaminant sources in Hn etiology. Of 820 animals examined, 91 contained neoplasms. Average incidences of Hn varied among stations from 0% (at several stations) to 37.9% at a station within Allen Harbor. Overall, the rate of Hn affliction was related to proximity to the NCBC landfill, indicating a possible association between *Mya* neoplasia and release of contaminants from this

site.

Chemical Markers Research

A review of past disposal practices and of the types of materials disposed at Calf Pasture Point indicated the hydrolyzation product of 1,3-dichloro-5,5-dimethylhydatoin to be a potential chemical marker useful in assessing the contributions of Calf Pasture Point to environmental contamination in Allen Harbor. The parent compound, referred to as Decontaminating Agent Non-Corrosive (DANC), was used by Naval Air Station (NAS) Quonset Point while the base was in operation repairing helicopters. H-DANC, produced when DANC is exposed to water, was quantified along with chemical markers of sewage, runoff, atmosphere, and petroleum source inputs in five selected sediments from Allen Harbor. The results of these analyses were used to evaluate the utility of the chemical approach to partitioning source contributions of chemical stressors.

The levels of marker compounds quantified in sediments indicated several potential origins of contamination to the harbor, including sewage, petroleum, and atmospheric sources. The pattern of sewage markers suggested direct input of fecal material, rather than input from municipal treatment facilities. Petroleum marker relationships implicated both direct introduction of high molecular weight petroleum mixtures (such as crankcase oil), and indirect input from pyrogenic sources. Atmospheric sources were implicated by the ratios of specific PAHs. H-DANC was not detected in any of the five samples analyzed, nor was a marker of roadway runoff. The absence of these two compounds should not be interpreted to suggest that Calf Pasture Point and surface runoff are insignificant contaminant sources, as chemical and/or biological degradation may have reduced their concentrations to levels below detection.

PHASE III APPROACH

The results obtained in Phases I and II indicated some degree of risk to ecological systems in Allen Harbor from chemical contaminants in the NCBC landfill. Phase III activities focused upon the direct quantification of this risk. Additional activities were

conducted to define the role of landfill contaminants in the etiology of hematopoietic neoplasia in Allen Harbor *Mya arenaria*. In conjunction with Phases I and II, this work provides the information necessary to support characterization of ecological risks associated with NCBC Davisville.

The approach established for quantification of ecological risk relied upon characterization of the responses of a number of benthic and water column species to direct exposure to landfill material. This effort consisted of the performance of laboratory exposure-response bioassays involving landfill seep water, sediments, and sediment extracts collected in close association with the landfill. The bioassays examined a variety of acute and chronic endpoints of survival, growth, and reproduction. Assay results were summarized into exposure-response models describing biological impact at any level of contamination. This approach is outlined in Norton *et al.* (1988).

The rationale for selecting seep water, whole sediment, and sediment extracts as surrogates for the landfill itself involved the feasibility of obtaining appropriate exposure media, as well as the validity of assays of landfill material with respect to marine systems. Previous reconnaissance activities conducted by TRC Environmental Consultants, the on-site contractor for the NCBC Davisville RI/FS process, indicated a low potential for collection of ground water within the landfill. Seeps were therefore selected as sources of water to obtain volumes sufficient for bioassay conduct. This medium likely represents the most immediate route of contaminant transport into Allen Harbor. Whereas EPA's Superfund Program has cautioned against the use of exposure media inappropriate to the species used in toxicity bioassays (Charters 1990), sediments were selected over landfill soils to examine risks to the marine system of the harbor. Utilization of sediment extracts permitted characterization of the effects of contaminants at all concentrations potentially available to biological systems.

Assays were selected for inclusion in Phase III based upon the following (unordered) criteria:

- involvement of species representative of the Allen Harbor benthic and water column systems,
- involvement of a range of taxonomic groups,

- involvement of endpoints addressing a range of ecological organization,
- potential for extrapolation to higher-level endpoints,
- ecological relevance of resulting data,
- suitability to quantifying effects to the selected exposure media,
- suitability to quantifying effects across exposure media,
- relevance of resulting data to results obtained in the previous two phases of the study, and
- feasibility of and familiarity with exposure-response assay protocols.

The species and endpoints of assays meeting these criteria and therefore utilized in Phase III are indicated in Table 1. For the most part, these assays were developed at ERLN to evaluate toxicity and effects of exposure media from marine and estuarine settings, and their utility has been validated in a variety of laboratory and field programs. ERLN Standard Operating Procedures have been developed for each assay and can be found in Mueller *et al.* (1992).

The second component of Phase III involved laboratory investigation into the etiology of soft-shell clam (*Mya arenaria*) hematopoietic neoplasia. High incidences of this disease, a disseminated sarcoma occurring in bivalves, were first observed by Farley (1969) in eastern and Pacific oysters. Since this time these malignant neoplasms, similar in nature to vertebrate leukemia, have been documented worldwide in 15 species of oysters, clams, cockles, and mussels (Peters 1988). During Hn, normal circulating hemocytes are replaced by round, non-aggregating, anaplastic cells which have lost their ability to adhere to glass, to form pseudopods, and to phagocytize and neutralize foreign particles (Beckmann 1989). They have a large nuclear to cytoplasmic ratio, a distinct nucleolus, and a high mitotic index with abnormal figures. As Hn progresses the number of aberrant cells increases, invading and destroying the soft tissues of the clam and leading eventually to death.

While more recent field surveys such as those conducted by Farley *et al.* (1986) have

indicated an increase in the prevalence of Hn, researchers have been unable to determine the etiology of this disease. It has been attributed to infectious agents, such as viruses, to pollution, and to transmission of neoplastic cells from introduced or transplanted affected organisms to healthy indigenous populations. Brown (1980), Appeldoorn *et al.* (1984), and Farley (1989) have successfully conducted transmission studies by holding healthy animals under head tanks containing neoplastic clams and through the inoculation of neoplastic cells into healthy animals. However, attempts to confirm the presence of a virus, originally isolated by Oprandy *et al.* (1981), have failed, as have several attempts to correlate Hn with environmental pollution (Mix 1979, 1986).

Table 1. Species and endpoints used in development of exposure-response models.

Exposure Medium	Species	Endpoints
Seep water	*Arbacia* (sea urchin)	fertilization larval development larval mortality
	Mysidopsis (mysid shrimp)	mortality
	Champia (red alga)	reproduction
	Mulinia (coot clam)	larval development
	Menidia (silverside minnow)	mortality
Sediment	*Ampelisca* (benthic amphipod)	mortality
	Mulinia (coot clam)	growth mortality
Sediment extract	*Arbacia* (sea urchin)	fertilization larval development larval mortality
	Mulinia (coot clam)	mortality
	Photobacterium (bacterium)	mortality

Hn was reported in Rhode Island soft-shell clams as long ago as 1976 (Brown *et al.* 1976, 1977) and again by Cooper *et al.* (1982a, 1982b). In Allen Harbor, incidences of Hn

as high as 23% have been documented (Munns *et al.* 1993). A single long-term laboratory study was conducted during this phase to determine the potential role of landfill contaminants in Hn etiology. Hn-free *Mya* were injected with hemolymph from non-affected and affected animals and exposed in the laboratory to either landfill or reference sediments for 90 days. Animals were examined for mortality, growth, and for the presence of Hn sarcoma cells. This experiment would provide information sufficient to assess risks to Allen Harbor clams of neoplasia development.

Phase III of the Risk Assessment Pilot Study began in October 1990. ERLN was the lead laboratory in this study with the cooperation and participation of NCCOSC. The remainder of this document describes the activities performed to address Phase III objectives and the results obtained through their conduct.

METHODS

The material in this section provides a description of methods used to address the objectives of Phase III. The two major activities in this phase, the establishment of exposure-response relationships and the evaluation of the role of landfill contaminants in development of *Mya* Hn are described separately. Because quality assurance/quality control procedures are common to all activities, their general description is given under a separate heading.

QUALITY ASSURANCE/QUALITY CONTROL

This project has been conducted in accordance with all ERLN quality assurance and quality control procedures outlined in the *Work/Quality Assurance Plan for Marine Ecological Risk Assessment Pilot Study* (ERLN/NOSC 1991), the *ERLN Standard Operating Procedures Manual* (ERLN 1991), the *Standard Operating Procedures and Field Methods Used for Conducting Ecological Risk Assessment Case Studies at: Naval Construction Battalion Center Davisville, RI and Naval Shipyard Portsmouth, Kittery, ME* (Mueller *et al.* 1992), and the *Quality Assurance Program Plan for the Environmental Research Laboratory -- Narragansett and Newport* (ERLN 1992). The first document addresses quality assurance steps undertaken for the specific activities of this project. The two Standard Operating Procedures (SOP) manuals describe the methods used to perform the biological, physical, and chemical assessments of this project. The last document describes general quality assurance requirements for research activities at ERLN. A copy of these documents have been added to the administrative record for NCBC Davisville, and may be obtained by contacting ERLN or NCBC Davisville.

All data generated during sample collection, preparation (*e.g.*, dry weight, wet weight, volume, *etc.*), and analysis were entered into computerized data bases for use in subsequent data reduction and statistical analysis. A description of the data management plan for this project is given in Rosen *et al.* (1988). In addition to describing quality

assurance/quality control (QA/QC) considerations with respect to data storage, transfer, and manipulation, this document provides a description of data base design and its relationship to the interdisciplinary data management strategy of ERLN. This document is also part of the administrative record, and may be obtained by contacting ERLN or NCBC Davisville.

A large portion of the QA/QC procedures used for this study were specific to each type of activity. For example, calibration of specific instrumentation is relevant only to the operation of that instrument. These procedures are described in the two SOP manuals cited above. However, important QA/QC descriptions are given where appropriate throughout the remainder of this Methods Section.

EXPOSURE-RESPONSE MODELS

Bioassays were performed to evaluate the effects of landfill exposure media on marine organisms and to establish exposure-response relationships for development of an ecological risk assessment model. Water emanating from landfill seeps, sediments associated with the landfill, and extracts of sediments associated with the landfill were used in the laboratory as exposure media. Prior to sample collection, data from Phases I and II were reviewed to select a site of maximum contamination. This was necessary to ensure the full range of exposure concentrations required to adequately describe the responses of test organisms to landfill contaminants. LANDM, located at a seep in the middle of the face of the landfill (see Munns *et al.* 1991, 1993 for descriptions of stations associated with the NCBC landfill), was chosen as the site for exposure media collection.

The basic protocol for the laboratory assays required serial dilutions of the landfill material with appropriate (*i.e.*, relatively uncontaminated) reference materials. Contaminant bioavailability in sediments has been shown to be influenced by such sediment attributes as total organic carbon (TOC) (*e.g.*, Di Toro *et al.* 1991), acid volatile sulfide (AVS) (*e.g.*, Di Toro *et al.* 1990), and grain size, which is related to the particle surface area available for contaminant sorption. Thus the diluting sediment for solid phase exposures needed to match LANDM in these attributes. Several sediments from relatively clean areas of Narragansett Bay and from Central Long Island Sound (CLIS) were evaluated for TOC, AVS, and grain

size. ASTM methods (1988) were adapted by Huffman Laboratories, Inc. (Golden, CO) for sediment TOC determinations. Total carbon concentrations were quantified by high temperature combustion of 0.1 to 1.0 g of sediment in a model CR12 Analyzer. Carbonate carbon was measured with a Coulometric 14D instrument as carbon dioxide. AVS in LANDM and potential diluting sediments were evaluated according to methods described in Boothman and Helmstetter (1993) and Johnston (1993). Volatile hydrogen sulfide released by HCl from aliquots of homogenized sediment samples was trapped with a sulfide anti-oxidant buffer solution and evolved S^{2-} was quantified with a sulfide ion-specific electrode. Sediment grain size was determined by a sieve and centrifuge method as described in ERLN SOP 1.01.005 (Mueller *et al.* 1992). A hydrogen peroxide solution was added to 12 g of dried sediment. The sample was sonicated, washed through a sieve, and centrifuged. The supernatant was decanted, distilled water was added, the silt plus clay fraction was resuspended and centrifuged several times to remove all clay particles. The remaining silt fraction was dried and weighed. The proportion of clay was determined by subtracting the weights of the sand and silt fractions from the weight of the total sample. Based on these analyses (Table 2), sediment from Potowomut Cove (POTO), a small riverine inlet to the north of Allen Harbor, was selected as the diluting sediment.

Table 2. Characteristics of sediments used in bioassays.

	LANDM	POTO
AVS (μM/g)	51.00	53.64
TOC (%)	3.74	3.16
Sand (%)	92.7	47.7
Silt (%)	4.0	40.4
Clay (%)	3.3	11.9

Exposure Media Collection and Preparation

Seep samples were collected from LANDM directly into Nalgene® bottles, transported on ice in insulated coolers, and refrigerated at 4°C until used. Serial dilutions of 5.8, 11.5, 23, 46, and 92% LANDM seep water were constructed with Narragansett Bay brine and deionized water prepared according to procedures described in ERLN SOP 1.01.004

(Mueller *et al.* 1992). Surficial sediments were collected at LANDM and at POTO with a large teflon coated spatula, transported in acid-stripped 2-g glass jars on ice, and refrigerated at 4°C until used. Serial dilutions of 12.5, 25, 50, and 100% LANDM sediments were constructed with POTO sediment on a volume basis. Following homogenization, these mixtures were allowed to "age" for 30 d under refrigeration at 4°C to permit equilibration of contaminants among sediment surfaces.

Extracts of LANDM sediment for use in laboratory exposure-response assays were prepared by sonicating samples (approximately 300 g wet weight) in acetonitrile and centrifuging three times. The supernatants were combined in pentane-extracted deionized water and back-extracted three times with pentane. The extracts were combined, dried over sodium sulfate, reduced twice in pentane, brought to dryness, and dissolved in dimethylsulfoxide (DMSO). Detailed methods for preparing sediment extracts are described in Munns *et al.* (1991) and Mueller *et al.* (1992). Final exposure concentrations of 0.001, 0.003, 0.006, 0.010, 0.013, 0.025, 0.05, 0.10, 0.15, 0.20, and 0.25% were created by diluting the original extract with reconstituted Narragansett seawater.

Laboratory Assays

The exposure media (prepared as described above) were evaluated over a range of concentrations in the laboratory bioassays indicated in Table 1. Detailed descriptions of the standard methods employed in each of these are given in Mueller *et al.* (1992). These tests are described briefly below, with references made to the corresponding SOP.

Seep water -- The effects of contaminants currently migrating from the landfill in seep water on sea urchin (*Arbacia punctulata*) fertilization were evaluated following ERLN SOP 1.03.005. In this test, gametes obtained from adults collected at reference field sites were artificially released in response to electrical stimulation and collected using a syringe with a blunted needle. One milliliter of eggs (2000/ml $\pm$ 200) was added for 20 minutes to 100 μl of a 5 X 10^7 cell/ml suspension of sperm which had previously been exposed to 5 ml of seep water dilutions in scintillation vials for 1 h at 20°C. Two ml of 10% formalin in seawater

were added at the conclusion of the test, and 100 eggs were observed by compound microscope (100X) in a Sedgwick-Rafter counting chamber for the presence of a membrane surrounding the egg, an indication of successful fertilization.

Seep water effects on *Arbacia* larval development and survival followed procedures given in ERLN SOP 1.03.007. Gametes were obtained and diluted as described above, and eggs and sperm were co-exposed for 48 h in 10 ml of seep water dilutions in scintillation vials at 20°C. Larvae were preserved by the addition of 2 ml of 10% formalin in seawater and were stained for microscopic observation with Rose Bengal in a 10% buffered formalin/seawater solution. Two hundred larvae were examined for development and mortality.

During the *Mysidopsis bahia* acute toxicity test (ERLN SOP 1.03.003), ten 1-5 day-old mysid shrimp (cultured according to methods described in ERLN SOP 1.01.003) were exposed at 20°C in two replicates of 200 ml of seep water dilutions for 48 h. Non-motile, opaque organisms were recorded at assay termination.

The effects of seep water on red algae (*Champia parvula*) reproduction were evaluated following ERLN SOP 1.03.001. One male and 5 female *Champia* branches, cultured in the laboratory as described in ERLN SOP 1.03.001, were exposed in 250 ml Erlenmeyer flasks to 100 ml seep water dilutions for 2 days at 23°C. After a 5-7 day recovery period, the number of mature cystocarps produced by each female was enumerated by stereomicroscopy.

During the *Mulinia* embryo-larval toxicity test, field-collected adults maintained in the laboratory were cooled to 4°C for 0.5-2 h and warmed to 25-28°C to induce spawning (ERLN SOP 1.03.008). Seven hundred and fifty embryos were exposed to 3 replicates of 10 ml of seep water dilutions in scintillation vials no more than 2 h after fertilization for 48 h at 22°C. Formalin preserved larvae were examined in Sedgwick-Rafter counting chambers by compound microscope (100X) for the presence of shells, an indication of normal development and therefore survival.

Survival effects on silverside minnow (*Menidia beryllina*) were determined for ten 7-11 day old *Menidia* larvae cultured according to methods described in ERLN SOP 1.01.003,

exposed at 20°C in 2 replicate 250 ml chambers for 96 h containing 200 ml seep water dilutions. Non-motile, opaque animals were recorded as dead (ERLN SOP 1.03.004).

Sediment -- Toxicity of sediments associated with the landfill was assessed to evaluate contaminant migration which may have occurred in the recent p[illegible] The 10-day amphipod (*Ampelisca abdita*) acute mortality tests was performed following ERLN SOP 1.03.002. Briefly, twenty immature amphipods collected from a nearby reference site were exposed in 1-quart jars to 200 ml dilutions of LANDM sediment for 10 days at 20°C. Each jar was monitored daily and at test termination for dead or moribund organisms.

Evaluation of sediment effects on *Mulinia* growth and mortality followed procedures described in Burgess and Morrison (in prep.). Survival and weight was measured in 10 1-mm laboratory-cultured *Mulinia* juveniles obtained from artificially spawned field-collected adults. Juveniles were exposed to 50 g of the landfill sediment dilutions in 150 ml beakers for seven days at 22°C. Mortality was quantified by the presence of open shells and through microscopic observation for bacteria infestation. Whole animal dry weight was calculated and compared to control or reference whole animal dry weight.

Sediment extracts -- To ensure a full range of contaminant concentrations for development of the exposure-response models, extracts of LANDM sediment were evaluated using four standard methods. In addition to effects on *Arbacia* fertilization, *Arbacia* larval development and survival, and *Mulinia* mortality (described above), assessments of sediment extract were conducted using Microtox® (*Photobacterium phosphoreum*) mortality as an endpoint. Microtox methods are described in detail in ERLN SOP 1.03.009. Briefly, suspensions containing 10^6 colony-forming units of the luminescent bacterium *Photobacterium phosphoreum* were exposed to dilutions of sediment extracts for 15 min at 15°C. Bioluminescence was monitored using a model 2055 Microtox Toxicity Analyzer (Beckman Instruments).

Model Development

Data obtained from laboratory assays were used to develop quantitative models of biological response to landfill media exposure. These exposure-response models utilize whole-waste concentrations as independent variables determining the level of endpoint response for each test species.

The model identified to describe the exposure-response relationships is based upon the assumption that thresholds exist in the sensitivities of organisms to contaminant concentration, and that due in part to inter-individual variability, these thresholds are log-normally distributed within the test population as a function of exposure concentration. Thus individuals may respond at different exposure concentrations and the overall test population's response to varying exposure concentrations can be modeled as a log-normal distribution (or as a Gaussian (normal) distribution if concentrations are log-transformed). This model describes an S-shaped logistic curve, the pattern classically observed in dose-response relationships. This pattern of response was observed in several of the assays described above (see Results). The approach taken here offers advantages over other logistic curve fitting procedures (*e.g.*, Barnthouse *et al.* 1987, Munns and Comeleo 1991), including its mechanistic theoretical basis, and that data obtained from reference (background or control) treatments can be incorporated directly into the model fitting procedure.

A nonlinear least-squares regression procedure described by Bruce and Versteeg (1992) was used to estimate parameters of the model:

$$R = \begin{cases} R_0\,\Phi[(\log(EC_x)-\log(C))/\sigma+Z_x] & C>0 \\ R_0 & C=0 \end{cases}$$

where R is the predicted biological response at exposure concentration C, R_0 is the biological response observed in reference or control treatments, Φ is the cumulative area under the standard Gaussian distribution, EC_x is the x^{th} percentile effects concentration, Z_x is the normal deviate above which x percent of the Gaussian distribution lies, and σ is the standard deviation of the Gaussian distribution. EC_x and σ can be thought of as parameters describing the position and slope of the cumulative Gaussian distribution, whereas R_0 describes the level

of response expected in the absence of contaminant exposure (*i.e.*, the intercept).

Assay data sets needed to meet two criteria of acceptance to be successfully modeled. The first of these was that the responses had to increase or decrease reasonably monotonically with increasing exposure concentration. Large deviations from this requirement would suggest the lack of cause-and-effect relationship between the two variables, and further would yield a poorly fitting model of little value for quantifying risk. The second criterion was that $\geq 50\%$ of the full response range needed to be present in the data set. A realized range of response less than this would not only suggest the lack of sufficiently high exposure concentrations, but also would yield unreliable estimates of the parameters EC_x and σ. Data sets failing this criterion also would yield a poorly fitting model of little value for quantifying risk.

The NLIN Procedure in SAS® (SAS Institute 1989) was used to determine estimates of R_0, EC_x, and σ resulting in the best description of each of the data sets meeting the above acceptance criteria. The resulting models could then be used to predict the toxicological responses of test organisms to whole-waste exposure.

MYA LABORATORY EXPOSURES

The experimental design employed in the laboratory assessment of the role of the NCBC Davisville landfill in neoplasia development involved a two-way design incorporating *Mya* exposed to landfill sediment or reference sediment, and the presence or absence of a suspected transmissible component. This design required injection of previously unaffected animals with hemolymph obtained from animals displaying the disease. Results obtained during Phases I and II indicated the FDA station in Allen Harbor to be a source of affected *Mya*. Clams were obtained from intertidal zone of this site at low tide with clam forks and garden rakes. One tenth of one milliliter of hemolymph was drawn from the posterior adductor muscle of each animal into a 1-cc tuberculin syringe with a 26-gauge needle and diagnosed by phase-contrast microscopy of hemolymph hemocytometer preparations. Affected hemolymph was injected into the siphon (Farley 1989) of disease-free 1-3 year old clams (evaluated as above) obtained from a commercial supplier for treatments involving the

presence of a transmissible component. The remaining clams received a placebo injection of disease-free hemolymph.

Inoculated *Mya* were planted in 10-gal aquaria containing 4 gal of sediment obtained from either the landfill (AH) or at a reference station in Narrow River (PR). Neoplasia was not observed at PR during the Phase II *Mya* survey (Munns *et al.* 1993), nor has it ever been reported at that site. Surficial sediment (top 2 cm) had been collected previously from the intertidal zones of these stations at low tide with a teflon-coated scoop, and was kept under refrigeration at 4°C until used. Each of the four treatments was replicated twice, for a total of eight aquaria. Following introduction of the inoculated animals (60/aquarium), aquaria were supplied with ambient temperature seawater flowing at 0.1 L/min. Animals were monitored for mortality and fed a suspension of the alga *Isochrysis* cultured as described in ERLN SOP 1.03.013 (Mueller *et al.* 1992) daily.

Fixed hemolymph cells were examined by bright-field microscopy every 30 days for 3 months according to the histocytological methods described by Farley *et al.* (1986) with several modifications. Briefly, cells were allowed to settle on 1% poly-L-lysine coated standard microscope slides for 30 min. Excess fluid was removed, slides were fixed in a 1% glutaraldehyde and 4% formaldehyde seawater solution, and stained with a standard histological Pap preparation. Pre- and post-exposure weights were measured to evaluate growth. Two-way analysis of variance (ANOVA) was conducted to determine the effects of sarcoma cell inoculations and contaminated sediments on Hn, mortality, and growth.

CHEMICAL ANALYSES

Analyses were performed to characterize organic and inorganic contaminants in sediment, water, and *Mya* tissue samples. These procedures are described here briefly and in detail in Munns *et al.* (1991). Seep water was collected in solvent-rinsed 1-L amber bottles for organic chemical analysis and in acid-washed 250-ml polyethylene bottles for trace metal analysis. This material was transported to ERLN on ice and refrigerated at 4°C until used. Organic contaminants were extracted from water samples with methylene chloride,

dried over anhydrous sodium sulfate, volume reduced, and separated into two fractions by silicic acid column chromatography. The f_1 fraction was analyzed for PCBs and pesticides using capillary gas chromatography (GC), and the f_2 fraction was analyzed for pesticides and PAHs using GC with electron capture detection (ECD) and gas chromatograph-mass spectrometry (GC-MS). For inorganic analyses, water samples were acidified with nitric acid, shaken, allowed to settle, subsampled into concentrated nitric acid, and analyzed by heated graphite atomization (HGA) atomic absorption (AA).

Sediments were homogenized, sonicated with acetonitrile, and centrifuged three times for organic analyses. The supernatants were combined and extracted with pentane. The extract was volume reduced and separated into the three fractions for quantification as described above. Homogenized sediments were freeze-dried, acidified with nitric acid, sonicated, and centrifuged for trace metal analyses. The supernatant was decanted, sonicated with nitric acid, centrifuged, and decanted. The resulting supernatant was analyzed on an inductively coupled plasma (ICP) emission spectrometer. Samples which required low detection limits were analyzed by AA as described above for water samples.

Mya were collected as described above, and were frozen at -20°C in plastic bags for chemical analyses. Homogenized tissue samples were rehomogenized with acetonitrile for organic analyses. The sample was centrifuged and decanted into deionized water three times. The supernatants were combined, extracted with pentane, and volume reduced. Silica gel chromatography was used to obtain three fractions for quantification as described above for water and sediment samples. Homogenized, freeze-dried, heated tissue samples were microwave digested in nitric acid, cooled, and vacuum-filtered for inorganic analyses. The filtrates were diluted with deionized water and analyzed by ICP (or AA, if necessary).

RESULTS

EXPOSURE-RESPONSE MODELS

Laboratory Assays

The presentation given below focuses upon the sensitivities of individual endpoint responses evaluated over the full range of exposure media concentrations used in each bioassay, with attention given to the utility of individual data sets in constructing exposure-response models. Comparisons of species' sensitivities are made where appropriate. Results are organized by exposure medium (seep water, sediment, or sediment extract). All bioassay data are given in Appendices H-J.

Seep Water -- The fertilization response of *Arbacia* exposed to landfill seep water decreased monotonically from near 100% in reconstituted Narragansett seawater to approximately 47% at the maximum concentration of 92% LANDM seep water (Figure 2), indicating that levels of contaminants in near full strength seep water were not sufficient to totally prevent successful fertilization. Exposure concentrations below 23% seep water had little effect on this endpoint.

Relatively little response to seep water was observed in *Arbacia* larval mortality: survival was near 80% at the maximum seep water concentration tested (Figure 3). However, developmental effects in this assay were measured at very low concentrations. The combination of larval mortality and abnormal development resulted in the near absence of normally developing larvae at concentrations of 46% seep water and above.

Seep water exposures were not acutely toxic to *Mysidopsis*, as measured by mortality (Figure 4). With exceptions of minor mortality ($\leq$10%) at concentrations of 0 and 23%, no survival effects were measured through the full range of seep water concentrations.

Sexual reproduction in *Champia* was affected by seep water concentrations as low as 5.8%, and the number of cystocarps produced by each female decreased monotonically thereafter (Figure 5). Little to no reproduction was observed at concentrations of 46% and

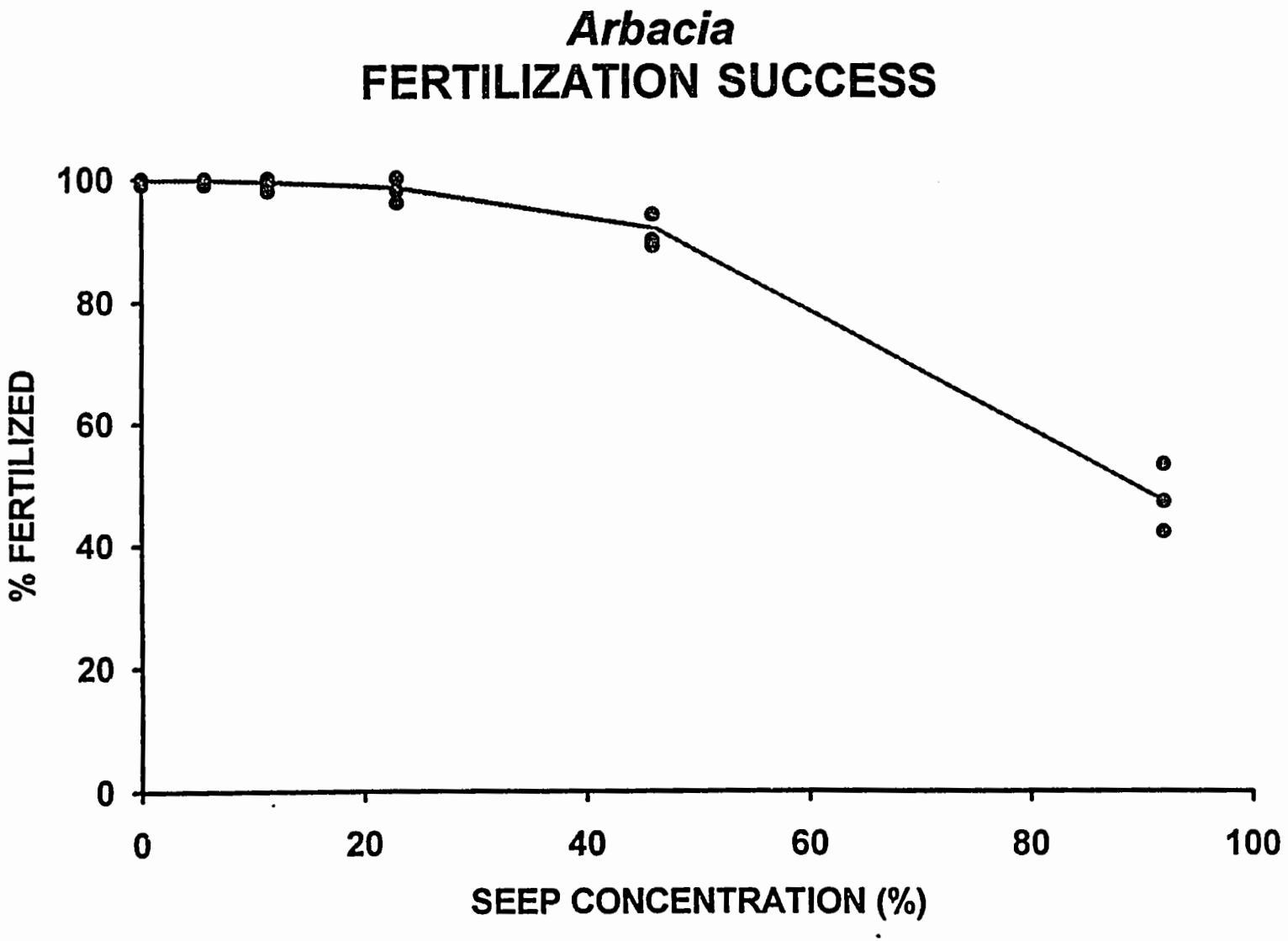

Figure 2. Fertilization Response of *Arbacia* to seep water exposure.

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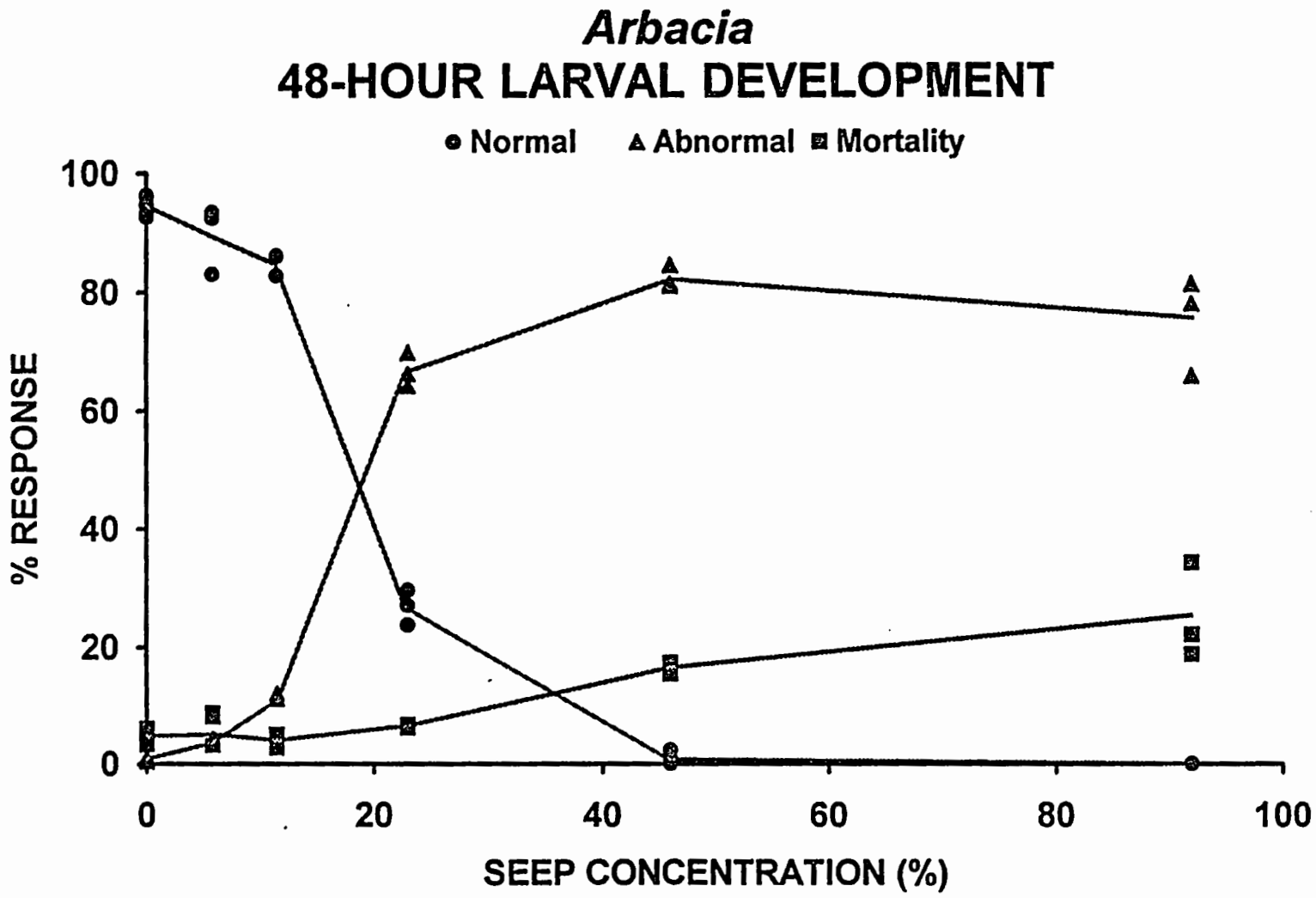

Figure 3. Larval mortality and development responses of *Arbacia* to seep water exposure.

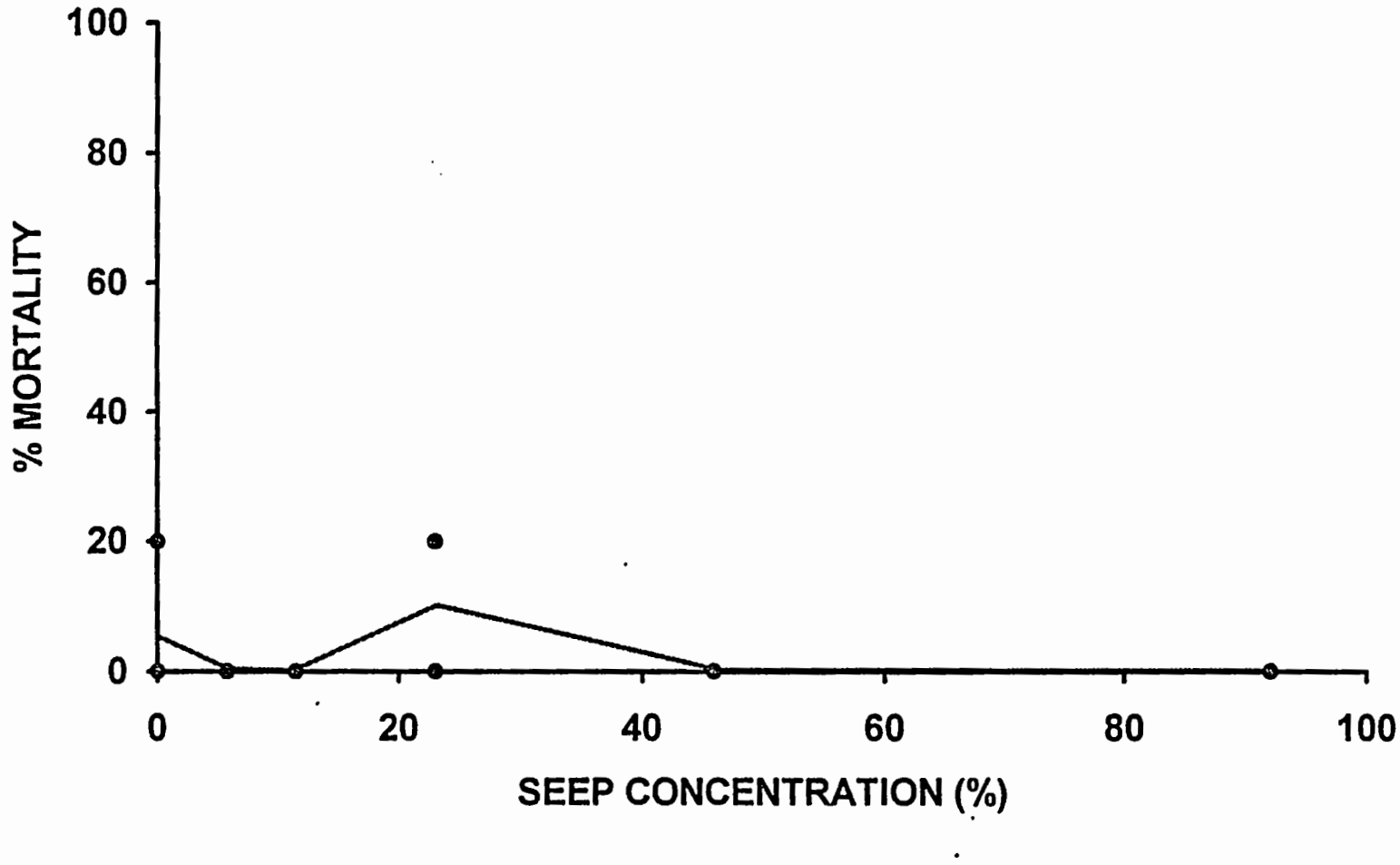

Figure 4. Acute mortality response of *Mysidopsis* to seep water exposure.

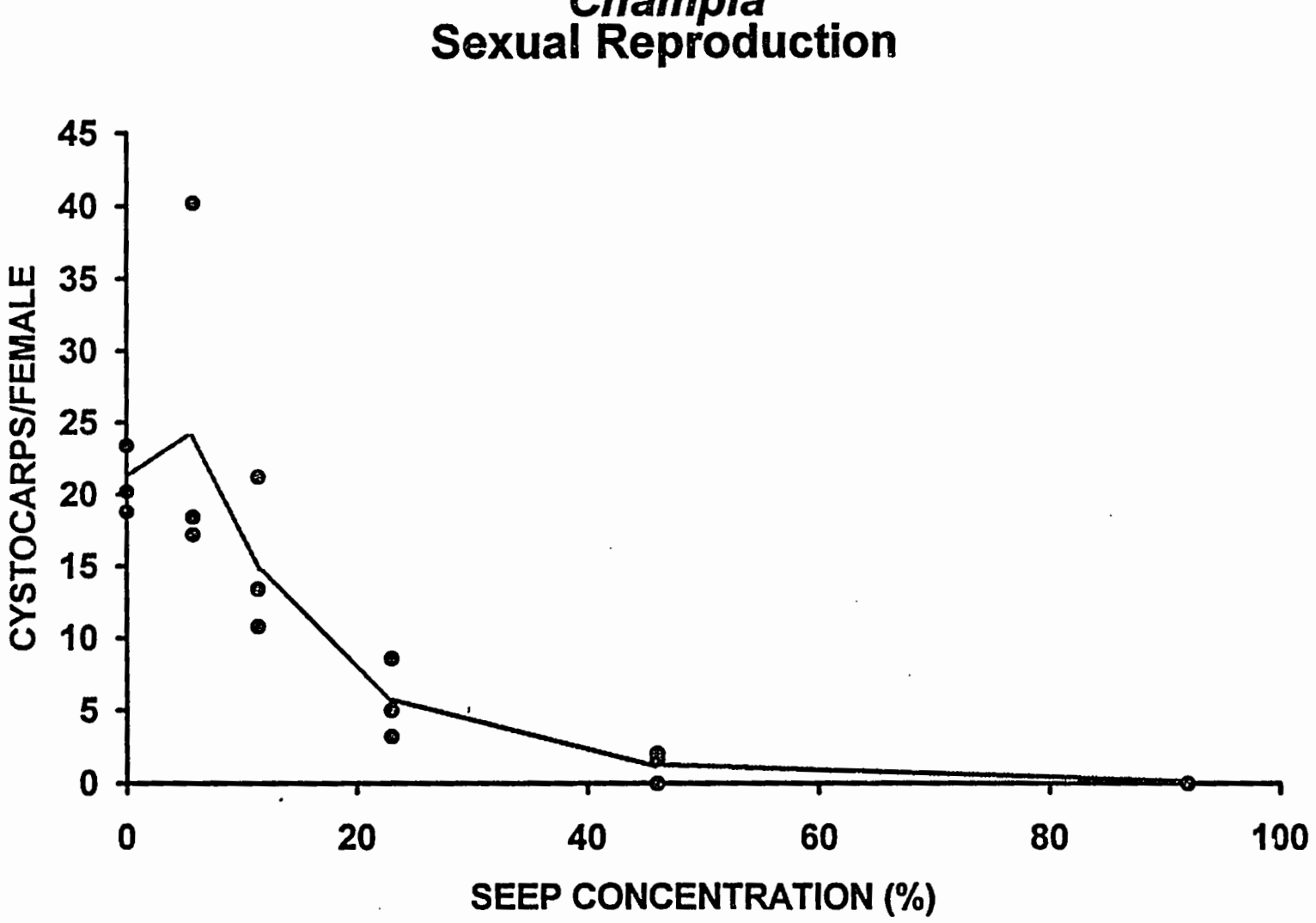

Figure 5. Sexual reproduction response of *Champia* to seep water exposure.

above.

Development of larval *Mulinia* shells, indicative of normal development and therefore larval survival, was impacted by seep water concentrations of 23% and greater (Figure 6). Apparent effects below this level (Figure 6) were not different from those experienced in the reference Narragansett seawater treatment in this test (see Appendix H).

As with the mortality response of mysid shrimp, survival of *Menidia* larvae was unaffected by seep water concentration (Figure 7). The apparent reduction in survival at low concentrations was evidently unrelated to contaminant concentrations in exposure medium, as no mortality was observed at concentrations higher than 11.5%.

In a comparison among species and endpoints, *Arbacia* development, *Champia* reproduction, and *Mulinia* larval shell development were most sensitive to seep water exposures. Acute mortality of *Mysidopsis* and *Menidia* larvae were not affected, showing no response at the maximum exposure concentration and suggesting that seep water was not acutely toxic to either species. Under the conditions of exposure used in these latter two assays, insufficient information was obtained for development of exposure-response models. Sensitivities of the *Arbacia* larval mortality and fertilization responses fell between these two extremes, with less-than-total effects being observed at maximum seep water concentrations. Although models of response to seep water exposure could be constructed for *Arbacia* mortality and fertilization, they would be incomplete with respect to risks associated with higher constitute contaminant levels than those existing in the LANDM sample used in these assays.

Sediment -- An initial 10-day *Ampelisca* acute mortality assay was repeated after the first test failed established QA criteria (reference mortality of greater than 10%). However, mortality in this first test was equivalent between the 0 and 100% LANDM treatments. A second test of these extreme concentrations was conducted in conjunction with an assay being performed as part of a separate project, in which mortality in the 100% POTO treatment and in the 100% LANDM treatment were both less than 10%. Thus, amphipod mortality was insensitive to exposure to landfill sediments in this assay, suggesting the lack of acute

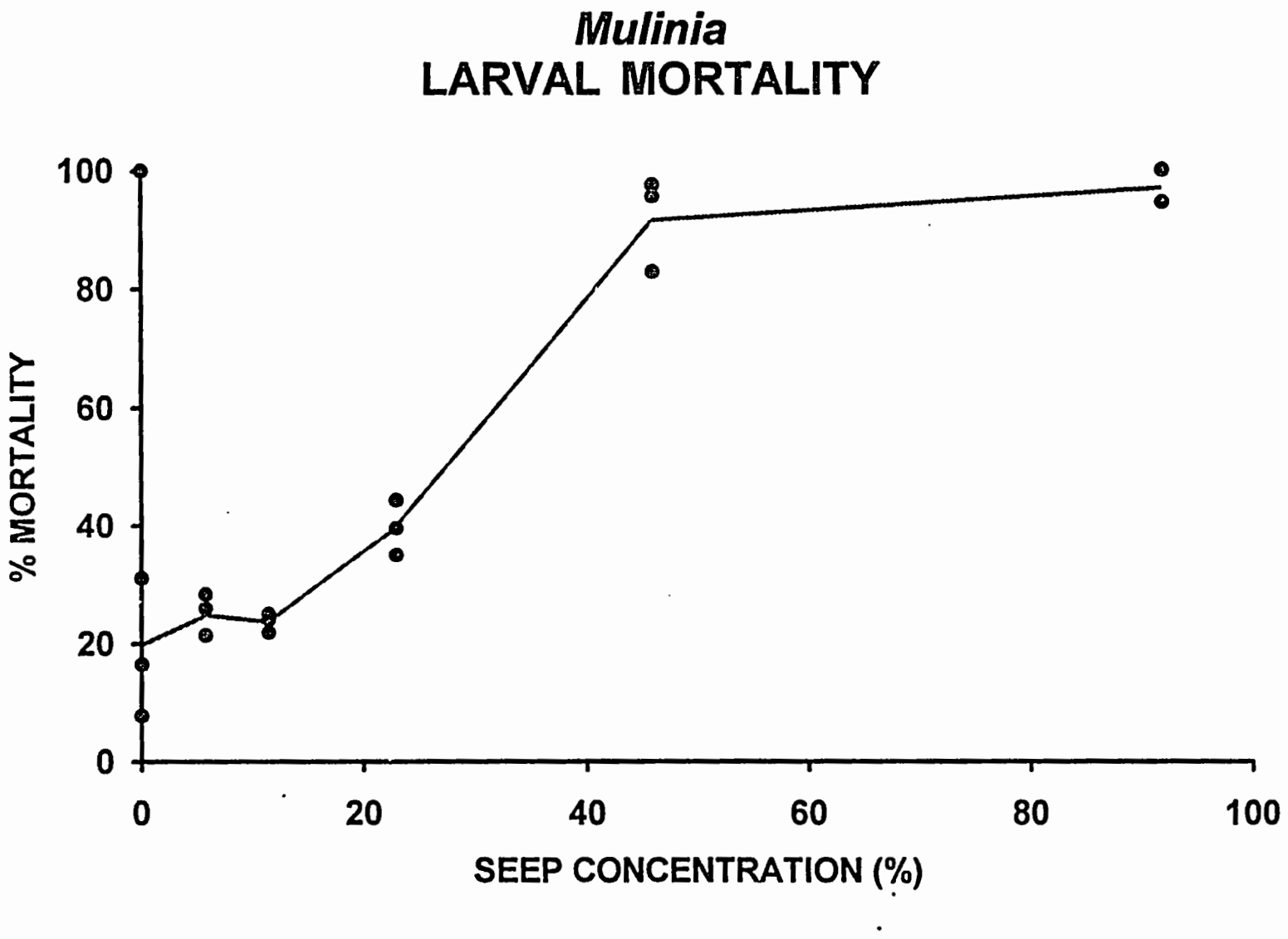

Figure 6. Embryo/larval toxicity response of *Mulinia* to seep water exposure.

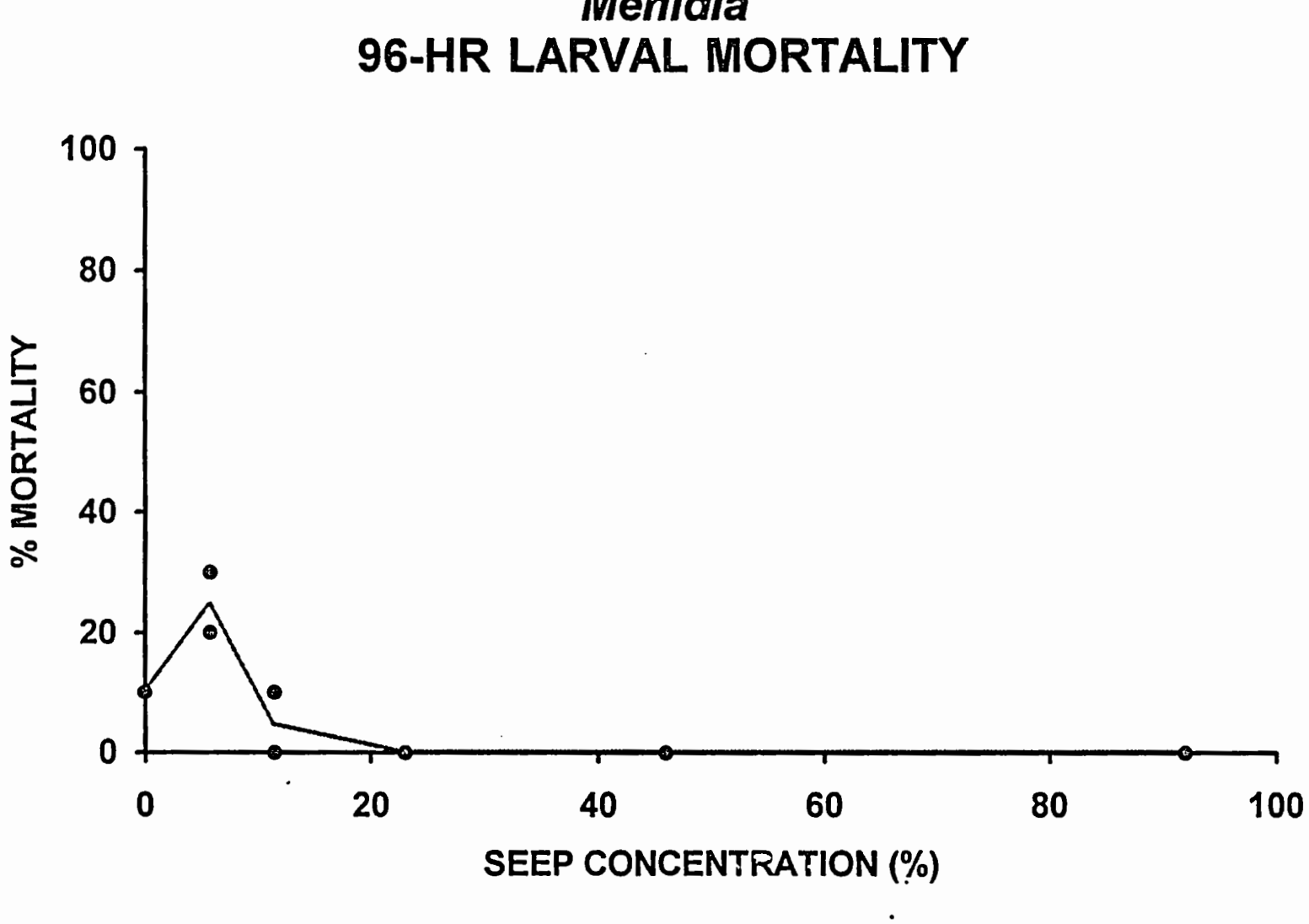

Figure 7. Larval mortality response of *Menidia* to seep water exposure.

toxicity to *Ampelisca*. These data are not displayed graphically, but can be found in Appendix I.

Mulinia 7-day mortality and growth tests also proved to be insensitive to solid phase sediment exposure (Figure 8 and 9). Insignificant mortality and no trends in growth were observed through the range of landfill sediment concentrations for development of exposure-response models. Thus none of the *Ampelisca* or *Mulinia* endpoints measured in sediment assays indicated this exposure medium to be toxic.

Sediment Extract -- The sea urchin sperm cell test was initially performed using extract dilutions in excess of 0.05%. These concentrations proved to be too high, so a second test was conducted using lower dilutions. Success of *Arbacia* fertilization in the second assay displayed a graded response to increasing concentrations of sediment extract (Figure 10), with near-maximum effects being observed at a sediment extract concentrations of 0.025% and above. Concentrations below 0.006% had little effect on fertilization success.

Abnormal development of *Arbacia* larvae increased dramatically at sediment extract concentrations greater than 0.05%, whereas larval mortality was affected at concentrations of sediment extract greater than 0.15% (Figure 11). As a result of these effects, normally developing larvae were absent in treatments above the 0.10% dilution.

As with the *Arbacia* discussed above, initial dilutions of sediment extract used in the *Mulinia* embryo/larval toxicity assay proved to be too high to adequately describe response, and a second test was conducted. Extract exposure conditions in this second test again resulted in minimal *Mulinia* larval shell development at all concentrations higher than 0% (Figure 12). However, examination of responses in the DMSO chemical control treatment indicated QA problems associated with the use of this compound as a carrier solvent: 88% of the animals did not develop shells in the DMSO-only treatment (Appendix J). The observed responses therefore were due to a solvent effect rather than to exposure to sediment extract contaminants, and the entire test was invalidated. Insufficient resources were available to explore use of other carrier solvents for this assay.

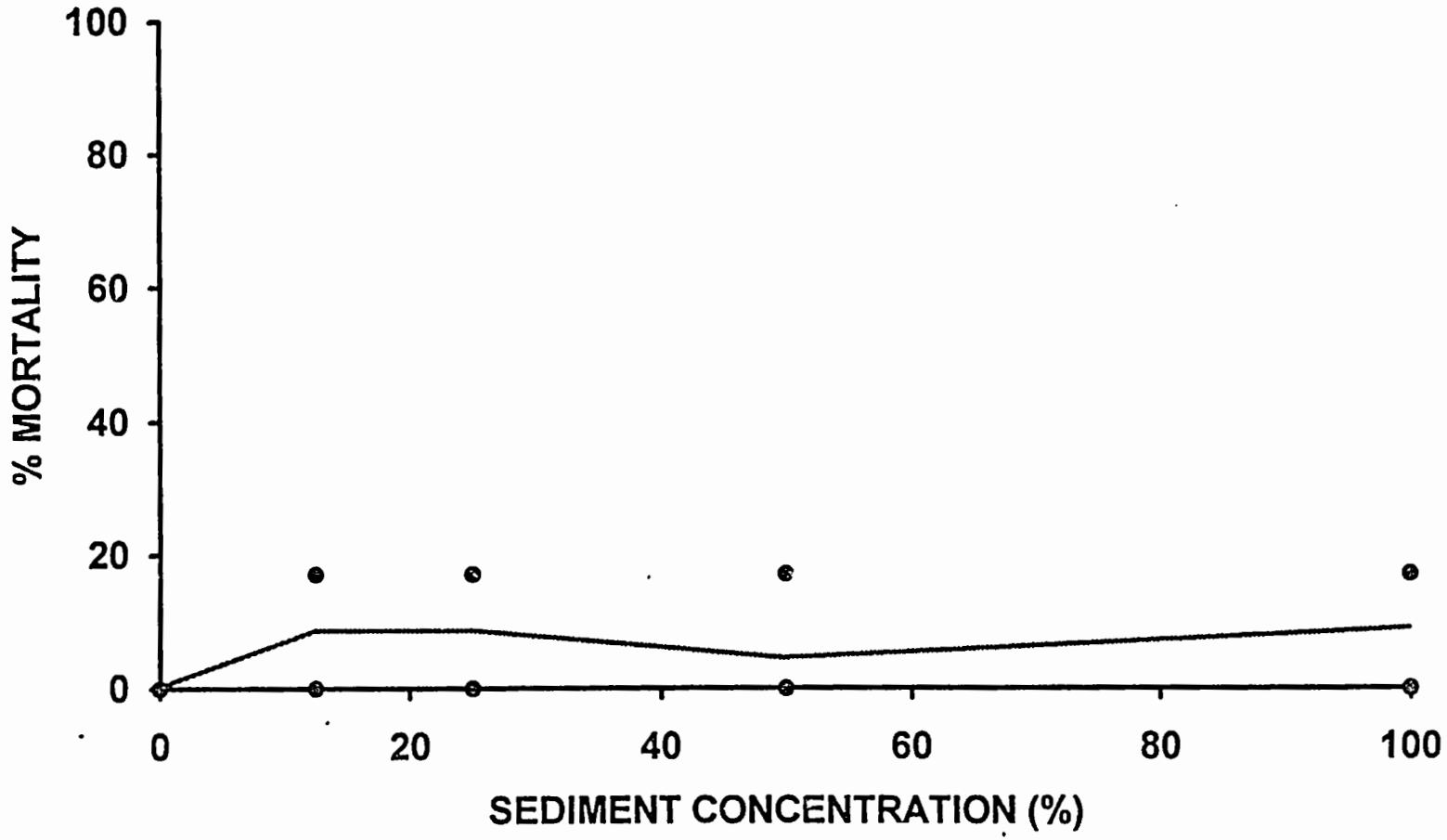

Figure 8. Seven-day mortality response of *Mulinia* to sediment exposure.

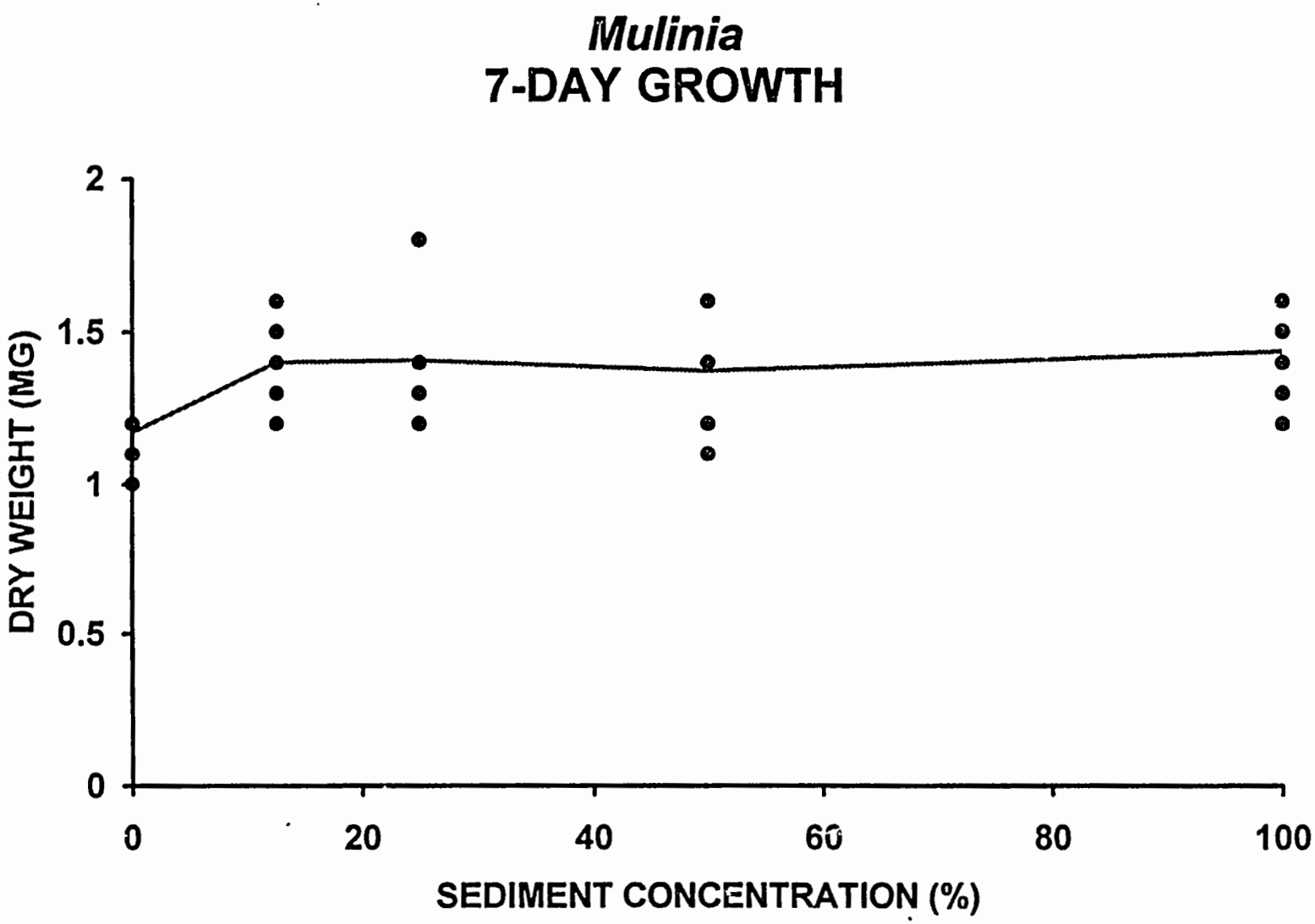

Figure 9. Seven-day growth response of *Mulinia* to sediment exposure.

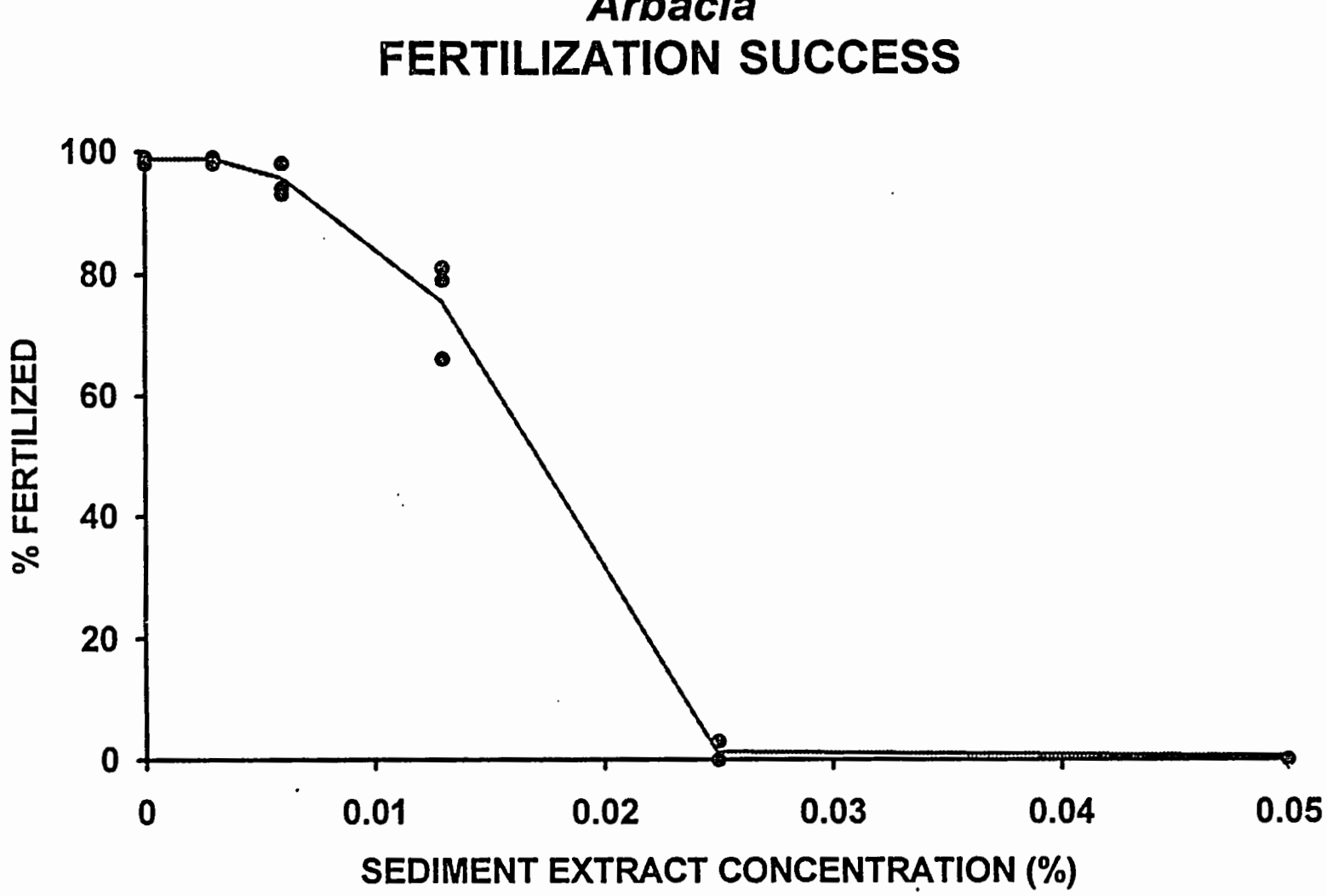

Figure 10. Fertilization response of *Arbacia* to sediment extract exposure.

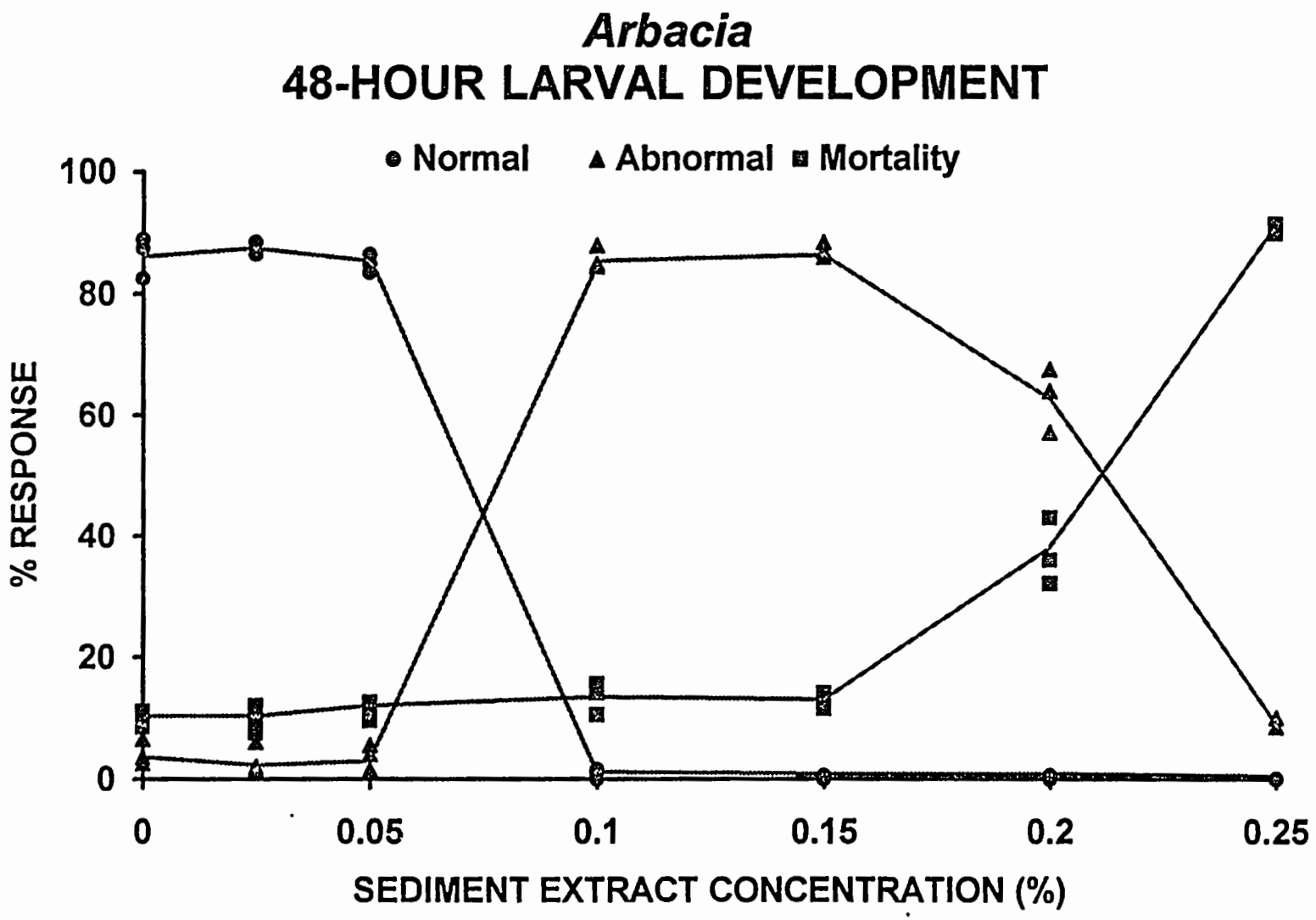

Figure 11. Larval mortality and development responses of *Arbacia* to sediment extract exposure.

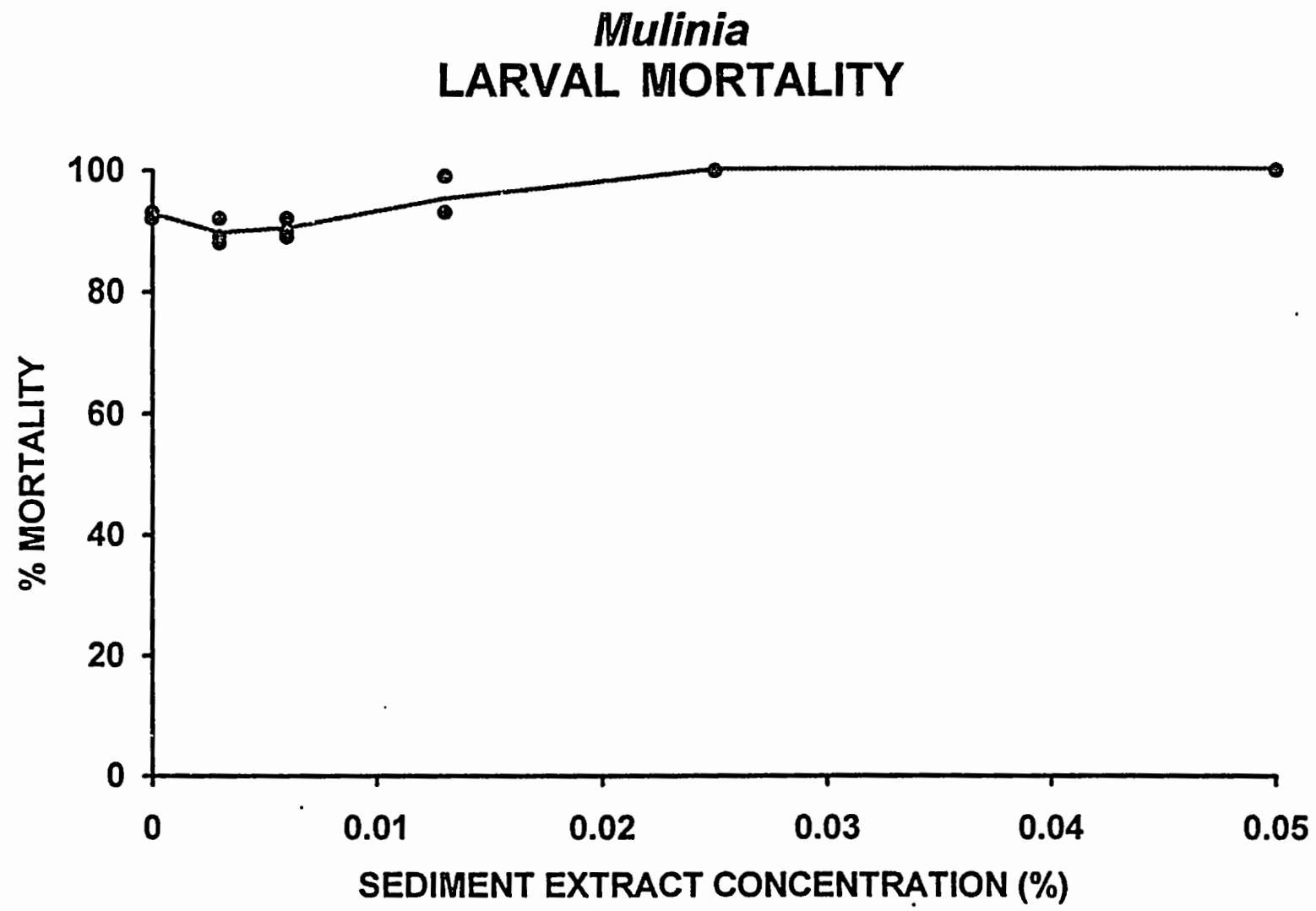

Figure 12. Embryo/larval toxicity response of *Mulinia* to sediment extract exposure.

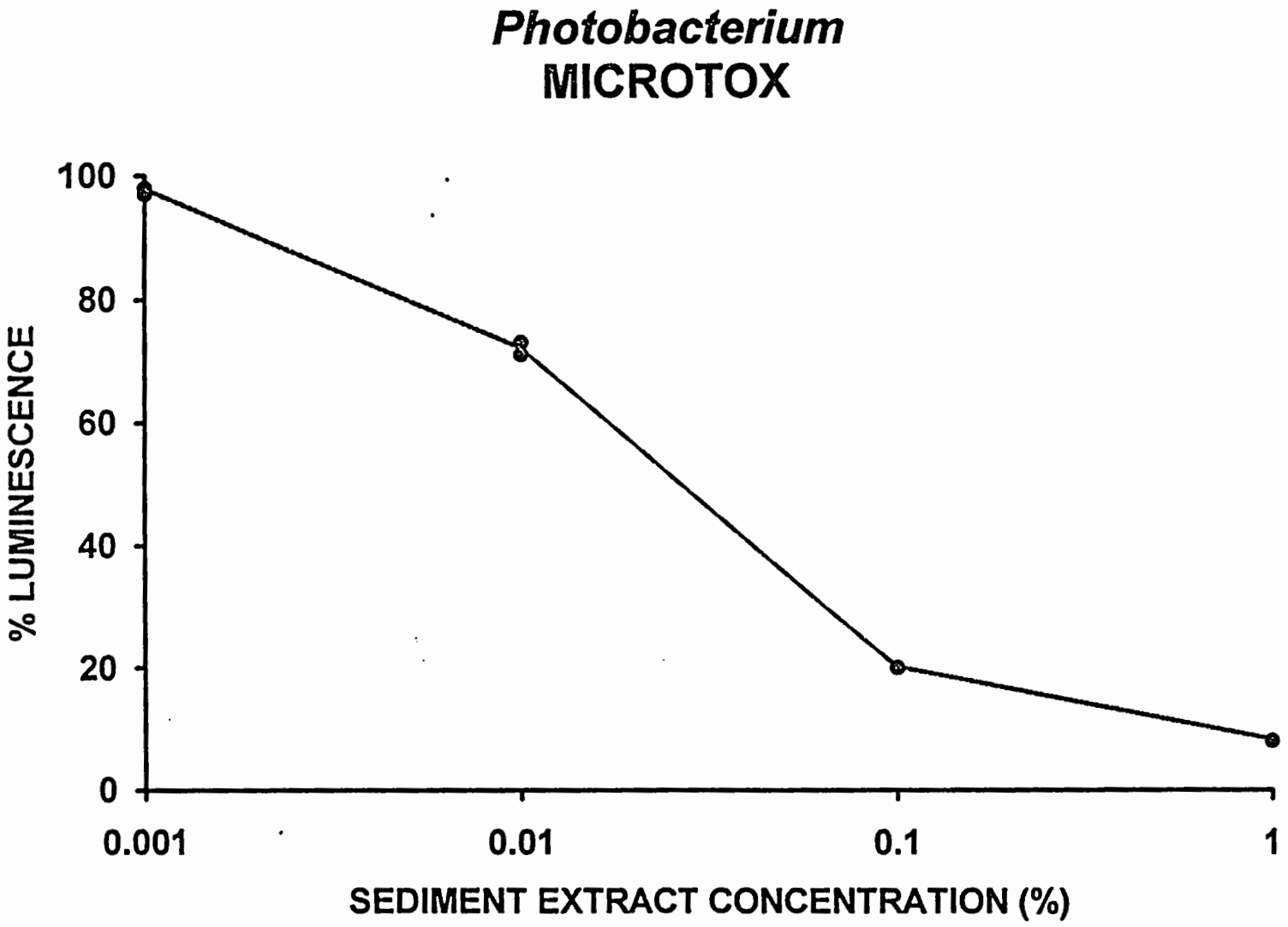

Figure 13. Bioluminescence response of *Photobacterium* to sediment extract exposure.

Photobacterium bioluminescence decreased geometrically with increasing extract concentration in the Microtox test (Figure 13), indicating a strong response between bacterium survival and contaminant exposure. This test was conducted using a dilution series different from the other extract assays to follow the standard operating protocols suggested by the manufacturer.

Overall, the *Arbacia* larval development assay was most sensitive to the suite of environmental contaminants present in landfill sediment extracts, although *Arbacia* fertilization and Microtox also were fairly sensitive. These three assays provided data sufficient to develop exposure-response models.

Model Development

Of the 15 species/endpoint/exposure medium data sets obtained in this study, six failed the criteria for exposure-response model development established earlier. Landfill sediments elicited no observable response in *Ampelisca* (mortality) or *Mulinia* (mortality, growth), whereas maximum exposure concentrations of seep water were insufficient to elicit *Arbacia*, *Mysidopsis*, or *Menidia* mortality responses suitably high for the model fitting procedure to be successful. Additionally, characterization of *Mulinia* growth in response to sediment extracts was confounded by solvent carrier effects, thus invalidating this assay. Although they contributed to the overall assessment of risks, these seven data sets were eliminated from further quantitative analysis.

Least-squares estimates of EC_{20}, σ, and R_0 for the remaining 8 assay data sets are provided in Table 3, along with model coefficients of determination (describing the percentage of total variance explained by the model). EC_{20} is presented to indicate exposure media concentrations at which small, but perhaps ecologically significant responses are expected to occur. For the most part, models were generated which fit the assay data reasonably well, as illustrated in Figures 14-20 and the R^2s listed in Table 3.

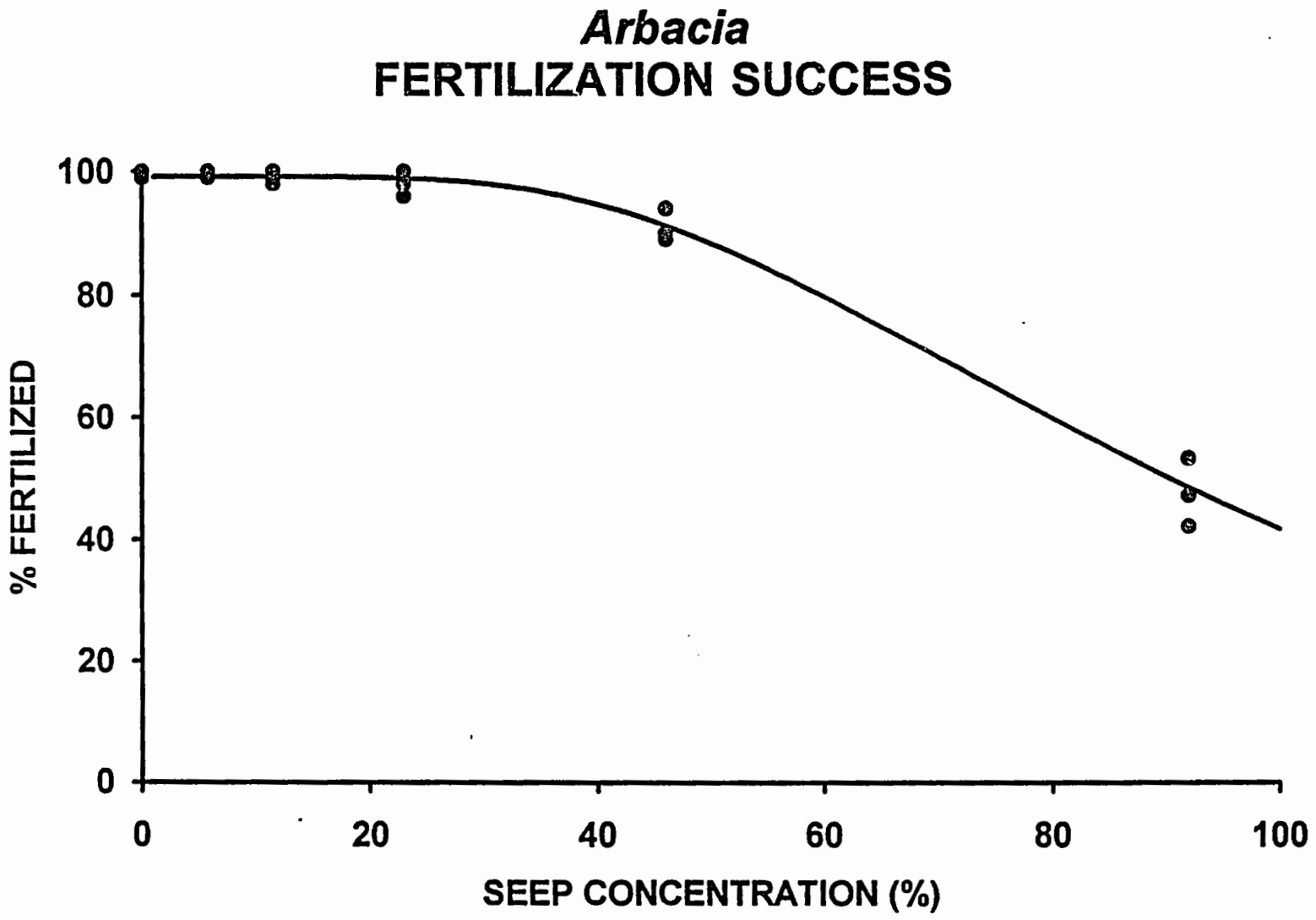

Figure 14. Whole media exposure-response model for fertilization response of *Arbacia* to seep water exposure.

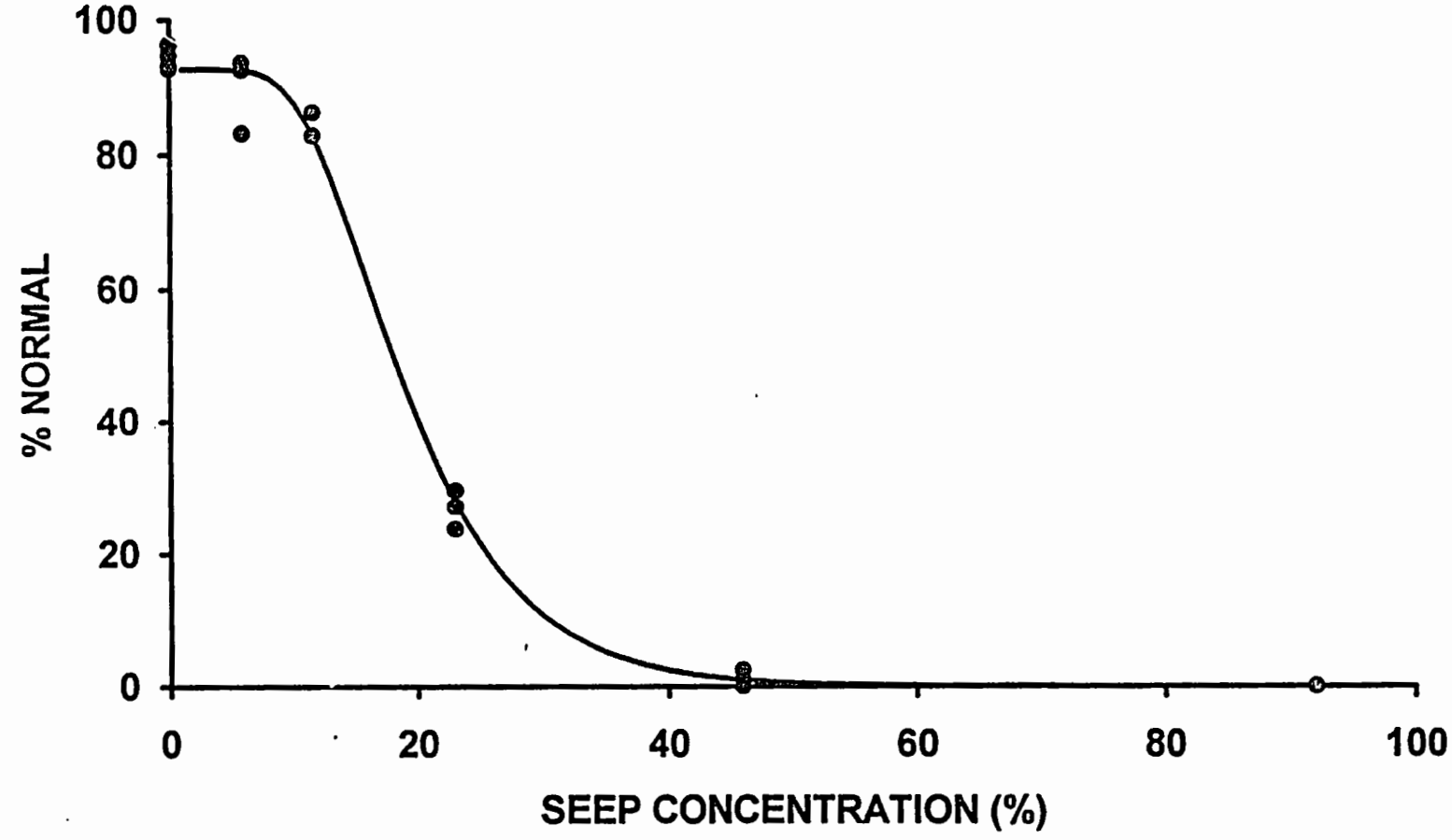

Figure 15. Whole media exposure-response model for larval development response of *Arbacia* to seep water exposure.

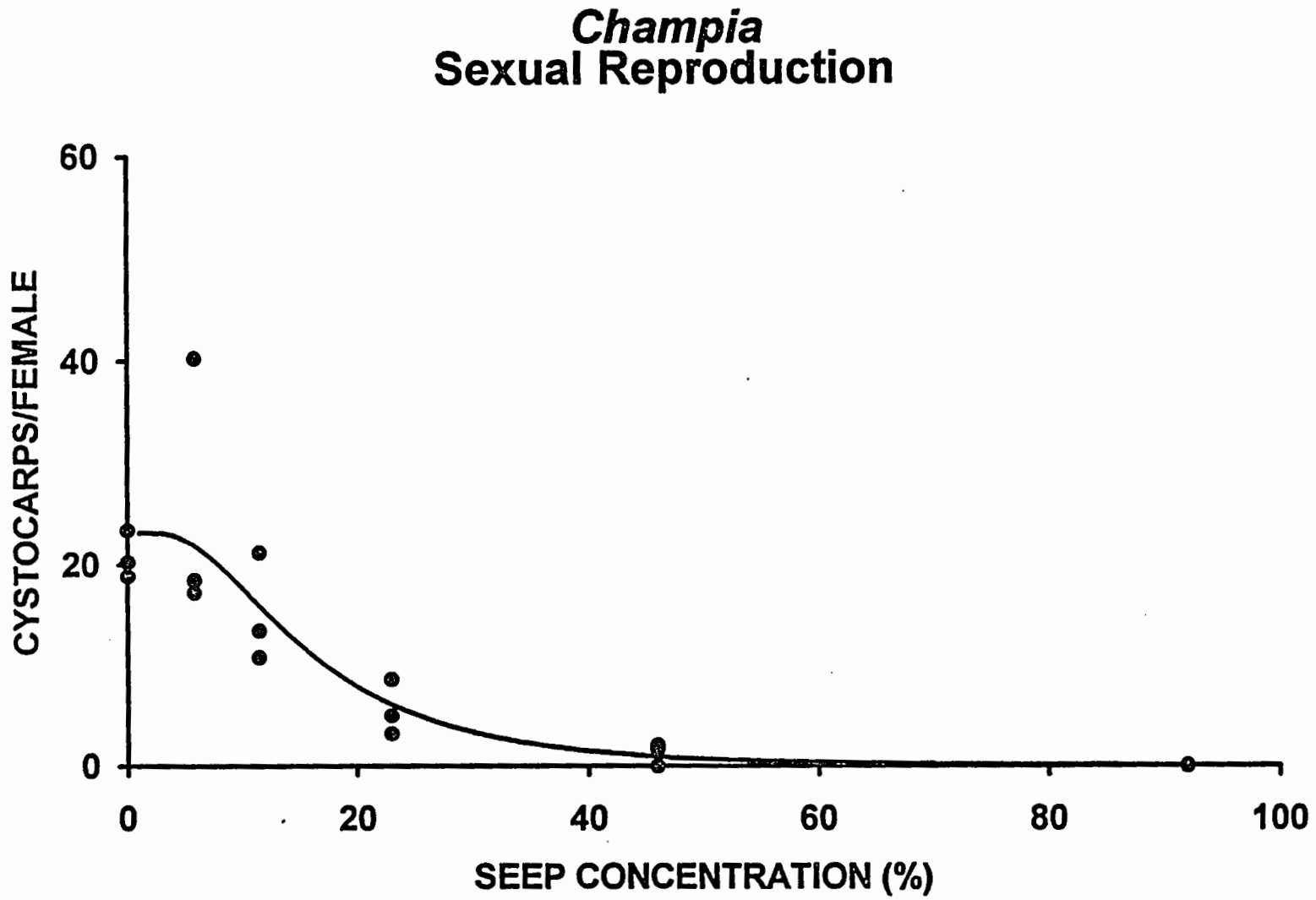

Figure 16. Whole media exposure-response model for sexual reproduction response of *Champia* to seep water exposure.

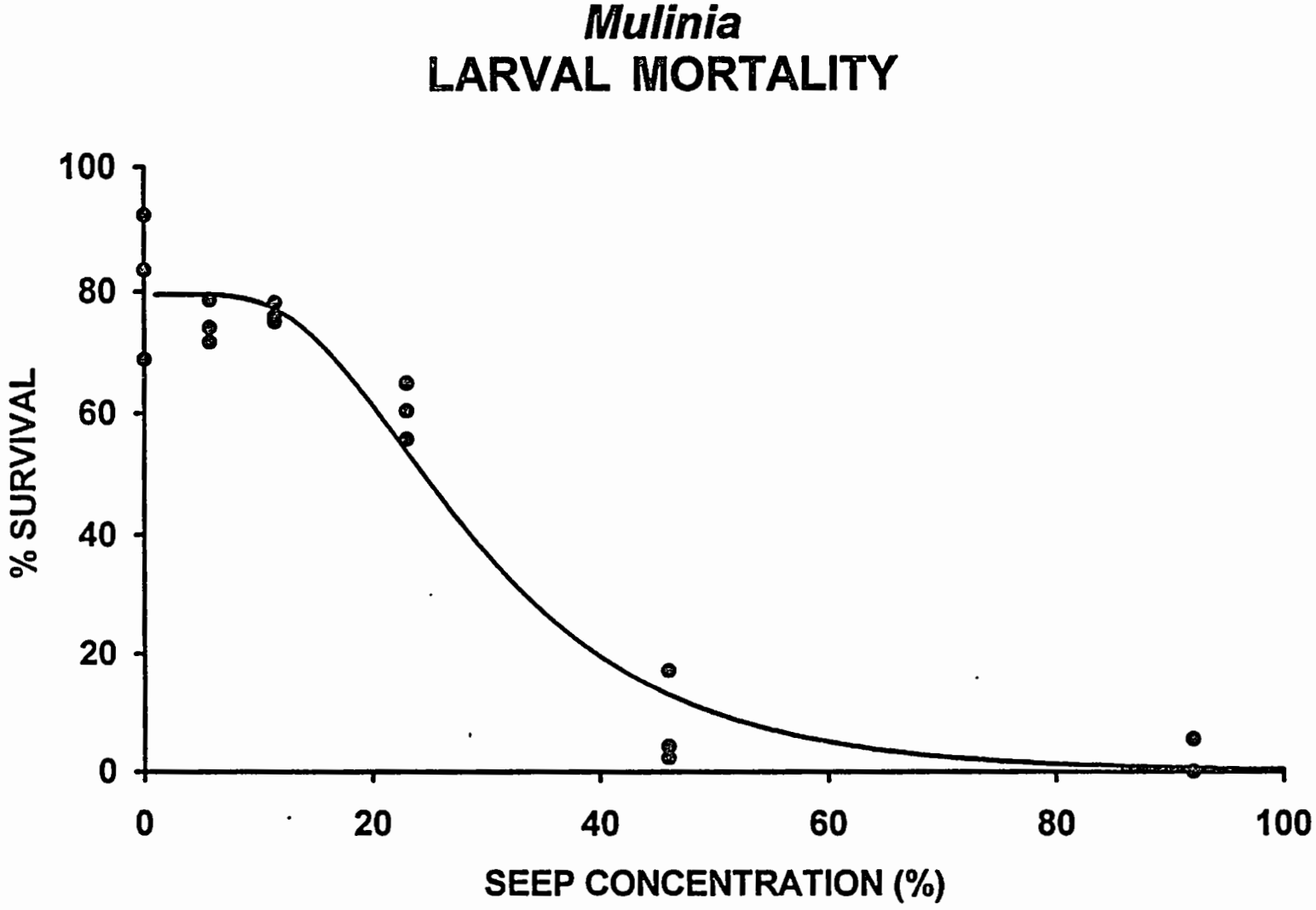

Figure 17. Whole media exposure-response model for larval survivorship response of *Mulinia* to seep water exposure.

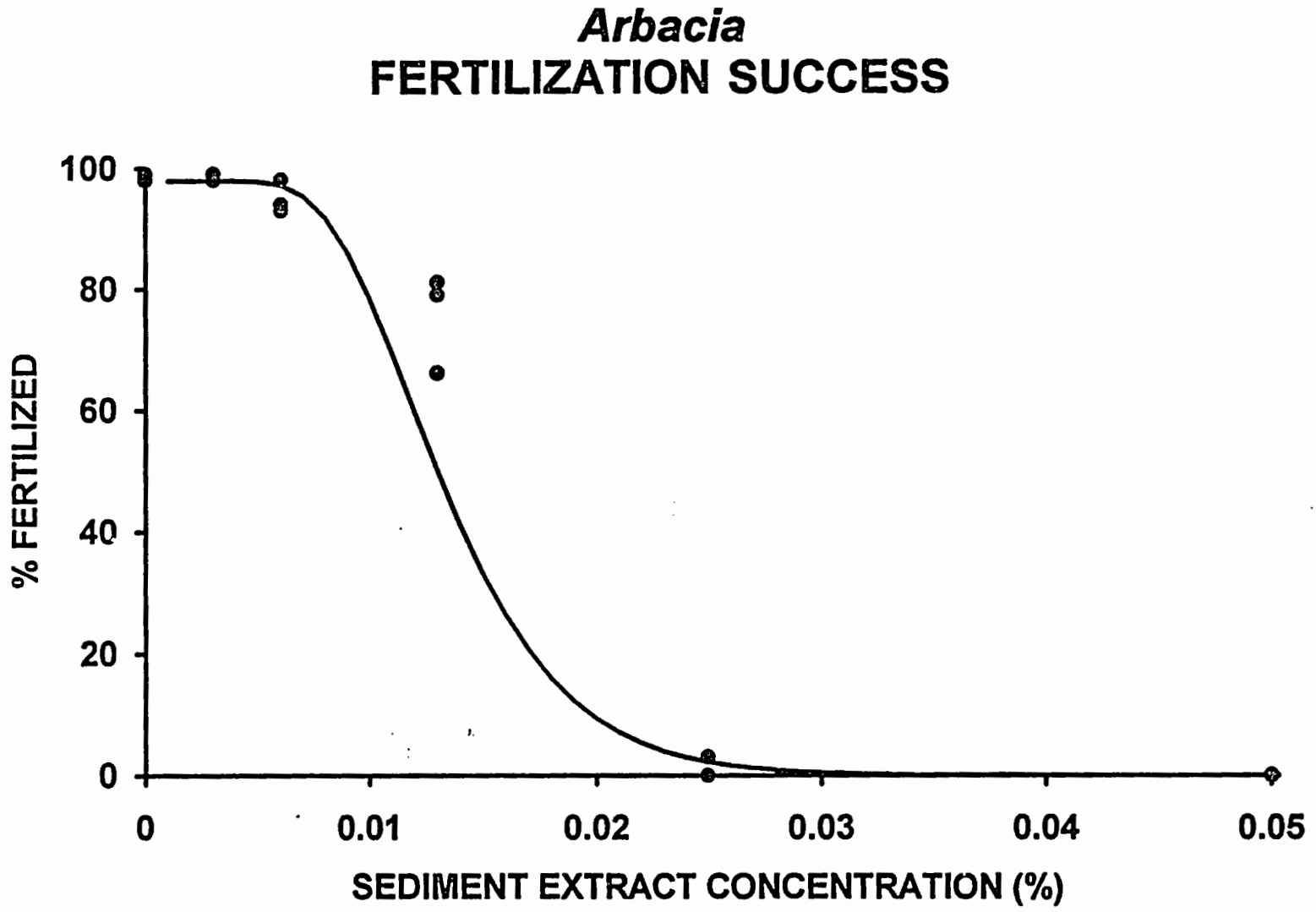

Figure 18. Whole media exposure-response model for fertilization response of *Arbacia* to sediment extract exposure.

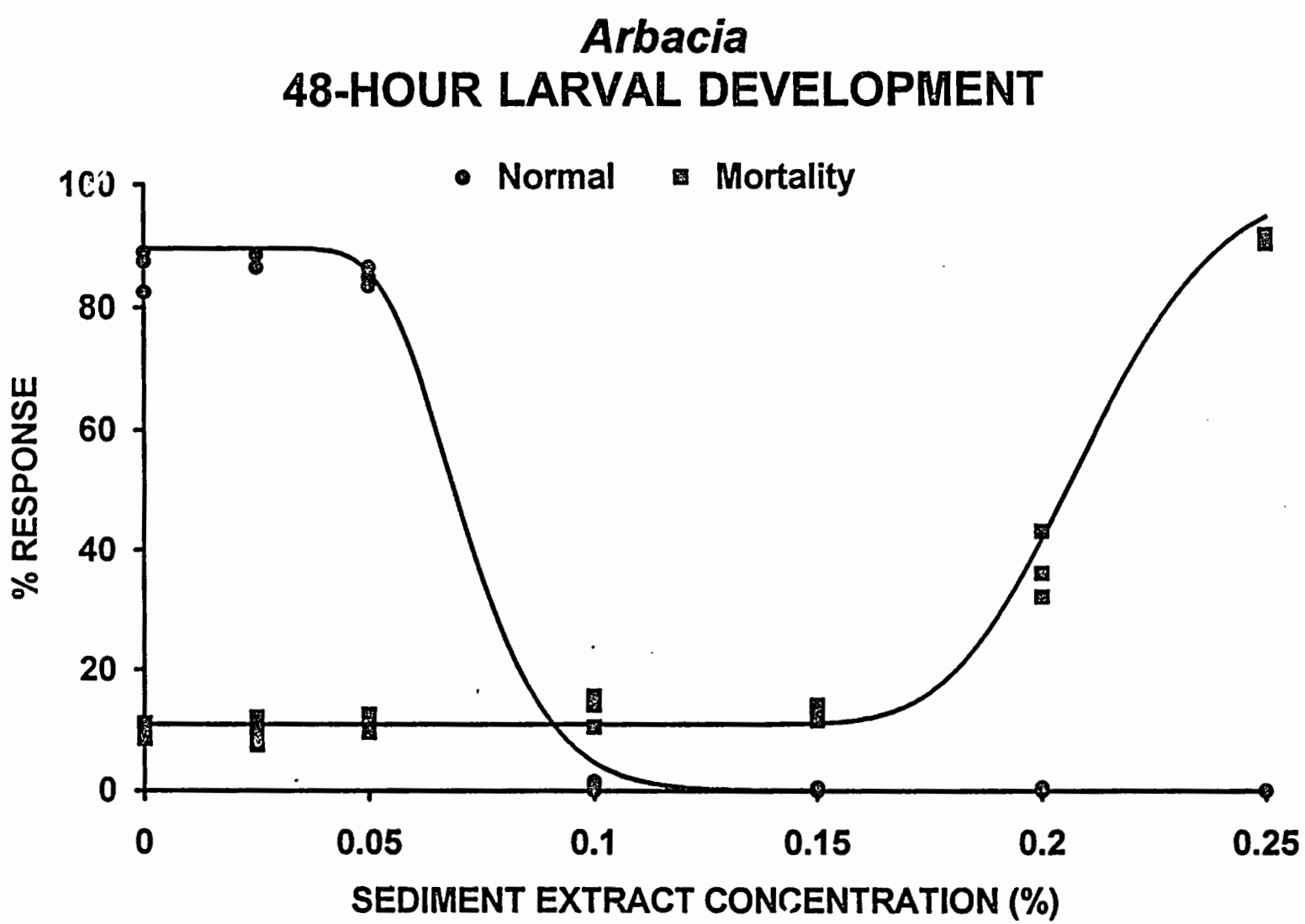

Figure 19. Whole media exposure-response model for larval development response of *Arbacia* to sediment extract exposure.

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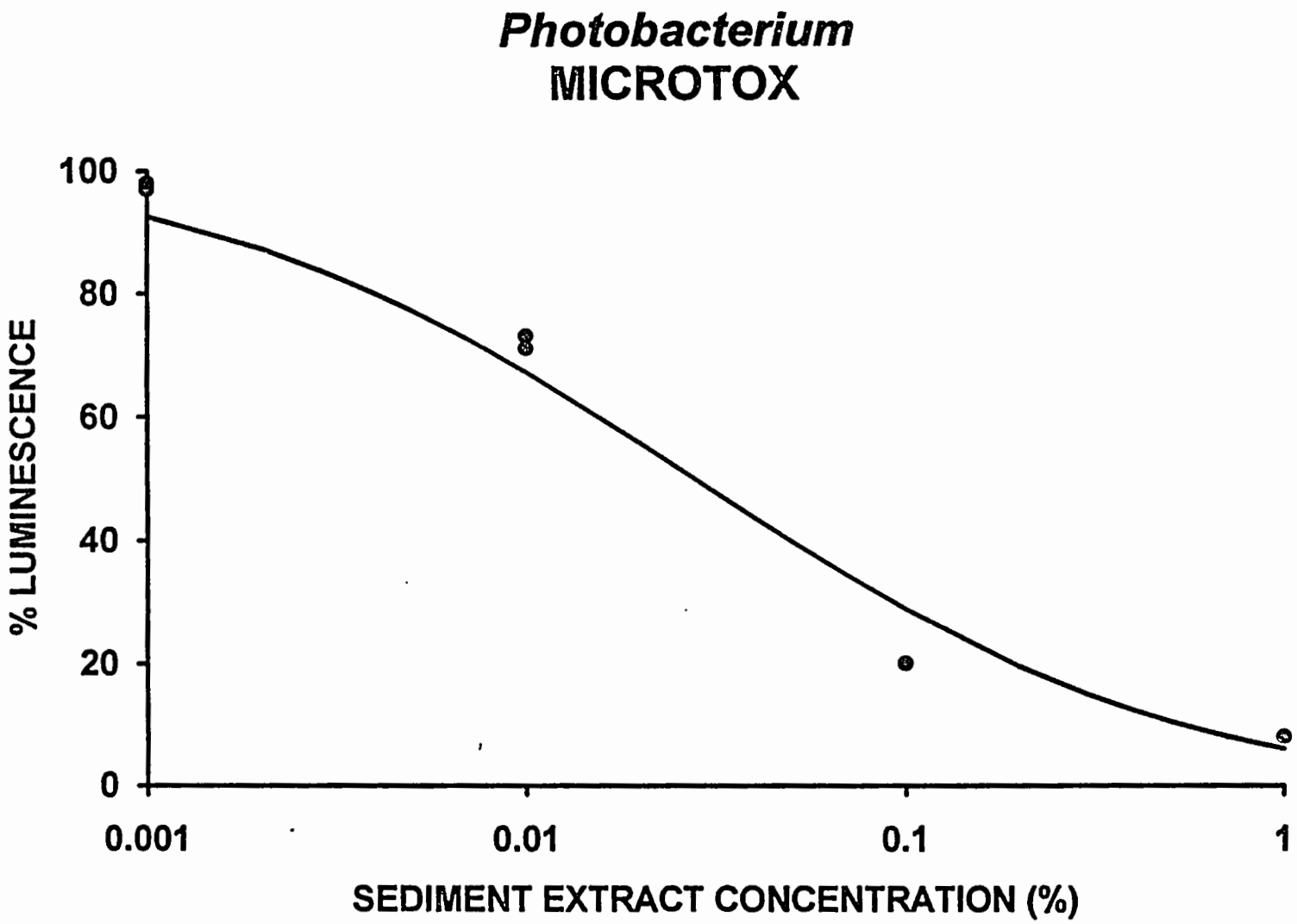

Figure 20. Whole media exposure-response model for bioluminescence response of *Photobacterium* to sediment extract exposure.

Table 3. Whole-media exposure-response model parameter estimates and coefficients of determination.

Exposure Medium	Species/Endpoint	EC_{20}[a]	σ	R_0[b]	R^2
Seep water	*Arbacia*/fertilization	60.3	0.21	99.2	999
	Arbacia/normal development	13.5	0.17	92.6	994
	Champia/reproduction	9.20	0.27	23.2	883
	Mulinia/mortality	19.1	0.21	79.5	931
Sediment extract	*Arbacia*/fertilization	0.01	0.14	97.9	972
	Arbacia/normal development	0.06	0.09	89.6	
	Arbacia/mortality	0.19	0.05	89.1	995
	Photobacterium/mortality	0.004	1.0	100	985

[a] In units of % seep water or % extract.
[b] Cystocarps/female for *Champia*, percent for all other.

MYA LABORATORY EXPERIMENT

Suggestively higher rates of neoplasia development and mortality in *Mya* inoculated with affected hemolymph and exposed to AH sediment were observed at the end of the 90-d laboratory exposure (Table 4). (In this table and the following text, references to inocula containing sarcoma cells are indicated by a "+" appended to the sediment treatment acronym, whereas inocula of unaffected hemolymph is referenced by a "-"). However, two-way ANOVA indicated no significant sediment or inoculation effects in any of the three biological endpoints measured. Because concentrations of contaminants in PR were substantially lower than those in AH (Appendices A-E), these results support observations made during Phase II (Munns *et al.* 1993) and other studies (*e.g.*, Mix 1979, 1986; but see

Walker *et al.* 1981) regarding the general lack of correlation between Hn and sediment chemistry.

Table 4. Mean responses of *Mya* endpoints in the 90-d neoplasia experiment.

	Treatment			
Endpoint	PR-	PR+	AH-	AH+
Neoplasia (%)	28.1	32.5	39.2	47.5
Mortality (%)	13.3	10.9	8.3	16.7
Growth (g)	-1.0	-1.2	-1.0	-1.6

It is possible that contaminants in PR sediments were more available to *Mya* than were those in AH treatment. In contrast to the initial efforts undertaken for the exposure-response assays to match sediment attributes which control contaminant bioavailability, the most important characteristics for the reference sediment used in the *Mya* laboratory exposure were low contaminant concentrations and the absence of neoplastic disease at the collection site. The diluting sediment used in the exposure-response bioassays (POTO) was unavailable for the *Mya* experiment because high incidences of neoplasia at been observed at Marsh Point (MP), located at the mouth of Potowomut Cove, during Phase I and II investigations (Munns *et al.* 1991, 1993). Bioaccumulation factors (BAFs) for most contaminants (with the exception of selected PAHs, Fe and DDT), were higher in clams exposed to PR (Table 5). While this implies that contaminants in PR sediments were more available for accumulation by the organism than contaminants in AH sediments, tissue concentrations in PR clams were lower than those of AH clams due to the overall higher levels of contamination found in the latter sediment.

Table 5. Range of bioaccumulation factors of sediment contaminants in the 90-d neoplasia experiment.

Contaminant Class	AH	PR
PCBs	0 - 1.27	36.5 - 74.5
PAHs	0 - 0.23	0 - 4.64
Pesticides	0.03 - 1.39	7.33 - 35.5
Metals	0.03 - 1.59	0.27 - 850

Despite these general

observations, the ubiquitous presence of Hn in all treatments renders suspect the assumptions of the experiment that no Hn should have occurred in the PR- treatment. Hn was not observed in *Mya* collected at PR during the field survey conducted in Phase II (Munns *et al.* 1993), so it is unlikely that some characteristic of that sediment initiated or promoted neoplasia development. Because of the lack of Hn in the large number of test animals screened at test initiation, it also is unlikely that these animals were affected prior to the laboratory exposure. One possibility is that hemolymph from animals diagnosed as being unaffected and used to inoculate the PR- and AH- animals was in the initial stages of Hn development and were contaminated with small numbers of sarcoma cells which went undetected in the initial screening. Cooper *et al.* (1982b) have demonstrated that diagnoses of mild cases of Hn by the histocytological technique used here to be accurate only 66 to 71% of the time. Perhaps the use of more sophisticated diagnostic techniques, such as those involving monoclonal antibodies developed by Reinisch *et al.* (1983) and Smolowitz and Reinisch (1986), would have minimized the potential for misdiagnosis of Hn.

QUANTIFICATION OF ECOLOGICAL RISK

RISKS OF TOXICOLOGICAL IMPACT

The exposure-response models developed through performance of laboratory bioassays provided insight to the potential effects of landfill-associated contaminants on a range of species and endpoints on a whole-medium basis. For example, assays involving sediment exposures indicated little to no acute toxicity to *Ampelisca* or *Mulinia*, whereas seep waters diluted down to 20% affected *Arbacia* larval development. This information is valuable in examining the risks to ecological systems in immediate association with the landfill, and could be used to evaluate remediation options. However, each exposure medium was a complex mixture of many environmental contaminants. This multiplicity of chemical stressors renders decisions regarding ecological risks posed by the landfill to the greater Allen Harbor system somewhat difficult, because the behaviors of individual contaminants differ in response to geochemical and biological processes acting in the harbor. The concentrations of individual contaminants in any given environmental sample likely do not represent simple dilutions of landfill media.

Risk Quantified by Toxic Unit Exposure-Response Models

To address this problem, a normalization procedure was employed involving the concept of a *toxic unit* (TU) which allowed more direct comparison of exposures associated with environmental samples with those of landfill media. EPA utilizes the TU approach in its water quality-based toxics control program (U.S. EPA 1991). In a general sense, a toxic unit is simply the ratio of a contaminant concentration to some biological benchmark concentration for that chemical (such as an LC_{50} or EC_{50}), and is often expressed as a percentage. As such, it is arithmetically similar to the risk quotients developed in Phase I (Munns *et al.* 1991). In the current analysis, however, contaminant-specific TUs were summed to derive a single, aggregated metric (ΣTU) of chemical contamination for each unique environmental sample. This approach assumes additivity in the toxic actions of

contaminants in complex mixtures, a conjecture of some debate (see Alabaster and Lloyd 1982, U.S. EPA 1991).

As presented earlier, insufficient toxicological response was observed in laboratory exposures to derive exposure-response models for whole sediments. However, these results and those obtained during Phases I and II (Munns *et al.* 1991, 1993) suggest ecological risks associated with whole sediment exposures to be small. Efforts therefore were directed towards development of TU-based models for seep water and sediment extracts only. For this analysis, TUs were calculated using established federal marine Water Quality Criteria (WQC) and the concentrations of contaminants measured in each exposure medium dilution series.

EPA has suggested that appropriate biological benchmarks be used for each specific endpoint (U.S. EPA 1991). This means that acute benchmarks be used with acute endpoints and chronic benchmarks be used with chronic endpoints. In this analysis, however, chronic WQC were used in all modeling efforts to yield conservative predictions of ecological response. WQC available (U.S. EPA 1987) for this exercise are indicated in Table 6. (The criterion shown for copper is actually the acute value, as no chronic value is given). The general paucity of available marine WQC can be viewed as a limitation to this analysis, although several of the contaminants listed in Table 6 were identified as potential contaminants of concern in Munns *et al.* (1991). The nature of chemical data available for TU calculations also restricted model development in that organic contaminants for which WQC are available were generally not detected in seep water media, whereas the method employed to develop sediment extracts isolated organic contaminants only. (In retrospect, a sediment elutriate procedure may have been more appropriate for isolating

Table 6. Federal marine chronic Water Quality Criteria used in ΣTU exposure-response model development.

Contaminant	Chronic WQC (μg/L)
cadmium	9.3
zinc	86
copper	2.9
lead	8.5
nickel	8.3
PCB	0.03
Chlordane	0.004
DDT	0.001

sediment contaminants.) Despite these limitations, efforts to develop TU-based models were generally successful. Contaminant-specific TUs for the two undiluted exposure media for which exposure-response models were developed are shown in Table 7.

Table 7. Contaminant-specific TUs for undiluted exposure media used in model development.

Exposure Medium	TU†								
	Cd	Zn	Cu	Pb	Ni	PCB	Chlordane	DDT	ΣTU
seep water	1.5	2.9	15.2[1]	0.5	2.3	nd[2]	nd	nd	22.3
sediment extract	np[2]	np	np	np	np	2.2	0.2	0.9	3.3

† Table entries are the number of toxic units, for each contaminant, quantified in the exposure medium. TUs for whole sediment were not calculated (see text for explanation). TUs for sediment extract are based on an extract concentration of 0.25%, the highest concentration used in sediment extract assays.

[1] Nominal values were used for copper concentrations in seep water in calculating contaminant-specific TUs, because measured concentrations above the 46% exposure media concentration did not agree with expected nominal levels (see Appendix A).

[2] nd = not detected
np = not present in medium

The TU models were constructed in a manner similar to those for whole exposure media, using nonlinear regression techniques, with the metric ΣTU replacing whole media concentration as the exposure term. Not surprisingly (as ΣTU is a direct function of the individual contaminant concentrations measured in each dilution series), fair success was achieved in obtaining models which described response data reasonably well (Table 8, Figures 21-27).

To some extent, both the whole-media and ΣTU exposure-response models developed in this study are assay-dependent: model parameter estimates depend not only upon the general responses of endpoints to exposure levels, but also upon the exact values measured in replicates of each exposure treatment. The replicate measures reflect some degree of inherent variability within the test population, as well as error (uncertainty) in part

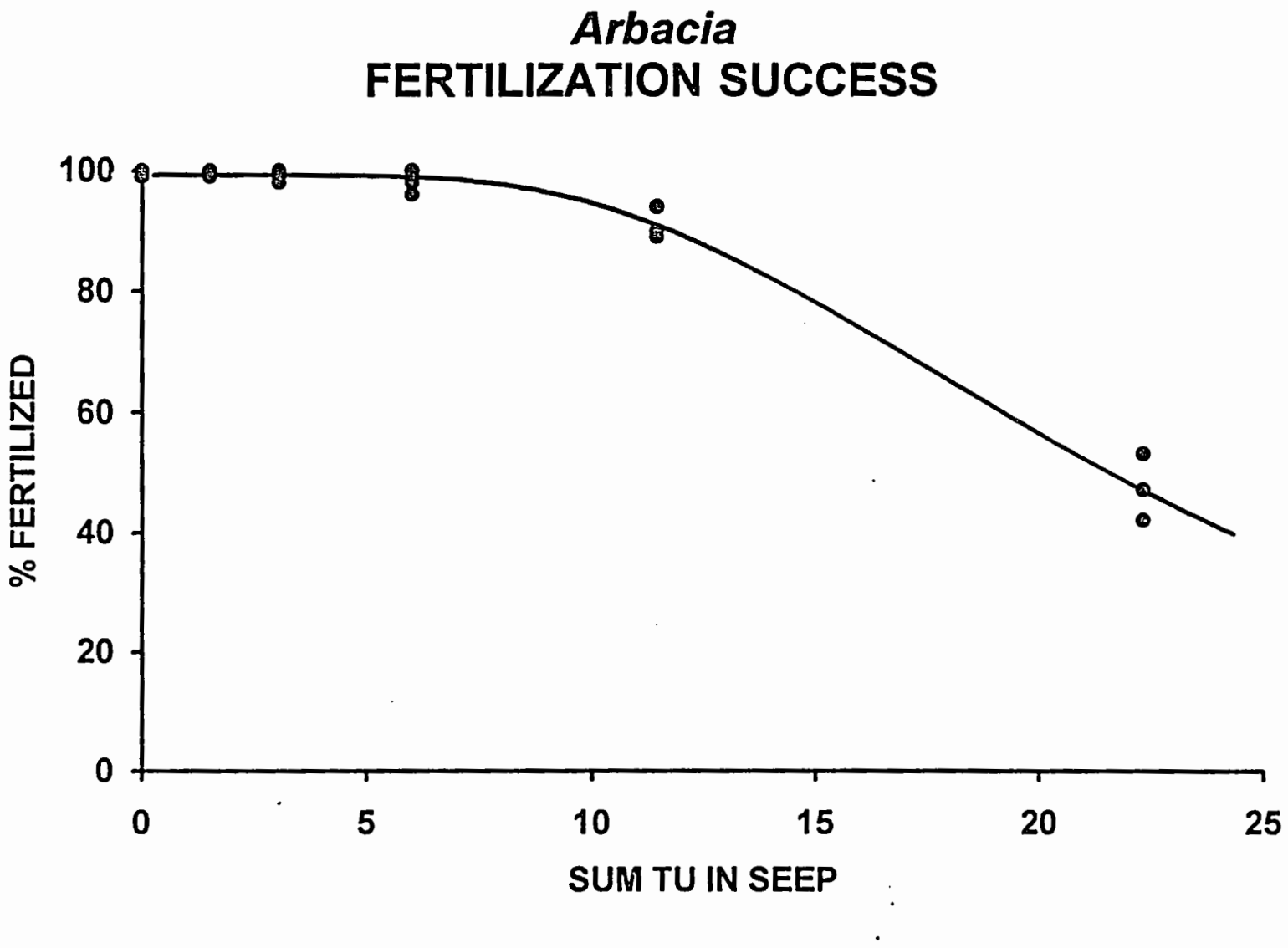

Figure 21. ΣTU exposure-response model for fertilization response of *Arbacia* to seep water exposure.

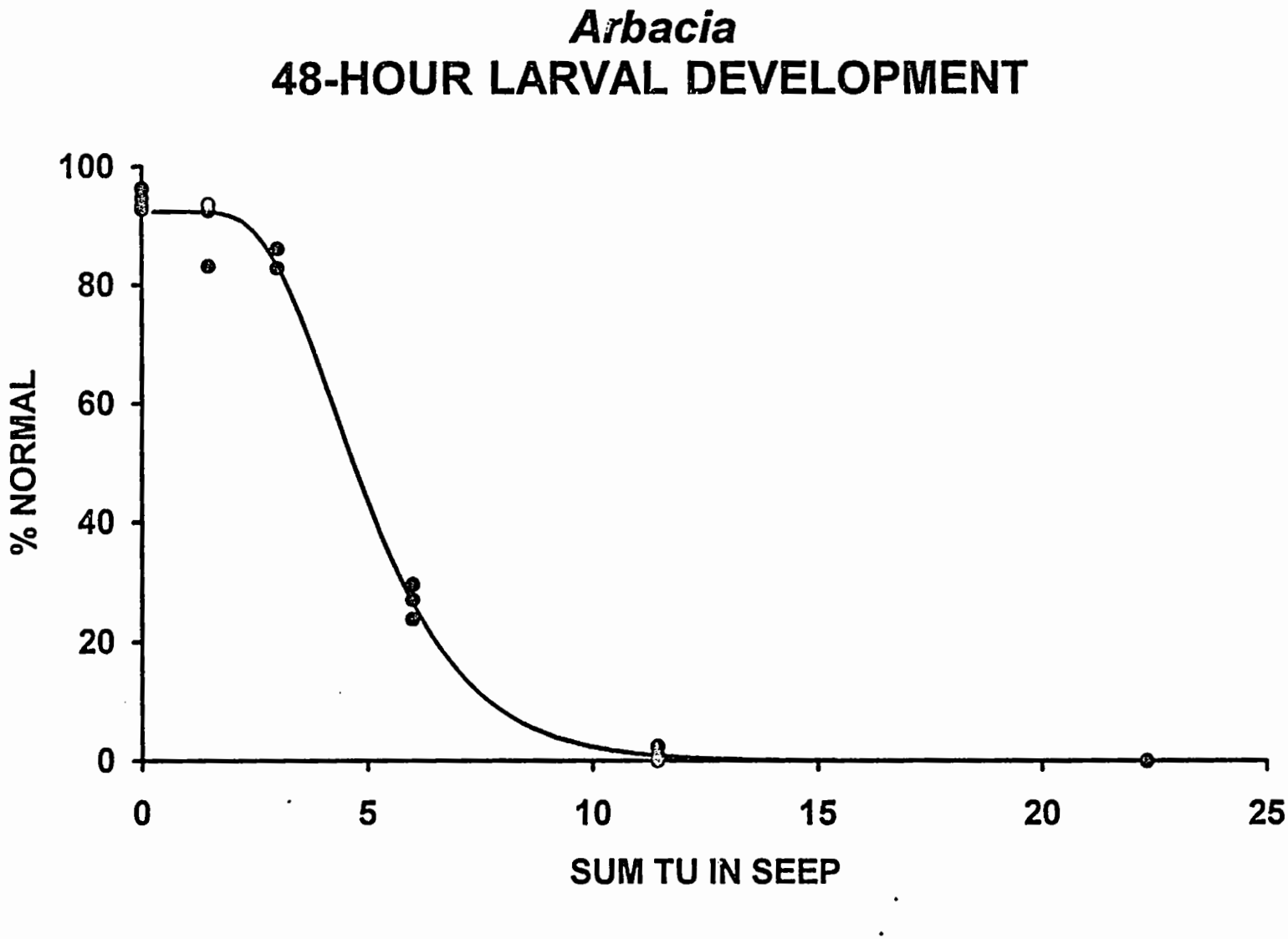

Figure 22. ΣTU exposure-response model for larval development response of *Arbacia* to seep water exposure.

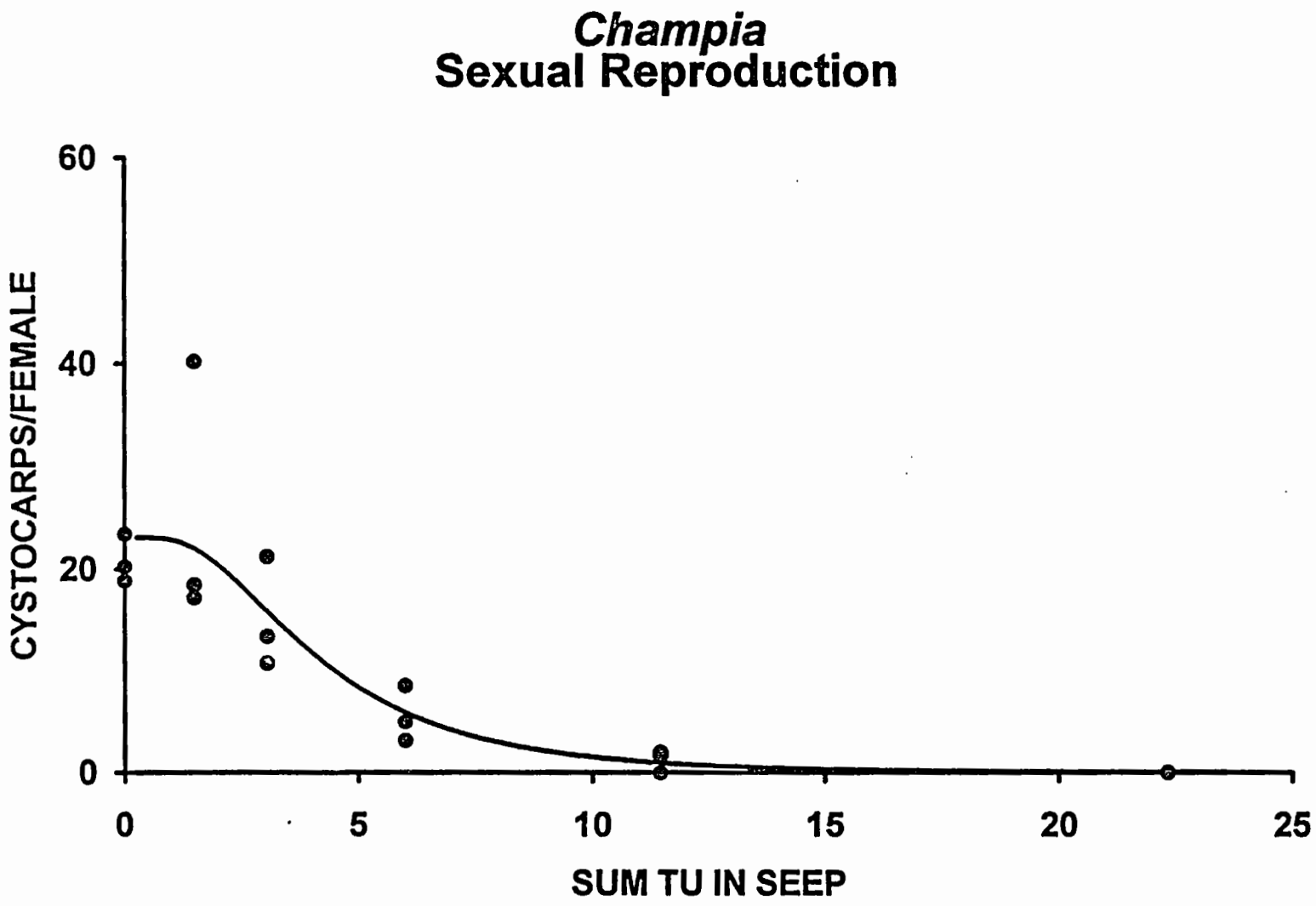

Figure 23. ΣTU exposure-response model for sexual reproduction response of *Champia* to seep water exposure.

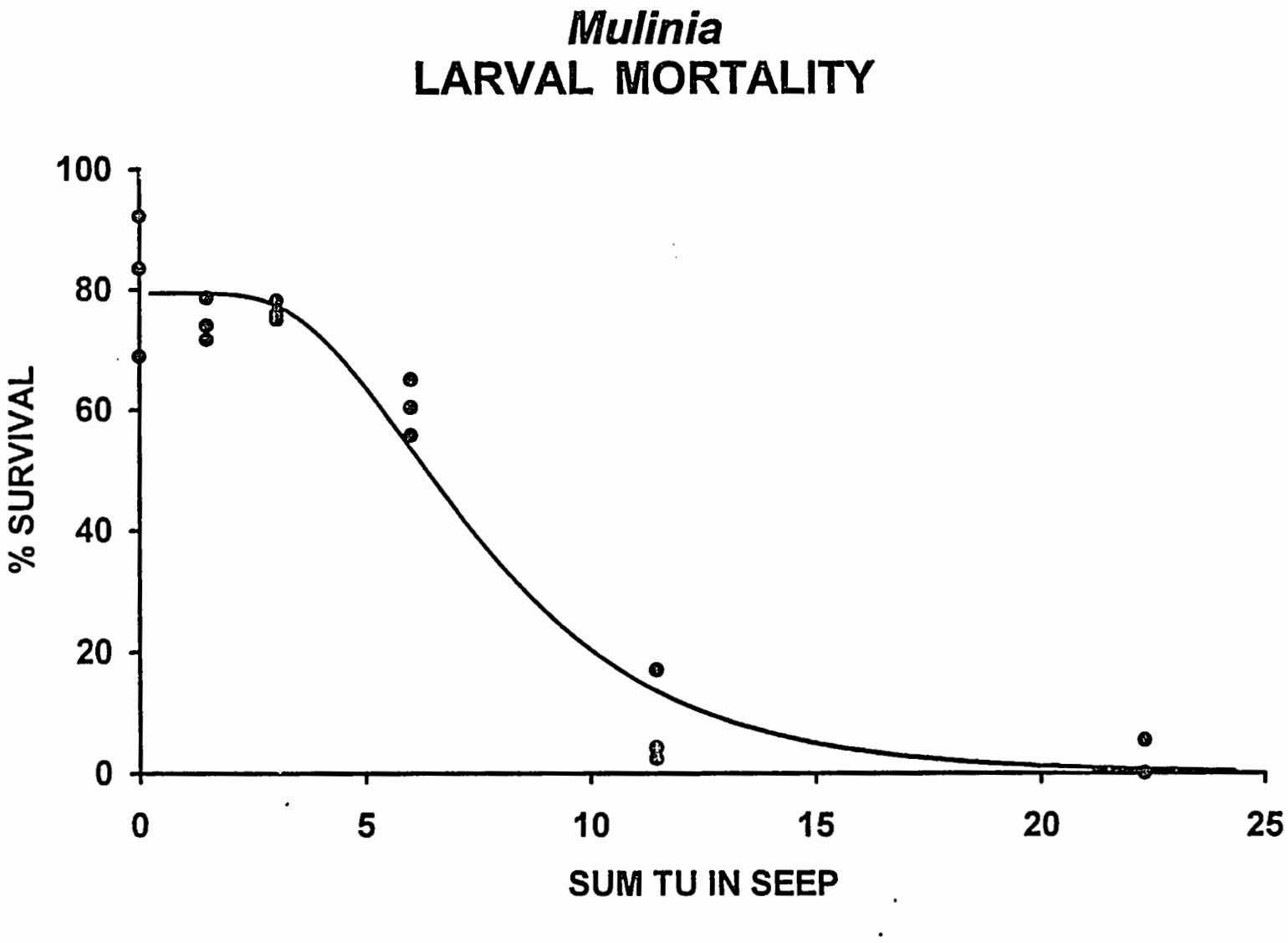

Figure 24. ΣTU exposure-response model for larval survival response of *Mulinia* to seep water exposure.

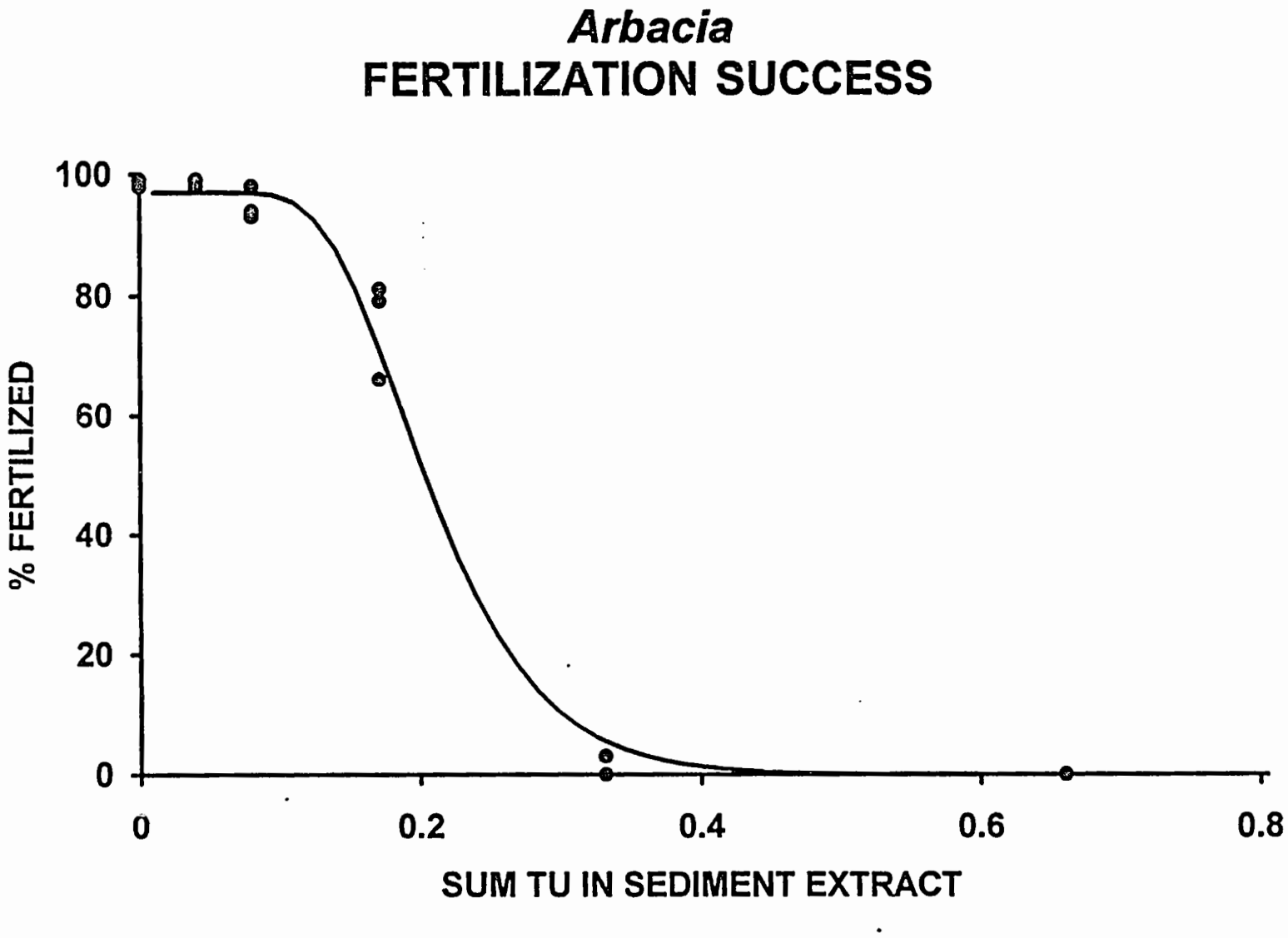

Figure 25. ΣTU exposure-response model for fertilization response of *Arbacia* to sediment extract exposure.

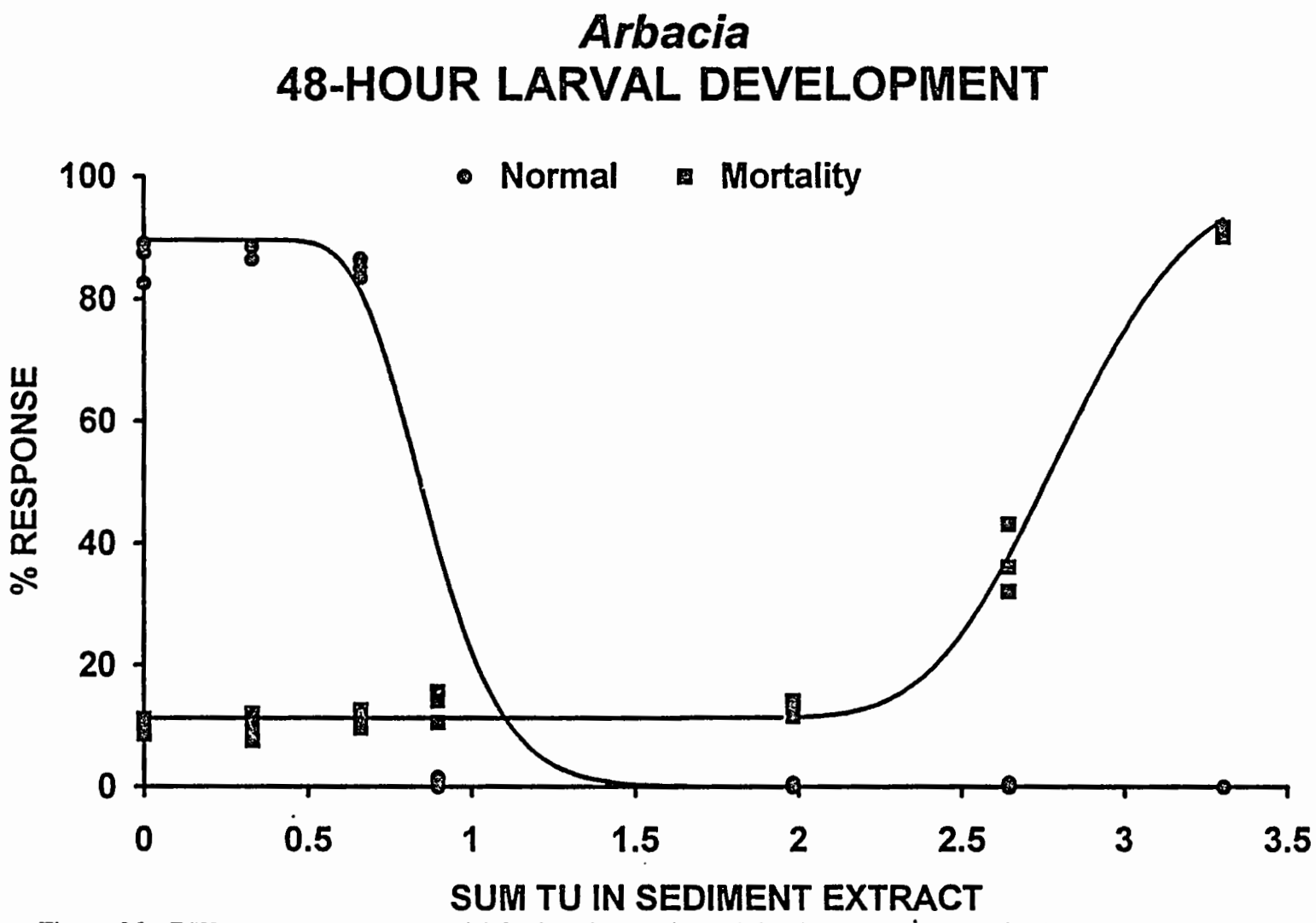

Figure 26. ΣTU exposure-response model for larval mortality and development response of *Arbacia* to sediment extract exposure.

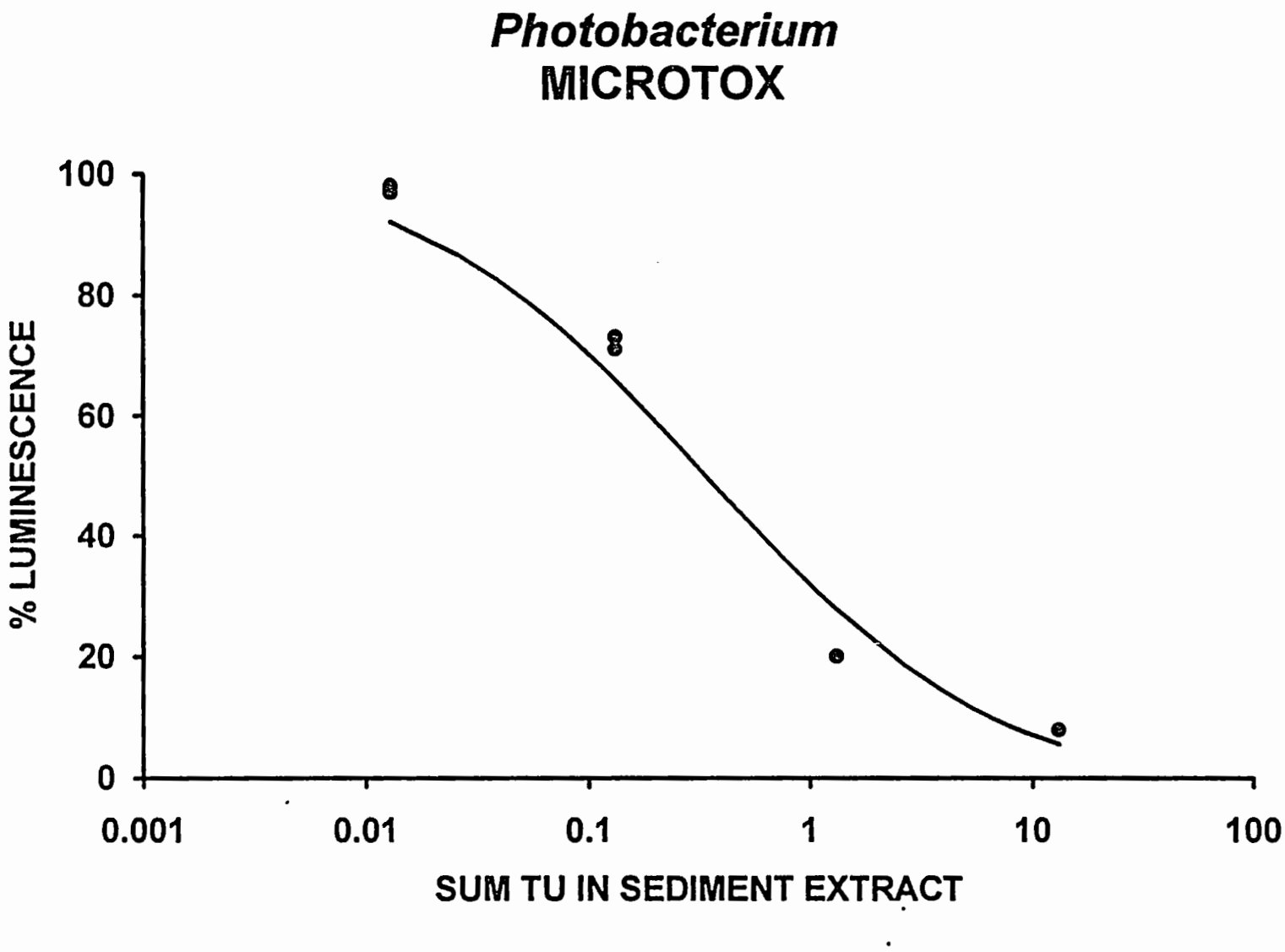

Figure 27. ΣTU exposure-response model for bioluminescence response of *Photobacterium* to sediment extract exposure.

Table 8. ΣTU exposure-response model parameter estimates and coefficients of determination.

Exposure Medium	Species/Endpoint	EC_{20}[a]	σ	R_0[b]	R^2
Seep water	*Arbacia*/fertilization	14.7	0.20	99.2	99.9
	Arbacia/normal development	3.58	0.16	92.3	99.4
	Champia/reproduction	2.46	0.26	23.1	89.7
	Mulinia/mortality	5.02	0.20	79.4	92.8
Sediment extract	*Arbacia*/fertilization	0.16	0.13	97.0	98.7
	Arbacia/normal development	0.73	0.09	89.6	
	Arbacia/mortality	2.55	0.05	88.7	99.9
	Photobacterium/mortality	0.049	1.0	100	98.7

[a] In units of ΣTU.
[b] Cystocarps/female for *Champia*, percent for all other.

determined by experimental design. Thus it is reasonable to question the adequacy of the models to describe or predict the responses of test populations (of the same species) different from those used the exposure-response assays, or the responses of organisms in Allen Harbor. To evaluate model validity, toxicity data were collated from Phases I and II (Munns *et al.* 1991, 1993) which could be compared with the predictions of the ΣTU models. Obviously, to be useful in this exercise, information concerning both exposure and the response of the appropriate species/endpoint needed to be available.

Of all biological measures obtained over the course of this project, only *Arbacia* fertilization data from water exposures met these criteria. Twelve exposure-response data pairs had been obtained as part of Phase II exercises (Munns *et al.* 1993). These were associated with both landfill seep (LANDN, LANDM, and LANDS), and runoff (SN, WC, NC) stations in the summer and fall of 1990. Validation of the *Arbacia* fertilization model proceeded by calculating ΣTU for each sample, and using the model in Table 8 (see Figure

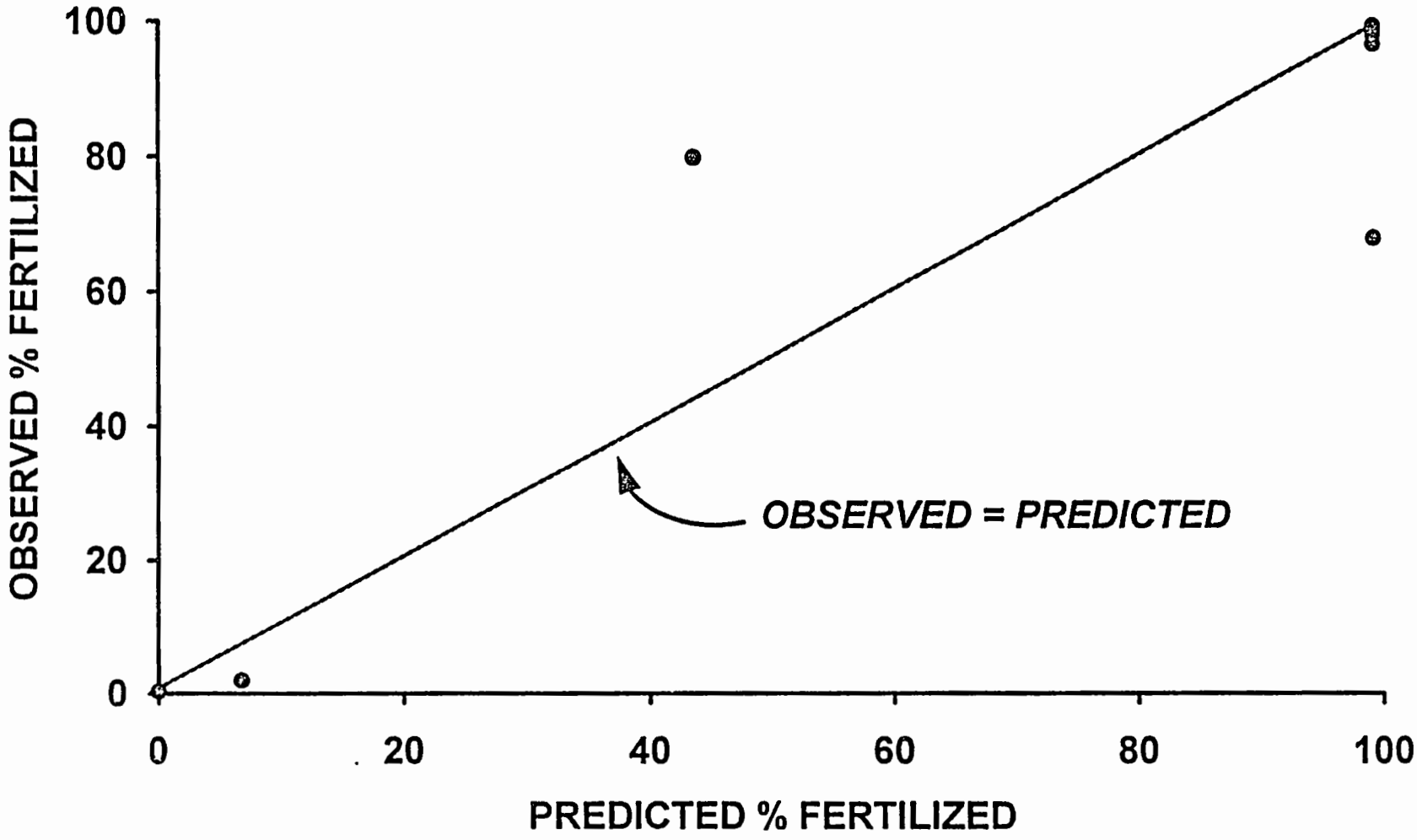

Figure 28. Comparison of predicted and observed *Arbacia* fertilization success.

21) to predict an associated fertilization response. These predictions were then compared with the response actually observed during Phase II. Of the 12 predictions, 10 (83%) accurately reflected the Phase II data, one overpredicted response, and one underpredicted response (Figure 28; five data points are obscured in this figure due to overlap with other points). The latter of the two mispredictions is the more troubling (risk managers likely would prefer to error on the conservative side), but is not surprising given the paucity of chemistry data utilized in model development. Thus the seep water-*Arbacia* fertilization model, at a minimum, appears to be useful in predicting risk within Allen Harbor. It should be noted, however, that insufficient data were available to adequately evaluate the model within the range of predicted partial (less than 90% but greater than 10%) effects.

Assuming the ΣTU exposure-response models to be reasonable predictors of biological response, the 8 models developed here should be valuable in quantifying risks directly associated with the Allen Harbor landfill. In this form, they are useful to environmental managers in at least two ways. With knowledge of the contaminant makeup of any particular site within the harbor, they can be used to establish associated ecological risk by summing the TU equivalents of chemical stressors, and estimating the probability of a particular ecological response using the appropriate model. Further, levels of remediation required to reduce risk (if deemed to be too high at a particular site) to some acceptable level can be established by restating the models in terms of an acceptable level of response and solving for the exposure term. (Defining acceptable levels of risk is beyond the scope of this project, and is best left up to the environmental and resource managers involved with the site.) Reductions in risk could theoretically be achieved by a remediation plan which decreases the concentrations of contaminants contributing the majority of risk (*i.e.*, those associated with the largest contributions to ΣTU).

Risk Quantified as Joint Probabilities

The ΣTU exposure-response models provide a means of estimating the degree of biological response expected given an understanding of contamination at a given site (say in immediate association with a landfill seep). Perhaps a more holistic approach to quantifying ecological risk in Allen Harbor would be to evaluate environmental conditions in the harbor

in its entirety, rather than on a site-by-site basis. To accomplish this, a probabilistic approach was applied which utilized the statistical distributions of exposure and expected biological effect to provide a direct, quantitative measure of ecological risk.

Conceptually, this procedure involves estimation of the joint probabilities of exposure and effects distributions, as illustrated in Figure 29. The graphic in the upper left-hand side of this figure represents the distribution of stressor concentration measured or modeled in space or time. Assumed to be log-normal, this distribution can be characterized by its associated geometric mean and standard deviation, and describes the probability of observing any particular stressor concentration within the bounds (again, spatial or temporal) of the risk assessment. The upper right-hand graphic illustrates an exposure-response model like those developed here. The EC_{50} and σ associated with this model also describe a statistical distribution, this time of the response thresholds to stressor concentration of the endpoint it models (as described previously). In moving from this depiction to the bottom graphic, this model, or cumulative distribution function, has been restated as a probability density function to correspond in form with the exposure distribution.

The area of overlap of these two distributions, shown at the bottom of Figure 29, defines the degree of risk expected within the system. It is within this region of existing stressor concentration that ecological effects are expected to occur. The probability that they will occur depends upon the probability of experiencing an exposure high enough to elicit an ecological response, or the joint probability of exposure and effects. The degree of overlap is therefore a quantitative measure of ecological risk.

In this type of analysis, overlap can be calculated directly by solving for the intersection area of the two distributions, or can be estimated using Monte Carlo simulation techniques. In this latter approach, the two distributions are artificially sampled (using a computer) in pair-wise fashion, and the value of the resulting exposure concentration is compared with that of the sampled effects threshold concentration. A biological response is expected to occur (and is scored as such) if the former is greater or equal to the latter. At the end of several such sampling events or iterations, the proportion of total iterations during which the biological threshold concentration was exceeded is calculated. Following standard sampling theory, this proportion estimates the probability of biological response, thus

RISK AS A JOINT PROBABILITY

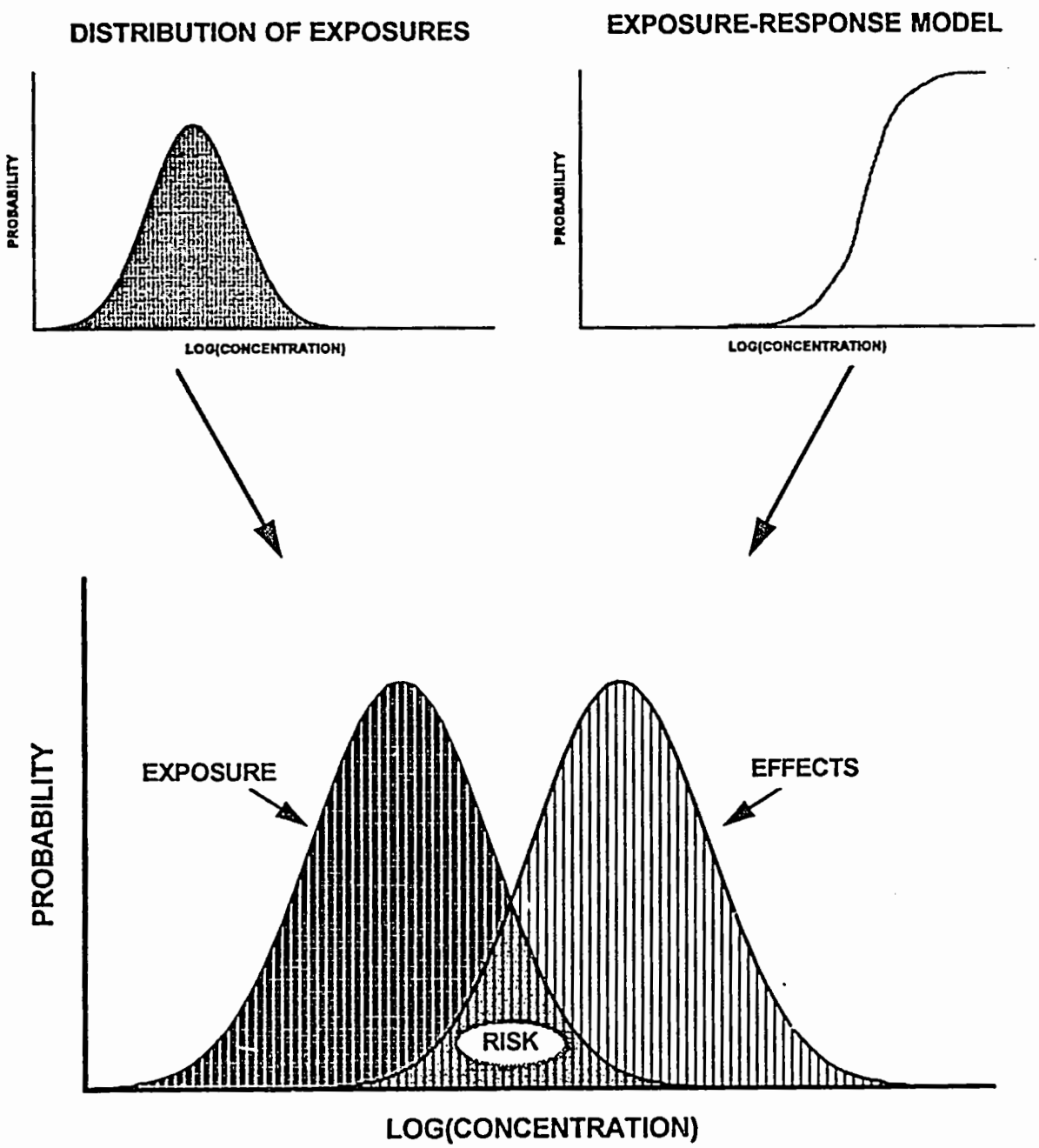

Figure 29. Characterization of ecological risk as a joint probability.

providing a probabilistic estimate of risk. As described, this method can be used to estimate probabilities associated with observing an ecological response, or with exceedence of some degree of response, such as an LC_{50} or EC_{50}.

To estimate risks to Allen Harbor pelagic and benthic systems, Monte Carlo simulations were conducted employing exposure data obtained throughout all three phases of this study, and the ΣTU exposure-response models derived for seep water and sediment extracts. Analyses of risks associated with these two systems differed somewhat due to the availability of data, and are described separately below.

Risk to pelagic system -- Due to reasons given elsewhere (Munns *et al.* 1991, 1993), little emphasis was placed in this study on characterizing pelagic exposure conditions through direct measurement of water column chemistry. However, chemistry data were available for water entering the harbor through landfill seeps and from runoff sources. These two data sets were used separately to quantify upper-bound risks (because they represent exposure media undiluted by harbor water) associated with each source.

Exposure distributions were derived for each source independently by calculating ΣTU for each sample (17 seep samples, 6 runoff samples), and determining the geometric means and standard deviations representing each source. Monte Carlo simulations were conducted with Crystal Ball® software using these distributions and the appropriate ΣTU exposure-response models, as described above. Five simulations, each involving 1,000 sampling iterations, were conducted to estimate joint probabilities. The five estimates of overlap were then averaged to yield an estimate of risk to each biological endpoint. Standard errors of these means were also calculated as a method of describing (minimal) uncertainty associated with each risk estimate.

Risks from landfill seep waters to the four endpoints ranged from 0.24 for *Arbacia* fertilization, to as high as 0.69 for *Champia* reproduction (Table 9). With the exception of *Arbacia* fertilization, risks to measured endpoints associated with landfill seeps were statistically higher than those from runoff sources (t-test, $P<0.05$). Although these estimates indicate the potential for negative ecological impact associated with both landfill and runoff sources, actual risks to the harbor pelagic system would be expected to be lower than these

estimates suggest, because both seep and runoff water would be diluted substantially upon mixing. Detailed water column measurements and/or transport studies would be necessary to fully characterize this risk.

Because these simulations were conducted to estimate total overlap between the exposure and effects distributions, the resulting risk estimates should be interpreted as describing the probability that *any* negative effect will occur. That is, in the case of *Arbacia* fertilization, there is a 24% chance that reduced fertilization success would be observed for sea urchin gametes in immediate proximity to the landfill. The magnitudes of such effects were not evaluated in this analysis.

Table 9. Mean probabilities of maximum risk to pelagic systems.

Endpoint	Seep	Runoff
Arbacia fertilization	0.243 (0.004)[a]	0.252 (0.004)
Arbacia development	0.644 (0.012)	0.688 (0.005)
Champia reproduction	0.688 (0.011)	0.498 (0.003)
Mulinia survival	0.529 (0.007)	0.403 (0.006)

[a] Standard error of the mean.

Risk to benthic system -- While full effects distributions could not be generated due to the lack of response observed in whole sediment assays (and thus an incomplete exposure-response description), it is straightforward to conclude minimal risks to Allen Harbor benthic system, at least as evaluated by *Ampelisca* and *Mulinia* responses, without actual calculation of joint probabilities. Such a conclusion is supported by *Ampelisca* assays results and general descriptions of the conditions of benthic populations obtained during the initial phase of this study (Munns *et al.* 1991). However, the sediment extract models provided an additional means of evaluating benthic risk. These models were used to calculate both upper-bound, maximum probabilities of risk, as well as more likely estimates which incorporated an understanding of contaminant bioavailability.

The extraction procedure was employed to isolate the entire quantity of nonionic

organic contaminants associated with the whole sediment. It was thus possible to relate contaminant concentrations in the whole sediment to those measured in the extract, and to use this relationship to derive expectations of the concentrations of contaminants in extract exposure media from harbor samples, had they been extracted. These sediment extract equivalents, quantified in terms of ΣTU, represent the maximum exposure potential of harbor samples assuming all contaminants to be bioavailable. Joint probability calculations employing sediment extract equivalents to describe the exposure distribution therefore yielded upper-bound probabilities of risk.

Sediment extract equivalents were calculated for 42 sediment samples collected in Allen Harbor throughout the course of this project (5 during Phase I, 37 during Phase II) as follows. Concentrations of relevant contaminants were normalized to those of the LANDM whole sediment sample used to generate the sediment extract exposure medium. These ratios were then multiplied by the contaminant-specific TUs determined for the LANDM extract sample to produce a TU estimate for each contaminant in each field sample. TUs were summed, and geometric means and standard deviations calculated to describe the statistical distribution of exposure. This distribution was then used in Monte Carlo simulations with sediment extract ΣTU exposure-response models. As described for simulations involving waters, five simulations involving 1,000 iterations each were conducted to characterize risk to modeled endpoints.

As indicated in Table 10, Allen Harbor sediments have the substantial potential to impact the four biological endpoints. Probabilities of maximum risk to *Arbacia* fertilization and development were estimated as unity, and maximum risks to *Arbacia* survival and *Photobacterium* survival were greater than 80%. These predictions are borne out to some extent by *Arbacia* fertilization and development endpoint data obtained during Phase I: Allen Harbor sediment extract concentrations as low as 0.2 and 0.05% were observed to impact these endpoints (Munns *et al.* 1991). Similarly, Allen Harbor sediment interstitial waters also impacted these endpoints at concentrations as low as 12.5% (Munns *et al.* 1991).

To aid in the interpretation of maximum risks calculated for Allen Harbor sediments, a similar analysis was conducted using sediment data obtained from Mount View (MV),

identified as a mid-Narragansett Bay reference station in Phases I and II (Munns *et al.* 1991, 1993). Results similar to those for Allen Harbor were obtained with one exception (Table 10): risk to *Arbacia* survival was calculated to be 0 at MV. Lower contaminant levels and the steepness of the exposure-response curve for this endpoint likely contributed to this result.

Table 10. Mean probabilities of maximum risk to benthic systems.

Endpoint	AH	MV
Arbacia fertilization	1.0 (0.0)[a]	1.0 (0.0)
Arbacia development	1.0 (0.012)	1.0 (0.005)
Arbacia survival	0.814 (0.009)	0.0 (0.0)
Photobacterium survival	0.844 (0.007)	0.769 (0.003)

[a] Standard error of the mean.

The above calculations assume all contaminants to be available to benthic organisms. Typically, however, some fraction of organic contaminants associated with sediments is bound in dynamic equilibrium to the organic carbon of those sediments, and is therefore unavailable to biota. Evidence suggests that for nonionic organics, exposure conditions are better represented by concentrations of contaminants measured in pore water than by bulk sediment chemistry (see Di Toro *et al.* 1991). Since the sediment extract equivalents employed above reflect harbor sample bulk chemistry, a more reasonable approach to evaluating risks would incorporate the bioavailability of organic compounds in estimating exposure distributions. Following the equilibrium partitioning approach described in Di Toro *et al.* (1991), concentrations of organic contaminants expected in harbor sediment pore water were calculated using the relationship:

$$C_d = C_s / (f_{oc} \times K_{oc})$$

where C_d is the concentration (μg/L) in pore water, C_s is the bulk concentration (ng/g) in the sediment, f_{oc} is the fraction of organic carbon in the sediment (see Appendix G), and K_{oc} is the partition coefficient for sediment organic carbon, expressed in terms of (ng chemical/g

organic carbon)/(μg chemical/L pore water). When K_{oc} is not known, it can be estimated from the chemical's octanol-water partition coefficient (K_{ow}) using the regression relationship (Di Toro *et al.* 1991):

$$K_{oc} = \text{antilog}\{0.00028 + 0.983[\log(K_{ow})]\}.$$

K_{ow}s (from MacKay *et al.* 1992 and U.S. EPA 1984) and K_{oc}s for the nonionic organic contaminants used in predicting pore water concentrations are given in Table 11.

Using the pore water concentrations predicted in this manner, TUs were calculated, summed, and geometric means and standard deviations calculated to describe the new statistical distribution of exposure from AH sediments. This distribution was then used in Monte Carlo simulations with sediment extract ΣTU exposure-response models. As with earlier risk estimation procedures, five simulations involving 1,000 iterations each were conducted to characterize risk to modeled endpoints.

With contaminant bioavailability taken into account, estimates of risks to benthic systems in Allen Harbor (Table 12) were substantially reduced from those calculated using sediment extract equivalents, particularly with respect to *Arbacia* development and survival. Some degree of risk was still indicated for *Arbacia* fertilization and the Microtox endpoint, however.

Thus, the discrepancy between the degrees of risk concluded from evaluations involving whole sediment and sediment extracts most likely reflects differences in the availability of organic contaminants in the two media. The extraction procedure is, by design, highly efficient at isolating organic contaminants from binding matrices in the sediment. Such factors as organic carbon reduce the availability of organic

Table 11. Partition coefficients used in pore water contaminant concentration calculations.

Contaminant	K_{ow}	K_{oc}
PCB (as Aroclor 1254)	6.47	6.36
Chlordane	5.54	5.45
DDT	5.75	5.65

contaminants in natural sediments, such that bulk chemistry measurements tend to overestimate actual exposure conditions. These contaminants are bioavailable in the sediment extract, however. Thus an analysis of sediment risks may be overly conservative when involving sediment extracts. It also is possible, however, that differences in sensitivity exist between endpoints of species used in whole sediment exposures and those of species involved in sediment extract model development.

Table 12. Mean probabilities of risk to benthic systems incorporating contaminant bioavailability.

Endpoint	AH
Arbacia fertilization	.755 (0.006)*
Arbacia development	0.317 (0.009)
Arbacia survival	0.083 (0.007)
Photobacterium survival	0.550 (0.010)

* Standard error of the mean.

A final word concerning uncertainties associated with the estimates of risk developed here is cogent. While attempts were made to quantify the uncertainties of the final risk calculations themselves through performance of multiple simulations, little regard was given to uncertainties introduced through exposure media sampling error, assay performance, and chemical analysis. The lack of quantification of these sources of error (and others) weakens the confidence with which conclusions can be drawn regarding this ecological risks to Allen Harbor. It also should be noted that the effects endpoints evaluated in this study generally are short-term in nature; the effects of some contaminants in the harbor may require longer time periods to manifest. Despite these caveats, the probabilities estimated above should serve to identify the degree to which ecological systems are at risk from landfill contaminants. A long-term monitoring program would provide information to verify this risk assessment and to assist in management of Allen Harbor.

RISK OF NEOPLASTIC DISEASE DEVELOPMENT IN *MYA*

Although a possible relationship between NCBC Davisville and hematopoietic neoplasia in *Mya arenaria* has been a focus of attention throughout the entire Risk Assessment Pilot Study, efforts undertaken in all three phases have failed to conclusively link disease etiology with chemical contamination at the facility. In a final evaluation of the risk of neoplastic disease development in relation to sediment contamination, attempts were made to correlate rates of Hn in *Mya* observed by histocytological techniques during the Phase II survey with corresponding exposure concentrations measured during Phases I, II, and III. Chemical information was available for nine stations visited during the survey: LANDM, LANDN, AH10, PR, SN, NC, CC, FDA, and MP (see Munns *et al.* 1991, 1993 for original station descriptions).

These analyses indicated no relationship between the incidence of Hn and sediment contamination. As an illustration of this, 23% of the clams examined at FDA during Phase II (Munns *et al.* 1993) were afflicted with Hn while contaminant levels were between 10 and 100 times lower than at SN, a station at which no Hn was diagnosed. Similar efforts by Brown (1980) and Mix (1986), among others, also have failed to link *Mya* neoplasia with sediment contamination. Additionally, Appeldoorn *et al.* (1984) were unable to relate Hn to sediment contamination in laboratory exposures. Recently, Chang *et al.* (1993) conclusively demonstrated Hn to be caused by a retrovirus. The general lack of observed association between environmental contamination and disease etiology in laboratory experiments and field studies strongly suggests the risk of *Mya* hematopoietic neoplasia development relative to chemical contamination at NCBC Davisville to be minimal.

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APPENDIX A

TRACE METAL CONCENTRATIONS

TABLE A-1. TRACE METAL CONCENTRATIONS (μg/L) IN SEEP WATERS

SAMPNUM	STATION	CONC(%)	Cu	Zn	Cr	Pb	Ni	Cd	Mn	Fe	As
798904	BRINE	0	ND	ND	5	ND	ND	ND	1	3	ND
798909	LANDM	5.8	3	29	ND	ND	1	ND	1	19	ND
798908	LANDM	11.5	6	62	ND	ND	2	ND	9	124	ND
798907	LANDM	23	11	90	ND	3	5	2	18	303	ND
798906	LANDM	46	16	152	ND	4	9	5	26	563	ND
798905	LANDM	92	16	249	3	4	19	14	13	252	ND
798902	LANDM	100	16	322	ND	5	28	21	59	ND	ND

TABLE A-2. TRACE METAL CONCENTRATIONS (μg/g) IN SEDIMENTS

SAMPNUM	STATION	CONC(%)	Cu	Zn	Cr	Pb	Ni	Cd	Mn	Fe	As
798900	AH	100	455	1110	109	515	58.5	6.52	186	49600	7.74
798910	PR	100	2.51	17.6	5.31	2.76	3.42	0.06	45.8	4400	0.67
798912	POTO	100	29.4	98.5	24.6	20.5	13.4	0.89	128	18200	4.83
798939	CLIS	100	53.2	155	59.9	34.1	26.2	0.05	499	27300	5.21
798915	LANDM	12.5	86.5	219	31.7	73.9	18.8	1.59	134	23800	5.29
798914	LANDM	25	120	349	40.0	118	24.4	2.07	145	25500	6.20
798913	LANDM	50	196	489	50.6	190	31.7	3.21	160	30800	6.15
798903	LANDM	100	290	708	63.9	275	39.4	4.26	169	37000	8.25

TABLE A-3. TRACE METAL CONCENTRATIONS (μg/g DRY WT) IN *Mya arenaria*

SAMPNUM	STATION	Cu	Zn	Cr	Pb	Ni	Cd	Mn	Fe	As
798901	SALTPOND	14.5	64.8	0.690	3.70	0.00	0.130	54.9	445	4.87
798918	AH	35.2	97.8	3.72	61.4	1.99	0.61	26.0	18700	12.3
798922	PR	11.0	43.1	2.10	5.39	0.93	0.49	21.6	1350	5.70

APPENDIX B

PESTICIDE CONCENTRATIONS

TABLE B-1. PESTICIDE CONCENTRATIONS (μg/L) IN SEEP WATERS

SAMPNUM	STATION	CONC(%)	HCB	alpha-BHC	gamma-BHC	alpha-Chlordane	gamma-Chlordane	p,p'-DDE	p,p'-DDD	p,p'-DDT
798904	BRINE	0	ND	ND	ND	ND	ND	ND	ND	ND
798909	LANDM	5.8	NM	NM	NM	NM	NM	NM	NM	NM
798908	LANDM	11.5	NM	NM	NM	NM	NM	NM	NM	NM
798907	LANDM	23	NM	NM	NM	NM	NM	NM	NM	NM
798906	LANDM	46	NM	NM	NM	NM	NM	NM	NM	NM
798905	LANDM	92	NM	NM	NM	NM	NM	NM	NM	NM
798902	LANDM	100	ND	ND	ND	ND	ND	ND	ND	ND

TABLE B-2. PESTICIDE CONCENTRATIONS (ng/g DRY WT) IN SEDIMENTS

SAMPNUM	STATION	CONC(%)	HCB	alpha-BHC	gamma-BHC	alpha-Chlordane	gamma-Chlordane	p,p'-DDE	p,p'-DDD	p,p'-DDT
798900	AH	100	3.80	0.33	0.24	2.10	3.55	8.03	58.5	43.6
798910	PR	100	0.05	0.02	0.06	0.06	0.05	0.05	0.17	0.08
798912	POTO	100	0.04	0.04	0.13	0.41	0.56	0.39	0.91	0.30
798939	CLIS	100	0.07	0.16	0.06	0.27	1.00	0.89	1.19	0.49
798915	LANDM	12.5	0.23	0.06	0.21	0.82	1.22	1.75	4.74	1.27
798914	LANDM	25	0.35	0.07	0.35	1.09	1.59	2.36	8.12	2.21
798913	LANDM	50	0.56	0.10	0.22	1.14	1.81	2.61	11.9	3.17
798903	LANDM	100	0.72	0.15	0.53	2.13	3.31	4.08	26.4	14.1

TABLE B-3. PESTICIDE CONCENTRATIONS (ng/g DRY WT) IN *Mya arenaria*

SAMPNUM	STATION	HCB	alpha-BHC	gamma-BHC	alpha-Chlordane	gamma-Chlordane	p,p'-DDE	p,p'-DDD	p,p'-DDT
798901	SALT POND	0.54	0.55	0.55	0.66	0.74	1.18	0.75	0.64
798918	AH	1.03	0.46	ND	0.73	1.14	1.35	2.18	1.23
798922	PR	0.66	0.71	0.64	0.44	1.39	1.46	0.88	0.81

APPENDIX C

POLYCHLORINATED BIPHENYL CONGENER CONCENTRATIONS

TABLE C-1. PCB CONGENER CONCENTRATIONS (μg/L) IN SEEP WATERS

SAMPNUM	STATION	CONC(%)	CB052	CB047	CB101	CB151	CB118	CB153	CB138	CB128	CB180	CB195	CB194	CB206	CB209
798904	BRINE	0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
798909	LANDM	5.8	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
798908	LANDM	11.5	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
798907	LANDM	23	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
798906	LANDM	46	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
798905	LANDM	92	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
798902	LANDM	100	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

TABLE C-2. PCB CONGENER CONCENTRATIONS (ng/g DRY WT) IN SEDIMENTS

SAMPNUM	STATION	CONC(%)	CB052	CB047	CB101	CB151	CB118	CB153	CB138	CB128	CB180	CB195	CB194	CB206	CB209
798900	AH	100	11.8	2.44	23.1	8.74	30.2	34.7	42.0	2.05	26.1	2.60	5.74	3.52	2.40
798910	PR	100	0.35	0.19	0.22	ND	0.20	0.20	0.20	ND	0.20	ND	ND	ND	ND
798912	POTO	100	0.76	0.30	0.99	0.20	1.30	1.00	1.30	ND	0.60	0.20	0.10	0.45	0.40
798939	CLIS	100	2.23	1.43	3.60	1.20	4.37	5.83	4.50	0.20	2.53	0.50	0.87	1.33	1.37
798915	LANDM	12.5	2.57	0.90	4.18	1.80	5.10	7.00	7.60	ND	6.00	0.50	1.30	1.30	1.00
798914	LANDM	25	3.77	1.40	6.62	2.80	7.70	11.0	11.6	ND	9.10	0.80	2.10	1.70	1.20
798913	LANDM	50	4.39	1.40	8.93	4.50	11.1	17.0	17.6	ND	14.9	1.50	3.30	2.50	2.80
798903	LANDM	100	6.20	1.83	13.4	6.43	18.5	25.9	26.7	1.13	22.7	2.17	5.30	2.97	1.73

TABLE C-3. PCB CONGENER CONCENTRATIONS (ng/g DRY WT) IN *Mya arenaria*

SAMPNUM	STATION	CB052	CB047	CB101	CB151	CB118	CB153	CB138	CB128	CB180	CB195	CB194	CB206	CB209
798901	SALT POND	9.05	2.13	7.41	1.40	5.50	6.43	4.43	0.23	2.13	ND	ND	ND	ND
798918	AH	6.54	3.11	11.1	3.80	7.40	14.1	10.3	ND	6.00	0.20	0.60	0.30	ND
798922	PR	20.9	9.92	16.4	2.10	13.9	11.7	7.30	0.40	9.20	ND	ND	ND	0.100

APPENDIX D

TOTAL POLYCHLORINATED BIPHENYL CONCENTRATIONS

TABLE D-1. TOTAL PCB CONCENTRATIONS (μg/L) IN SEEP WATERS

SAMPNUM	STATION	CONC(%)	Aroclor 1242	Aroclor 1254	TOTAL PCB
798904	BRINE	0	ND	ND	ND
798909	LANDM	5.8	NM	NM	NM
798908	LANDM	11.5	NM	NM	NM
798907	LANDM	23	NM	NM	NM
798906	LANDM	46	NM	NM	NM
798905	LANDM	92	NM	NM	NM
798902	LANDM	100	ND	ND	ND

TABLE D-2. TOTAL PCB CONCENTRATIONS (ng/g DRY WT) IN SEDIMENTS

SAMPNUM	STATION	CONC(%)	Aroclor 1242	Aroclor 1254	TOTAL PCB
798900	AH	100	39.0	1400	1440
798910	PR	100	ND	7.70	7.70
798912	POTO	100	ND	34.7	34.7
798939	CLIS	100	6.49	117	123
798915	LANDM	12.5	ND	243	240
798914	LANDM	25	ND	378	380
798913	LANDM	50	ND	608	610
798903	LANDM	100	5.92	1030	1040

TABLE D-3. TOTAL PCB CONCENTRATIONS (ng/g DRY WT) IN *Mya arenaria*

SAMPNUM	STATION	Aroclor 1242	Aroclor 1254	TOTAL PCB
798901	SALT POND	ND	76.3	76.3
798918	AH	ND	226	230
798922	PR	99.6	247	350

APPENDIX E

POLYCYCLIC AROMATIC HYDROCARBON CONCENTRATIONS

TABLE E-1. PAH CONCENTRATIONS (μg/L) IN SEEP WATERS

SAMPNUM	798904	798909	798908	798907	798906	798905	798902
STATION	BRINE	LANDM	LANDM	LANDM	LANDM	LANDM	LANDM
CONC (%)	0	5.8	11.5	23	46	92	100
Fluorene	ND	NM	NM	NM	NM	NM	ND
Phenanthrene	ND	NM	NM	NM	NM	NM	0.011
Anthracene	ND	NM	NM	NM	NM	NM	ND
Sum MW178-C1	ND	NM	NM	NM	NM	NM	ND
Sum MW178-C2	ND	NM	NM	NM	NM	NM	ND
Sum MW178-C3	ND	NM	NM	NM	NM	NM	ND
Sum MW178-C4	ND	NM	NM	NM	NM	NM	ND
Fluoranthene	ND	NM	NM	NM	NM	NM	0.034
Pyrene	ND	NM	NM	NM	NM	NM	0.041
Benz[a]anthracene	ND	NM	NM	NM	NM	NM	0.015
Chrysene	ND	NM	NM	NM	NM	NM	0.021
Sum MW228	ND	NM	NM	NM	NM	NM	0.044
Tinuvin 328	ND	NM	NM	NM	NM	NM	ND
Tinuvin 327	ND	NM	NM	NM	NM	NM	ND
Sum Benzofluoranthenes	ND	NM	NM	NM	NM	NM	0.060
Benzo[e]pyrene	ND	NM	NM	NM	NM	NM	0.029
Benzo[a]pyrene	ND	NM	NM	NM	NM	NM	0.022
Perylene	ND	NM	NM	NM	NM	NM	ND
Indeno[123-cd]pyrene	ND	NM	NM	NM	NM	NM	0.017
Dibenz[ah]anthracene	ND	NM	NM	NM	NM	NM	ND
Benzo[ghi]perylene	ND	NM	NM	NM	NM	NM	0.025
Sum MW276	ND	NM	NM	NM	NM	NM	0.044
Sum MW278	ND	NM	NM	NM	NM	NM	0.012
Sum MW302	ND	NM	NM	NM	NM	NM	0.015
Coronene	ND	NM	NM	NM	NM	NM	ND
LOD	0.007						0.007

TABLE E-2. PAH CONCENTRATIONS (ng/g DRY WT) IN SEDIMENTS

SAMPNUM	798910	798912	798900	798903	798913	798914	798915	798939
STATION	PR	POTO	AH	LANDM	LANDM	LANDM	LANDM	CLIS
CONC (%)	100	100	100	100	50	25	12%	100
Fluorene	15	4.3	144	153	34.1	35.1	19.7	13.2
Phenanthrene	132	40.7	346	955	394	398	229	155
Anthracene	19.7	9.09	265	346	109	106	62.5	36.9
Sum MW178-C1	52	40.5	595	507	198	186	95.8	130
Sum MW178-C2	32.4	37.2	351	300	135	122	67.9	118
Sum MW178-C3	13.1	22.1	102	140	77.8	59.9	37.5	61.6
Sum MW178-C4	3.24	12.8	40.3	51.5	27.8	21.1	14.2	25.2
Fluoranthene	170	118	561	1350	845	887	534	413
Pyrene	143	104	815	1240	778	808	445	448
Benz[a]anthracene	47.6	38.5	1350	963	422	477	201	180
Chrysene	54.1	51.9	1190	1120	484	444	225	243
Sum MW228	116	108	3110	2390	1040	1060	488	487
Tinuvin 328	6.28	302	388	495	387	326	334	2.67
Tinuvin 327	1.44	56.1	60.7	81.2	68.5	60.8	57.6	ND
Sum Benzofluoranthenes	91.9	105	2670	1890	853	866	413	537
Benzo[e]pyrene	33.2	39.2	1100	677	310	303	149	216
Benzo[a]pyrene	47.3	42.8	1330	859	368	384	173	250
Perylene	13.4	29.5	432	286	129	132	69.8	83.6
Indeno[123-cd]pyrene	28	33.7	544	473	216	203	109	191
Dibenz[ah]anthracene	8.76	10.6	187	186	83.9	78.9	40.1	55.9
Benzo[ghi]perylene	29.9	34.6	540	477	222	207	118	213
Sum MW276	80.1	91.2	1440	1290	588	552	304	525
Sum MW278	32.1	38.6	547	637	270	249	142	203
Sum MW302	61	70.3	800	907	421	377	234	440
Coronene	9.65	13	90	103	54.4	49.6	35.3	74.7
LOD	0.608	1.16	1.15	1.77	1.32	1.32	1.45	1.83

TABLE E-3. PAH CONCENTRATIONS (ng/g DRY WT) IN *Mya arenaria*

SAMPNUM	798901	798918	798922
STATION	SALT POND	AH	PR
CONC (%)	100	100	100
Fluorene	ND	ND	ND
Phenanthrene	13.6	23.2	8.56
Anthracene	ND	ND	ND
Sum MW178-C1	13.1	19.2	14.1
Sum MW178-C2	12.5	29.6	25.2
Sum MW178-C3	ND	13	14.8
Sum MW178-C4	ND	ND	ND
Fluoranthene	33.4	68.4	26.6
Pyrene	33.9	72.9	34.4
Benz[a]anthracene	11	26.4	7.67
Chrysene	23.4	47.1	16.5
Sum MW228	35.8	79.9	28.9
Tinuvin 328	14.1	36.2	13.3
Tinuvin 327	7.93	14.1	6.68
Sum Benzofluoranthenes	29.5	91	25.7
Benzo[e]pyrene	23.1	53.6	22.4
Benzo[a]pyrene	10.3	33.4	9.79
Perylene	5.48	17.1	ND
Indeno[123-cd]pyrene	ND	19.7	ND
Dibenz[ah]anthracene	ND	6.79	ND
Benzo[ghi]perylene	11.2	32.3	12.1
Sum MW276	16.3	52.3	16.8
Sum MW278	ND	7.2	ND
Sum MW302	ND	ND	ND
Coronene	ND	ND	ND
LOD	6.21	6.67	6.15

APPENDIX F

ACID VOLATILE SULFIDE CONCENTRATIONS IN SEDIMENTS

TABLE F-1. AVS CONCENTRATIONS (μMOL/g DRY WT) IN SEDIMENTS

SAMPNUM	STATION	CONC (%)	AVS
98912	POTO	100	53.64
98903	LANDM	100	51.0

APPENDIX G

TOTAL ORGANIC CARBON CONCENTRATIONS IN SEDIMENTS

TABLE G-1. TOC CONCENTRATIONS IN SEDIMENTS

SAMPNUM	STATION	CONC (%)	Dry Loss (%)	Carbonate (%)	Total C (%)	Organic C (%)
798912	POTO	100	55.83	<0.02	3.16	3.16
798903	LANDM	100	44.41	<0.02	3.74	3.74
798400	LANDM	100		<0.02	1.86	1.86
798406	LANDS	100		0.08	1.57	1.49
798412	WC	100		0.03	5.38	5.35
798414	SN	100		<0.02	1.07	1.07
798416	LANDN	100		0.04	3.13	3.09
798419	BI	100		0.03	1.50	1.47
798435	MV1	100		0.12	3.99	3.87
798436	MV1	100		0.06	3.71	3.65
798437	MV1	100		0.46	0.56	0.10
798438	MV1	100		0.12	3.75	3.63
798444	AH7	100		0.04	2.81	2.77
798445	AH7	100		0.04	3.12	3.08
798446	AH7	100		0.07	3.76	3.69
798447	AH7	100		0.17	3.18	3.01
798453	AH2	100		0.04	3.20	3.16
798454	AH2	100		<0.02	4.26	4.26
798455	AH2	100		0.02	2.37	2.35
798465	LAB	100		0.11	1.65	1.54
798466	LAB	100		0.09	1.09	1.00
798467	LAB	100		0.15	1.23	1.08
798468	LAB	100		0.12	1.35	1.23
798635	AH7	100		0.03	3.21	3.18
798636	AH7	100		0.06	3.33	3.27
798637	AH7	100		0.04	3.43	3.39
798644	AH2	100		0.04	3.26	3.22
798645	AH2	100		0.02	2.88	2.86
798646	AH2	100		0.03	3.36	3.33
798653	AH2	100		0.53	3.17	2.64
798654	MV1	100		0.21	4.06	3.85
798655	MV1	100		0.88	2.42	1.54
798665	LAB	100		0.18	1.30	1.12
798666	LAB	100		0.08	1.23	1.15
798667	LAB	100		0.08	1.24	1.16
798760		100		<0.02	1.97	1.97
798731	WC	100		<0.02	1.71	1.71
798732	SN	100		<0.02	0.77	0.77
798794	MV1	100		0.47	3.21	2.74
798805	NC	100		<0.02	3.50	3.50
798806	WC	100		<0.02	2.53	2.53
798807	SN	100		<0.02	0.79	0.79
798824	LAB	100		0.11	1.68	1.57
798825	LAB	100		0.30	2.01	1.71
798826	LAB	100		0.11	1.21	1.10

TABLE G-1 (cont). TOC CONCENTRATIONS IN SEDIMENTS

SAMPNUM	STATION	CONC (%)	Dry Loss (%)	Carbonate (%)	Total C (%)	Organic C (%)
798828	AH7	100		0.02	3.04	3.02
798829	AH7	100		0.02	2.73	2.71
798830	AH7	100		0.05	3.59	3.53
798832	AH2	100		0.02	2.79	2.77
798833	AH2	100		0.02	3.17	3.15
798834	AH2	100		0.03	3.48	3.45
798836	MV1	100		0.36	3.70	3.34
798837	MV1	100		0.32	3.50	3.18
798838	MV1	100		0.36	3.05	2.69
798900	AH	100		0.08	4.67	4.59
798903	LANDM	100		0.07	3.77	3.70
798910	PR	100		<0.02	0.56	0.56
798912	POTO	100		0.10	2.98	2.88
798913	LANDM	100		0.05	2.63	2.58
798914	LANDM	100		0.04	2.75	2.71
798915	LANDM	100		0.15	2.66	2.51
798939	CLIS	100		0.17	1.81	1.64
798945	AH7	100		0.02	1.55	1.53
798946	AH7	100		0.15	1.47	1.32

APPENDIX H

SEEP WATER BIOASSAY RESULTS

TABLE H-1. *Arbacia punctulata* FERTILIZATION TEST ON SEEP SAMPLES

SAMPNUM	STATION	CONC (%)	REPLICATE	% FERTILIZATION
798904	BRINE+DI	0	1	100
798904	BRINE+DI	0	2	100
798904	BRINE+DI	0	3	99
56886	NSW	0	1	99
56886	NSW	0	2	100
56886	NSW	0	3	100
798909	LANDM	5.8	1	99
798909	LANDM	5.8	2	100
798909	LANDM	5.8	3	100
798908	LANDM	11.5	1	100
798908	LANDM	11.5	2	98
798908	LANDM	11.5	3	99
798907	LANDM	23	1	98
798907	LANDM	23	2	100
798907	LANDM	23	3	96
798906	LANDM	46	1	89
798906	LANDM	46	2	94
798906	LANDM	46	3	90
798905	LANDM	92	1	42
798905	LANDM	92	2	53
798905	LANDM	92	3	47

TABLE H-2. *Arbacia punctulata* 48 HOUR LARVAL DEVELOPMENT TEST ON SEEP SAMPLES

SAMPNUM	STATION	CONC (%)	% NORMAL	% MORTALITY	%ABNORMAL
	NSW	0	96.3	3.3	0.4
	NSW	0	93.3	5.7	1.0
	NSW	0	93.3	5.9	0.8
	NSW	0	94.7	4.8	0.5
	NSW	0	92.7	6.1	1.2
	NSW	0	96.2	3.3	0.5
798909	LANDM	5.8	93.5	3.2	3.3
798909	LANDM	5.8	83.1	8.7	8.2
798909	LANDM	5.8	92.5	3.1	4.4
789908	LANDM	11.5	82.8	5.1	12.1
789908	LANDM	11.5	86.1	2.7	11.2
789907	LANDM	23	29.6	6.2	64.2
789907	LANDM	23	23.8	6.4	69.8
789907	LANDM	23	27.1	6.7	66.2
798906	LANDM	46	0.0	15.4	84.6
798906	LANDM	46	2.4	16.5	81.1
798906	LANDM	46	1.0	17.4	81.6
798905	LANDM	92	0.0	34.2	65.8
798905	LANDM	92	0.0	22.0	78.0
798905	LANDM	92	0.0	18.7	81.3

TABLE H-3. *Mysidopsis bahia* 48 HOUR ACUTE TEST ON SEEP SAMPLES

SAMPNUM	STATION	CONC (%)	REPLICATE	% MORTALITY
	NSW	0	1	0.0
	NSW	0	2	0.0
	NSW	0	3	0.0
	NSW	0	4	20.0
798904	BRINE+DI	0	1	0.0
798904	BRINE+DI	0	2	0.0
798904	BRINE+DI	0	3	0.0
798904	BRINE+DI	0	4	0.0
798909	LANDM	5.8	1	0.0
798909	LANDM	5.8	2	0.0
798909	LANDM	5.8	3	0.0
798909	LANDM	5.8	4	0.0
789908	LANDM	11.5	1	0.0
789908	LANDM	11.5	2	0.0
789908	LANDM	11.5	3	0.0
789908	LANDM	11.5	4	0.0
789907	LANDM	23	1	20.0
789907	LANDM	23	2	20.0
789907	LANDM	23	3	0.0
789907	LANDM	23	4	0.0
798906	LANDM	46	1	0.0
798906	LANDM	46	2	0.0
798906	LANDM	46	3	0.0
798906	LANDM	46	4	0.0
798905	LANDM	92	1	0.0
798905	LANDM	92	2	0.0
798905	LANDM	92	3	0.0
798905	LANDM	92	4	0.0

TABLE H-4. *Champia parvula* SEXUAL REPRODUCTION TEST ON SEEP SAMPLES

SAMPNUM	STATION	CONC (%)	REPLICATE	CYSTOCARPS
	NSW	0	A1	22
	NSW	0	A2	27
	NSW	0	A3	30
	NSW	0	A4	22
	NSW	0	A5	38
	NSW	0	B1	28
	NSW	0	B2	20
	NSW	0	B3	14
	NSW	0	B4	19
	NSW	0	B5	17
	NSW	0	C1	18
	NSW	0	C2	12
	NSW	0	C3	24
	NSW	0	C4	10
	NSW	0	C5	16
798904	BRINE+DI	0	A1	18
798904	BRINE+DI	0	A2	17
798904	BRINE+DI	0	A3	17
798904	BRINE+DI	0	A4	30
798904	BRINE+DI	0	A5	19
798904	BRINE+DI	0	B1	12
798904	BRINE+DI	0	B2	19
798904	BRINE+DI	0	B3	21
798904	BRINE+DI	0	B4	11
798904	BRINE+DI	0	B5	31
798904	BRINE+DI	0	C1	15
798904	BRINE+DI	0	C2	29
798904	BRINE+DI	0	C3	16
798904	BRINE+DI	0	C4	26
798904	BRINE+DI	0	C5	31
798909	LANDM	5.8	A1	36
798909	LANDM	5.8	A2	42
798909	LANDM	5.8	A3	31
798909	LANDM	5.8	A4	42
798909	LANDM	5.8	A5	50
798909	LANDM	5.8	B1	16
798909	LANDM	5.8	B2	14
798909	LANDM	5.8	B3	14
798909	LANDM	5.8	B4	17
798909	LANDM	5.8	B5	25
798909	LANDM	5.8	C1	17
798909	LANDM	5.8	C2	20
798909	LANDM	5.8	C3	22
798909	LANDM	5.8	C4	16
798909	LANDM	5.8	C5	17

TABLE H-4(cont). *Champia parvula* SEXUAL REPRODUCTION TEST ON SEEP SAMPLES

SAMPNUM	STATION	CONC (%)	REPLICATE	CYSTOCARPS
789908	LANDM	11.5	A1	12
789908	LANDM	11.5	A2	11
789908	LANDM	11.5	A3	21
789908	LANDM	11.5	A4	9
789908	LANDM	11.5	A5	14
789908	LANDM	11.5	B1	6
789908	LANDM	11.5	B2	14
789908	LANDM	11.5	B3	7
789908	LANDM	11.5	B4	14
789908	LANDM	11.5	B5	13
789908	LANDM	11.5	C1	22
789908	LANDM	11.5	C2	29
789908	LANDM	11.5	C3	19
789908	LANDM	11.5	C4	20
789908	LANDM	11.5	C5	16
789907	LANDM	23	A1	5
789907	LANDM	23	A2	1
789907	LANDM	23	A3	0
789907	LANDM	23	A4	7
789907	LANDM	23	A5	3
789907	LANDM	23	B1	3
789907	LANDM	23	B2	9
789907	LANDM	23	B3	1
789907	LANDM	23	B4	4
789907	LANDM	23	B5	8
789907	LANDM	23	C1	2
789907	LANDM	23	C2	12
789907	LANDM	23	C3	9
789907	LANDM	23	C4	13
789907	LANDM	23	C5	7
798906	LANDM	46	A1	2
798906	LANDM	46	A2	0
798906	LANDM	46	A3	1
798906	LANDM	46	A4	5
798906	LANDM	46	A5	0
798906	LANDM	46	B1	0
798906	LANDM	46	B2	0
798906	LANDM	46	B3	0
798906	LANDM	46	B4	0
798906	LANDM	46	B5	0
798906	LANDM	46	C1	4
798906	LANDM	46	C2	1
798906	LANDM	46	C3	2
798906	LANDM	46	C4	2
798906	LANDM	46	C5	1
798905	LANDM	92	A1	0

TABLE H-4(cont). *Champia parvula* SEXUAL REPRODUCTION TEST ON SEEP SAMPLES

SAMPNUM	STATION	CONC (%)	REPLICATE	CYSTOCARPS
798905	LANDM	92	A2	0
798905	LANDM	92	A3	0
798905	LANDM	92	A4	0
798905	LANDM	92	A5	0
798905	LANDM	92	B1	0
798905	LANDM	92	B2	0
798905	LANDM	92	B3	0
798905	LANDM	92	B4	0
798905	LANDM	92	B5	0
798905	LANDM	92	C1	0
798905	LANDM	92	C2	0
798905	LANDM	92	C3	0
798905	LANDM	92	C4	0
798905	LANDM	92	C5	0

TABLE H-5. *Mulinia lateralis* EMBRYO / LARVAL TOXICITY TEST ON SEEP SAMPLES

SAMPNUM	STATION	CONC (%)	REPLICATE	% NO SHELL
	NSW	0	1	14.7
	NSW	0	2	18.0
	NSW	0	3	25.3
798904	BRINE+DI	0	1	7.8
798904	BRINE+DI	0	2	16.5
798904	BRINE+DI	0	3	31.1
798909	LANDM	5.8	1	28.3
798909	LANDM	5.8	2	26.0
798909	LANDM	5.8	3	21.4
789908	LANDM	11.5	1	25.0
789908	LANDM	11.5	2	24.0
789908	LANDM	11.5	3	21.9
789907	LANDM	23	1	39.6
789907	LANDM	23	2	44.3
789907	LANDM	23	3	35.0
798906	LANDM	46	1	82.9
798906	LANDM	46	2	95.7
798906	LANDM	46	3	97.6
798905	LANDM	92	1	94.6
798905	LANDM	92	2	100.0
798905	LANDM	92	3	100.0

TABLE H-6. *Menidia berylina* 96 HOUR LARVAL SURVIVAL TEST ON SEEP SAMPLES

SAMPNUM	STATION	CONC (%)	REPLICATE	% MORTALITY
	NSW	0	1	10.0
	NSW	0	2	10.0
798909	LANDM	5.8	1	30.0
798909	LANDM	5.8	2	20.0
789908	LANDM	11.5	1	10.0
789908	LANDM	11.5	2	0.0
789907	LANDM	23	1	0.0
789907	LANDM	23	2	0.0
798906	LANDM	46	1	0.0
798906	LANDM	46	2	0.0
798905	LANDM	92	1	0.0
798905	LANDM	92	2	0.0

APPENDIX I

SEDIMENT BIOASSAY RESULTS

TABLE I-1. *Ampelisca abdita* SEDIMENT BIOASSAY ON SEDIMENT SAMPLES

SAMPNUM	STATION	CONC (%)	REPLICATE	% MORTALITY
798912	POTO	100	1	5.0
798912	POTO	100	2	0.0
798903	LANDM	100	1	15.0
798903	LANDM	100	2	5.0
798903	LANDM	100	3	10.0

TABLE I-2. ***Mulinia lateralis*** **7 DAY SEDIMENT TOXICITY TEST**

SAMPNUM	STATION	CONC (%)	REPLICATE	% MORTALITY	GROWTH	Δ GROWTH
sand			1	0.0	1.4	0.9
sand			2	0.0	1.3	0.9
sand			3	0.0	1.4	0.9
sand			4	0.0	1.7	1.1
sand			5	0.0	1.3	0.9
sand			6	17.0	1.5	1.0
798910	PR		1	0.0	1.4	0.9
798910	PR		2	0.0	1.8	1.2
798910	PR		3	33.0	1.4	0.9
798910	PR		4	0.0	1.4	0.9
798910	PR		5	0.0	1.5	1.0
798910	PR		6	0.0	1.4	0.9
	NJT		1	0.0	1.6	1.1
	NJT		2	17.0	1.3	0.9
	NJT		3	17.0	1.5	1.0
	NJT		4	0.0	1.6	1.1
	NJT		5	0.0	1.4	0.9
	NJT		6	0.0	1.6	1.1
798939	CLIS		1	0.0	1.1	0.7
798939	CLIS		2	0.0	1.3	0.9
798939	CLIS		3	17.0	1.0	0.7
798939	CLIS		4	0.0	1.4	0.9
798939	CLIS		5	17.0	1.6	1.1
798939	CLIS		6	17.0	1.5	1.0
798912	POTO	0	1	0.0	1.0	0.7
798912	POTO	0	2	0.0	1.2	0.8
798912	POTO	0	3	0.0	1.2	0.8
798912	POTO	0	4	0.0	1.1	0.7
798912	POTO	0	5	0.0	1.2	0.8
798912	POTO	0	6	0.0	1.2	0.8
798915	LANDM	12.5	1	17.0	1.5	1.0
798915	LANDM	12.5	2	0.0	1.2	0.8
798915	LANDM	12.5	3	0.0	1.3	0.9
798915	LANDM	12.5	4	17.0	1.6	1.1
798915	LANDM	12.5	5	0.0	1.4	0.9
798915	LANDM	12.5	6	17.0	1.3	0.9
798914	LANDM	25	1	17.0	1.4	0.9
798914	LANDM	25	2	17.0	1.4	0.9
798914	LANDM	25	3	0.0	1.8	1.2
798914	LANDM	25	4	0.0	1.3	0.9
798914	LANDM	25	5	17.0	1.2	0.8
798914	LANDM	25	6	0.0	1.3	0.9
798913	LANDM	50	1	0.0	1.6	1.1
798913	LANDM	50	2	0.0	1.6	1.1
798913	LANDM	50	3	17.0	1.2	0.8
798913	LANDM	50	4	0.0	1.2	0.8
798913	LANDM	50	5	0.0	1.1	0.7
798913	LANDM	50	6	0.0	1.4	0.9
798903	LANDM	100	1	0.0	1.5	1.0
798903	LANDM	100	2	17.0	1.2	0.8
798903	LANDM	100	3	17.0	1.4	0.9
798903	LANDM	100	4	17.0	1.6	1.1
798903	LANDM	100	5	0.0	1.3	0.9
798903	LANDM	100	6	0.0	1.5	1.0

APPENDIX J

SEDIMENT EXTRACT BIOASSAY RESULTS

TABLE J-1. *Arbacia punctulata* FERTILIZATION TEST ON SEDIMENT EXTRACTS

SAMPNUM	STATION	CONC (%)	REPLICATE	% FERTILIZATION
798947	NSW	0.000	1	98.0
798947	NSW	0.000	2	99.0
798947	NSW	0.000	3	98.0
798946	DMSO	(0.05)	1	100.0
798946	DMSO	(0.05)	2	99.0
798946	DMSO	(0.05)	3	97.0
798945	BLANK	(0.05)	1	98.0
798945	BLANK	(0.05)	2	95.0
798945	BLANK	(0.05)	3	96.0
798944	LANDM	0.003	1	99.0
798944	LANDM	0.003	2	98.0
798944	LANDM	0.003	3	98.0
798943	LANDM	0.006	1	98.0
798943	LANDM	0.006	2	94.0
798943	LANDM	0.006	3	93.0
798942	LANDM	0.013	1	66.0
798942	LANDM	0.013	2	81.0
798942	LANDM	0.013	3	79.0
798941	LANDM	0.025	1	0.0
798941	LANDM	0.025	2	0.0
798941	LANDM	0.025	3	3.0
798940	LANDM	0.050	1	0.0
798940	LANDM	0.050	2	0.0
798940	LANDM	0.050	3	0.0

TABLE J-2. *Arbacia punctulata* 48-HOUR LARVAL DEVELOPMENT TEST ON SEDIMENT EXTRACTS RUN 1

SAMPNUM	STATION	CONC (%)	REPLICATE	% NORMAL	% MORTALITY	% ABNORMAL
798950	NSW	0	1	82.5	11.0	6.5
798950	NSW	0	2	87.5	9.0	3.5
798950	NSW	0	3	89.0	8.5	2.5
798948	BLANK	(0.25)	1	81.0	13.5	5.5
798948	BLANK	(0.25)	2	84.5	13.0	2.5
798948	BLANK	(0.25)	3	84.0	12.5	3.5
798949	DMSO	(0.25)	1	82.5	13.0	4.5
798949	DMSO	(0.25)	2	89.5	9.0	1.5
798949	DMSO	(0.25)	3	87.0	12.0	1.0
798938	LANDM	0.025	1	86.5	12.0	1.5
798938	LANDM	0.025	2	88.5	9.5	4.0
798938	LANDM	0.025	3	86.5	7.5	4.0
798937	LANDM	0.05	1	86.5	12.0	1.5
798937	LANDM	0.05	2	83.5	12.5	4.0
798937	LANDM	0.05	3	85.0	9.5	5.5
798936	LANDM	0.1	1	1.5	10.5	88.0
798936	LANDM	0.1	2	1.0	14.0	85.0
798936	LANDM	0.1	3	0.0	15.5	84.5
798935	LANDM	0.15	1	0.5	12.5	87.0
798935	LANDM	0.15	2	0.0	11.5	88.5
798935	LANDM	0.15	3	0.0	14.0	86.0
798934	LANDM	0.2	1	0.5	32.0	67.5
798934	LANDM	0.2	2	0.0	36.0	64.0
798934	LANDM	0.2	3	0.0	43.0	57.0
798933	LANDM	0.25	1	0.0	91.5	8.5
798933	LANDM	0.25	2	0.0	90.0	10.0
798933	LANDM	0.25	3	0.0	91.5	8.5

TABLE J-2 (cont). *Arbacia punctulata* 48-HOUR LARVAL DEVELOPMENT TEST ON SEDIMENT EXTRACTS RUN 2

SAMPNUM	STATION	CONC (%)	REPLICATE	% NORMAL	% MORTALITY	% ABNORMAL
798947	NSW	0	1	77.5	13.0	9.5
798947	NSW	0	2	74.0	16.0	9.5
798947	NSW	0	3	75.5	13.0	11.5
798945	BLANK	(0.05)	1	74.0	14.0	12.0
798945	BLANK	(0.05)	2	78.0	12.0	10.0
798945	BLANK	(0.05)	3	71.5	12.5	16.0
798946	DMSO	(0.05)	1	85.0	8.5	6.5
798946	DMSO	(0.05)	2	78.5	10.5	11.0
798946	DMSO	(0.05)	3	85.0	6.0	9.0
798944	LANDM	0.003	1	72.5	20.5	7.0
798944	LANDM	0.003	2	87.0	5.5	7.5
798944	LANDM	0.003	3	89.5	3.0	7.5
798943	LANDM	0.006	1	79.0	11.0	10.0
798943	LANDM	0.006	2	71.0	19.0	10.0
798943	LANDM	0.006	3	84.0	5.5	10.5
798942	LANDM	0.013	1	83.5	11.5	5.0
798942	LANDM	0.013	2	88.5	8.5	6.0
798942	LANDM	0.013	3	83.5	6.5	10.0
798941	LANDM	0.025	1	78.5	6.5	15.0
798941	LANDM	0.025	2	77.5	11.5	11.0
798941	LANDM	0.025	3	85.0	9.5	5.5
798940	LANDM	0.05	1	86.0	7.5	6.5
798940	LANDM	0.05	2	79.0	11.5	9.5
798940	LANDM	0.05	3	76.0	9.5	14.5

TABLE J-3. ***Mulinia lateralis*** **EMBRYO / LARVAL TOXICITY TEST ON SEDIMENT EXTRACTS**

SAMPNUM	STATION	CONC (%)	REPLICATE	% NO SHELL
	NSW	0	1	26.0
	NSW	0	2	30.0
	NSW	0	3	34.0
	BLANK	(0.05)	1	84.0
	BLANK	(0.05)	2	93.0
	BLANK	(0.05)	3	87.0
	DMSO	(0.05)	1	93.0
	DMSO	(0.05)	2	92.0
	DMSO	(0.05)	3	93.0
798944	LANDM	0.003	1	92.0
798944	LANDM	0.003	2	89.0
798944	LANDM	0.003	3	88.0
798943	LANDM	0.006	1	90.0
798943	LANDM	0.006	2	92.0
798943	LANDM	0.006	3	89.0
798942	LANDM	0.013	1	99.0
798942	LANDM	0.013	2	99.0
798942	LANDM	0.013	3	93.0
798941	LANDM	0.025	1	100.0
798941	LANDM	0.025	2	100.0
798941	LANDM	0.025	3	100.0
798940	LANDM	0.05	1	100.0
798940	LANDM	0.05	2	100.0
798940	LANDM	0.05	3	100.0

TABLE J-4. MICROTOX ASSAY ON SEDIMENT EXTRACTS

SAMPNUM	STATION	CONC (%)	REPLICATE	BIOLUMINESCENCE
798932	BLANK	(0.001)	1	100.0
798931	LANDM	0.001	1	97.0
798931	LANDM	0.001	2	98.0
798930	BLANK	(0.01)	1	98.0
798930	BLANK	(0.01)	2	101.0
798929	LANDM	0.01	1	73.0
798929	LANDM	0.01	2	71.0
798928	BLANK	(0.1)	1	100.0
798928	BLANK	(0.1)	2	99.0
798927	LANDM	0.1	1	20.0
798927	LANDM	0.1	2	20.0
798926	BLANK	(1)	1	102.0
798926	BLANK	(1)	2	99.0
798925	LANDM	1	1	8.0
798925	LANDM	1	2	8.0

APPENDIX K

MYA NEOPLASIA EXPERIMENT RESULTS

TABLE K-1. LABORATORY EXPOSURE OF *Mya arenaria*.

SAMPNUM	STATION	REPLICATE	INJECTED	%HN	MORTALITY	GROWTH	SE
798922	PR	1	+	28.3	15	-0.71	0.237
798922	PR	2	+	36.7	6.7	-1.61	0.282
798922	PR	1	-	25	16.7	-1.29	0.230
798922	PR	2	-	31.1	9.8	-0.74	0.359
798918	AH	1	+	60	18.3	-0.64	0.210
798918	AH	2	+	35	15	-2.55	0.313
798918	AH	1	-	39	6.8	-0.34	0.395
798918	AH	2	-	39.3	9.8	-1.76	0.226

APPENDIX L

GRAIN SIZE ANALYSES OF SEDIMENTS

TABLE L-1. GRAIN SIZE ANALYSIS.

SAMPNUM	STATION	CONC (%)	% SAND	% SILT	% CLAY
798912	POTO	100	47.7	40.4	11.9
798903	LANDM	100	92.69	4.05	3.26